Rigor mortis in the heart and the state of the cardiac cavities after death / by J.A. MacWilliam.

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By J. A. MACWILLIAM, M.D., *Professor of Physiology in the University of Aberdeen.* (Two Figures in Text.)

(*From the Physiological Laboratory of the University of Aberdeen.*)

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THOUGH the occurrence of rigor mortis with the general features of that condition—acidity, &c.—in the mammalian heart has been long known, little seems to have been ascertained as to its exact characters, its order of development, relative intensity, and duration in the different parts of the heart, the conditions which influence it, &c.

My observations have been made from time to time over a period of many years and mainly on the hearts of cats, rabbits, guinea-pigs, and rats[1], though in some instances the hearts of men and oxen have been examined.

Methods. In many instances the heart has been exposed prior to death and the development of rigor (after death induced in various ways) directly observed; in other cases the thorax has not been opened till some time after death. Means have always been taken to prevent accidental escape of blood from the vascular system. The amount of blood in the various chambers of the organ has often been determined by weighing the clots extracted from each cavity, or when it was desired to determine the amounts of blood in the right and left sides respectively before coagulation had occurred the great vessels were

[1] All experiments on animals were conducted under anæsthesia (chloroform &c.).

ligatured close to the heart and then the contents of the right and left sides were separately evacuated and measured. Care was taken to close the coronary arteries at their origin. The intra-cardiac pressure was estimated after death on the two sides of the heart by the following methods:—

(*a*) by a Hg. manometer connected with the ventricular cavity by a tube either passed through the ventricular wall or introduced through the auricle.

(*b*) by observing the height to which the blood rose in a vertical tube of small calibre pushed through the cardiac wall into the cavities; the tube had a sharp bevelled point and was washed out with 25 % $MgSO_4$ solution just before using. A minute snip was made through the epicardium to permit the entrance of the point of the tube. Both in this method and the preceding (*a*) care has to be taken to avoid displacement of blood by mechanical pressure on the heart, &c., to observe the exact elevation of the cardiac chambers where the observations are made, and in (*b*) to allow for the capillarity of the tube used.

Tracings of the contraction of the left ventricle as it enters into rigor were obtained by tying a cannula into the aortic orifice of a heart just excised and connecting the cannula with a mercurial kymograph in the usual way, the tracing being inscribed on a slow drum.

Post-mortem Intra-cardiac Pressure.

Brunner[1] many years ago described the existence of a slight positive pressure in the vascular system after death.

In recent years there have been conflicting views on this point. Bayliss and Starling[2] found, in accordance with Brunner's observation, a definite positive pressure amounting in the dog to 5—10 mm. Hg.

On the other hand L. Hill and Barnard[3] have stated that in the dead animal when the flow of fluid has entirely ceased and the conditions have become static the mean pressure measured in the aorta and vena cava is zero.

My own observations have invariably shown the presence of a slight but appreciable pressure in the cardiac chambers after death—in the rabbit pressures of 2—3 mm. Hg. when tested with the manometer and

[1] *Zeitschr. f. rat. Medicin.* 1853.

[2] This *Journal*, xvi. p. 166. 1894.

[3] *Ibid.* xxi. p. 345. 1897.

25—40 mm. blood when determined by a vertical tube as already described; and somewhat higher pressures in the cat. I have found the pressures equal on the two sides of the heart after death induced in many different ways—asphyxia, cardiac paralysis caused by various poisons, &c. (Cases where there has been great loss of blood, mechanical obstruction, &c. are of course not included in these statements.)

Graphic Records of the Contraction of the Left Ventricle in Rigor.

When the pressure in the interior of the left ventricle is recorded in the way stated the left ventricle is found to exhibit considerable expulsive force when it contracts on entering into well-marked rigor—getting up a pressure of 25—30 mm. Hg. (cat) in the kymograph; this pressure may be maintained at practically the same level for a considerable time and then at a somewhat lower level for a further and more prolonged period extending over very many hours, &c. The rise of pressure is usually rapid in the first few minutes after contraction has begun; then it developes very much more slowly up to its maximum. In the intact heart (ox) movement of blood in the coronary veins on the anterior surface of the ventricles may be distinctly seen at this time.

