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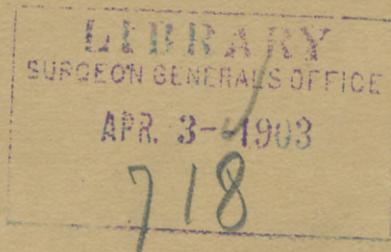
VOL. I.—MARCH 1, 1898.—No. II.

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THE INFLUENCE OF THE HEART-BEAT ON  
THE FLOW OF BLOOD THROUGH THE  
WALLS OF THE HEART.

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[FROM THE LABORATORY OF PHYSIOLOGY IN THE HARVARD MEDICAL  
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THE  
American Journal of Physiology.

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THE INFLUENCE OF THE HEART-BEAT ON THE  
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[From the Laboratory of Physiology in the Harvard Medical School.]

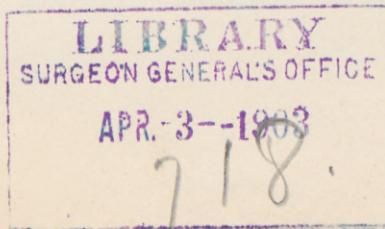
IN 1689 J. Baptista Scaramucci<sup>2</sup> published two hypotheses which have played a great part in the history of the physiology of the heart: the first, that the deeper coronary vessels are squeezed empty by the contraction of the muscle fibres which surround them; and the second, that the coronary vessels are refilled from the aorta during the diastole of the heart. In 1707, Stroem<sup>3</sup> added to these the hypothesis that the coronary vessels are filled in diastole because their mouths are closed in systole by the semilunar valves. Thus, almost at the outset, the penetrating conception of Scaramucci was linked with an obstinate and overshadowing error, destined to be a source of controversy for nearly two hundred years.<sup>4</sup> Thebesius,

<sup>1</sup> The first account of my observations on the compression of the intramural vessels by the systole of the heart was published in the Journal of the Boston Society of Medical Sciences, No. 10, March 30, 1897. Most of the facts have been presented also to the American Physiological Society, in Washington, May 5, 1897, and to the British Association for the Advancement of Science, Toronto, 1897.

<sup>2</sup> SCARAMUCCI: *Diario Parmense*, 1689. Quoted by HALLER: *Element. physiolog.*, 1778, lib. iv, s. v, p. 459.

<sup>3</sup> STROEM: *Nova theoria machinæ animalis*. Quoted by HALLER: *loc. cit.*

<sup>4</sup> A sufficient account of the literature of this subject has already been given by CERADINI, G.: *Il meccanismo delle valvole semilunari del cuore*. Milan, 1871; *Der Mechanismus der halbmondförmigen Herzklappen*. Leipzig, 1872. See also REBATEL, F.: *Recherches expérimentales sur la circulation dans les artères coronaires*. Paris, 1872.



Vieussens, Morgagni, Boerhaave, and other famous eighteenth century men took sides for and against the theory of Stroem, and not until the century had passed, and many observers of the first rank had shown that the mouths of the coronary arteries are often beyond the reach of the semilunar valves, and that the pulse in these vessels is synchronous with the pulse in the aorta, did opinion come to rest on the filling of the coronary arteries in both systole and diastole. The calm that followed was brief indeed. About 1840, Marshall Hall attempted to revive the old belief, but was answered by the experiments of Kleefeld. Five years after Kleefeld, the controversy broke out afresh. Brücke on the one side and Hyrtl on the other, neither knowing that he was repeating arguments and observations that already filled many pages of cardiac literature, fought over the old ground, drew many with them into an extended and often unprofitable discussion, — and reached the old conclusion. Once more physiological opinion settled to the belief that the coronary arteries are filled during systole as well as diastole, — a position since rendered impregnable by the observations of Ceradini and the experiments of Rebatel and of Martin and Sedgwick.

Throughout this long discussion, the primary hypothesis of Scarabucci, namely, that the deeper coronary vessels are emptied by the squeeze of the fibres contracting around them, received but scant attention. Thebesius,<sup>1</sup> in the celebrated inaugural dissertation in which he gave the first accurate description of the cardiac veins that bear his name, had said that in “no way could the arterial blood be forced into the vessels of the heart, unless during diastole; because in systole, the contraction of the fibres is so intense that all blood would be forced out, from the arteries no less than from the veins, — a condition that actually can be observed in the hearts of amphibia — frogs and others — which appear all white and bloodless when contracted, but are red and swollen with blood when relaxed in diastole;” and such reasoning was accepted by many who forgot that the heart of the frog is almost wholly wanting in bloodvessels, and that the red color of the full ventricle is due to the blood which fills the ventricle, seen through its translucent walls.

After the middle of the present century, experiment grew bolder and speculation began to yield place to direct observation. Hyrtl,<sup>2</sup> in 1855, trying to prove that the coronary arteries were filled in both

<sup>1</sup> THEBESIUS: *De circulo sanguinis in corde*. Leiden, 1708, p. 14.

