On the law which regulates the frequency of the pulse / by A.H. Garrod.

GARROD, A.H.

The Law which regulates the Frequency of the Pulse.

London, H.K. Lewis, 1872.

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ON

THE LAW WHICH REGULATES

Cat. 54

THE

FREQUENCY OF THE PULSE

BY

A. H. GARROD, B.A. (CANTAB).

LONDON:
H. K. LEWIS, 136 GOWER STREET.
1872.

ON THE LAW WHICH REGULATES THE FREQUENCY OF THE PULSE.

The paucity of mechanical theories to explain the frequency of the pulse, probably arises from the very general assumption, that in all cases when the rapidity of the heart's beat is caused to vary, the action of nerves with special powers of retarding or quickening it, is brought into play; and the relation of heart power to work to be performed, has not been introduced into the problem.

The theory of energy has of late spread so far and wide the necessity for finding in all cases where work is done, a sufficient source for the production of that work, in one form or other, that a vague statement to the effect that heart frequency depends solely on nerve action, is far from sufficient for the requirements of physiologists. It is now necessary to shew that with different amounts of work to be performed in the circulation, different supplies of nutrient substance must be presented to the motor organ, just as in the steam-engine the amount of fuel must be varied according to the work required from the machine.

When the microscope revealed the existence of a well-marked muscular coat to the smaller systemic arteries it became evident that the different diameters of those vessels consequent on the degrees of contraction of their walls, varied the amount of force necessary to propel the blood through them;

and these variations have been considerably studied of late. Dr. Marey of Paris, the introducer of the sphygmograph, has, in his most scientific treatise "on the Circulation of the Blood,"* strongly drawn attention to this subject, and he has worked out a theory respecting the law regulating the frequency of the pulse, which is based mainly on the variations in arterial resistance.

This theory of Marey's it will be necessary to recapitulate here, and to examine the facts on which it rests. The following is the law in the two forms in which he gives it.

1. "The heart beats so much the more frequently, as it experiences less difficulty in emptying itself."

2. "The frequency of the pulse varies inversely as the arterial tension."

As reasons for the accuracy of this law are given—

1st. The analogy of other intermittent muscular movements, as the following—A man can walk a certain distance quicker, the less he is loaded. Or this—The hand can be moved alternately backwards and forwards more quickly in air than in the more resisting fluid, water.

2nd. The pressure can be made to change by variations in the amount of blood in circulation, and by modifications in the degree of arterial or capillary resistance, both of which vary the pulse rate in the manner required by the theory.

To prove the effects of different amounts of blood

* *Physiologie Medicale de la Circulation du Sang.* Paris, 1863.

in circulation, the experiments of Hales are quoted in which he found that loss of blood increased the frequency of the pulse.

To prove the effects of varied arterial or capillary resistance many satisfactory and original results are referred to, among them, the effect of compressing the abdominal aorta, or the femorals, which retards the pulse; the effects of cold baths, according to Drs. Bence Jones and Dickinson, when the pulse was greatly reduced in frequency; the quickened pulse following successive additions of warm clothing over the body is also proved.

From these latter results it is clear that Marey assumes that by varying the capillary resistance the blood pressure is also varied at the same time, but this assumption is not necessarily true in a circulation that is maintained by a pulsating motor organ, whose rate is variable, as can be easily shewn by an analogy from electricity, which is a useful one in many ways to students of the circulation and is quite worth being worked out by each. It is this—Suppose a battery connected, through a break-and-make key, to a long uniform insulated line or telegraph cable, insulated at the other end, and connected with a static galvanometer.

First connect the two parts by the key and thereby charge the line, and then break connection; upon this the charge will fall in tension slowly, and this fall may be observed on the galvanometer; when the tension has fallen one half, reconnect and break again. It is evident that if this process be repeated, a definite current is maintained between the cable and the surrounding bodies to which it

leaks. If the line be now halved in length, whereby the resistance is doubled, and again insulated at the free end, it is evident that by again breaking and making contact as before, when the tension is halved, the maximum tension will not be changed. So with the circulation, if the resistance in the arterial peripheral vessel is varied and the length of the pulsation depends on the time of fall in tension only, the pressure does not vary, if the vascular capacity is constant.

It is thus seen that the blood pressure need not depend on the arterial resistance, but if the pressure does not vary, the pulse rate must do so.

A desire to arrive at the genuine value of this theory of Marey's, led me to make experiments similar to his own, as to the accuracy of his fundamental facts. My observations were divided into two series, to find,—

1st. Whether the pulse rate was related to the capillary resistance.

2nd. Whether the pulse rate depended on the pressure of the blood in the arteries.

