EXPERIMENTS AND CONSIDERATIONS ON HAEMODYNAMICS.

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EXACTLY thirty years ago Donders[1] made some critical and experimental contributions to Haemodynamics. In his usual clear argumentative style Donders discussed the relations of the lateral pressure in the different parts of the vascular system as deduced from observations on the flow of fluids through tubes. In the clearest manner he contended that some of the views set forth in Volkmann's Haemodynamics[2] were erroneous, a contention of all the more importance at that time, because it was seen that Volkmann's book would long be the basis of further investigations into the circulation of the blood. Most willingly do I, at the outset of this essay, adopt the language which Donders then employed, and I say, with the great physiologist, that if I must occasionally differ in opinion from men who in this domain, are far above all praise of mine, I do this without desiring in any way to diminish the great respect to which their merits entitle them.

Even in Manuals of physiology and pathology we occasionally meet with views on the relations of the circulation which are not compatible with our notions of hydrostatics and hydrodynamics. Nor is the physiologist to blame for this.

In the course of his investigations problems often arise which must be deduced from similar problems in physics or chemistry; but when for their solution he consults these auxiliary sciences, he finds no solution until in these domains he investigates the matter for himself. The processes in physics or chemistry with which the physiologist has to deal, are generally of a most complicated character; and the physicist or chemist is not to be blamed for not trying to solve the most complicated matters first. Thus the physiologist is often obliged to attempt the

[1] Donders. *Arch. f. Anat. u. Physiol.* 1856, p. 433.
[2] Volkmann. *Haemodynamik.* Leipzig, 1850.

solution himself, and hence, to combine with his physiological investigation one in chemistry or physics.

These difficulties are sure to occur most frequently in every investigation into the relations of the circulation. All physiologists agree that the relations we meet with in the circulation of the blood must be deduced from the passage of fluids through tubes; and how greatly would not it avail, if the solution of the latter problem were at hand, or, even if the matter had been thoroughly and successfully investigated? As this is not the case, we are sometimes led astray. What has been discovered by experiments in physics is often applied to an organism. Thus a very dangerous path is entered into. We ought first to be accurately acquainted with the facts in their relations in the organism itself, and when we have arrived at an explanation and thorough understanding of these relations, we must have recourse to the auxiliary science. If we do not adopt this plan, but the reverse, we run great risk of thrusting physiological phenomena into some previously conceived physical notions.

Let us not forget that in living organisms we have to do with living tissue. However much we may desire to express what we observe in the organism in a more concise form, in other words, to reduce the laws which there obtain to a formula, the data required for this purpose have nowhere been collected. To give a single example, if we wish to express in formulae the laws which govern the circulation of the blood we ought to know, (to mention but one factor) the coefficient of elasticity of the living wall of the vessels over the whole vascular system. But when we wanted to employ such a coefficient, we should find that we knew very little about that of the dead tissue, and all but nothing about that of the living one. Hence we are generally obliged to content ourselves with relative statements, when we wish to give absolute ones.

When Volkmann[1] found, in the case of a fluid flowing from a reservoir through a non-elastic tube of unequal width, that, in the passage of the fluid from a narrower to a wider part, the pressure at the end of the narrower part is lower than at the beginning of the succeeding wider part, he called this: "negative Stauung." Donders justly disputed the propriety of this name, and gave a simple and plausible explanation of the phenomenon. It was thus shewn by an experiment that a fluid flowing through a tube may at a certain part of the tube indicate a pressure lower than that indicated further on in the direction

[1] *op. cit.* p. 45.

of the stream; in other words, paradoxical as it may seem, that fluid can flow from a place of lower to one of higher pressure. Hence it follows that, if a fluid flows in a certain direction, we cannot a priori say whether the pressure at a given point will be higher or lower than or equal to that at a point more remote.

Yet still in our time we find it given as a fundamental principle of the blood-circulation that the blood flows in a definite direction, because the pressure in the artery is greater than in the vein. In the "Handbuch der Physiologie, herausgegeben von L. Hermann, 1ster. Theil, Physiologie der Blutbewegung von A. Rollett," which may undoubtedly be considered as a standard work on Physiology, we find verbatim at page 224: "Es ist nun in der That die Aufgabe der Herzventrikel, die Druckdifferenz zwischen Arterien und Venen auf einer nahezu constanten Höhe zu erhalten, und so wie in unserm Schema, ist auch beim Blutlauf die Strombewegung des Blutes auf diese Druckdifferenz zurückzuführen."

It is this principle which Weber[1] enunciates and which is constantly accepted in spite of Volkmann's observations and Donders' explanation[2]. We do in fact find that the pressure of the arterial blood is higher than that of the venous; but this difference is not the cause of the blood-current, is only one of the most constant phenomena; and after Volkmann's observations we must accept the possibility that the pressure of the blood in a blood-vessel at a certain distance from the heart may be lower than in one at a farther distance.

To this subject I shall refer later. I have mentioned it now merely as an example of inaccurate representation with respect to the relations of the circulation. Before proceeding to these relations themselves, I wish to premise a few observations on the passage of fluid through tubes.

I. On the passage of fluid through tubes.

a. On the constant flow of fluid through a non-elastic, horizontal, straight tube of equal width. When a fluid flows from a reservoir to which such a tube has been attached laterally, the relations of the fluid may be examined both experimentally and theoretically. As soon however as we wish to apply the principles there found to the passage of fluid through a tube of unequal width, and other cases still to be mentioned, difficulties present themselves, because the phenomena cannot

[1] E. H. Weber. *Arch. f. Anat. u. Physiologie*, 1851, p. 497.

[2] Where I myself previously, in discussing the blood-current in the lungs, spoke of difference of pressure between arteriae and venae pulmonales, I added in a note an explanation of what I understood by this difference of pressure.

be deduced from these principles. Yet it occurs to me that the relations we meet with in the last mentioned cases may be very easily inferred from the case of the tube mentioned at the head of this paragraph. For this purpose a simple representation of the relations obtaining in this case is necessary; and these may without any difficulty be inferred, with some modification, from what Donders has communicated to us on the subject.

When fluid flows by a lateral opening from a reservoir, in which it is maintained at a constant height, then, apart from the loss of the actual energy of the particles of the fluid caused by the change in the direction of the moving particles, the velocity of the issuing fluid is $v = \sqrt{2gH}$ where H is the height of the column of water in the reservoir (Torricelli's Theorem). A fluid mass m which thus flows out, has an actual energy $T = \frac{1}{2} mv^2, = mgH$. And even when, after its passage through the aperture, this mass of fluid is no longer under the influence of gravity, it is capable of doing work until $\frac{1}{2} mv^2$ is consumed. This quantity of energy lies in the mass m itself, and the height of pressure H, as soon as the mass has passed the aperture, exercises no further influence on the labour which it can perform; or in other words, the force, which gave it this energy, has ceased to act on it.

If we apply a tube to the aperture, and again exclude the loss of energy necessary to give the particles of fluid the direction from the reservoir into the tube, then the mass m will pass from the aperture, and thus into the tube, with the same quantity of energy, T. The presence of the tube can exercise no influence on this amount. The mass m is, however, now forced to move through the tube, and must in its passage overcome resistance[1]; the energy existing in the mass cannot thus be entirely employed to produce velocity; a part of it must be used to overcome the resistance in the tube. The mass of fluid, on reaching the end of the tube, will thus have lost a part of its energy and will there flow out with the actual energy $= \frac{1}{2} mv_1^2$, where $v_1 < v$.

In the passage of the fluid through the tube there must be consumed an energy of $T - \frac{1}{2} mv_1^2$. As the fluid may be considered incompressible, the velocity during the whole passage through the tube will be the

[1] I will not enter upon the question of the different kinds of resistance encountered by fluids in their passage through tubes. Though with respect to the flow of fluid this is a most important matter, and up to this moment there is much in it that has not been explained, I have not had up to the present time an opportunity of investigating the matter by experiments.

same, and may be expressed as $v_1 = \frac{A}{\pi r^2}$ (where r is the radius of the section of the tube, and A the amount of fluid which in the unit of time flows out at the end of the tube). At the end as well as at the beginning of the tube the velocity will therefore be v_1; and the actual energy $\frac{1}{2} mv_1^2$, which the mass of fluid m has at the end of the tube, it will have possessed through the whole of its passage. The difference just adduced in the energy of the mass of fluid at the beginning and at the end of the tube, $T - \frac{1}{2} mv_1^2$, cannot therefore have been present as actual energy, but must have accumulated in another form. It has been deposited in the mass of fluid as potential energy. Over the whole of its passage through the tube the mass of fluid will thus have an energy $> \frac{1}{2} mv_1^2$; only at the end of the tube is this exactly $= \frac{1}{2} mv_1^2$. This surplus of energy in the mass of fluid must therefore be indicated.

We know that, in fluids in movement, not only velocity but also pressure may be indicated. Now the so-called hydrodynamic pressure is exactly the expression of the potential energy present in the mass of fluid. What is the force which this potential energy gives to the mass of fluid? Mechanics teaches us, that a particle possesses potential energy only as long as a force works on it. The pressure of the column of fluid in the reservoir may have given rise to this condition, yet this can no longer be the force in question. I have just shewn, that, as soon as the mass of fluid m has passed through the aperture, the influence of the height of pressure in the reservoir has ceased to act on it. The whole energy is in the mass m itself. The force in question can be nothing else than the tension of the fluid. Fluid is indeed considered to be incompressible, but this merely means that with an increase of tension the diminution in volume is so minute, that we have no means of accurately determining the slight modifications in the result thereby produced. Although the density of the fluid will be different in places where the pressure differs, it follows from what I have just said that the difference in density may be so slight that we can safely disregard it. Still wherever the fluid shews a higher pressure the tension becomes greater. Hence, as much of the energy of the mass of fluid m, as cannot at a given moment be operating as actual energy, and cannot therefore express itself as velocity, will so long be detained as potential energy, that is to say as tension of the mass of fluid. If we attach piezometers to the tube we see the expression of this tension as the so-called lateral pressure or rather hydrodynamic pressure.