<sup>2</sup> HYRTL: *Ueber die Selbststeuerung des Herzens*. Vienna, 1855, p. 9.

systole and diastole, severed a coronary artery in the living rabbit, cat, and dog, and declared positively that only the upper segment spurted in systole, — a statement confirmed by Perls.<sup>1</sup> In 1876, Klug<sup>2</sup> drew a ligature about the rabbit's heart at the auriculo-ventricular junction while the heart was in full systole, and again while in diastole. He then coagulated the blood in the cardiac vessels by holding the organ some time in dilute sulphuric acid, and compared thin sections of the ventricle with regard to the amount of blood in their walls. The vessels of the heart ligated during diastole were filled with blood, while those of the heart ligated during systole contained little blood. But neither of these experiments can be said to be of value in our present inquiry: for the observation of Hyrtl, though accurate for his purpose, which was to determine which limb of the severed artery "spurted," is otherwise incorrect; and the method of Klug is open to objections based upon facts discovered since his time.

With regard to Hyrtl's work, it is true that the distal segment of a severed coronary artery does not "spurt," but it is also true, as will be shown in detail in the description of the writer's experiments, that blood is forced out of the distal segment with each contraction of the ventricle. The quantity which thus escapes is extremely small, but this is because the amount of blood contained in the distal segment of a severed "terminal" artery is always necessarily small. The anastomosis with neighboring vessels is too slight to permit of collateral circulation, and only a free collateral circulation can cause the distal end of a severed artery to bleed profusely.

Turning to Klug's experiment, let us consider first the heart ligated in systole. Klug slowed the heart in order to be sure of the moment of ligation. The reader will not need to be reminded that when the beat of the mammalian heart is considerably slowed by exhaustion, or by artificial means, as in Klug's method, the cavity of the ventricle is seldom, if ever, fully emptied. The observations of Pratt<sup>3</sup> have shown the ease with which the veins in the heart wall are filled from the cavity of the ventricle through the vessels of Thebesius. It is clear that even if a ligature could be drawn tight around the auriculo-ventricular junction in the precise fraction of a second during which the mammalian heart remains fully contracted, the relaxation of the

<sup>1</sup> PERLS, M. : *Archiv für pathologische Anatomie*, 1867, xxxix, p. 189.

<sup>2</sup> KLUG, F. : *Centralblatt für die medicinische Wissenschaften*, 1876, p. 134.

<sup>3</sup> PRATT, F. H. : The nutrition of the heart through the vessels of Thebesius and the coronary veins. *American journal of physiology*, 1898, i, p. 86.

ventricle after the tying of the ligature would fill its capillaries from the ventricular cavity, so that the amount of blood in the ventricular walls when the heart came to be examined would in no wise correspond to the amount present in the walls at the height of their contraction. If the heart is permitted to beat at its usual rapid rate, the ventricular cavities may be fully emptied at each stroke; but the time for the tying of the ligature is then so short that it is obviously impossible to be sure whether the ligation is made in full systole or a little before or after systole. If it were possible to be sure of the moment of ligation, and to make certain that the ventricular cavities were empty at that moment, and that the ligature shut off the auricles entirely, — the mural capillaries could still be filled when the heart relaxes from the blood in the large superficial coronary vessels, which are not within the grasp of the contracting fibres and cannot be compressed by them. In the heart ligated in diastole, it cannot be determined whether the blood found in the intramural vessels was present there at the moment of ligation, or entered the walls afterward through the veins of Thebesius or the superficial coronary vessels. Finally, the plunging of the fresh heart, warm from the body, into a coagulating bath of sulphuric acid, may so change the tonus of the ventricle as to alter materially the amount of blood in its capillaries. These sources of error render the observations of Klug unavailable.

It is Rebatel<sup>1</sup> whom we must thank for the first fruitful experiment in this field. Chauveau had given him the circulation in the coronary arteries as the subject of his inaugural dissertation, and had suggested that a T-tube should be placed in the right coronary artery of the horse and connected with an hæmodromograph, which should write a curve of the quickness of flow in the coronary artery, while at the same time a curve of the tension in the aorta should be recorded for purposes of comparison. Rebatel secured these curves, and saw at a glance that the beginnings of the upstrokes in the aortic and the coronary curves coincided exactly, showing that the blood-wave is synchronous in the two arteries, and that the coronary arteries are filled during systole. He saw also that the primary increase in the rapidity of current in the coronary artery was followed by a second augmentation "corresponding exactly to the moment when the aortic tension is least, *i. e.*, to the diastole of the heart. The first augmen-

<sup>1</sup> REBATEL, F. : Recherches expérimentales sur la circulation dans les artères coronaires. Paris, 1872.

tation," Rebatel asserts, "is evidently due to the propulsion imparted to the column of liquid by the contraction of the ventricle; the second current may be due to the entrance of a new wave from the aorta

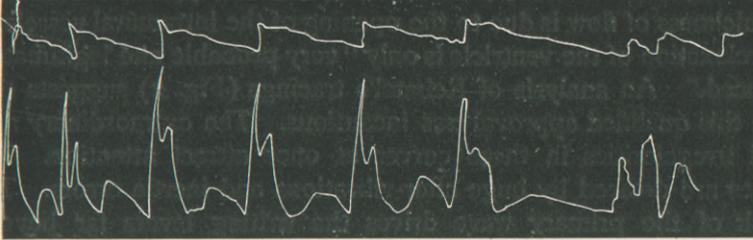


FIG. 1. Curves of the tension in the aorta (upper tracing) and the quickness of flow in the right coronary artery of the horse, simultaneously recorded (Rebatel's Fig. 3, page 25).

into the coronary artery, or to a sudden diminution of the peripheral resistance in the intramural vessels (p. 27)." To determine the origin of the second current, the tension and the quickness of flow in the coronary artery were recorded simultaneously. It was then seen that the tension curve was like that of every other artery, and presented no secondary rise or other feature that could account for the secondary augmentation in the quickness of flow. Thus led to a variation in the peripheral resistance, Rebatel concluded that the primary blood-wave