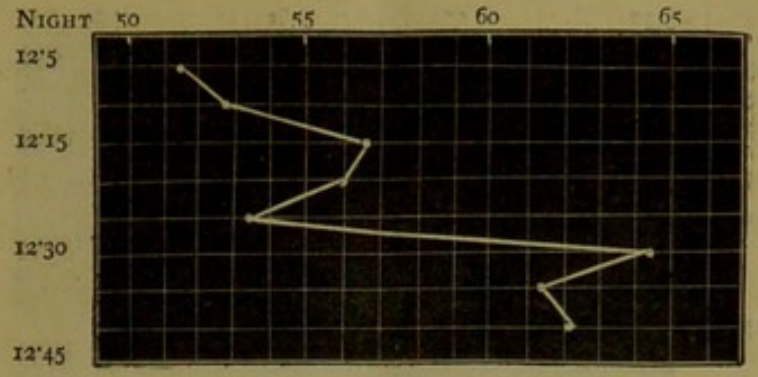
These points will be considered separately.

1st. *The relation of the pulse rate to the arterial resistance.*

The effect of exposing the surface of the body to the influence of different temperatures, whereby, as it has been my endeavour to prove elsewhere,* variations in the calibre of the cutaneous vessels are produced, was carefully examined and the following tables embody my results, the curves being those of changes in pulse rate.

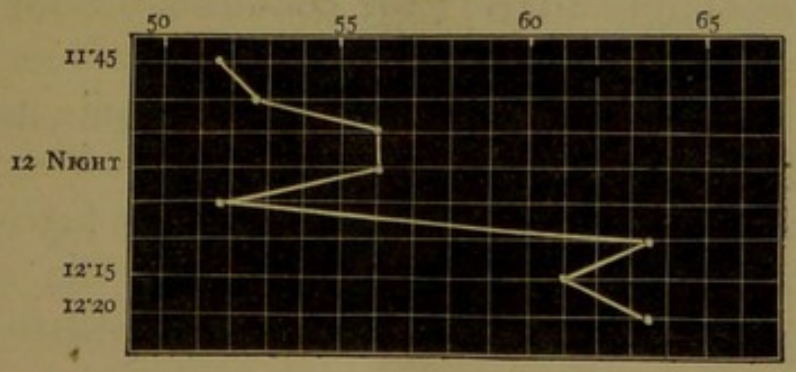
* *Proceedings of the Royal Society*, 1869, p. 419.

Experiment I. Temperature of the air 51·5° F.



Nude at 11·57 P.M. Lay down on floor, carpeted, on right side, at 11·58 P.M., with head on foot stool. Did not feel cold. Got up and put on night shirt and jumped into bed at 12·20; a skin glow came on at 12·29. Same position maintained in bed as when on carpet.

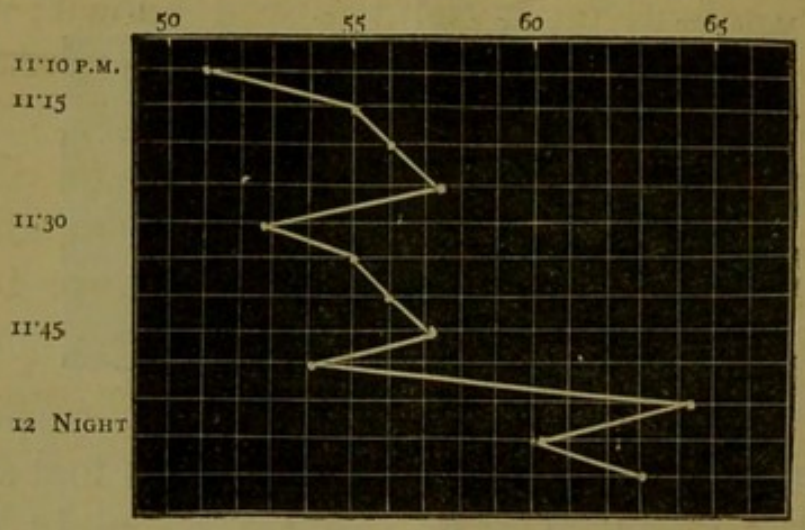
Experiment II. Temperature of air 50° F. Nude



at 11·40 P.M. and lay down on right side. Experiment conducted exactly as the last. Got up at 12 night, and in bed in less than a minute. A glow came on at 12·6.

Experiment III. Temperature of air 52·5° F. To show that the change in pulse-rate did not depend on the effort of getting into bed. Experiment conducted exactly as the first. Nude, lying on right side, at 11·7 P.M. Got up at 11·26 P.M. put

on night shirt, took it off again and lay down again



on floor. Got up again at 11·45′5″ and went into bed, in night shirt. A glow came on at 11·54′5″ P.M.