This consideration is of importance, because it follows that, if there

is a cock at the beginning of the tube, and the fluid flows steadily through the tube, the mass of fluid on the sudden closing of the cock will continue to move not only in consequence of the actual energy $\frac{1}{2} mv_1^2$, but also under the influence of its potential energy, which then passes into actual energy. If the pressure in the reservoir were the force which imparted to the particles of the fluid their potential energy, then, on the closing of the cock, that is to say on the removal of this force, the fluid would move only under the influence of the actual energy present in it.

The relations of hydrodynamic pressure in the passage of the fluid through the tube are well known. It regularly diminishes and, at the end of the tube, if we disregard the atmospheric pressure, is zero. If we take the latter into consideration, this pressure at a given point in the tube can, according to Neumann, be represented by

$$p = b + p_0 - \frac{p_0}{l} x$$

where b is the pressure of the atmosphere, p_0 the pressure at the beginning of the tube, l the length of the tube, and x the distance between the point selected and the beginning of the tube, from which it follows that if $x = l$, $p = b$. If p is the pressure in a certain section at a distance x from the beginning of the tube, u the velocity in a point of this section at a distance r from the axis of the tube, and η a coefficient of friction, then, according to Neumann, $\frac{dp}{dx} = \frac{\eta}{r} \frac{d\left(r \frac{du}{dr}\right)}{dr}$. Since the second member of this equation is independent of x and hence so far constant, p will be formulated as follows:

$$p = Ax + B \text{ (where } A \text{ and } B \text{ are constants);}$$

in other words, p will be a linear function of x. Hence, the pressure over the whole of the tube may be represented by a straight line; it steadily diminishes from the beginning to the end.

Considering then, that the velocity in the different sections of the tube is, as we have seen, the same, the actual energy of the mass of fluid m will everywhere be $\frac{1}{2} mv_1^2$, the potential energy at every point of the tube will be just equal to the resistance that must still be overcome; this is generally expressed by saying: the height of pressure at every point of the tube is equal to the height of resistance.

This however is true only in this special case of a tube everywhere of the same width. It ceases to be so as soon as we have to do with one of unequal width.

b. On the constant flow of fluid through a non-elastic, horizontal, straight tube of unequal width. The argument adduced at the beginning of the former paragraph (*a*) is also applicable to the flow of a fluid from a reservoir through this kind of tube. At the beginning of the tube the mass of fluid m has the energy T, which in the absence of the tube is expressed by $\frac{1}{2} mv^2$. This mass, however, must overcome the resistance in the tube, and it has at the end of the tube the velocity $v_1 < v$; it thus has an actual energy $= \frac{1}{2} mv_1^2$. Hence in the tube $T - \frac{1}{2} mv_1^2$ of energy has been consumed. In what form this energy thus consumed will be present in the fluid depends upon in what part of the tube the fluid is. With the movement of the mass of fluid m somewhat of the energy in it will be continually consumed, and the total energy of the mass must diminish from the beginning to the end of the tube, to be at the end exactly $\frac{1}{2} mv_1^2$; but as the diminution depends on the resistance experienced, and this is not, as in a tube of equal width, everywhere the same throughout, the diminution will not occur regularly. Where the resistance is greatest, in the narrowest part for instance, the diminution of T will be greatest. Nor will the actual energy of the mass m be everywhere in the tube the same, since the velocity at a given point $\left(v_x = \frac{A}{\pi r_x^2}\right)$ is not everywhere the same, but depends on the magnitude of r_x. A must here, of course, in every section be equally great, and then it immediately follows from the above formula, that the velocity in a wider part will be less than in a narrower, and that the actual energy of the mass m will be less in the wider than in the narrower part. If we call the total energy at a certain point of the tube T_{x_1} and the actual energy at that point $\frac{1}{2} mv_{x_1}^2$ then the difference $T_{x_1} - \frac{1}{2} mv_{x_1}^2$ must again be present here as potential energy, that is to say as tension in the mass of fluid. If we denote this potential energy by P_{x_1}, then $P_{x_1} = T_{x_1} - \frac{1}{2} mv_{x_1}^2$, or[1] $T_{x_1} = \frac{1}{2} mv_{x_1}^2 + P_{x_1}$.

At a point x_2, a little farther in the tube, T_{x_2} will also $= \frac{1}{2} mv_{x_2}^2 + P_{x_2}$; and of course T_{x_2} is $< T_{x_1}$.

Now two cases are possible, when a narrowing or widening takes place,—viz.

[1] We have the right to make this equation, because we may add together potential and actual energy. The mass m, which in general has an actual energy of $\frac{1}{2} mv^2$, will be able to perform a labour $= ks$. If the mass m has an equal amount of potential energy, this may also be represented by ks, and differs only so far from the actual energy that with the latter the path s is actually traversed, while with the potential energy the same path may be traversed.

1st. A wider part passes into a narrower. Let us call the total energy of the mass m just before the end of the wider part T_{x_1} and just after the beginning of the narrower part T_{x_2}, then

$$T_{x_1} \text{ is} > T_{x_2},$$

that is $$\tfrac{1}{2} m v_{x_1}^2 + P_{x_1} \text{ is } > \tfrac{1}{2} m v_{x_2}^2 + P_{x_2},$$

and since $$v_{x_1} \text{ is} < v_{x_2},\ \tfrac{1}{2} m v_{x_1}^2 \text{ is} < \tfrac{1}{2} m v_{x_2}^2;$$

whence, by subtraction

$$P_{x_1} \text{ is} > P_{x_2}.$$

The potential energy will, therefore, be greater in the wider part of the tube than in the narrower; and if we then determine the hydrodynamic pressure we must find it greater in the first case than in the second. Whether this difference will be great or small depends, of course, on the difference in magnitude of v_{x_1} and v_{x_2} and on that of T_{x_1} and T_{x_2}. We have first considered the mass of fluid in the wider, and then when it has reached the narrower part; but just at this transition, especially when it is sudden, the mass of fluid has had to overcome a special resistance, into the nature of which we will now not enter, but which is occasioned by what we usually call eddies (tourbillons) of the fluid. To overcome this special resistance a special amount of energy has, of course, been consumed, and on this account the difference between T_{x_1} and T_{x_2} will be greater than if the particles of the fluid had been able to make a rectilinear movement. With greater changes in the lumen this special resistance will be greater, and moreover v_{x_1} will be much smaller than v_{x_2}, for which two reasons P_{x_1} will be much greater than P_{x_2}.

2nd. A narrow part passes into a wider. T_{x_1} is the total energy of the mass m just before the end of the narrower part, T_{x_2} that at the beginning of the wider. Here again

$$T_{x_1} \text{ is} > T_{x_2},$$

$$\text{or } \tfrac{1}{2} m v_{x_1}^2 + P_{x_1} \text{ is } > \tfrac{1}{2} m v_{x_2}^2 + P_{x_2},$$

but, since now $$v_{x_1} \text{ is} > v_{x_2},\ \tfrac{1}{2} m v_{x_1}^2 \text{ is} > \tfrac{1}{2} m v_{x_2}^2;$$

whence by subtraction

either $$P_{x_1} > P_{x_2} \text{ or } P_{x_1} = P_{x_2} \text{ or } P_{x_1} < P_{x_2}.$$

Hence with respect to the magnitude of the potential energy, and so also to that of the hydrodynamic pressure to be observed in both parts of the tube, three cases are possible. Which of these three will occur depends on the relations of the change from v_{x_1} to v_{x_2} and from T_{x_1} to T_{x_2}.

If in this transition from one part to the other there were no eddies, and thus no special resistance to overcome, then T_{x_1} and T_{x_2} would

differ but little from each other, and on any considerable widening P_{x_1} would be $< P_{x_2}$. Since, however, eddies arise, T_{x_1} will always be considerably greater than T_{x_2} and the possibility of the two other cases is present. With any considerable widening, however, the influence of the large decrease of v_x is greater than that of the decrease of T_x, as experiment shews. In most of these cases we find $P_{x_1} < P_{x_2}$. Here, then, we have one of those cases (we shall meet with more) where a fluid flows from a place of lower to one of higher pressure.

Unreasonable then is Jacobson's[1] contention with Donders. Jacobson discusses the case in which a narrower part passes into a wider, and the wider part is at the end of the tube. At the end of the narrower part the hydrodynamic pressure was already next to nothing, and in the following wider part there was no higher pressure shewn than in the preceding part; there was indeed hardly any pressure at all indicated, not even when he narrowed the egress more than a third.