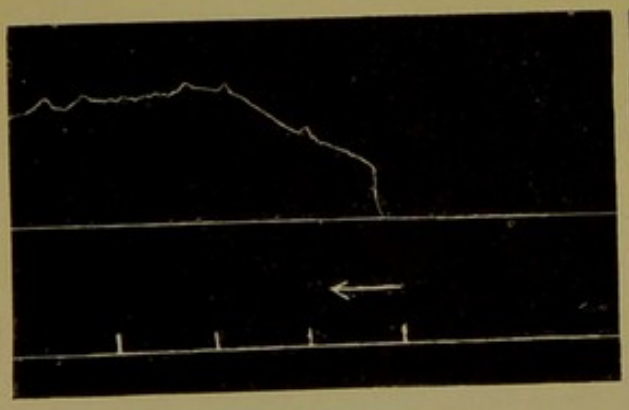

Fig. 1.

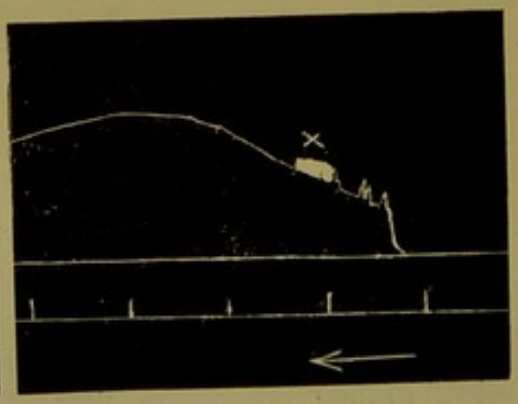

Fig. 2.

Fig. 1. Cat's heart; manometer connected with L.V. Secondary oscillations well marked on the rise of pressure caused by the ventricle contracting as it goes into rigor. Time tracing shows half-hours.

Fig. 2. Cat's heart. Arrangement similar to preceding experiment. A series of quick contractions and relaxations resembling ordinary systoles and diastoles on a small scale is seen on the tracing below the mark ×, after the pressure had risen considerably, and some slow secondary oscillations had occurred.

A remarkable feature seen in many tracings is the occurrence of very well-marked minor oscillations of pressure showing as secondary waves on the curve (Fig. 1.) These may occur during the rise of pressure, or for some time after it has reached its maximum, or in both these periods. These minor oscillations are each of considerable duration—their rise and fall extending over several minutes; the phases of ascent and descent are approximately equal as a rule. These oscillations succeed one another in an irregular fashion; sometimes only one in half-an-hour, at other times several may occur in close succession and with a fairly regular rhythm within the same period. They obviously depend on slow variations in the state of contraction of the ventricular walls. They only occur, as far as I have seen, in the earlier stages of rigor, *e.g.* within 1—2 hours after death.

It is worthy of note in some cases that after the appearance of some well-developed slow oscillations of the kind described the ventricle may execute a series of very different contractions and relaxations—short in duration, quick in succession, and evidently resembling ordinary systoles and diastoles in miniature. (Fig. 2.)

Thus the ventricular muscle while shortening in the contraction of rigor, or soon after it has reached its maximum shortening, can show slow contraction and relaxation, and after showing some of the slow oscillations, can again exhibit a succession of quick contractions and relaxations on a small scale; of course the two kinds of secondary oscillation may depend upon the action of different sets of fibres in the ventricular wall.

It is only in some hearts that the oscillations described above are seen; in very many instances there is no sign of them. I have not seen them at all in tracings of rigor made by a lever laid across the ventricles of an excised heart resting on a horizontal surface. The lever rises as the ventricular wall goes into rigor but the tracing shows no secondary oscillations as far as I have seen. Of course the conditions are very different here; the contracting ventricle is not in the state of tension present in the manometer experiment.

The relation of shortening of the muscular fibres to loss of their excitability seems to be quite different in the heart from what is described as occurring in skeletal muscle, viz., that there is a previous loss of excitability before any contraction takes place. In the heart on the other hand the ventricular wall may show decided evidence of excitability when contraction has already gone on to a considerable extent. In this respect the cardiac muscle rather resembles skeletal muscle subjected to

the influence of gradual heating; here excitability may persist for some time after contraction has set in[1].

There is no necessary parallelism between cardiac and skeletal muscle as regards the occurrence of rigor, its intensity, &c. In cases where the heart shows little or no sign of rigor the skeletal muscles may be strongly rigid.