From these observations it is apparent that the effect of simply altering the condition of the cutaneous vessels, by varying their relations to external agencies, varies the pulse rate in a definite manner; and thermometric results shew that on warming the skin, as by covering it with bad conductors, the vessels are increased in calibre and the arterial resistance reduced. These experiments therefore shew that reducing the resistance quickens the pulse.

Marey's own observations, specially as they are recorded mostly by the graphic method, are of themselves sufficiently convincing on this point. He compressed the abdominal aorta of a horse, per rectum, and found the pulse thereby rendered much slower. The same result followed compression of the human femoral arteries.

The quickened pulse produced by the Turkish

bath, (in one case reaching the extreme rapidity of 172 in a minute on myself,) is well known; as is the slow one following a cold bath, as shewn by Drs. Bence Jones and Dickinson.

From these many facts, all tending in one direction only, it may be stated that the rapidity of the pulse varies inversely as the resistance to the flow of blood from the arteries.

2nd. *The relation of the pulse rate to the amount of blood in circulation, or to the blood pressure in the arteries.*

The following experiments were made—

Experiment IV. An old donkey which had been standing for more than half an hour in the room in which the experiment was conducted, had at 7·30 a.m. a pulse of 34 a minute. At 7·40 half an ounce of chloral hydrate was given it in 2 oz. of water,

At	Pulse a minute.	
7·50′	46	standing unsteadily as if intoxicated.
7·52′		it fell down asleep.
7·55′	43	
7·59′	40	
8·8′	48	a tap having been put in the jugular vein but no bleeding having occurred.
8·12′	52	Bleeding slowly from jugular.
8·15′	67	minute after minute, the animal having lost altogether about one pint or a little more of blood.
,,	64	
,,	62	
,,	60	
8·19′	59	Bleeding freely.
8·20′	52	Bleeding ceased.
8·21′	49	,, ,,
8·22′	48	,, ,,
8·22′ 30″		Bleeding resumed
8·23′ 30″	49	Bleeding. Resp. 11·5

At		Pulse a minute.	
8·24′	15″		Bleeding ceased after loss of another pint and half.
8·24′	30″	43	Bleeding ceased.
8·25′	15″	42	No bleeding. Resp. 11·5
8·26′		42	,,
8·28′	15″	42	Bleeding freely.
8·30′	30″	42	,, ,, Resp. 13·
8·34′	30″	37	,, ,,
8·36′		38	,, ,,
8·40′		37	,, ,,
8·42′		35	,, ,, Resp. 14·
8·45′		36	,, ,, Resp. 16·

and from this time until 9·10′, by which time more than half a pailful of blood had been lost and the carotid pulsations were very feeble, the pulse remained at 35·5 to 35 in a minute with the respirations varying from 12 to 13 in the same time, and the loss of blood being continuous throughout.

The animal did not move once through the whole experiment.

Experiment V. A terrier dog had 30 grains of chloral given it in two doses, 15 grains first and another 15 grains about an hour afterwards. This did not render it quite comatose, so it sniffed chloroform until insensible, when a hæmadynamometer was connected with one of its carotids, and a pressure of 6·6 inches was immediately registered, which was steadily maintained, undulating with the respiration.

	Resp. in a minute.	Pulse in a minute.	Pressure.
	40	186·5	6·45 inches
In 3 minutes	42	192	6·3 ,,
2	40	188	6·4 ,,

	Resp. in a minute.	Pulse in a minute.	Pressure.
In 2 minutes	...	184	6·4 inches
2	49	182	6·6 ,,
1	52	164	6·85 ,,
1	...	159	7· ,,
1	...	150	7· ,,
1·5	70	138	6·9 ,,
4	...	142	6·7 ,,
5	...	149	6·8 ,,
6	...	156	5·8 ,,
7	...	168	6·4 ,,
6	...	102	1·7 ,,
10	...	129	2·1 ,,
5	...	138	1·9 ,,
2	...	141	2·1 ,,
3	64	142	2·1 ,,
5	...	138	1·9 ,,

In this experiment the bleeding occurred from the carotid, and took place between the pulse countings, which were traced on a revolving drum. The great fall in pressure indicates the excessive bleeding in one case. In others a much less quantity of blood was lost on each occasion.

There was no bleeding after the fall in pressure to 1·7 inches and from that time the pressure and pulse became less and less till the animal died.

Experiment VI. A rabbit was made comatose by 15 grains of chloral, and was bled to death, the whole operation taking half an hour. An hæmadynamometer was connected with the carotid and the blood was lost from the jugular of the same side, in drops continuously. The pressure at first, at about 6 inches, fell at the end of the experiment, to less than one inch, when death occurred. The following are the pulse rates taken at equal intervals during the half hour.