As I have just shewn, this depends only on the change of v_x with respect to that of T_x. At the end of the narrower part (in Jacobson's case) almost all resistance is overcome, the difference $T - \frac{1}{2} m v_1^2$ is already small, and if the mass has reached the wider part, the difference has become almost nil, if the wider part is not long. Thus the relations are here as unfavourable as possible to get $P_{x_1} < P_{x_2}$. Experimentally this relation will be most clearly observed, when we cause the fluid to flow directly from a reservoir into a narrower tube which passes into a wider. If the latter part is short, Jacobson's case will ensue. If however the latter part is constantly made longer, while all the rest remains the same, we shall at last, when the wider part has attained a certain length, see $P_{x_1} < P_{x_2}$. As the wider part becomes longer, since the resistance increases with the length (according to Poiseuille), the actual energy of the mass of fluid m, at the end of the tube, $\frac{1}{2} m v_1^2$ will be smaller in proportion as the wider part is longer. Since $v_1 = \frac{A}{\pi r_1^2}$ (r_1 is the radius at the end of the tube) A will become smaller if v_1 becomes smaller while r_1 is constant. The quantity of fluid A, which passes through every section of the tube in the unit of time, will thus, upon a lengthening of the wider part, become smaller; from which it follows, that in the narrower part the velocity also will decrease, when the wider part is lengthened. Since the resistance of friction decreases with the velocity, as also that which arises from the

[1] Jacobson. *Anat. f. Anat. Physiol.* 1860 and 1862.

eddies, the amount of energy required to overcome the resistance decreases with the velocity. Hence in proportion as the wider part becomes longer, the difference between T_{x_1} and T_{x_2} will become smaller; and, as this difference becomes smaller, the chance that P_{x_1} becomes smaller than P_{x_2} will become greater, if in this case the proportion between v_{x_1} and v_{x_2} is not too much changed, which case will presently be discussed. In order that P_{x_1} should be $< P_{x_2}$, it is not absolutely necessary that the wider part should be followed by a narrower, as was the opinion of Volkmann. Yet it follows from the above that this will be most favourable to P_{x_1} becoming $< P_{x_2}$.

What is applicable to the tube of equal width, is also applicable here, viz.: that the potential energy exists as tension in the mass of fluid. Thus where P_{x_1} is $< P_{x_2}$ the fluid flows from a place of lower to one of higher pressure. A difference of density in the fluid at the two places, though extremely slight, will ensue.

With a view to its application to the vascular system it is perhaps not superfluous to point out that to the hydrodynamic pressure we must add algebraically the hydrostatic pressure, even in a horizontal tube. This may be easily shown by experiment. For this purpose I employed a tube such as is shown in Fig. 1, and caused water from a

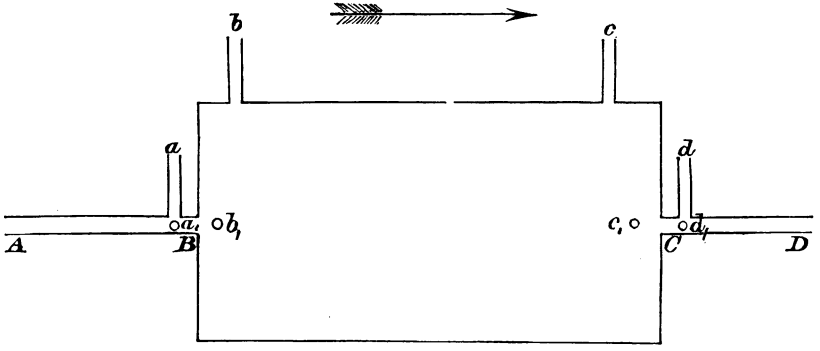


FIG. 1.

reservoir to flow through it. At *a*, *b*, *c*, and *d* vertical glass tubes were placed to measure the pressure. By keeping, as far as was possible, the level in the reservoir at a constant height, and by opening, to a greater or less extent, a cock which was attached to the beginning of the tube, I was able to obtain and compare with each other different heights of pressure at *a*, *b*, *c*, and *d*.

The dimensions are: diameter AB = diameter CD = 1 centimeter. Diameter of the part BC = 23 centimeters. Length of BC = 30 centimeters. Horizontal distance $ab = 12\frac{1}{2}$, $bc = 22\frac{1}{2}$, $cd = 11$ centimeters.

TABLE I.

The heights of pressure are in each case measured from the level in the vertical tube to the upper side of the horizontal tube at the point indicated by the letter.

Number of observations	Height of pressure in cm. at a	Height of pressure in cm. at b	Height of pressure in cm. at c	Height of pressure in cm. at d
1	$116\frac{1}{4}$	107	$106\frac{3}{4}$	$106\frac{3}{4}$
2	119	111	$110\frac{3}{4}$	110
3	$109\frac{3}{4}$	101	$100\frac{3}{4}$	101
4	92	$82\frac{3}{4}$	$82\frac{1}{2}$	$82\frac{3}{4}$
5	66	56	$55\frac{3}{4}$	$60\frac{1}{4}$
6	$42\frac{1}{2}$	$32\frac{3}{4}$	$32\frac{3}{4}$	38
7	26	16	16	$22\frac{1}{2}$
8	43	33	33	39
9	69	60	$59\frac{3}{4}$	63
10	$90\frac{3}{4}$	$81\frac{3}{4}$	$81\frac{1}{2}$	82
11	$105\frac{1}{2}$	$96\frac{1}{2}$	$96\frac{1}{4}$	97
12	116	107	$106\frac{3}{4}$	107

From the table it appears, that the pressure on the wall of AB at a is greater than that on the wall of BC at b. But when the fluid is not in motion there will be a difference in pressure at these two places. On AB the atmospheric pressure will be exerted, on the wall at b the atmospheric pressure minus the pressure of a column of water, the height of which is equal to the vertical distance from the wall at a to that at b. If now the fluid is allowed to flow, and ascends in a to h_1, in b to h_2 above the wall of the respective part of the tube, the pressure in a will be $F + h_1$ (F equal to the atmospheric pressure); and since before the fluid began to flow, there was a pressure equal to F, the pressure caused by the flow of the fluid, the hydrodynamic pressure, will be $F + h_1 - F = h_1$.

In b the fluid has risen to h_2, and the pressure on the wall is indicated by $F + h_2$. Before the fluid began to flow, there was here a pressure of $F - q$, (q is equal to the vertical distance between the walls of AB and BC). The pressure caused by the flow of the fluid, the hydrodynamic pressure, is hence $F + h_2 - (F - q) = h_2 + q$. In order, therefore, to compare the hydrodynamic pressure in the two parts of the

tube, the height of the column of water, which is equal to the vertical distance between the two points to which the piezometers have been applied, must be algebraically added to the pressure observed. It then appears, that the hydrodynamic pressure at every point of a certain section of the tube is the same, while the hydrostatic pressure at those points of this section which do not lie in a horizontal plane, is different. Now, since the observed pressure is the algebraic sum of both, this will be found to be different if we determine it in another horizontal plane of the same section. Where the parts of the tube differ but little in diameter, the difference will of course be slight. Jacobson in his observations perhaps overlooked this. From this relation it may even follow, that, if in a wider part of the tube (for instance, if the wider part comes last, as in Jacobson's case) the hydrodynamic pressure is very slight, the pressure to be observed on the upper side of the tube, which is found by subtracting q from the hydrodynamic pressure, may become negative, that is lower than the atmospheric pressure. If a piezometer is applied to this point, air will be aspired.

The above relations explain to us the fact, that in Table I. the pressure observed in c is lower than in d. To ascertain the hydrodynamic pressure, q must in each case be added to the numbers of c. If we apply these corrections of the numbers of Table I. by adding 11 cm. to the numbers of b and c, we then obtain the real hydrodynamic pressure. They then become :

TABLE II.

Number of observations	Height of pressure in cm. at a	Height of pressure in cm. at b	Height of pressure in cm. at c	Height of pressure in cm. at d
1	$116\frac{1}{4}$	118	$117\frac{3}{4}$	$106\frac{3}{4}$
2	119	122	$121\frac{3}{4}$	110
3	$109\frac{3}{4}$	112	$111\frac{3}{4}$	101
4	92	$93\frac{3}{4}$	$93\frac{1}{2}$	$82\frac{3}{4}$
5	66	67	$66\frac{3}{4}$	$60\frac{1}{4}$
6	$42\frac{1}{2}$	$43\frac{3}{4}$	$43\frac{3}{4}$	38
7	26	27	27	$22\frac{1}{2}$
8	43	44	44	39
9	69	71	$70\frac{3}{4}$	63
10	$90\frac{3}{4}$	$92\frac{3}{4}$	$92\frac{3}{4}$	82
11	$105\frac{1}{2}$	$107\frac{1}{2}$	$107\frac{1}{4}$	97
12	116	118	$117\frac{3}{4}$	107

It now really appears that the hydrodynamic pressure at a is smaller than at b, that at c it is greater, even considerably greater, than at d. By this it has not however been experimentally proved, that the fluid flowing from AB to BC goes from a point of lower pressure to one of higher. If we, however, bear in mind, that the hydrodynamic pressure at every point of a given section of the tube must be the same, then, in the tube BC at the height where AB enters, in other words, in the same horizontal plane in which the tube AB lies, the hydrodynamic pressures are as they are indicated in Table II. b. Seeing that in the same horizontal plane the hydrostatic pressure remains the same, it follows hence, that at B, before its passage into the wider part, the total pressure is really lower than in the wider part itself. The numbers in column a and b Table II. give, at the same time, the total pressure in AB at a, and in BC in the vertical section b at the same height as a.

But it also appears from experiment that this view is correct when we apply our piezometers at AB and BC in the same horizontal plane. The following table indicates this. The piezometers were applied to the same tube as in figure 1, but now laterally at a_1, b_1, c_1 and d_1, and then bent rectangularly upwards.

TABLE III.

The heights of pressure are now measured from the median plane of the horizontal tube.