Rigor in the heart is not prevented by freezing in the way that I have found effective in the case of the post-mortem contraction of excised arteries. A recently-excised (contracting) heart frozen for 4—5 hours was found to show well-marked rigor after thawing—the cavity of the left ventricle was almost obliterated while the muscle became markedly rigid.

Similarly when rigor has been established in the heart it is not removed by freezing for 4—5 hours, nor by treatment with potassium sulphocyanide solution, nor by heating to 50°—55°—methods which are effective in abolishing post-mortem contraction of the arteries.

State of the Heart after Death from Various Causes.

Asphyxia. Rigor mortis plays an essential part in determining the state of the heart and the distribution of the blood in the heart and great vessels which have long been recognised as characteristic of death by asphyxia. Many causes have been adduced to account for the fulness of the right heart and its vessels and the emptiness or comparative emptiness of the left side, *e.g.* the greater distensibility of the right heart, the influence of violent respiratory movements and muscular spasms in determining an accumulation of blood in the great systemic veins and the right heart, an alleged strong contraction of the pulmonary arterioles opposing the discharge of blood from the right ventricle, diminished suction power of the left ventricle, &c.; also the influence of rigor mortis on the left ventricle after death, though no explanation seems to have been offered as to why the right ventricle should behave differently to the left in this respect.

Most of the alleged causes may be set aside as inadequate or unessential. The greater distensibility of the right heart would naturally apply equally in cases of death from causes other than asphyxia and would obviously not account for the left heart being nearly empty. The influence of violent respiratory movements and muscular spasms

[1] Brodie and Richardson, *Phil. Trans.* B. 191, p. 143. 1899.

is evidently unessential since these can be excluded by the previous administration of curare without materially altering the condition found after death. The assumed contraction of the pulmonary arterioles may be shown not to be an effective cause by evidence of various kinds (study of pressure in left auricle, &c.), and so with the diminished suction power of the left ventricle. Indeed any cause merely leading or tending to an accumulation of blood on the right side of the heart prior to death must be an unsatisfactory explanation. For though some discordant statements[1] have been made on the point, it is quite certain that at death both sides of the heart are distended with blood—both auricles and ventricles[2]. The pressure in the right and left chambers has, in my experiments, been found to be equal—usually a pressure of 25—40 mm. blood in the rabbit, and a somewhat higher pressure in the cat.

Conflicting statements have been made as to the amounts of blood in the two sides of the heart immediately after death by asphyxia. One observer[3] gives the relative proportions on the right and left sides as two to three, while another[4] describes a very great preponderance on the right side—in one experiment (dog) he found 16 oz. on the right side and only $2\frac{1}{2}$ oz. on the left.

In my experiments the amount on both sides was invariably found to be relatively large; there was always found to be more on the right side than on the left—commonly in the proportion of three to two—*e.g.* in a rabbit I found 9 c.c. on the right side and 6 c.c. on the left[5]; in another (smaller) rabbit 4·5 c.c. on the right and 3 c.c. on the left, &c.[6]

The greater amount of blood on the right side is evidently due to

[1] Sir George Johnson (*An Essay on Asphyxia*, London, 1889; *Lancet*, Aug. 10, 1889; *Proc. Roy. Soc.* XLIX. p. 144, 1891) describes the cavities of the left heart as becoming distended in the earlier stage of asphyxia when the systemic pressure is very high, and then diminishing in size, and becoming empty at the end of asphyxia, by which time the cavities of the right heart have become largely distended—on account (as he affirms) of extreme contraction of the pulmonary arterioles.

[2] Roy and Adami (*Phil. Trans.* 183 (1892) B, p. 288) relying on records obtained by their myocardiograph, describe the tonicity of the auricles as being increased in asphyxia (after section of the vago-sympathetics) and the expansion in diastole diminished, while in the ventricles the expansion in diastole is increased.

My own tracings, made by different methods, invariably show marked distension of the auricles as well as the ventricles in asphyxia; and the change visible on direct inspection of the heart is too extensive and striking to leave any room for doubt on the point.

[3] Burdon-Sanderson, *Handbook for the Physiological Laboratory*, London, 1873, p. 323.

[4] Johnson, *loc. cit.*, London, 1889, p. 15.