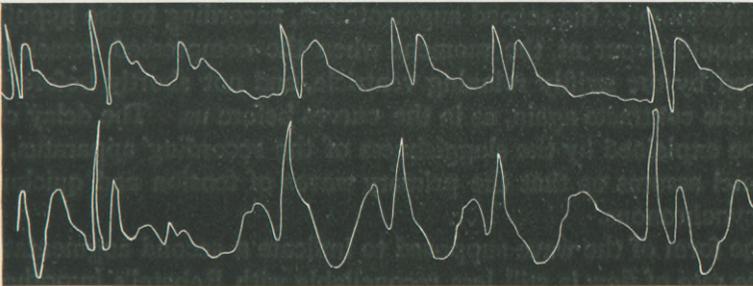


FIG. 2. Curves of the tension and quickness of flow in the right coronary artery of the horse, simultaneously recorded (Rebatel's Fig. 8, page 29).

penetrates with difficulty into the intramural branches during systole, because of their compression by the contracting cardiac muscle, but when the relaxation of the ventricle opens the peripheral vessels, the pent stream rushes suddenly forwards, and thus produces the second, or diastolic, rise in the curve of the hæmodromograph.

Rebatel himself does not accept this hypothesis unreservedly. His attitude is that of M. Marey,<sup>1</sup> who, on being shown the curves, admitted that the first proposition, namely, the filling of the coronary arteries in systole, is incontestable, while the second, namely, that the increase in quickness of flow is due to the opening of the intramural vessels by the relaxation of the ventricle is only "very probable and legitimately deduced." An analysis of Rebatel's tracings (Fig. 2) suggests that even this qualified approval was incautious. The extraordinary artificial irregularities in these curves at once attract attention. The curves are defaced by huge after-vibrations and inertia errors. The shock of the primary wave drives the writing levers far beyond the real maximum of the upstrokes; there is then a sharp rebound, which, in several instances, carries the writing points far below the correct level of the curve. Evidently the lever of the hæmodromograph, once set in motion by the sudden and violent changes in pressure and velocity consequent on the beat of the heart, has continued to swing. Serious as these faults are, they are by no means the chief reason for doubting the correctness of Rebatel's reasoning. According to him, the second augmentation is caused by the relaxation of the ventricle opening the compressed intramural vessels. But this relaxation occurs in the first half of the cardiac cycle, as shown by the position of the dicrotic notch in those of his tension curves that are written clearly enough to make the dicrotic notch visible. Hence, the maximum of the second augmentation, according to the hypothesis, should occur at the moment when the compressed vessels are opened by the swiftly relaxing ventricle, and not shortly before the ventricle contracts again, as in the curves before us. The delay cannot be explained by the sluggishness of the recording apparatus, for Rebatel assures us that the primary waves of tension and quickness are synchronous.

The form of the wave supposed to indicate a second augmentation of the rate of flow is still less reconcilable with Rebatel's hypothesis. The alleged increase in velocity quickly reaches its maximum, and is always succeeded by a rapid fall, greater in many of the tracings than the rise which precedes it. In the absence of any change in the blood-pressure either in the aorta or in the coronary arteries, it is impossible to understand how this slowing in the blood stream can take

<sup>1</sup> M. Marey, que nous remercions d'avoir bien voulu examiner nos tracés, admet, ainsi que nous, la première proposition comme incontestable, et la seconde comme très-probable et légitimement déduite (p. 31).

place. If a reservoir containing water kept at a constant level and provided with an elastic outflow tube is raised 130 cm. above the mouth of the tube, thus giving a constant pressure like that of the blood in the coronary arteries, shown to be constant by Rebatel's tension curves, and the mouth of the tube compressed, as the tubes of the coronary system are said by Rebatel to be compressed, and then released, while the water flowing out during two brief successive periods is measured, — it will be found that the outflow per unit of time, or in other words the velocity, is even a little greater in the second period than in the first. His second augmentation should, therefore, not have been followed by a marked slowing in the rate of flow.

Thus, seeing that Rebatel's second augmentation of velocity is not synchronous in his own curves with the relaxation said to be its cause, and perceiving that the form of the curve offered by him in evidence is physically improbable under the conditions premised by him, we may conclude that his results do not prove his assumption that the intramural vessels of the heart are compressed in systole.

I have spoken thus fully of Rebatel's work both because of its intrinsic interest and because his are the only recorded experiments that bear directly upon the problem in hand. It is true that Martin and Sedgwick,<sup>1</sup> ten years after Rebatel, recorded simultaneously curves of the blood-pressure in the carotid artery and in a branch of the left coronary artery; but their tracings were taken with a mercury manometer, and show nothing more than the synchronism in the primary pulse-wave, finer details being obscured by the inertia of the mercury.

## I.

My own observations upon the characters of the coronary pulse began Sept. 16, 1895, with the record of the pressure-curve in the carotid and left coronary arteries in the dog. It seemed *a priori* probable that variations in the peripheral resistance in the coronary arteries would be visible in the pressure-curve, provided it were written with a sensitive manometer.