Number of observations	Height of pressure in cm. at a_1	Height of pressure in cm. at b_1	Height of pressure in cm. at c_1	Height of pressure in cm. at d_1
1	$128\frac{1}{2}$	$130\frac{3}{4}$	$130\frac{1}{2}$	120
2	106	108	$107\frac{3}{4}$	$98\frac{1}{2}$
3	$80\frac{1}{2}$	$82\frac{1}{2}$	$82\frac{1}{4}$	75
4	30	31	$30\frac{3}{4}$	$26\frac{1}{2}$
5	$130\frac{3}{4}$	133	$132\frac{1}{2}$	$121\frac{1}{2}$

Most important are the relations of the pressures in AB and BC when the part of the tube CD possesses a greater diameter while AB and BC remain the same. In Table IV. the heights of pressure are indicated, while at a_1, b_1, c_1, and d_1, piezometers have again been attached, but the tube CD has now a diameter of 4 centimetres.

TABLE IV.

The heights of pressure are measured from the median plane of the horizontal tube at a_1, b_1, c_1, and d_1.

Number of observations	Height of pressure in cm. at a_1	Height of pressure in cm. at b_1	Height of pressure in cm. at c_1	Height of pressure in cm. at d_1
1	$56\frac{1}{2}$	$61\frac{3}{4}$	$61\frac{1}{2}$	$62\frac{1}{2}$
2	$34\frac{1}{2}$	$40\frac{1}{2}$	40	40
3	$21\frac{1}{2}$	$27\frac{1}{2}$	$27\frac{1}{2}$	$27\frac{1}{2}$
4	16	22	22	$21\frac{3}{4}$
5	$25\frac{1}{4}$	$31\frac{1}{2}$	$31\frac{1}{4}$	$31\frac{1}{4}$
6	$39\frac{1}{2}$	$44\frac{1}{2}$	$44\frac{1}{4}$	$44\frac{1}{4}$
7	$53\frac{1}{2}$	$59\frac{1}{2}$	$59\frac{1}{4}$	$59\frac{1}{4}$
8	$80\frac{1}{2}$	$84\frac{3}{4}$	$84\frac{1}{2}$	$84\frac{1}{2}$
9	$100\frac{1}{4}$	104	$103\frac{3}{4}$	$103\frac{3}{4}$
10	$116\frac{1}{2}$	$119\frac{1}{2}$	$119\frac{1}{4}$	$119\frac{1}{4}$
11	$24\frac{1}{2}$	31	31	31 [1]

Here we see that the pressure is considerably higher at b_1 than at a_1. The difference is greater than when the tube CD had a smaller diameter. On the other hand the difference between c_1 and d_1 has become much smaller; it was often even smaller than $\frac{1}{4}$ centimetre. Superficially considered this does not agree with our discussions above. We then argued that if the latter part of the tube was a wider part, the difference of pressure between the preceding narrower and the following wider part would be greater, if this latter part was made longer, and the resistance thus increased there. On the shortening of this part the difference of pressure will thus decrease. In the experiments from which the Tables are taken, the wider part BC again passes into a narrower CD, thus the most favourable condition to obtain $P_{x_1} < P_{x_2}$. Hence, the narrower CD becomes, we should expect the difference between P_{x_1} and P_{x_2} to be all

[1] With respect to all the Tables given I must observe, that numbers occurring there have not any absolute value, but can serve only for comparing the pressures in the different parts of the tube AD; and I must, at the same time, add that CD was not a short tube, as might be supposed from the diagram, but a rather long one. In comparing the pressures on the passing of a narrower into a wider part, and the reverse, this is of no importance, if we do not assign an absolute value to the numbers. The level in the reservoir was moreover not constant, and the influences of temperature have not been regarded. Hence the levels in the piezometers constantly made slight oscillations, and the measurements could not be made within $\frac{1}{4}$ cm. Thus if the levels in two succeeding piezometers, for instance in b_1 and c_1, differed by less than $\frac{1}{4}$ cm., we find in the Tables the same numbers.

the greater; and we see, on comparing Table IV. with Table II. that if in Table IV. the last part CD is taken wider, the difference between P_{x_1} and P_{x_2} increases (the height of pressure at a_1 and b_1 really gives us the magnitude of P_{x_1} and P_{x_2}).

The narrowing which is made to get the difference $P_{x_1} < P_{x_2}$ as great as possible has also a limit; for, though by that narrowing the amount A of fluid which escapes becomes smaller, and thus also the velocity in all the sections of the tube and the resistance of friction thereby slighter, by which the difference between T_{x_1} and T_{x_2} becomes smaller, yet the difference between v_{x_1} and v_{x_2} is thereby changed, as I have observed above. Now seeing that $\frac{1}{2}mv_{x_1}^2$ must be subtracted from T_{x_1} and $\frac{1}{2}mv_{x_2}^2$ from T_{x_2} in order to obtain P_{x_1} and P_{x_2}, the second power in which v occurs will exercise much influence.

Suppose for instance the radius of the narrower part $r_{x_1} = 2$, of the wider part $r_{x_2} = 4$, then $v_{x_1} = \frac{A}{4\pi}$ and $v_{x_2} = \frac{A}{16\pi}$.

If the aperture of exit, or, as in our case, the tube CD be widened, then the quantity A which escapes increases; suppose this widening to be so large that A becomes $2A$, then v_{x_1} will have become $= \frac{A}{2\pi}$ and $v_{x_2} = \frac{A}{8\pi}$. In the formula of the actual energy $\frac{1}{2}mv^2$, v occurs in the second power, and we must thus compare

$$\tfrac{1}{32}\left\{\frac{A}{\pi}\right\}^2 m, \quad \tfrac{1}{512}\left\{\frac{A}{\pi}\right\}^2 m \text{ and } \tfrac{1}{8}\left\{\frac{A}{\pi}\right\}^2 m, \quad \tfrac{1}{128}\left\{\frac{A}{\pi}\right\}^2 m.$$

The difference between formulae 1 and 2 is much smaller than between formulae 3 and 4; and if we disregard the changes which T_{x_1} and T_{x_2} also undergo when A changes, then the difference between P_{x_1} and P_{x_2} will be smaller when A flows through the tube than when $2A$ flows through it.

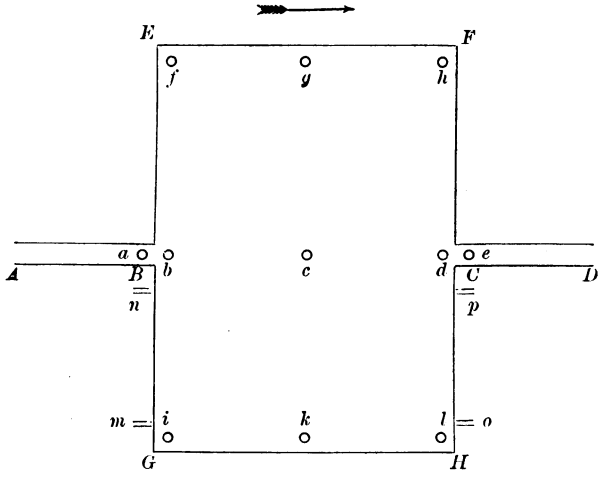
We have however already seen, that under these changes the difference between T_x and T_{x_2} also changes, and thus the only question is: What is the mutual relation between these changes? By accurate experiments, which I have at present no opportunity of making, it would be possible to determine with what lumen of CD the difference between P_{x_1} and P_{x_2} will be the greatest, when the height of pressure in the reservoir remains the same, and the parts of the tube AB and BC do not change.

As I have already observed, all these conditions can only be traced by

accurate observations upon the different resistances, and the eddies with different velocities. Nor will I now dwell any longer on these very important relations, but merely show that in some cases when a fluid flows through a horizontal tube the pressures in different points of the same horizontal plane of a given section may be different. I caused the fluid to run through a flat tube (all the tubes of which I have hitherto spoken were round).

The tube was as in the figure annexed. The round tube AB (diam.

FIG. 2.



$=2\frac{1}{4}$ cm.) passes into the large flat tube $BEFCHG$ (height$=2\frac{1}{4}$ cm.), and this again into the round tube CD (diam.$=2\frac{1}{4}$ cm.). In the vertical direction, thus, the tube undergoes no change in size, but only in a horizontal one. AB and CD were attached to the middle of EG and FH. $EG=FH=44\frac{1}{2}$ cm. $BC=EF=GH=28$ cm.

At $a, b, c, d, e, f, g, h, i, k$ and l vertical glass tubes were inserted; at m, n, o and p lateral tubes were affixed and bent upwards. Altogether then there are fifteen piezometers. The part of tube CD is much longer than indicated in the figure. The following Table gives the heights of pressure, which are measured from the upper side of the flat tube.

TABLE V.

Heights of pressure in cm.