[5] The post-mortem pressure here was 4 mm. Hg in each ventricle.

[6] Sometimes the difference is much less than this.

the greater distensibility of the right heart; since the pressure on the two sides is found to be equal.

After death by asphyxia rigor occurs early and strongly in the left ventricle. In the rat I have seen rigor commencing as soon as 9 minutes after occlusion of the trachea, in the rabbit in 12 minutes, and in the cat in 15 minutes—though as a rule the periods are much longer than these. It seems probable that the diminution or emptying of the left ventricle described by Johnson (*op. cit.* 1899), as occurring at the end of asphyxia may have been really due to the early onset of the contraction of rigor.

While the expulsive power of the left ventricle after asphyxia can be demonstrated by means of a manometer in the way already described the exterior of the ventricle also shows changes when going into rigor—its apex becomes more pointed, its surface paler, &c.

Why is the right ventricle not emptied by rigor mortis like the left? In asphyxia the right ventricle dies slowly and gradually—later than the left. Rigor comes on late, it is gradual in its onset, often markedly unequal in distribution, and relatively slight in intensity. In the larger animals marked differences may be found in different portions of the muscular walls of the right ventricle, *e.g.* the mesial wall may be much more rigid than the rest of the ventricle. As the force exerted in rigor is confined to the period between the beginning of the attempt to shorten and its completion, it is obvious that if different sets of fibres go into rigor at different times no considerable expulsive force can be developed, even if the contractile effort was very much stronger than it is, as a rule, in the right ventricle after asphyxia. Further, the lateness of the occurrence of rigor is unfavourable to the expulsion of blood from the cavity, on account of changes occurring in the blood itself after a time, increased adhesiveness and subsidence of the corpuscles, coagulation, &c.

In some cases after death by asphyxia rigor comes on much earlier than usual in the right ventricle and is much more strongly developed. When this happens the blood is expelled just as ordinarily happens in the left ventricle, so that some time after death *both* ventricles are found to be almost empty.

Exp. 1. Rabbit, Chloroform. Natural respiration.
Trachea closed.
4 min. after. Respiration has ceased; cardiac pulsation visible through skin.
7 min. Heart-beat can still be felt; slowed.
20 min. Thorax opened. Whole heart distended.
30 min. Distinct evidence of rigor in L.V.

20 hours. Both ventricles contracted. When cut across the cavities of both were found to be almost obliterated; a little reddish serum escaped. A considerable amount of clotted blood in R.Au.; little in L.Au.

Exp. 2. Rat. Chloroform. Natural respiration.
Asphyxia by closure of trachea.
5 min. after cardiac impulse ceased to be felt through chest wall, the thorax was opened. Whole heart distended. Ecchymoses in lungs.
90 min. Both ventricles rigid and contracted; cut into—both almost empty. R.Au. more distended than L.Au.

In some instances—in young animals at least—in which the left ventricle lives longer than usual after asphyxia and the onset of rigor in the heart is later than usual, neither ventricle may be emptied that there may be no decided preponderance in the contents of either 24 hours after death.

Exp. 3. Rabbit (young). Chloroform. Natural respiration.
Trachea closed.
1½ min. Respiration stopped.
2¼ min. No respiration. Cardiac pulsation visible; slowed.
3½ min. Slight respiratory movements.
4 min. No sign of respiration.
12 min. Thorax opened. Whole heart distended, but R.Au. larger than L.Au. No contraction at first, but soon rhythmic contraction occurred in all parts, though feeble and ineffective.
22 min. R.Au. and systemic veins beating more vigorously—104 per min. R.V. distended—shows no movement. Contraction still visible in L.V.
1 hour. R.Au. and systemic veins beating; no contraction in any other part; R.Au. seems to contain somewhat more blood than L.Au.
24 hours. Auricles contain much blood (coagulated), R.Au. more than L.Au. Ventricles feel rigid. Cut across—considerable blood-clot in each; no decided difference in amount.

It is well known that in skeletal muscle activity of a muscle prior to death favours the development of rigor very markedly, and it is conceivable that in ordinary asphyxia the great strain thrown upon the left ventricle while it is working against asphyxial rise of blood-pressure might have an influence upon the subsequent development of rigor in the heart. There is also the question of possible over-stretching of the thin-walled right ventricle, &c. But it is clear that such influences are not at all essential for the production of the characteristic p.m. condition of the organ. For asphyxia induced while the usual rise of pressure is absent leads to results in the heart after death essentially similar to those usually seen when the asphyxial rise has occurred in the ordinary way though the L.V. may not be emptied.