The heart of a dog anaesthetized with ether was exposed, the ramus descendens of the left coronary artery ligated about two centimetres from its origin, and a cannula tied into the central end. The

<sup>1</sup> MARTIN, H. N., and W. T. SEDGWICK: *Journal of physiology*, 1882, iii. p. 165.

cannula was then connected by thick-walled but flexible rubber tubing to a glass tube, which led to a sensitive Hürthle membrane manometer, placed on the level of the artery. Evidently a manometer thus situated must receive the pressure-changes in the ramus circumflexus of the left coronary artery and in the branches given off by the ramus descendens in the first part of its course, *i. e.*, between its origin and the cannula. A second manometer recorded simultaneously the changes of pressure in the carotid artery. But the hope of securing a curve from the coronary arteries differing from the pressure-curve

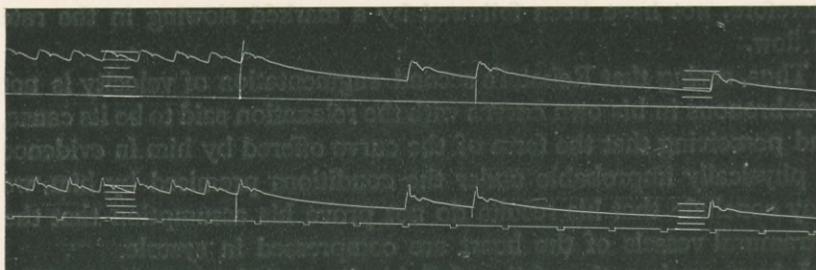


FIG. 3. Sept. 16, 1895. Curves of the blood-pressure in the left coronary artery (upper tracing) and the carotid artery (lower tracing) of the dog, recorded simultaneously. One half the original size. The horizontal line below each curve is the line of atmospheric pressure. In the case of the carotid artery, the atmospheric pressure line served also for the record of the time, in fifths of a second. The intervals of the graduation-scales correspond to a pressure of 20 mm. Hg. On raising the pressure in the Hürthle manometers to 100 mm. Hg, as here recorded, and then opening the chamber of the manometer to the pressure of the atmosphere, the writing points returned accurately to the line of atmospheric pressure, — this line in the pressure-scale being thus twice drawn. The vertical lines are synchronous ordinates. During the latter part of the curves, the heart was slowed by vagus excitation.

of other arteries was not realized. The most careful scrutiny of the two curves taken during the ordinary contractions and during the slowing of the heart by vagus excitation (see Fig. 3) failed to reveal any noteworthy difference, except that the pulse-wave reaches the coronary artery sooner than the carotid, depending of course on the nearness of the former vessel to the heart.

The first fully satisfactory evidence of the effect of the contraction of the ventricle on the flow of blood through the walls of the heart was secured during the writer's experiments on extirpated portions of the ventricle of the dog and cat. When a piece of the mammalian ventricle is kept beating by supplying it with defibrinated blood

through its nutrient artery at a constant pressure, each beat can be seen to force the blood out of the severed vessels in the margins of the fragment. The details of several of these experiments are as follows: —

*Experiment March 29, 1897.* A dog weighing 11 kilos, anæsthetized with morphia and ether, was bled from the left carotid artery, and the blood whipped, filtered through glass wool, and diluted with an equal volume of 0.8 per cent normal saline solution. Normal saline of the same strength was meanwhile allowed to flow into the right jugular vein. After a short interval, the dog was again bled from the carotid artery. A second injection of saline solution was followed by a third bleeding. The product of these bleedings was mixed, and placed in a reservoir at the temperature of the body. The heart was now extirpated, and a cannula tied into the ramus descendens of the left coronary artery not far from the apex of the left ventricle. That part of the apex which could be fed through the cannula was then excised. Both apex and basal portion fibrillated. The septum was removed. The piece of ventricle secured was 28 mm. in length (*i. e.* the direction from the base to the apex), 23 mm. broad opposite the end of the cannula, and 27 mm. broad at the somewhat flattened tip of the apex. The ventricle measured from base to apex 70 mm. The cannula was now connected with the blood reservoir and the apex perfused with blood. In a few moments regular and strong contractions set in. Curves were recorded with an ordinary muscle lever. The flow of blood from the veins was increased during each systole. The experiment was stopped after the apex had contracted one hour and forty minutes. During a part of this time the preparation was in a bath of blood at the temperature of the body.

*March 30, 1897.* On the morning of this day, a cannula was placed in the ramus descendens of a dog prepared as in the foregoing experiment, and most of the left ventricle and all of the right ventricle and septum except a fringe near the arteria descendens cut away. The portion remaining was fed through the cannula with defibrinated dog's blood, and beat strongly and at first quite regularly. It was observed that the outflow from the veins was increased at each systole. Distinct pulsations synchronous with the contractions of the heart-fragment were observed in the vena descendens at the point where it crosses the auriculo-ventricular groove. A cannula was tied into this vein, and a pulsation of the liquid in the cannula noted.

*April 5, 1897.* A pulse synchronous with systole was observed in the liquid in a cannula placed in the coronary artery of a piece of dog's ventricle fed with defibrinated blood from a reservoir at a constant pressure.