	In 10 seconds.	In 10 seconds.
	42·5	42·75
	41·9	43·
	44·	42·
	43·	42·
	42·75	42·5
	41·	41·5
	43·	42·
Bleeding began	46·	42·4
	44·5	41·
	45·5	41·75
	44·75	42·
	43·25	42·
	44·	41·
	45·	41·
	43·5	42·
	43·9	40·6
	43·	42·
	42·	42·
	43·	39·

Experiment VII. A rabbit under the influence of 15 grains of chloral

When	pressure	4·8	inches.	Pulse 136	in a minute.
,,	,,	4·3	,,	,, 133	,,
,,	,,	2·8	,,	,, 127	,,
,,	,,	1·7	,,	,, 132	,,
,,	,,	2·8	,,	,, 133	,,
,,	,,	2·4	,,	,, 158	,,
,,	,,	1·9	,,	,, 144	,,
,,	,,	1·1	,,	,, 133	,,
,,	,,	·75	,,	,, 136	,,
,,	,,	·9	,,	,, 127	,,

death from loss of blood.

From these experiments it is evident that the pulse does not increase in frequency with loss of blood, as it did not do so in any one of them.

In Experiment IV. the pulse rate rose on making

the incision in the skin necessary to expose the jugular vein and continued to do so shortly after bleeding commenced, but soon diminished, and after reaching 36 a minute remained perfectly constant notwithstanding a continuous and considerable loss of blood from the vein until the animal was almost exsanguinated.

With the rabbits the difficulty in keeping them completely under the influence of the hypnotic, with the tendency to struggle, makes the results less uniform, but in all the cases there was a fall in pulse rate, not a rise, accompanying the reduction in blood pressure. This fall, which was not very great, may result from the cooling of the surface, consequent on the lessened circulation.

From these observations it may be concluded that variations in the amount of blood in circulation do not vary the rapidity of the pulse, and consequently, that the pulse rate is not dependent on the blood pressure as Marey supposed.

The next question was—What law as to the frequency of the heart's beats would satisfy these two above proved facts, namely the dependence of the pulse length on the arterial resistance and its non-dependence on arterial blood pressure?

The method adopted by Mr. Fleeming Jenkin for detecting the insulation of long cables at different times occurred to me as being subject to exactly similar laws, the time of fall of cable charge from tension to half tension, which he employs, varying directly as the leakage, and as that only.

Can it be that the heart always recommences to beat when the tension falls a certain invariable

proportion, and then only? This theory it was my next object to analyse, and the different elements into which it resolves itself were, and will be now considered separately.

First, as to the full meaning of the term,—a uniform circulation. A uniform circulation is one in which the quantity of fluid flowing through all cross sections of the circulating system is the same; for if the flow through one part were less than through another, there would be a tendency for the fluid to accumulate in front of the obstruction, which is incompatable with the premises.

As a consequence of this, the heart must always recommence to beat directly as much blood has left the capillaries as was sent out from it in the previous pulsation, and therefore the length of the pause or diastole must depend on the relative capacities of the heart and of the arterial system, and on the rapidity of the flow of blood through the capillaries.

At this point the work of Poiseuille respecting the flow of fluids through capillary tubes is invaluable. He found* that, other things being the same, the flow of fluids through capillary tubes varies directly as the pressure. These results were verified by a Committee of the Academy of Science; and, by an entirely different method, I have been enabled to do the same on the vessels of the animal system.

My method was the following in a particular

* *Recherches expérimentales sur le mouvement des liquides dans les tubes de très-petits diamètres. Rapport de l'Académie des Sciences. Comptes Rendus.* Tome IV. 1842.

case.—The kidneys of a deer, with the aorta and renal vessels intact, were removed from its body and placed for some time in water at 100 F.; the aorta was ligatured just below the origin of the renal arteries, and a uniform glass tube was tied into it just above them. Water at 100 F. was poured into the tube and it distended the organs; the tube was maintained full by a continuous supply which was suddenly stopped and the time of fall of the column from tension to half tension at different initial pressures observed, and it was always found that it took exactly the same time to fall from 40 inches to 20 inches as from 20 inches to 10 inches, thus verifying the law.

This law being thus true it is evident that if the capacity of the arterial system, including the left ventricle, varies directly as the pressure, then the heart must always recommence to beat when the arterial tension has fallen a certain proportion; for with double pressure, and consequently double amount of blood, the time of flow through the capillaries is constant, as the flow varies directly as the pressure; and with double resistance and unvaried pressure the time of flow is double also, for the heart pumps again when as much has gone from the capillaries as it has sent into the arteries and the relative capacities of the heart and arteries do not vary according to the assumption.

But does the capacity of the arterial system vary as the pressure?