Exp. 4. Cat. Chloroform. Large dose of curari injected (subcutaneously) while artificial respiration was kept up. Artificial stopped after a little time. No rise in blood-pressure; heart stopped with both sides distended.

2 hours p.m. Feeble contractions (irregular in sequence) seen in R.V. Rigor present in L.V.

24 hours p.m. R.V. contains a great deal of blood; faint signs of rigor in it. R.Au. much blood. L.V. firm and contracted; L.Au. has little blood.

48 hours p.m.
72 „ „ } condition much the same.

96 hours p.m. R.V. less distended than before but still large ventricles cut across—R.V. and R.Au. contain large clots; L.V. almost empty—only traces of clot.

In a rabbit where no rise of pressure occurred when asphyxia was induced, the clots obtained from the cavities of the heart 20 hours after death were as follows—stated in milligrammes—L.V. 490, R.V. 1120, L.Au. 220, R.Au. 210. (This result is exceptional in one respect—in there not being the usual preponderance in the R.Au., as compared with the L.Au.)

Chloroform. A. When an animal is killed by the gradual administration of dilute chloroform vapour by inhalation in the usual way the respiration stops, as a rule, some time before the weakened heart has ceased to beat; the whole heart at length fails with all its chambers largely distended.

Some hours after death the cardiac condition is that usually found after asphyxia.

Exp. 5. Rabbit chloroformed till respiration stopped.

Thorax opened. Whole heart distended; still showing rhythmic contraction (ineffective).

15 min. after stoppage of respiration. L.V. apparently contracted to some extent, though still responding to mechanical stimulation.

2 hours. Rhythmic contraction in R.Au.; occasional feeble contraction in R.V. L.V. is much smaller than before. L.Au. as well as R.Au. and R.V. much distended. Pressure in R.V. and L.V. equal = 38mm. blood.

24 hours. R.H. distended; large clot in R.V., L.V. rigid and almost empty; small clot in L.Au.

B. When the heart is paralysed by an overdose of chloroform prior to the cessation of respiration it ceases to beat with its walls relaxed and all its chambers distended with blood; this is the case whether the chloroform has been given by inhalation in the intact animal, or whether it has been administered by artificial respiration after the thorax has been laid open and the heart submitted to direct inspection. When an overdose has been rapidly given by inhalation, deep slow respiration often goes on (in the cat) for a considerable time (minutes) after all signs of cardiac action have ceased to be perceptible.

On examination some hours after death the condition of the heart is commonly found to be greatly changed from the state of general distension seen at death. The left ventricle may be markedly contracted in rigor and nearly empty, while the right side of the heart is gorged, *i.e.*, the condition usually recognised as being characteristic of asphyxia has developed (Exps. 6 and 7).

In other cases rigor is well marked in both ventricles and both are found to be nearly empty.

Exp. 6. Cat. Overdose of chloroform given by inhalation during quick respiration. Collapse occurred; heart-beat ceased to be perceptible and exposed axillary artery suddenly became pulseless and empty; while respiration continued regularly, becoming slow and deep, for more than two minutes—artificial respiration being performed occasionally. No recovery of circulation.

12 min. after collapse. Thorax opened. Whole heart distended; faint movements in auricles.

15 min. Signs of commencing rigor in L.V.

30 min. Pronounced rigor in L.V.

45 min. L.V. firmly contracted and apparently empty. R.V. much distended. Aus. both large; R. somewhat larger than L.

3 hours. The characteristic asphyxial condition is present.

48 hours. Ventricles cut open. Large clot in R.V. L.V. almost empty.

Exp. 7. Rat. Chloroform. Overdose of chloroform given by artificial respiration—until all signs of heart action abolished. Thorax opened—whole heart found to be distended.

1¼ hour later. L.V. in rigor, empty or nearly so. R.V. distended. R.Au. (still beating) has more blood than L.Au.

Such results as these indicate the impossibility of determining with certainty from post-mortem examination of the heart whether respiratory or cardiac failure had been the determining cause of death. For what is usually regarded as an asphyxial condition of the heart is found not only when the respiration has ceased first, but also in many instances in which cardiac paralysis preceded respiratory failure.