*April 9, 1897.* The circumflex area of the left ventricle of a cat's heart was fed with defibrinated cat's blood at a constant pressure through a cannula

placed in the ramus circumflexus. A vein on the surface of the ventricle was incised, and a little stream of normal saline solution allowed to flow over the opening, so as to prevent the blood collecting there. By this means a clear view of the wound in the vein and the escaping blood was secured. The discharge from the vein was then seen to be distinctly greater with each contraction of the ventricle. The superficial veins in a fragment of the auricle left attached to the preparation were observed to be nearly obliterated by each systole of the auricle. The pulse in these auricular veins could not have been caused by the rhythmic contractions of the coronary sinus, for the pulse in the veins continued after their separation from the sinus. Moreover, a similar pulse, noted in the superficial ventricular veins, ceased when the ventricle stopped beating, although the coronary sinus continued to contract.

The effect of the contraction of the heart on the contents of the intramural vessels can also be demonstrated in the living animal, as the next experiment will show.

*April 12, 1897.* A dog weighing 24 kilos was anæsthetized with morphia and ether, and the heart exposed by the resection of a part of the first five ribs on the left side. A branch of the vena descendens was incised about midway between the base and the apex of the ventricle, and a small stream of warm 0.8 per cent normal saline solution allowed to flow over the spot in order that the wound and the quantity of blood escaping from it might be readily seen. The vagus was now divided in the neck, and the peripheral end stimulated with induction shocks of such a strength that the ventricle was not continuously inhibited, but still gave occasional beats. Each time the ventricle contracted, the blood gushed from the vein. The increased outflow appeared absolutely synchronous with the contraction.

An eye-witness of this experiment could hardly have been persuaded that the gush of blood from the vein in systole was due to the transmission of the arterial pulse wave through the capillaries into the veins, yet it seemed advisable to answer this possible objection by direct experiment.

*July 22, 1897.* The experiment of April 12 was this day repeated, and again each contraction of the ventricle caused a greatly increased outflow from the vein. The vagus inhibition being prolonged, the heart swelled greatly, and the occasional contractions which broke through the inhibition were very strong. Each of these powerful contractions caused the blood to spurt from the vein. The heart was now excised, and the aorta connected with a reservoir of defibrinated dog's blood much diluted with 0.8 per cent

NaCl solution. The pressure in the reservoir was about 100 mm. Hg, so that, as soon as the connection with the aorta was made, the blood from the reservoir filled the artery, closed the semilunar valves, and passed through the coronary vessels. The perfused heart beat for a few minutes with considerable strength. With each beat the wound in the vein spurted, as an artery spurts when severed in the living animal.

The following experiments show that the squeezing of the vessels by the contracting muscle fibres makes itself evident in the arteries as well as in the veins. In this connection, it should be remarked once more, that the coronary arteries are "terminal arteries." In the absence of a collateral circulation, any pulsation observed in the distal segment of a coronary artery after its ligation is probably due to the compression of the intramural vessels by the contraction of the heart, and not to the transmission of an arterial pulse through collateral branches from other arteries.

*November 18, 1897.* A dog was anaesthetized with morphia and ether, and the heart exposed by the resection of the first five ribs on the left side. The ramus descendens was then ligated about 20 mm. from its origin. The artery was now incised 10-12 mm. distal to the ligature. A little blood escaped from the wound. On stimulating the vagus so that the ventricle contracted only occasionally, and allowing a small stream of warm normal saline solution to flow over the opening, it was possible to see plainly that each beat forced blood out of the artery. There was no visible delay between the beat and the outflow. The artery was now tied a few millimetres distal to the wound. The slight flow from the artery then ceased altogether, but during each systole a little blood appeared at the mouth of the wound in the artery.

The next day, a very high constant pressure was suddenly made in the aorta of a living dog, so that the semilunar valves were kept closed for a time, the pressure on their aortic side being greater than the maximum pressure in the left ventricle. The coronary circulation was fed during this time not by the beat of the ventricle but by the blood in the pressure-reservoir. Nevertheless, each beat of the ventricle forced blood out of the incision made in a coronary vein on the surface of the ventricle and out of a wound made in the arteria descendens distal to a ligature which had been placed around it. The details are as follows:—

*November 19, 1897.* The great vessels and heart of a dog anaesthetized with morphia and ether were exposed by resecting five ribs on the left and

three ribs on the right side and removing the upper part of the sternum. Cannulas were placed in the right and left carotid arteries. The right subclavian artery was ligated at its origin, and the left subclavian artery and the aorta prepared so that they could be clamped at the proper moment. The cannula in the left carotid artery was connected with a reservoir containing 0.8 per cent NaCl solution at a pressure of 140 mm. Hg. The cannula in the right carotid was connected to a mercury manometer, which showed a maximum pressure of 51 mm. Hg (the heart being rather feeble from long exposure). A vein on the surface of the left ventricle was now incised. The venous blood escaped from the wound in weak jets synchronous with the contractions of the ventricle, which were infrequent enough to permit the outflow to be seen distinctly. The stopcock between the carotid artery and the reservoir of saline solution under pressure was now opened and the left subclavian artery and the aorta clamped. The pressure in the manometer connected with the right carotid artery then rose to more than double its former maximum height. The semilunar valves were kept shut by this very high pressure in the aorta. The left ventricle, unable to open the semilunar valves, became greatly distended. An observation on the outflow from the incised artery and vein was made the moment the high pressure in the aorta closed the semilunar valves, before there could possibly have been time for the heart-beat to change sufficiently to overcome a pressure nearly three times as great as the former maximum arterial pressure, if indeed it could ever have done so. The blood still emerged from the vein in gentle systolic jets. The wound in the artery merely oozed, but the quantity escaping was distinctly greater in systole.