This is a point which it is very difficult to prove. With regard to the heart the following facts bear on it. By connecting a syringe with the coronary

arteries, or by tying it into the aorta and pumping backwards, it can be shown that increasing the pressure in the coronary arteries increases the capacity of the ventricles. Also in many post-mortem examinations the heart is found with the ventricular cavities fully obliterated, and as they are not then in action, the capacity of the heart and the pressure in it are at a minimum together. This is all the direct evidence that it is in my power to bring on this point.

With regard to the arterial system and its capacity, the absence of blood in the arteries after death has been known from time immemorial, and if their capacity varied directly with the pressure, it is evident that that must be the case, both capacity and pressure being at a minimum.

A direct method of determining this point having occurred to me, the following description will illustrate it. In a rabbit one of the carotids was put in communication with a kymographion ; and during the time the recording drum was revolving, the chest was suddenly opened and the ventricles cut across transversely. The pressure fell rapidly to zero, and it is clear that the fall must have arisen from the escape of the blood through the peripheral vessels, as the aortic valves would close immediately. The curve of descent would take a definite form, which is easily expressed in mathematical language, if the capacity diminished as the pressure. Unfortunately the time required to open the chest, and other difficulties connected with the operation, prevented my results from being of much value, and Dr. Michael Foster suggested to me

that the same object would be attained if the heart were made to stop by the action of the interrupted current on the pneumogastric nerve. Mr. Martin of Christ's College, Cambridge, kindly sent me some traces thus taken, and one of the two which are suitable for measurement, entirely conforms with the law that the capacity of the vessels varies directly as the blood pressure, assuming Poiseuille's law to be correct. The other curve does not exactly fulfil the requirements, but varies very little from them. When further opportunity occurs, I hope to repeat these experiments on a larger scale.

It can also be shown in other ways that the arteries do not obey the laws of ordinary elastic tubes. They are covered by a dense, scarcely elastic, fibrous coat which limits their distension, and they are surrounded by organs and muscles which are pressing on them in all directions. So it may be said at least that they do not vary in capacity as simple elastic tubes, and that the difference is towards their varying directly as the pressure.

However, the indirect evidence proves that the capacity of the arterial system, the ventricle included, varies directly as the pressure: for the facts above considered as to the frequency of the pulse depending on the resistance, and *not at all* on the pressure, can only be explained on this assumption.

If the direct evidence as to the capacity of the vessels had been contradictory, it is true that it would have been necessary to assume some error in the method of conducting the pulse experiments; but as above shown, it it quite in the right direction and only lacks partial direct verification.

So much in the verification of theories connected with Physiology must depend on the way in which collateral facts are explained by them that it will be advisable now to consider some of them, and these considerations will be divided into two sections,—1st. The explanation of the known variations in pulse rate in health, and 2ndly, The explanation of the cardiograph laws.

1st. *Variations in Pulse rate in Health.* With regard to these points, as on this theory change in pulse rate can only depend on change in arterial resistance it is evident that Marey's law will, upon his supposition as to the relation between blood pressure and arterial resistance, explain the phenomena equally well.

The following are some of the best known :

The effects of Respiration on the Pulse rate.

Physiologists, though not completely agreed as to the effects of respiration on the pressure of the blood in the arteries, all acknowledge that during inspiration the pulse quickens, and during expiration it gets slower, whether the pressure rises or falls. The theory under consideration clearly shows that this must be so, for during inspiration the expansion of the chest must reduce the pressure in the intra-thoracic aorta, and consequently its contained blood must fall in tension more rapidly than if the chest were motionless, and the more rapid tension fall causes increase in pulse rate. In expiration the opposite occurs, diminution in chest capacity reduces the size of the aorta and consequently delays the time of fall of tension and therefore slows the pulse.

If other remote effects of respiration tend to modify the pressure in the vessels, it is evident that they would co-exist with the above and influence it but slightly, explaining the existence of the experimental discrepancies.

The effect of position of the body on the pulse rate.

The experiments of Dr. Guy led him to explain the differences in pulse rate following change of position as depending on the amount of muscular effort necessary to maintain the positions assumed, and his explanation, assuming that muscular effort of itself can change pulse rate, is very complete. It is curious that the theory under consideration gives an interpretation of the same facts, though very different from that of Dr. Guy.

The following are the most essential facts—The pulse is quickest while standing erect, slowest while lying, intermediate while sitting, slow while standing leaning and while supported entirely as by being bound to a wheel in any position.

The following is the explanation. While standing, the only soft parts of the body which support the weight of the body are the soles of the feet, and the weight is transmitted to them through non-vascular and rigid tissues, cartilage and bone. Consequently the blood flows freely through almost all the vascular system unobstructed. But while lying, most of the weight is supported by highly vascular tissues, as the shoulders, arms, thighs and legs, and consequently much of the circulatory system is greatly reduced in capacity from the compression it experiences, and considerable resistance to the flow of blood is introduced into the

system, the fall of tension is retarded, and the pulse therefore rendered slower.