Number of observations	*a*	*b*	*c*	*d*	*e*	*f*	*g*	*h*	*i*	*k*	*l*	*m*	*n*	*o*	*p*
1	$56\frac{3}{4}$	$57\frac{1}{4}$	$54\frac{1}{2}$	$57\frac{3}{4}$	$29\frac{1}{4}$	56	55	57	$56\frac{3}{4}$	$54\frac{3}{4}$	$57\frac{3}{4}$	$57\frac{3}{4}$	$56\frac{1}{2}$	$57\frac{1}{2}$	60
2	$56\frac{3}{4}$	$57\frac{1}{4}$	$55\frac{1}{2}$	$57\frac{3}{4}$	29	57	55	$57\frac{1}{4}$	$57\frac{1}{4}$	$55\frac{1}{2}$	58	$57\frac{3}{4}$	$56\frac{3}{4}$	$57\frac{3}{4}$	60
3	$59\frac{3}{4}$	$60\frac{1}{4}$	$57\frac{3}{4}$	61	$30\frac{1}{4}$	60	59	$60\frac{1}{4}$	60	$57\frac{3}{4}$	61	$60\frac{3}{4}$	$59\frac{3}{4}$	$60\frac{3}{4}$	$63\frac{1}{4}$
4	$78\frac{1}{2}$	$79\frac{3}{4}$	$75\frac{1}{2}$	80	$37\frac{3}{4}$	$79\frac{1}{4}$	$76\frac{1}{2}$	$78\frac{3}{4}$	78	75	78	$77\frac{3}{4}$	$75\frac{3}{4}$	77	81
5	$110\frac{3}{4}$	$111\frac{1}{4}$	$108\frac{1}{2}$	$111\frac{3}{4}$	$83\frac{1}{4}$	$111\frac{1}{4}$	$110\frac{1}{4}$	111	112	$109\frac{1}{2}$	$112\frac{1}{4}$	$112\frac{1}{2}$	$111\frac{1}{2}$	$112\frac{1}{2}$	115
6	$117\frac{3}{4}$	$118\frac{1}{2}$	116	$118\frac{1}{2}$	$86\frac{3}{4}$	$117\frac{3}{4}$	$115\frac{3}{4}$	$118\frac{1}{4}$	118	$115\frac{3}{4}$	$118\frac{3}{4}$	$118\frac{1}{2}$	$117\frac{1}{2}$	$118\frac{3}{4}$	$121\frac{1}{4}$
7	$117\frac{1}{4}$	118	116	$119\frac{1}{4}$	87	$117\frac{1}{4}$	$115\frac{3}{4}$	$117\frac{3}{4}$	$117\frac{1}{4}$	116	$118\frac{3}{4}$	$118\frac{1}{4}$	$117\frac{1}{4}$	$119\frac{1}{4}$	$121\frac{1}{4}$

The relations of the heights of pressure are here very remarkable. In every observation b is higher than a, the same phenomenon that we observed in the case of the round tubes at the passage from a narrower into a wider part. In the same way d is always higher than e. We see further that in the flat tube itself the heights of pressure have a peculiar proportion to each other. Of b, c and d, of f, g and h, of i, k and l, the middle one is always the lowest, while p always stands the highest of all. Still more clearly than from the readings of these numbers do these differences of levels come out when we look at the fifteen piezometers at the same time.

c. On the constant flow of fluid through a curved non-elastic tube. Let us first consider the case in which the tube is everywhere of the same width and lies horizontal; then the relations of velocity and hydrodynamic pressure will be the same as in a straight tube, with this difference, however, that at the bends there is increased resistance in consequence of eddies arising there.

In proportion as these bends are greater the resistance there will increase. If, in addition to this, widenings and narrowings occur, then what has been said in paragraph b is also applicable here. What is most important to us is the case in which the bends occur not only in a horizontal but also in a vertical or oblique direction, since the hydrostatic pressure can then exercise a great influence on the pressure in the different parts of the tube. Let us then for simplicity's sake consider the case of a tube everywhere of the same width, of which the bends are not acute, and are in a vertical direction, and let us disregard the eddies; we may then imagine the two following cases represented in figure 3 and figure 4. The parts of the tube AB and DE lie in the same horizontal plane, so also in the other case A_1B_1 and C_1D_1, and at b, c and d, and at b_1, c_1 and d_1 piezometers have been attached.

If we make these tubes of solid india-rubber so that they are flexible, while the lumen, under the pressure to be applied, undergoes no change worth mentioning, then we may consider them as non-elastic tubes. Now if the parts BC and CD, and B_1C_1 and C_1D_1 are long we may by bending them, bring C or C_1 to different heights. Bends in an oblique direction do then indeed thereby occur, but, as we shall immediately see, this has no influence on the experiment, and we may thus provisionally take only the vertical bends into consideration. Experiment shews that the shifting of the heights of C and C_1 exercises no influence on the flow of the fluid in the tubes, on the velocity, or on the hydrodynamic

pressure. Upon the shifting of C and C_1 the levels in b and d, and in b_1 and d_1 do not change. The vertical distance from the level in c or c_1 to B or B_1 also remains constant. It is only the distance from the level

FIG. 3.

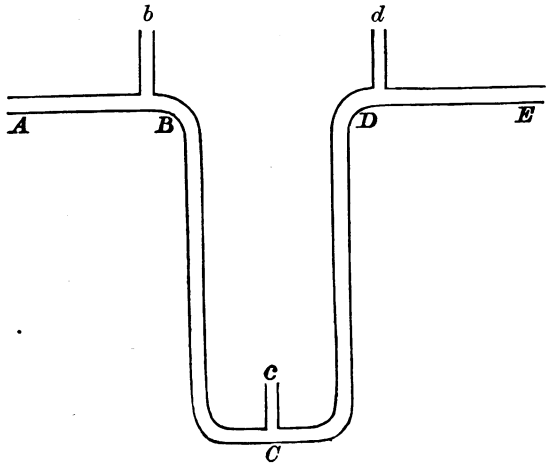
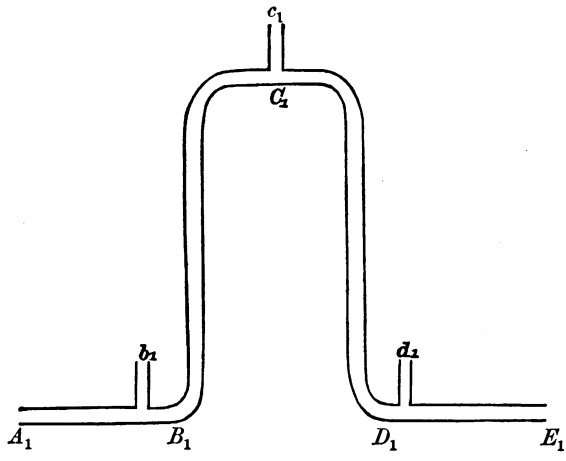


FIG. 4.



of c or c_1 to C or C_1 that changes; in other words, the pressure on the wall in C or C_1 changes with the transposition of C or C_1 but the hydrodynamic pressure remains the same there. In the experiment which I made, C was placed alternately above and below $ABDE$, so that both the above cases were comprised in one experiment.

The following Table exhibits the relations of the height of pressure. The level in the reservoir remained as constant as possible.

TABLE VI.

Heights of pressure in cm.

	b	*c*		*d*
Number of observations	Height of pressure measured from the level to the wall at *B*	Height of pressure measured from the level to the wall at *C*	Height of pressure measured from the level to the wall at *b* = *d*	Height of pressure measured from the level to the wall at *d*
1	141½	30¾	91	37¼
2	141½	13¼	91¼	37¼
3	140½	134	91	37½
4	140½	119	91	38
5	140½	43	91½	38

Hence it appears that the pressure measured from the wall at *c* varies according as *C* is shifted, yet remains constant when measured from the wall at *b* or *d*. The explanation is simple enough. When the fluid is at rest the atmospheric pressure alone will be exerted in *b* and *d*, or in b_1 and d_1. In *c*, on the other hand, there is the atmospheric pressure plus the weight of a column of water equal to the vertical distance *bc*, and in c_1 the atmospheric pressure minus the weight of the column b_1c_1. If the fluid now flows, the hydrodynamic pressure will then be added, in the same way as in paragraph *b*, where we have considered this subject in speaking of a wide part of a tube.

If *C* be placed very low the sum of hydrostatic and hydrodynamic pressures at this point may be greater than at *b*, and if *C* be placed very high this sum may be smaller than at *d*. Thus we have here again two cases in which fluid flows from a place of lower to one of higher pressure; in the first case from *b* to *c*, in the second from c_1 to d_1. We find an example of the latter case in Table VI., Nos. 1 and 2. Both to the working of the reservoir and to the flow in the tube, it is thus quite indifferent as to what height *C* or C_1 is placed, if the bends, the lumen, and the length of the tube are not altered. There is of course a limit to the raising of C_1 with respect to the height of the column of water in the reservoir. If C_1 be placed so high that the height of the column of water in the vertical distance b_1c_1 is greater than the atmospheric

pressure, then, if the water be at rest, Torricelli's vacuum will ensue in c_1 (we of course suppose that the fluid does not issue from the tube and bubbles of air take its place). The level in B_1C_1 will stand at such a height, that the column of water from B to this level forms an equilibrium with the atmospheric pressure. Now, if the height of the column of water in the reservoir is less than the vertical distance from this level to c_1, then this vacuum will remain, and the fluid will not flow over C_1. Of course if the algebraic sum of hydrodynamic and hydrostatic pressure at c_1 be negative (that is less than the atmospheric pressure) no piezometer can be placed at c_1 because the air would enter the tube there. In this case we must apply an open or closed manometer. Finally it is clear that if either in its ascending or descending part the tube makes an oblique bend, we must, in order to measure the height of the column of fluid which is the measure of the hydrostatic pressure, always take the vertical distance between the two extreme points of the bend; in other words, we must measure the rectilinear distance between these two points and the angle which this line forms with a horizontal line, and multiply that rectilinear distance into the sine of the angle. The application of these matters to the vascular system is very interesting, as we shall see.

d. On the constant flow of fluid through a branching non-elastic tube. The influence of branching on the flow of fluid is, alas! but very little known. At every division eddies arise. As to the relative proportions of actual and potential energy we can draw few conclusions from the experiments made. Jacobson found that the angle at which the division occurs exercises an influence on the pressure observed in the branch. Moreover the lumen is, of course, of influence. The mode of branching is generally such that the collective lumen of the branches is greater than the lumen of the main tube. The velocity in the branches will then be in proportion to the velocity in the main tube, the same as when a narrower part of a tube passes into a wider.