The emptying of the intramural vessels by the systolic squeeze of the fibres around them has been repeatedly observed in this Laboratory in the course of experiments on the extirpated heart of the cat, and has recently been admirably demonstrated by my friend, Mr. F. H. Pratt, by suspending a strip of the cat's heart, fed through one of the coronary arteries, in a large vessel of normal saline solution. The experiment is so instructive that it seems worth while to describe in this place a simple method by the aid of which the phenomena may be very easily shown.

A cat is anæsthetized with ether and cannulas placed in the left carotid artery and the right jugular vein. The animal is now bled from the artery. When the blood no longer flows except in drops, the artery is clamped, and 0.8 per cent NaCl solution at a temperature of 37° C. allowed to flow slowly into the jugular vein. When the blood vessels are well filled with saline solution, the cat is bled

again. The blood drawn in the first bleeding is diluted one half with the normal saline solution. The defibrinated blood mixture from both bleedings is then placed in a Mariotte tube, 30 cm. long and 3 cm. in diameter, of 190 c.c. capacity. The Mariotte tube opens below into a vertical glass tube about 5 mm. in diameter, on the end of which is a cannula provided with a stopcock. The cannula is inserted in the ramus descendens or the ramus circumflexus of the left coronary artery and all the heart cut away except that part of the ventricle supplied by the chosen artery. The fragment of the ventricle is now suspended in a very large beaker, filled with warm normal saline solution. When the Mariotte tube, the connecting tube, and the cannula are filled with the defibrinated blood, the height of the liquid column is about 65 cm., giving a blood-pressure of about 50 mm. Hg in the coronary artery. On opening the stopcock between the cannula and the upright tube, the blood circulates through the coronary artery and its branches, and the fragment of ventricle presently begins to beat. With each contraction the blood shoots from the severed vessels in the margins of the fragment some distance into the surrounding liquid, making a funnel-shaped cloud in the clear saline solution.

## II.

Having thus demonstrated the pressure which the muscular fibres in the heart exercise upon the intramural vessels during systole, it remains to consider to what extent this constriction and subsequent relaxation assist the flow of blood through the heart walls. That they do assist the flow of blood through the heart walls seems *a priori* probable; it is, indeed, difficult to imagine how the periodical squeezing of vessels communicating on the one hand with the aorta, a reservoir in which the pressure is always relatively very high, and on the other with outflow channels in which the pressure is always relatively very low, could fail to drive the blood towards the point of low pressure, *i. e.* into the veins. But these premises do not justify the conclusion that the systolic compression of the intramural vessels increases the total volume of the coronary circulation. This is quite another problem, and one which cannot be answered from the data thus far brought forward. It has just been demonstrated that the circulation through the intramural vessels is diminished during the contraction of the fibres around them. The emptying of the vessels

and their subsequent refilling is favored by this same rhythmic contraction. Which of these factors has the upper hand? Does the check which the circulation through the walls sustains during systole diminish the total volume of blood passing through the wall per minute, or is the lessening more than made up by the favorable factors—the emptying of the intramural vessels and their easier refilling? The experiments next to be described afford a partial answer to this question.

In February, 1896, while studying with Messrs. Magrath and Kennedy the relation of the volume of the coronary circulation to the frequency and force of the ventricular contraction in the isolated heart of the cat, I observed that the heart took more blood through the coronary arteries from a reservoir under constant pressure when contracting than when at rest. The same observation was made again, later in that year, when at work with Miss Hyde on the effect of the distention of the ventricle on the flow of blood through the walls of the heart. The fact is very well demonstrated by Fig. 4.

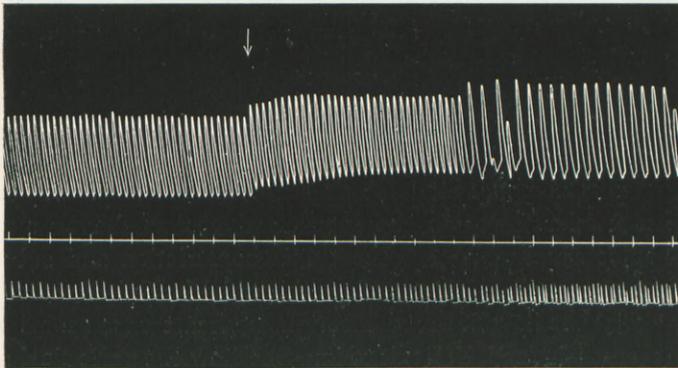


FIG. 4. Showing the increase in the volume of the coronary circulation consequent on an increase in the force of ventricular contraction. The uppermost tracing is the pressure in the left ventricle of the isolated heart of the cat, recorded by a Hürthle manometer; the next is the time, in seconds; and the lowermost is the record of the drops of blood flowing through the coronary vessels. The arrow points to the distention of the ventricle, which shortly calls forth beats of greater force. From an experiment performed with Miss I. H. Hyde.