In sitting, an intermediate condition is the result and an intermediate rate of pulse is produced.

Leaning while standing, and entire support on a wheel, both by introducing resistance from compression of soft parts, tend to make the pulse slow.

Thus, according to Dr. Guy's assumption the slow is the normal pulse, and the quick the induced; upon the fall of tension theory the reverse is the case. The occurrence of bed-sores and the paleness of a compressed part prove that pressure disturbs the uniformity of the circulation.

The rapid pulse after a meal, during digestion, depends on the relaxation of the vessels of the alimentary canal while its functions are being performed.

In a paper on Cardiograph tracings* from the human chest wall, published in the 5th vol. of the *Journal of Anatomy and Physiology* I have endeavoured to substantiate a law respecting the elements of the heart's beat, which may be thus enunciated—

The heart's beat consists of two parts, which for

* Since writing the paper referred to, a further comparison of tracings has shewn me that in the slow pulses taken while lying, I mistook the primary systolic rise for the auricular, and was so led to the conclusion that the length of the cardiac intervals depended in some measure on the position of the body. This is incorrect, as subsequent measurements shew me, and the length of the first part does not vary with the position of the body; the proper equation for finding the cardiac first part under all circumstances being $xy=20\sqrt{x}$.

any given pulse rate do not vary in their ratio to one another; but the length of the first part varies inversely as the square root of the rapidity of the pulse.

A second series of measurements of the cardio-arterial intervals, published in the Proceedings of the Royal Society, have further verified the law just stated, and in the rest of this paper it will be assumed as proved. No theory respecting the circulation throws light on its significance; but the one which it has been my endeavour to demonstrate above gives a very satisfactory explanation of it, which will now be considered in detail.

First, the heart's beat consists of two parts, which for any given pulse rate do not vary in their ratio to one another. It having been proved previously that the pulse rate does not depend on the blood pressure, and, as shown now, the length of the first part of the heart's beat not varying when the pulse length is constant, it is evident that the length of the first part of the pulse beat does not depend on the blood pressure in any way.

Again, the first part of the pulse beat is compound, for it is the interval between the commencement of the cardiac or ventricular systole and the closure of the semilunar valves; therefore it may be divided into the systole and the valve-closure interval.

Physiologists have laid very little stress on this valve-closure interval, it generally being considered as instantaneous. But in the study of cardiograph tracings it is to be remembered that the distances between events occurring within one-fiftieth of a

second of one another can be appreciated without much difficulty, and there is every *a priori* reason for believing that this interval has a longer duration than that. In my paper on the Cardiograph trace, reasons have been given for the belief that in quick pulses the commencement and the end of this valve-closure interval are indicated by separate and distinct changes of direction in the curve, and its length as obtained by measuring from these points agrees entirely with that required from arguments to be mentioned further on. It may be called the *diaspasis*, that is, the period during which the heart is being opened out by the regurgitation of blood from the arteries.

The length of the combined systole and diaspasis not depending at all on the pressure, and it being constant for any pulse rate, it is infinitely probable that the systole and diaspasis separately are independent of the pressure, and this is extremely interesting, as it gives a further insight into the mechanism of the heart. For, in order that the duration of the diaspasis should not vary with different blood pressures, it is evident that with higher pressures there must be greater obstruction to the heartward flow of blood, otherwise the valves would then close more quickly. And this is exactly what would be expected from the combination of Mr. Bryan's observations concerning the shape of the heart, and Brücke's theory of the active diastole of the ventricles.* According to the latter

* Mr. Bryan's paper is in the *Lancet*, Feb. 8th, 1834.

Brücke's theory appeared in *Sitzungberichte der Wiener Akad, der Wiss.* 1854, vol. xiv. p. 345. A paper on the same sub-

author the cardiac muscular tissue has no inherent power of opening out the ventricles, but remains inactive after systole, during diaspasis in fact, until the regurgitation from the aorta has closed the aortic valves and so uncovered the orifices of the coronary arteries, immediately upon which, the resulting sudden turgescence of the heart's walls makes them open up. Mr. Bryan has shown that during systole the whole heart alters its position as a result of its change in shape during contraction, and recovers it during diastole; therefore the greater the force of contraction the more will it alter its shape, and the more difficult will it be for it to resume the original one, which has to be done partly by the regurgitating arterial blood; but the greater the blood pressure, the greater will be the facility for overcoming this greater work, which two, as must be the case, vary together. This argument explains how the diaspasis need not vary in length with different blood pressures.