To what extent in this case, however, T_x will decrease, depends on special and still unknown resistances. The resistance in the branches themselves undoubtedly increases if, at least, the lumen of each branch in itself is smaller than that of the main tube, as is the case in the vascular system. As the branches here at last become extremely small, capillary in fact, the resistance increases enormously. The fluid issuing from the reservoir will thus have lost very much of its energy by the time it has flowed through these capillary tubes.

In conclusion let it be here observed, that if a tube, whether with or without branches, narrowings, widenings, or bendings, returns to itself and the fluid flowing through it makes a circuit, the atmospheric pressure has no longer to be taken into consideration.

A reservoir can no longer be used for a stream of this kind. We may in such a case provide for a flow of fluid in the tube by inserting in the tube a double suction pump with a system of valves. On the upper surface of the pistons there must be no atmospheric pressure; this we can effect by expelling the air from the suction-cylinders above the surface of the pistons. The fluid may then for a time be made to flow continuously, by pressing at the same time one piston downwards, and the other upwards, with a certain pressure.

e. On the constant flow of fluid through an elastic tube. To this tube, whether everywhere of equal width or with narrowings, widenings, divisions, or bends, everything is applicable that has been said in the preceding paragraphs. We have but one observation to make in this case. Since the wall is elastic, the lumen of the tube will depend on the pressure which is exerted on that wall. Suppose that the atmospheric pressure is exerted on the external surface of the tube, that the tube opens free into the air, and that the fluid in the tube is not in motion, then the tube will in its different parts have a certain lumen, since the atmospheric pressure is exerted on both the inside and outside, and we may disregard the hydrostatic pressure. If some parts of the tube are rather wide, or if there are vertical or oblique bends, then this hydrostatic pressure will, of course, have to be taken in consideration, and will give a definite lumen to the tube at these parts. If the fluid now flows through the tube, the lumen will be increased by the hydrodynamic pressure which thus arises, and in general will be most increased at those points where the hydrodynamic pressure is greatest. As the lumen at any point grows wider, the wall of the tube is stretched, and the dilatation will continue until the stretching has established an equilibrium with the hydrodynamic pressure at this point. The magnitude of the dilatation at any part will depend not only on the magnitude of the hydrodynamic pressure, but also on the coefficient of the elasticity of the wall at the part.

When this equilibrium has once been established, the lumen remains constant, the flow of fluid is constant, and everything that has been said in paragraphs *a*, *b*, *c*, and *d* is applicable here. Only, while the tube is being dilated, a certain amount of energy will be employed,

and this will be detained in the wall as potential energy. The velocity in the different parts of the tube will, of course, not become constant until the lumen has become constant. The size of the lumen is hence in this case of great importance, and we must therefore know the conditions on which the lumen depends. These are three: 1st, the external pressure on the wall of the tube; 2nd, the total internal pressure on the wall; 3rd, the coefficient of elasticity of the wall.

At the close of this discussion of the constant flow of fluid, in which we have always caused the fluid to flow from a reservoir of constant height, I have still to observe that all that has been said will apply also to the cases where the fluid flows through the tube under the influence of constant suction. Mechanically considered, there is no difference between the force of suction and of pressure. We must consider the force of suction as a negative force of pressure. The potential energy will in these cases be present in the tube not as higher but, on the contrary, as lower tension; it may, when it is diminished to a greater extent than the hydrostatic pressure is increased, even become negative, i.e. lower than the tension of fluid subject to the influence of the atmospheric pressure only. The fluid will then be rarefied, though it may be but slightly. We generally express this by saying that there is a negative pressure in the fluid.

f. On the flow of fluid through a non-elastic tube, under the influence of a force acting rhythmically. We may suppose the fluid to flow from a reservoir through a tube, while at the origin of the tube there is a cock, which is suddenly opened at regular intervals, always kept open the same length of time, and then suddenly closed again. During the time it is open, everything that has been said of non-elastic tubes in paragraphs *a*, *b*, *c* and *d* is applicable here also. After the sudden closing, the fluid will keep moving under the influence of the energy existing in it; beyond the cock a vacuum will arise, the fluid will after a while come to rest, and then move in an opposite direction, that is towards the cock. We will disregard this last phenomenon by supposing that the pause between each closing and the next opening of the cock is always just so long that the energy in the fluid which proceeds from the preceding opening of the cock has been totally consumed; in other words, that the fluid has again been brought to rest. Velocity and pressure in the fluid during the time the cock is open will be the same as if the cock were always open. The quantity of fluid which in this interval flows through will depend on the time

during which the cock is open: let us suppose the time to be t seconds. The quantity then flowing through in one minute will depend on the number of times the cock is opened in a minute; suppose n times. If now during every interval that the cock is open Q fluid flows through, then in one minute nQ fluid will flow through the tube. If the cock is opened $2n$ times, this quantity will be $2nQ$. In both cases the velocity and pressure will be quite the same, since we have supposed that the influence of a preceding opening of the cock has already totally disappeared at the next opening. The mean pressure, however, which the tube experiences during one minute will depend on the number of times the cock is opened. On the cock being opened n times, the tube experiences this pressure n times a minute, and on its being opened $2n$ times, this same pressure will be felt on the wall of the tube $2n$ times a minute. We may thus speak of a mean pressure as we speak of a mean velocity of the fluid. Both these will then vary with the number of times the cock is opened.

g. On the flow of fluid through an elastic tube under the influence of a force acting rhythmically. What has been said in paragraph *f* applies only so long as the wall of the tube is not elastic; if it is really elastic the conditions are changed. If as before the elastic tube is attached to a reservoir and there is a cock at the origin of the tube, then the fluid will as before flow through when the cock is opened. If the cock remains open then what has been said in paragraph *e* is applicable.

Before, however, this stationary condition has occurred, waves have arisen in the tube. After the sudden closing of the cock, the fluid moves on under the influence of the energy it has acquired, while that part of the energy which was detained as tension in the wall of the tube now contributes to the movement of the fluid, and thus gives rise to waves. I will not notice the waves which arise from the mere opening and the closing of the cock, because I wish to confine myself to the movement of the stream.

If as before the cock be open t seconds, then a certain quantity of fluid will flow through the tube. This quantity is not quite equal to that which during the same interval has flowed through the cock. If the cock be opened, and the fluid begins to flow through the tube, the latter will be distended by the hydrodynamic pressure, in the same way as is described in paragraph *e*. This increased lumen of the tube must be filled with fluid, and this quantity of fluid will be detained in the tube;

in other words, the capacity of the tube will be increased. After the closing of the cock the fluid thus detained will flow out again, until the tube returns to its previous condition, and regains the capacity which it had before the cock was opened. Thus, if the cock is open t seconds, and if in this interval Q fluid flows through the cock, then there will flow out from the tube $Q - M$; M being the quantity of fluid detained, and thus serving to denote the increase in the capacity of the tube. This quantity M flows from the tube after the cock has been closed.

Immediately after the cock has been opened, the velocity of the flowing fluid will in any part of the tube be less than if the tube were non-elastic. In proportion as the tube becomes wider the coefficient of elasticity of the wall of the tube increases, the velocity at the widening decreases, and the velocity of the flowing fluid continually approaches that which it ultimately reaches when the stream has become constant.

The quantity of fluid which in one minute flows through the tube will depend on a variety of circumstances. Let us suppose that the cock is opened n times a minute, and that after each opening of the cock the tube again returns to its state of rest, then nQ fluid will flow out through the mouth of the tube in one minute. In the pause which occurs before the cock is opened for a second time, there will always be an opportunity for the fluid M which has been detained in the tube to flow out. This flowing out of M will require a certain time, and thus, to obtain the result just described, the pause between every two openings of the cock must be greater than, or at least equal to, the time required for M to flow out. If the number of times the cock is opened every minute becomes greater, while the time during which it is open remains the same, namely t seconds, then the cock will at last be opened at a moment at which the quantity M has not yet entirely flowed out; in other words, the tube will contain a certain quantity of fluid resulting from the previous opening of the cock. Hence, at this moment, there will be a certain tension of the wall of the tube. The fluid flowing through the cock will thus find a wider tube, but at the same time one the wall of which is in a state of distension. The coefficient of elasticity of the wall of the tube will at this moment be higher than when the wall is in the state of repose. The hydrodynamic pressure of the flowing fluid will occasion a smaller distension of this already dilated tube than when the fluid found the tube in the state of rest. The increase of the capacity of the tube will now therefore not

be so great, and hence the quantity of fluid detained will be less than when the cock was previously opened. If the quantity of fluid detained be smaller, the quantity which flows out must be greater, from which it follows that the velocity has increased, if we disregard for a moment the increase of r.