In this experiment the extirpated heart of a cat was fed with warm defibrinated cat's blood from a reservoir at constant pressure through a cannula in the ascending aorta, all the branches of that vessel except the coronary arteries having previously been tied. The blood

passed from the coronary artery into the right ventricle, and thence through a glass tube, drop by drop, onto an aluminium plate fastened upon the lever of a Marey tambour.<sup>1</sup> The variation in the air pressure in the tambour occasioned by the falling drops was transmitted through a connecting tube to a second tambour, provided with a small chamber, thin membrane, and very light moving parts, and recorded by its writing lever upon the smoked paper of a kymograph. With this record of the number of drops of blood passing through the coronary vessels was written the pressure in the left ventricle, the cavity of which was filled with normal saline solution and connected with a sensitive Hürthle membrane manometer. A side branch led from this cannula to a Mariotte flask placed higher than the heart and filled with normal saline solution. When the stopcock leading to this flask was opened, the pressure in the left ventricle rose, as shown by the rise in the base line of the curve. After a few seconds the stimulus of the increased intracardiac pressure caused the ventricle to beat with greater force and the volume of the coronary circulation became greater, — and this in spite of a diminished frequency of contraction. Later, the pressure in the ventricle was lowered to that of the atmosphere, the ventricle contracted less vigorously, and the volume of the coronary circulation was correspondingly reduced.

A diminution in the volume of the coronary circulation in consequence of lessening the frequency of contraction is demonstrated by Fig. 5. The uppermost curve in this figure records the pressure in the left ventricle of the isolated heart of the cat, fed through the aorta and coronary vessels with defibrinated cat's blood at a constant pressure and temperature. The ventricle was filled with saline solution and connected with a Hürthle membrane manometer. The second curve was written by the armature of an electro-magnet placed in the primary circuit of a du Bois-Reymond inductorium. The heavy white line records the stimulation of the peripheral end of the vagus nerve with a weak induced current; the individual strokes of the armature are blended, owing to the slow speed of the smoked paper. The third curve marks the number of drops of blood flowing through the coronary vessels, the recording apparatus being that used for the experiment illustrated by Fig. 4. The fourth curve marks the time in seconds. The weak excitation of the vagus diminished the

<sup>1</sup> For the details of this method and a discussion of its sources of error, see MAGRATH and KENNEDY: *Journal of experimental medicine*, 1897, ii, p. 13.

frequency of ventricular contraction, but left the force unchanged. The volume of the coronary circulation lessened when the frequency of contraction lessened, and was restored with the restoration of the former frequency.

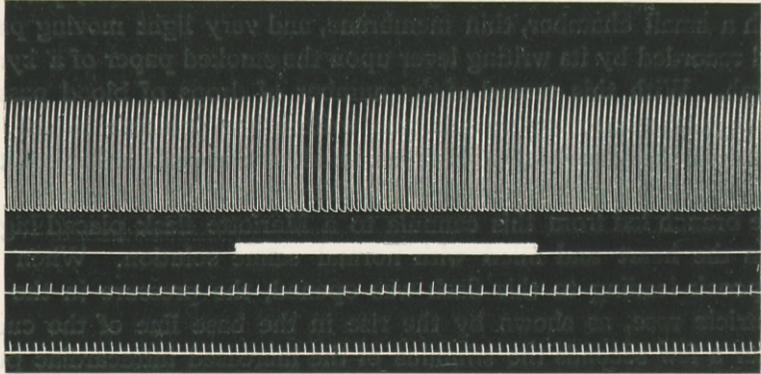


FIG. 5. March 26, 1896. Showing the lessening in the volume of the coronary circulation consequent on a lessening of the frequency of the heart-beat. The uppermost tracing is the pressure in the left ventricle of the isolated heart of the cat; the line below was drawn by the writing point of an electro-magnet placed in the primary circuit of the inductorium, the broad, white band indicating the duration of vagus stimulation; the next curve records the number of drops of blood passing through the coronary vessels; and the lowermost tracing is the time, in seconds. The weak excitation of the vagus finally lessens the frequency of contraction,—at once the volume of the coronary circulation is also lessened.

It would seem, then, that an increase in either the force or the frequency of the contractions of the heart increases the volume of blood passing through the coronary circulation by means of the periodical emptying of the intramural vessels,—yet it would not be prudent to accept this conclusion unreservedly. Two possible sources of error suggest themselves. The recorded changes in the volume of the coronary circulation may depend upon alterations in peripheral resistance in consequence of changes in the tonus of the heart muscle, or they may be due to changes in the vascular tonus in consequence of the action of vasomotor nerves. The first of these sources of error may be excluded with considerable certainty. The base line of the intraventricular pressure curves in Figs. 4 and 5 gives no evidence of changes in tonus; Fig. 5 is particularly convincing. The possible action of vasomotor nerves cannot be wholly excluded. Yet the pronounced synchronism between the changes

in frequency and the changes in the volume of the coronary circulation in Fig. 5 points toward a mechanical explanation, and seems to warrant the statement that the increase in the volume of the coronary circulation which accompanies an increase in the force or frequency of the heart-beat is probably to be explained by the periodical emptying of the intramural vessels by the contraction of the heart.

### III.

It is conceivable that the emptying of the intramural vessels by the contraction of the heart may favor the flow of blood through the heart walls in two ways: first, by the diminished resistance which the empty patulous vessels should offer to the inflow of blood from the aorta when the heart relaxes; and second, by the suction which might accompany the sudden expansion of the compressed vessels,—expanding either by virtue of their intrinsic elasticity, or because of the pull of the surrounding tissues upon their walls, as the heart quickly regains its diastolic form. It will be best to begin with the second problem, namely, the possible suction of the relaxing heart muscle.