Next, with regard to the systole. As the first part of the heart's beat varies as the square root of the length of the beat, and as the diaspasis, a part of that first part, does not vary with the blood pressure, upon which alone it can depend, it is necessary that the other component of that interval must vary more than as the square root of the pulse length. And to find how much more quickly, it is necessary to obtain the actual length of diaspasis. Careful measurements of a cardiograph trace, beating 102 in a minute, give the ratio of the systole to

ject by myself will be found in the *Journal of Anatomy and Physiology*. May. 1869.

the whole beat as 1 to 3·1915, and that of the first part to the whole beat as 1 to 2·0, which leaves the ratio of the diaspasis to the beat as ·187 to 1, or the diaspasis length as ·00183 of a minute. A similar length of diaspasis is found from quicker pulses.

The great interest attaching to these figures is that, when with the diaspasis equal to ·00183 of a minute, the ratio of the systole to the diastole is enquired into, it is found that there is a very simple relation between them, and that after subtracting this diaspasis of constant length, there remains the systolic varying as the square root of the diastolic period, and with no other diastolic length is so simple a ratio obtainable, which is all-important, because it will be seen that the systole must depend directly on the previous diastole.

Next, considering the systole itself; the fact above demonstrated, that its length does not depend on the blood pressure is extremely important and can only be explained by assuming that when the pressure rises, the circulation through the coronary vessels increases to a sufficient extent to enable the heart to get through the extra work it has to perform without altering the duration of its action, or in more precise terms, the nutrition of the walls of the heart must vary directly as the blood pressure in the aorta.

But the systolic length varies as the square root of the diastolic, in other words, the longer the time of nutrition of the heart, the longer the systole. This at first sight seems an anomaly, but the theory that the pulse rate depends on the fall in

tension only, presents a most complete explanation, and so throws great light on cardiac action in general.

Consider the heart as a pump working against a certain pressure, and filling an elastic reservoir with a certain resistance to the outflow of its contents. Varying the pressure has been shown to have no effect on the lengths of the different parts of the pulsation for the reasons given above; and it has next to be considered how it is that varying the resistance changes the lengths of the elements of the revolution. This pump, directly its muscular fibres begin to contract, exerts its full pressure, for there is nothing to prevent it doing so. But during the previous diastole it was supplied by blood at a certain definite pressure and for a definite time, both of which factors limit the force of the systole. Consequently the ventricles produce directly their full systolic pressure and maintain that pressure until they are empty. But it is evident that the time necessary for emptying them of a definite amount of blood under these conditions must depend on the rapidity of the flow from the capillaries, for when the flow is halved the systolic time must be doubled, if no other force come into play; in other words the length of cardiac systole is a function of the arterial resistance; and the pulse rate has also been shown to be a function of the same, upon the fall of tension theory.

It has been proved that the systole varies as the square root of the diastole, not directly with it, as might be supposed. This clearly shows that the time of diastole influences the length of the systole

and shortens it, in other words strengthens the heart, according to the law that may be stated thus, the nutrition of the heart varies as the square root of the time during which the coronary circulation is maintained.

It will strike some as peculiar that no mention has yet been made of the influence of the nervous system on the heart. But it appears to me that the facts which have been brought forward have not called for any special reference to it. May not the law it has been my endeavour to prove, be but an expression of that action in the healthy body? For it must depend on a somewhat complicated mechanism, as is shewn by the fact that it is almost impossible to contrive a self-acting engine which would pulsate in accordance with its requirements.

As is well known, the effect on the kymographion trace, of stimulating the depressor nerve, is greatly to amplify the oscillations, and at the same time to lower the mean pressure; while cutting the pneumogastrics produces the reverse effects. The larger oscillations of the hæmadynamometer column in the former case, shew that the proportionate tension-fall and the time of pulsation are both greatly increased, and from previous considerations it is evident that these are necessarily associated, when as now, no influence is being exerted on the peripheral vessels.*

* In rabbits the normal fall of tension as judged by the hæmadynamometer trace is about $\frac{1}{20}$th of the whole, while when the depressor is stimulated it may increase to $\frac{1}{10}$th or more.

Further, these amplified oscillations must be attended with an abnormal enlargement of the ventricular cavities during diastole, for the time intervening between the beats being increased, the amount of blood which flows through all segments of the circulation between any two pulsations, must be also more considerable. Having arrived so far, it is extremely interesting to observe how an augmentation in the degree of cardiac dilitation during diastole, as a cause, will include and correlate all the peculiarities which are observed when the depressor nerve is thus operated on; and it is not unreasonable therefore to suppose that this is the direct effect of its action. As the quantity of blood contained in the heart at the end of diastole has been shewn to depend on the circulation through the coronary vessels, it is evident that the explanation of any variations in the capacity of the ventricles must be referred to changes in the cardiac walls themselves. Just as the degree of rigidity of an india-rubber tube through which a current of water is flowing, can be made to vary by changing the diameter of the orifice from which the fluid is allowed to escape, so the turgescence of the ventricular walls, or what is the same thing, the amount of active diastole of the heart, can be altered, by varying the diameter of the small arteries of the coronary system, their contraction producing a greater, and their dilitation by facilitating the flow of blood through the capillaries, a less degree of diastolic enlargement of the ventricular cavities.