Morphia. Intravenous injection of a large overdose of morphia speedily arrests the heart in a state of general distension. Some hours afterwards both ventricles may be found to be firmly contracted in rigor and nearly empty.

Exp. 8. Cat. Chloroform. Large dose of morphia acetate. Thorax opened and heart found to be acting very feebly and inefficiently; its excitability as tested by electrical stimulations was greatly depressed; it soon stopped beating—in distended condition.

3 hours p.m. Rigor evident in heart. Ventricles are contracted and appear to be nearly empty; auricles contain a fair amount of blood.

24 hours p.m. Ventricles firmly contracted. Little blood in auricles.

48 hours p.m. No change.

96 hours p.m. Distribution of blood unchanged. Ventricles cut across—cavities almost entirely empty—only a few small shreds of clot in right ventricle.

Potassium Salts. When the heart is paralysed by injection of potassium salts into the jugular vein the respiration survives the heart for a little time. In many animals (cat, &c.) a sufficient dose of potassium salt at once throws the heart into long-persisting fibrillar contraction—causing speedy death.

Rigor is usually later in developing and less marked than usual, and some hours after death (24, &c.) much blood is found on both sides of the heart; the L.V. contains much clot, though not as a rule as much as the right.

Exp. 9. Rabbit. Chloroform. Killed by injection of potassium bromide solution into vena cava inferior in abdomen. Thorax opened—heart distended; ineffective waves of contraction.

45 min. after injection. Heart motionless. No sign of rigor.

24 hours after injection. Large clots in both ventricles, though R.V. is the larger. Rigor very strongly marked in skeletal muscles.

Exp. 10. Rat. Chloroform. Injection of KBr sol. into inf. cava. Respiration went on for a little time. Heart exposed—distended and showing fibrillar contraction.

20 hours later. Clots of very considerable size in both ventricles. Rigor well marked in skeletal muscles.

Muscarin and Pilocarpin. Sudden death from an overdose of either of these drugs injected into the jugular vein is usually followed by a post-mortem condition of the heart pretty similar to that induced by potassium salts, though the amount of blood in the L.V. may be less than after potassium salts. In both cases rigor is well marked in the skeletal muscles.

Lactic Acid. When the heart is rapidly paralysed by lactic acid it stops in a condition of general distension, but after death it assumes the appearances usually regarded as characteristic of death by asphyxia.

Exp. 11. Rabbit, chloroform and chloral. Lactic acid injected into jugular vein caused speedy abolition of all external signs of heart action, while the respiration became asphyxial in character and soon ceased. Thorax opened at once. Whole heart distended and motionless. Ventricles respond feebly to direct (mechanical) stimulation; no visible response in auricles.

20 hours later. Right side of heart distended, containing large clots, L. side contains very little blood, the L.V. being almost empty. Here an asphyxial distribution of blood is present though primary cardiac failure had undoubtedly occurred.

Ammonia. Injection of ammonia into the jugular vein speedily causes paralysis and distension of the whole heart. After death an asphyxial distribution of the blood developes.

Exp. 12. Rabbit; chloroform and chloral. Heart paralysed by injection of Liq. Ammon. Fort. (B.P.) into jugular vein; respiration stopped shortly afterwards. Thorax opened at once. Heart motionless and distended. Pressure in R. and L. Vs. found to be equal = 25 mm. blood. Later, rigor developed in L.V.

24 hours p.m. R. side distended, large clots in V. and A.; L.V. almost empty, only a few shreds of clot on musculi papillares, &c.; small clot in L.Au. Rigor seems to have passed off from L.V. earlier than usual.

Exp. 13. Similar procedure and results except that the post-mortem pressure (equal on both sides as before) was 35 mm. blood.

Hæmorrhage. Rigor is well marked in both ventricles, which are usually contracted and empty or nearly so. The auricles contain less blood than usual—especially the left. The intravascular pressure being lower than usual contraction will more easily discharge blood from the heart.

Exp. 14. Rabbit, chloroform. Right carotid artery cut. Rapid respiration and speedy death.

10 min. p.m. Thorax opened and heart exposed. Whole organ flaccid and almost entirely motionless; rhythmic action has ceased. Both ventricles contain much blood; the R.Au. is fairly filled, while the L.Au. is very small.