Suppose the fluid be at rest. The cock is opened for the first time. Q flows through the cock and M is detained; thus $Q-M$ escapes through the tube. The cock is closed. M strives to flow through, but before M has wholly escaped, the cock is again opened. During this pause only N has flowed out instead of M. Thus $M-N$ has been detained in the tube. Disregarding now a slight alteration in the quantity Q, which for the second time flows through the cock, Q will again flow through the cock. The tube is again dilated by the hydrodynamic pressure, and increases in capacity; but as the wall is still in a distended state, and has a higher coefficient of elasticity than when the cock was opened for the first time, the increase in the capacity of the tube will not be M, but M_1 and M_1 will be $< M$. The increase in capacity, in terms of that of the state of rest, will now be $M-N+M_1$. In the next pause a certain quantity of fluid will flow out, viz. N_1, thus $M-N+M_1-N_1$ is detained. If the cock be opened for the third time, then an increase of capacity M_2 will take place, and in the next pause N_2 will flow out. Before opening the cock for the fourth time, the increase in the capacity of the tube, in terms of that of the state of rest, will be $M-N+M_1-N_1+M_2-N_2$. Here M is $> N$, $M_1 > N_1$, $M_2 > N_2$; in other words, the capacity of the tube constantly increases, and this will last until $M_n = N_n$, that is to say, until the increase of capacity during the time the cock is open has become equal to the decrease during the ensuing pause, so that the quantity of fluid detained while the cock is open flows out again during the pause. In proportion as the number of times the cock is opened every minute is increased, the pauses between every two openings of the cock will diminish, and thus there will be less time left for N to flow out. The cock, in this case, will have to be opened a greater number of times before the condition has arisen in which $M_n = N_n$; the number of terms of $(M+M_1+M_2+\ldots)$ $-(N+N_1+N_2+\ldots)$, which represents the entire increase of the capacity of the tube will thus be greater.

Suppose now that the state of equilibrium has been brought about, that M_n has become equal to N_n, and that at every opening of the cock Q again flows through, the cock being opened n times a minute; then in each minute nQ will flow through every section of the tube. The

mean velocity reckoned per second at a point x of the tube will then be[1]

$$v_x = \frac{nQ}{60\,\pi r_x^2}.$$

We can now consider the fluid as flowing constantly with this mean velocity. The reservoir in this case has an actual height H, and if the cock were always open, a certain quantity of fluid A would in the unit of time flow through, and A would be greater than $\frac{nQ}{60}$. In order now, that this last quantity might flow constantly through the cock, the reservoir would require a height H_1 where H_1 is $< H$. The fluid, which flowing from a reservoir of the height H, acquires a certain mean velocity when the cock is opened n times a minute, may be considered to flow constantly through the same tube with the above mean velocity from a reservoir of the height H_1.

The total energy of the mass of fluid m must also be considered with respect to this height of pressure H_1. If the cock were always open, this energy at the beginning of the tube, T_0, might be expressed by mgH; in the present case, however, it is mgH_1. In the same way T_x, somewhere in the course of the tube, must be calculated with respect to H_1. Thus the hydrodynamic pressure

$$P_x = T_x - \tfrac{1}{2}\,mv_x^2 = T_x - \tfrac{1}{2}\,m\left(\frac{nA}{60\,\pi r_x^2}\right)^2.$$

P_x is thus, as we generally express it, the mean hydrodynamic pressure.

This P_x establishes an exact equilibrium with the tension in the wall of the tube, which since the first times of opening of the cock has gradually arisen. As we have just seen, upon the distension of the wall, the tension has arisen from the very first opening of the cock until M_n has become equal to N_n. A certain quantity of energy has been used to produce this tension. Potential energy has thus been accumulated in the wall, and the possibility of this taking place is afforded by the fact that so long as M is not equal to N a certain quantity of fluid is detained. No energy is of course expended in merely retaining this fluid in the tube, and there must therefore be an accumulation. The energy thus accumulated is the energy of the fluid mass $(M + M + M_2 + \ldots M_{n-1})$ $-(N + N_1 + N_2 + \ldots N_{n-1})$. The fluid mass m flowing from the opening of the cock has an energy $= mgH_1$. The above-mentioned accumulated energy is thus represented by $[(M + M_1 + M_2 + \ldots M_{n-1}) - (N + N_1$

[1] I hardly need observe, that r_x is the mean radius of the widening and the narrowing which the tube at this point undergoes by the detention of M_n and the escape of N_n.

$+ N_2 + \ldots N_{n-1})]\, gH_1$. This energy is present wholly as potential energy, as tension in the wall of the tube; but, as appears from what has preceded, is not equally distributed over the whole tube. Where the hydrodynamic pressure increases, as the fluid is being accumulated in the tube, more energy is accumulated in the wall of the tube.

Considered in this way, we now obtain the exact meaning of P_x. It is the potential energy (manifesting itself as hydrodynamic pressure) of a fluid mass m, which, under the influence of a reservoir at the height H, flows through an elastic tube while the cock is opened n times a minute (which is equivalent to the influence of the reservoir at the height H_1, if the cock always remains open), while the dilated state of the tube owing to the accumulation of the fluid mass

$$(M + M_1 + M_2 + \ldots M_{n-1}) - (N + N_1 + N_2 + \ldots N_{n-1})$$

must be considered as the quiescent state of the tube with respect to the flowing fluid mass. The pause between each two openings of the cock is just sufficient to cause the tube every time to return to this quiescent state. We have thus obtained the same conditions as those which were discussed at the outset of this paragraph, where the pause after every opening of the cock was so long that the influence of the preceding opening had totally disappeared, and the tube had therefore returned to the state of rest; only with this difference, that the quiescent state of the tube is different from what it was then, in as much as this quiescent state is the dilated state resulting from the accumulation of the above-mentioned fluid mass, and the wall has also a higher coefficient of elasticity. If the quantity of fluid which flows through, when the cock is open, be Q, then M_n accumulates every time, and thus $Q - M_n$ flows out of the tube. This M_n being equal to N_n is conveyed out of the tube during every pause.

When this M_n has once become equivalent to N_n, no more energy accumulates, for the quantity of energy, which by the detention of M_n has accumulated while the cock is open, is in the pause again used for N_n to flow out. As soon as $M_n = N_n$, the whole energy T of the fluid mass m, is used for the current; and thus we may here apply all that has been said concerning the constant flow of fluid, but we must suppose the height of the reservoir to be H_1.

Now suppose the number of times the cock is opened every minute to be doubled, becoming thus $2n$. We again disregard the slight alteration in the quantity of fluid flowing through the cock, and we therefore continue to call this quantity Q. If the state of equilibrium has again

arisen, then $2nQ$ fluid will every minute flow through every section of the tube. In this state of equilibrium, the tension of the wall of the tube is of course greater than when the cock is opened n times a minute. To prove this we have only to apply the same argument we have just used. If $2nQ$ fluid flows through every minute, then, reckoned per seconds[1], $v_y = \frac{2nA}{60\pi r_y^2}$ and thus $P_y = T_y - \frac{1}{2} m \left(\frac{2nA}{60\pi r_y^2}\right)^2$.

We can therefore, in all these cases, consider the current of fluid as constant, with a mean velocity and a mean hydrodynamic pressure. The distended wall is, as it were, the piezometer for the flow of fluid. This distension has hence no influence on the flow itself, if the state of equilibrium has been established. It is only the greater lumen of the tube which has any bearing on the stream. Leaving aside the comparatively small difference between r_y and r_x, it follows from the above formulae, and those previously mentioned, that $v_y > v_x$. The velocity has thus increased with the increased number of times the cock has been opened, and if we suppose $r_y = r_x$ then the velocity has been doubled. As however $r_y > r_x$ this will not be accurate, but the velocity v_y, will be something smaller than $2v_x$. The difference between v_y and $2v_x$ will be so much greater as the lumen of the tube has the more increased.

Now, in order that $\frac{2nQ}{60}$ may flow through every second, if we again consider the current to be constant, the height in the reservoir must be H_2, H_2 being greater than the H_1 we have previously considered. Owing to this, T_y will also have a value other than the T_x previously considered, and T_y will be greater than T_x. Only when we can accurately express T_y and T_x in formulae, which will not be possible until we know accurately the influence of the different resistances in the tube, shall we know the proportions between P_x and P_y. Thus, if the number of times the cock is opened increases, we must consider the supposed constant flow of fluid to be under the influence of a greater height of pressure in the reservoir. Though we do not know the value of P_x and P_y, unquestionably P_y will be greater than P_x, as every experiment will prove. Hence it follows, that velocity and hydrodynamic pressure will increase with the number of times the cock is opened.

Moreover, if we take the hydrostatic pressure into account, and if we

[1] In these formulae y has no definite value. It only indicates on the cock's being opened $2n$ times a minute, the same point in the tube, that x does on its being opened n times.

call the potential energy occasioned by it in any part of the tube, Z_x, then the total potential energy of the fluid mass m (which will be totally manifest in a piezometer) is D_x, which is equal to the algebraic sum $P_x + Z_x$.

We have seen above on what the lumen of the elastic tube depends. If we suppose the pressure on the outside of the wall to be equal to the atmospheric pressure, we can then briefly express the dependence of the lumen by $R_x = F[E_x, (P_x + Z_x)]$. We have further seen in the course of our discussions that P_x depends on the lumen of the tube; thus we may say $P_x = f(R_x)$. E_x also depends on the distension of the wall, and therefore $E_x = \phi(R_x)$[1]. The great difficulty in tracing experimentally the nature of the dependence, lies just in the reciprocal dependence of the different factors, as appears from these formulae.

When by means of a manometer, affixed laterally to an elastic tube through which fluid flows, we determine the pressure, we do not thereby determine the magnitude of the hydrodynamic pressure, but we obtain the expression of $D_x = P_x + Z_x$. The manometer simply takes over the tension of the wall at the place to which it is applied.

Having set forth this view, I wish to proceed to the relations of the circulation of the blood.

II. On blood-pressure.

In most cases we do not employ the determination of blood-pressure in order merely to measure the absolute height of the blood-pressure, but in order that from the variations which we observe in the blood-pressure we may arrive at conclusions with respect to the definite relations of the circulation. Have we the right to do so?