From the above argument therefore, the ampli-

fied range of pressure and time depending on change in heart capacity, and the change in capacity being caused by modification in the calibre of the smaller coronary arteries, it is almost a logical necessity that the function of the depressor nerve is to regulate the degree of tonicity of those vessels, and Dr. Brown Séquard, from entirely different facts, has also published it as his belief that the pneumogastrics contain fibres which contract the small coronary vessels.*

It will be noticed that throughout this paper it has been assumed that the systole never recommences until the ventricular cavities are completely filled, that is, until a pressure equilibrium has been arrived at in the interior of the heart. Perhaps it is the absence of pressure which admits of the heart recontracting, but this is a doubtful point, and until more is known as to the mechanism of muscular action in general, it is probable that the question as to the reason why the heart recommences to beat at a particular moment will remain unsettled. Sir J. Paget,† when he pointed out the relation of rhythmic nutrition to rhythmic action of nerves and muscles, laid the foundation for a scientific treatment of the subject, and the law which it has been my endeavour to substantiate, is only a precise method of expressing that relation.

The following summary of the main features in

* See *Principles of Human Physiology*. By Dr. Carpenter, 1869, p. 219. Foot-note.

† *Croonian Lecture*. Royal Society, 1857.

the circulation, as they appear to me, may assist in explaining some of the previous arguments.

The circulation of the blood is maintained by the repeated contraction of the heart. Each cardiac revolution is divided into three parts, the systole, the diaspasis, and the diastole. The following laws hold with regard to the length of these intervals.

I. The systole together with the diaspasis, or in other words, the first cardiac interval varies as the square root of the whole revolution.

II. The systole varies as the square root of the diastole.

III. The diaspasis is constant.

The amount of work that the heart has to perform in maintaining the circulation depends on two sets of changes which may occur in the system; 1. Variations in the blood pressure. 2. Variations in the resistance to the outflow of that fluid from the arteries.

As the capacity of the arteries including the ventricles, varies directly as the blood pressure, and as the flow of blood from the capillaries does the same, the frequency of the heart's beats is dependent on the resistance to the capillary outflow, and not at all on the blood pressure; in other words, the heart always recommences to beat when the blood pressure in the systemic arteries has fallen a certain invariable proportion.

Variations in blood pressure result from: 1. Absorption into, and excretion from the vascular system, of fluids. 2. Changes in the capacity of the arterial system, which occur on the contraction or

relaxation of the muscular arteries. 3. Changes in the amount of available blood, which result from the hæmastatic dilitation of some of the yielding vessels on altering the position of the body. As changes in the first of these cannot be very sudden, and those in the latter are never very considerable, the mean blood pressure in health varies but little during short intervals.

Variations in peripheral resistance result from: 1. Different degrees of tonicity or patency of the muscular arteries. 2. Different resistances in the venous system. The former may occur independently in one or other system of vessels, as the cutaneous or the alimentary; also mechanically from pressure on a part of the body. The latter are insignificant in health.

The heart depends for its power of doing work on chemical properties in the blood it pumps into the systemic vessels, and as the blood reaches it direct from those vessels, the cardiac intramural circulation varies with the changes in the former; and the length of the systole varying only as the square root of the time of diastole, the degree of cardiac nutrition varies directly as the systemic blood pressure, and as the square root of the diastolic time. The coronary arteries supplying the whole heart, the work done by the right ventricle is governed by that done in the left; thus the supply of blood in the left auricle is always rendered sufficient for the requirements of the systemic circulation; though, as there is no reason for believing that the resistance in the pulmonary vessels varies with that of the systemic, there must be some

peculiarities in the former circulation (which may explain the variations in the ratio of the number of pulse beats to respirations, in some cases).

The auricular contraction is a very small force and its function is most probably to close the tricuspid and mitral valve.

The heart commencing its systole as a whole, it is highly probable that the impulse for action is given by a force which affects both ventricles; such is found in the coronary circulation and the active diastole produced by means of it.

In conclusion I have to present my best thanks to Dr. Michael Foster, Professor Sanderson, and Professor Pritchard of the Royal Veterinary College, for the opportunities they have afforded me in trying the experiments above detailed; without their assistance it would have been impossible for me to have put the law of the relation of blood pressure to pulse frequency on any satisfactory basis.

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