45 min. p.m. L.V. in very firm rigor; apparently empty. R.V. also rigid and seems almost empty. L.Au., small (motionless). R.Au., contracting rhythmically; fairly full.

24 hours p.m. Both ventricles contracted and rigid; cut across—L.V. is empty and R.V. almost empty. R.Au., fair amount of blood; L.Au., small.

Pulmonary Embolism. This was produced by the injection of air or lycopodium spores (in normal saline) into the jugular or superior vena cava. The obstruction of the pulmonary vessels speedily led to a great accumulation of blood in the right heart, the pulmonary artery, and the great systemic veins.

Rigor was well marked in the left ventricle after death; the right remained full of blood.

Exp. 15. Rabbit. Chloroform. Natural respiration. Air into jugular vein. Speedy collapse. Thorax opened. Lungs very pale and anæmic; did not bleed when wounded. Heart almost entirely arrested. Both ventricles contain much blood, though R.V. more than L.V. R.Au. and venæ cavæ greatly distended. L.Au. is small.

20 hours after. Great preponderance of blood on R. side. Small clot and a little serum in L.V. and L.Au.; rigidity well marked in L.V. Large amount of clot in R.V. and R.Au.

Exp. 16. Rabbit. Chloroform. Thorax opened; artificial respiration. Injection of 1 c.c. ·75 % NaCl solution with some lycopodium powder stirred in it into vena cava superior. Speedy development of asphyxial symptoms with great distension of right pulmonary artery and right heart, while the left heart looks almost empty.

2½ min. Heart seems to have stopped.

4 min. Still an occasional respiratory movement. Both auricles beating at good rate; whole heart gives a beat now and then. R. Au. distended; L. Au. apparently empty.

6 min. Ventricles beating at 20 per min. Auricles much more rapidly.

9 min. Ventricles give a beat occasionally; L. V. now contains a good deal of blood. Condition of auricles unchanged, still beating.

23 min. Auricles still beating. Ventricles contract when stimulated but not otherwise. Contraction of R. Au. distinctly precedes that of L. Au.

24 hours. Large clot in R. V.; much less blood in L. V., which is rigid.

Conclusions.

Rigor mortis in the heart differs from that in the skeletal muscles (*a*) in some of its characters, and (*b*) in regard to the conditions which influence its appearance and intensity.

The hypothesis of a relation between the time of onset of rigor and the length of the nerves connecting the muscle with the central nervous system does not hold good in the heart.

The onset of rigor in the left ventricle is attended by contraction (or retraction) of considerable force—sufficient to get up a pressure of 30 mm. Hg. (in the cat) in a manometer connected with its cavity. In some cases slow minor contractions and relaxations occur after the persistent contraction of rigor is more or less fully developed.

While rigor is the chief factor in determining the post-mortem condition of the heart as regards fulness or emptiness of the different chambers, &c., the time of occurrence of coagulation of the blood, and the post-mortem intravascular pressure are also concerned.

After death there is always a positive blood-pressure on both sides of the heart, and this pressure is equal on the two sides. (Cases of hæmorrhage and mechanical interference excluded.)

In cases where the cardiac muscle dies very slowly and goes on discharging energy long after the circulation has ceased, rigor is commonly less strongly developed, and both ventricles may contain much blood 24 hours after death.

After death from paralysis of the heart induced in various ways, the state of the heart (some hours afterwards) varies widely—(*a*) both ventricles may be contracted and practically empty (morphia, &c.), (*b*) the right ventricle may be distended and the left almost empty—what is usually regarded as an asphyxial distribution of the blood being present (chloroform, lactic acid, ammonia), or (*c*) both ventricles may contain much blood (potassium salts, &c.).

After death by asphyxia, while the most common post-mortem condition is the one which has long been recognised as characteristic of this condition—right side distended, left side comparatively empty. The L.V. is emptied by early and strong rigor; the R.V. is not emptied because rigor is late in appearance, and weak, and unequal in distribution, etc. But in some cases of death by asphyxia both ventricles are found contracted and empty, and in other cases both contain much blood.

After death by chloroform it is impossible to determine with certainty from the post-mortem examination of the heart whether failure of the respiration or of the heart had occurred first.