The method by which this problem was attacked consists in suddenly connecting the distal portion of a coronary artery of the strongly beating heart with a small reservoir of blood at the atmospheric pressure. If each compression of the deeper branches of the artery were followed by an expansion sufficient to cause a noteworthy suction, the blood in the reservoir should be drawn into the artery; for this blood is the sole source of supply throughout the experiment, the “terminal” nature of the coronary arteries preventing any material backflow from collateral branches. It will be seen from the experiments about to be cited that no appreciable suction can be demonstrated in the larger coronary arteries, even when a very sensitive minimum valve is interposed between the artery and the reservoir in order to prevent the possible masking of the suction by rising pressures accompanying the contraction of the ventricle.

*April 14, 1897.* The heart and great vessels of a dog anæsthetized with morphia and ether were exposed by the removal of a part of the chest wall. A glass cannula, 177 mm. long and 3.5 mm. in diameter, bent near the end as illustrated by Fig. 6, and furnished with a stopcock and a side branch leading to a minimum manometer, as shown in Fig. 7, was connected with a

reservoir containing warm defibrinated dog's blood, obtained in the manner described in the Exp. March 29, page 153. The pressure in the blood reservoir

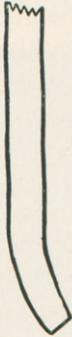


FIG. 6. Lower end of glass cannula for perfusing the ramus circumflexus in the living animal.

was maintained at a constant level of about 80 mm. Hg. (The exact reading of the mercury manometer connected with the reservoir was inadvertently omitted from the protocol.) The minimum valve and its manometer were filled with 0.8 per cent NaCl solution, and the cannula and the connecting tubes with defibrinated dog's blood. The long cannula was now rapidly passed through the innominate artery, aorta, and left coronary artery into the ramus circumflexus, which it filled completely, and the stopcock leading to the blood reservoir opened. The stopcock leading to the minimum manometer had previously been closed. The defibrinated blood entered the artery at about the normal temperature and pressure and maintained a satisfactory circulation. The heart continued to beat strongly and regularly. The blood reservoir was now suddenly shut off, and the stopcock leading to the minimum manometer as suddenly opened. The contents of the manometer passed slowly into the artery,

but on comparing the level of the liquid in the manometer, with that of the heart it was found that the manometer was higher than the heart. The slow emptying of the manometer may therefore have been due to gravity. The experiment shows at least that there is no strong suction, otherwise the manometer would have been emptied rapidly.

*April 15, 1897.* The foregoing experiment was repeated, but no suction could be demonstrated.

*April 16, 1897.* The experiment was varied by tying a cannula into the ramus descendens, opened for the purpose on the surface of the ventricle near the origin of the artery, and connecting this cannula with a minimum manometer, filled, as before, with normal saline solution. But no suction could be found.

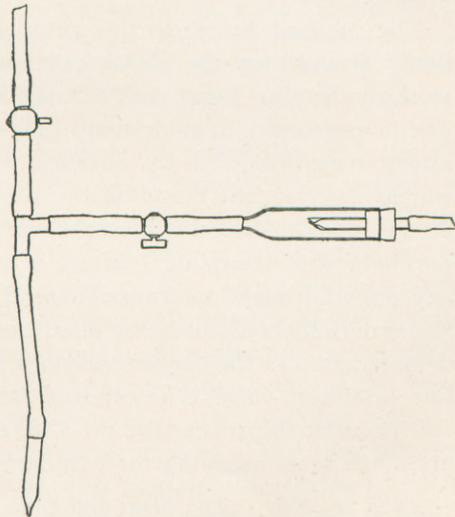


FIG. 7. Minimum valve and cannula, one-fourth actual size.

But no suction could be found.

April 23, 1897. A cannula tied into the ramus descendens of a dog's heart was furnished with a T-tube, one limb of which led through a minimum manometer to a nearly horizontal glass tube filled with normal saline solution, while the other led to a reservoir from which the descendens area was supplied with warmed, defibrinated dog's blood at about the normal pressure (Fig. 7). While the heart was beating well, the descendens area being fed from the pressure flask, the latter was suddenly cut off and the stopcock leading to the minimum valve tube opened. There was no suction, although the conditions of the experiment were all favorable to its discovery.

Experiments similar to that of April 16 on the dog have been tried on four cat's hearts (Nov. 11-17), but also without finding any suction.

It should be remarked that these are all negative results. Against a single positive result they would be worthless. Yet I am obliged at present to conclude that the relaxation of the heart wall does not produce a suction in the larger coronary vessels.

Having failed to demonstrate any suction in the coronary arteries during the diastole of the heart, it is necessary to accept the alternative explanation of the favorable influence of the heart-beat on the flow of blood through the heart-walls, namely, the diminished resistance which the empty patulous vessels offer to the inflow of blood when the heart relaxes.

#### SUMMARY.

1. Curves of the blood-pressure in the carotid and the coronary artery, recorded simultaneously by two sensitive membrane manometers, reveal no noteworthy difference in the form of the pulse-wave.
2. The intramural branches of the coronary vessels are compressed by the contraction of the muscle fibres around them.
3. The volume of blood passing through the coronary vessels is increased by an increase in either the force or the frequency of the heart-beat.
4. It is probable that this increase in the volume of blood passing through the coronary vessels is accomplished largely through the periodical emptying of the intramural vessels by the systolic squeeze of the fibres around them.
5. The emptying of the intramural vessels by the contraction of the heart favors the flow of blood through the heart-walls chiefly by the diminished resistance which the empty patulous vessels offer to the inflow from the aorta when the heart relaxes.
6. The relaxation of the heart-walls does not produce a noteworthy suction in the larger coronary vessels.