No one doubts that in the circulation of the blood we have to deal with relations analogous to those which are observed when a fluid flows through an elastic tube. If by means of a piezometer, or, as is usual in the vascular system, by means of a manometer we determine the pressure under which the blood stands, we determine the total pressure of fluid at that point, we determine D_x; and we have just seen that this pressure depends on circumstances existing at the point x itself. Thus, when from what we observe in the blood-pressure we draw conclusions concerning the relations of the circulation in places other

[1] I must observe, that these formulae express nothing else than dependence.

than that in which we determine this pressure, we must satisfy ourselves, that the variation observed in the pressure is not occasioned by any circumstance at the point itself and that the variation is produced from a variation in P_x.

We have now seen that $D_x = P_x + Z_x$, and that P_x depends on the lumen of the tube at the point x; and further that the lumen itself is dependent in a manner indicated by the formula $R_x = F[E_x, (P_x + Z_x)]$. Thus the first requisite for arriving at a conclusion concerning definite relations of the circulation from variations in the pressure at the point x is, that (1) R_x, (2) E_x and (3) Z_x are constant.

Let us see how far these conditions have been complied with in the usual method of determining the blood-pressure by the manometer of Ludwig's Kymographion. We do not propose to discuss the determination of the pressure in the capillaries, since the pressure in them cannot be determined in this way. We have therefore to dwell only on determinations of pressure in arteries and veins.

First condition: *constant lumen.* We generally connect the cannula of the manometer direct with the central end of an artery. We thus determine, as Donders observed, the pressure in the trunk from which this artery originates. That this may be true, the artery must have a strictly horizontal course, in other words the opening of the cannula must be in the same horizontal plane as the place in the trunk where the artery originates. (See further on.) Occasionally a T piece is used, by means of which the stream at the point of observation is not broken, and the pressure at the point itself is measured.

In this latter case the lumen remains constant at the point where we determine the pressure, for it is the lumen of the metal T piece. In the former case this is not perfectly true. With every variation of pressure in the trunk and this is the pressure which we are determining, the lumen will there change and this must react on the pressure itself. Fortunately, however, this change in the lumen is extremely slight, because the coefficient of elasticity of the arterial wall is high. The very slight changes in the lumen, which arise from difference of pressure, will exercise an influence on the pressure at that point, so small in comparison with the variations of pressure themselves, that we may safely neglect it. Only in case of very low arterial pressure and thus of small tension of the wall, can this change in the lumen be of importance.

This factor is of more importance in the veins. Here the coefficient of elasticity is much smaller, and the mean blood-pressure much

lower than in the arteries, and variations in pressure can thus give rise to important changes in lumen. We generally, however, in determinations of venous pressure, make use of a T piece, and in this case the difficulty is removed, because the lumen then remains constant. In the experiments I have still to speak of, I have generally used a T piece in the vein. I have occasionally applied the method which we generally use in the arteries, and bound the cannula in a lateral branch of a vein. Though this latter method is less accurate, yet it appears even with it, that, in the presence of the great variations in pressure we have to deal with, the inaccuracy occasioned by the varying lumen is thrown into the background.

In determining small variations of pressure, however, this really becomes of importance. Suddenly occurring small variations of pressure are always inaccurately indicated by the manometer. Properly speaking, every apparatus which we apply to determine the pressure of fluid occasions some disturbance in the actual state of things; this is the case even with an absolutely constant stream, but still more so with a stream like that of the blood, which is constantly undergoing variations. Every time a rise of pressure is indicated by the piezometer or manometer, a certain quantity of blood flows into the apparatus, and thus from the blood-vessel; the reverse takes place when a fall in the pressure is indicated. This must, of course, influence the flow at that point. Yet, for the registration of the pressure of blood the manometer is, as yet, the best apparatus. The Tonometer described some time ago by Talma[1] labours under this defect, that the lumen at the point where the pressure is determined, is constantly varying.

Second condition: E_x *constant.* What has been said of the first condition is also applicable to this, because with every variation of lumen E_x also varies. Yet something else must be added, which has a special interest with respect to the veins. In determinations made with a T piece E_x remains constant. If, however, in the jugular vein for instance, the pressure be determined by means of a lateral branch, and if the jugular vein at this point be at first quite free, then by the subsequent drying up of the outer coat of the vein, or by its becoming covered with blood-coagula from the wound, which then, as it were, form part of the coats, and especially if these coagula are dried up, the condition of the coats and with it E_x will be altered.

Third condition: Z_x *constant.* This is a very important factor. Z_x will, of course, remain constant if the animal remains in the same

[1] Talma. *Pflüger's Arch.* Bd. XXIII.

position. On a change of position Z_x may change, and a variation observable in the manometer will therefore be no measure of a variation in P_x. Moreover, the opening of the cannula into the blood-vessel must remain at the same height during the whole determination. (See later on the influence of gravity on P_x.)

If however we take these conditions into consideration, we have in the manometer an apparatus from whose variations we may draw conclusions concerning variations in P_x. What conclusions can we then draw as to the circulation of the blood, when we have thus come to a conclusion that P_x varies?

For this purpose it is necessary to gain an accurate representation of the circulation with respect to what we have said in Part I.

The blood flows from the action of the cardiac muscle. Whereas, in Part I, the pressure of the water-column in the reservoir is the force which propels the fluid, here it is the pressure exerted on the blood in the heart by the contraction of the cardiac muscle. The cardiac muscle contracts rhythmically. We may consider the force of the contraction as remaining constant during the systole, and should this not be really the case, we have then to consider what is the mean of the force exerted during the whole systole. During the systole, the blood flows from the heart in the same way as from a reservoir of a constant height during the time the cock is open. We may now suppose that the systole is always of the same duration, just as we previously did with respect to the cock being open, for instance t seconds. The quantity that flows through while the cock is open, and which we have called Q, is hence now the quantity of blood which the ventricle contains. The number of times the cock was opened every minute, which we called n, is now the number of contractions per minute. Thus nQ blood flows every minute through the arterial orifice. The pressure to which the blood in the ventricle is subjected during the systole is not to be considered as the pressure under the influence of which the blood flows. For just as in the case of the reservoir when the cock was opened a certain number of times, so here too we have to deal with a reduced height of pressure H_1, which is smaller than the real pressure prevailing in the ventricle during the systole. The blood, moreover, flows through a repeatedly branching elastic tube the collective lumen of the divisions of which constantly increases, while the lumen of each division becomes at last extremely small, capillary in fact. The resistances therefore greatly increase in the course of the tube. At last the blood returns to the heart. There is thus a pure circular flow.

The blood is nowhere in contact with the atmosphere, nowhere does the atmospheric pressure exercise any direct influence on the blood itself. In determining the blood-pressure we see that the manometer generally indicates a certain pressure higher than that of the atmosphere. (In stating the height of the blood-pressure we do not generally add the atmospheric pressure.) This is intelligible when we remember that the wall of the elastic tube (the blood-vessels) which in many places is very thin, stands under an external pressure, which is almost everywhere equal at least to the atmospheric pressure, and that the pressure which is exerted on the outer surface of the heart is only a few mm. *Hg* less than the atmospheric pressure.

We will now at first disregard Z_x (the hydrostatic pressure), and consider that the blood all flows in a horizontal direction. This is nearly always the case when we determine the pressure of blood in an animal lying on its back. We shall presently return to this subject.

Just as in the elastic tube attached to the reservoir, so in the blood-vessels we have to deal with velocity and hydrodynamic pressure. On the proportions of the two in the different parts of the vascular system I shall not here expatiate. Seeing that everything that we laid down in paragraph *g* is applicable here, we cannot indicate the velocity and hydrodynamic pressure until we have obtained an accurate knowledge of the magnitude of the muscular force of the heart, the dimension of the tube in all its parts, the number of ramifications, and the angles at which they occur, the magnitude and variations of the coefficient of elasticity in the different parts of the tube, and, last but not least, the extent of the different resistances in the different parts of the tube.

Thus we may consider, as in paragraph *g*, the blood-current as constant at n heart-beats a minute. Under what conditions will this remain so? Suppose the blood to be at rest. The heart begins to work. At every systole Q blood is pumped into the arteries. On applying the arguments of paragraph *g*, it follows that the capacity of the blood-vessels will constantly increase until M_n becomes equal to N_n, that is, if we for the moment think of the arteries only, until the quantity of blood pumped into the arteries during the systole, again flows into the capillaries during the succeeding pause of the cardiac action. As soon as this state of things has been reached, a condition of equilibrium has been established, in which the greatest quantity of blood has accumulated at those points where the hydrodynamic pressure has given rise to the greatest distension. In this state of equilibrium nQ blood will flow through every

section of the arterial system in a minute, and seeing that the blood again flows back to the heart through the capillaries and veins, nQ must also flow through every section of the whole system per minute. Proceeding from this principle, the explanation of the phenomena observed in every disturbance of this state of equilibrium, follows as a matter of course. The mean velocity in every section of the vascular system may be easily calculated. It is $v_x = \frac{nQ}{60\ \pi r_x^2}$ (reckoned per seconds); r_x is the sum of the radii of the blood-vessels which at the point x compose the area of the blood-vessels. P_x cannot be calculated, since we cannot calculate T_x. It is to be found by experiment only.

Now we find the hydrodynamic pressure P_x to be greater in the arteries than in the veins. This is, of course, not the cause of the flow of blood from arteries to veins. The real cause why the blood flows in this way is, that T_x is greater in the arteries than in the veins. In general we have, under normal circumstances, no sudden widenings in the vascular system, and we shall therefore nearly always find the hydrodynamic pressure at any given point greater than that at a point somewhat more remote in the direction of the stream. In a wide cylindrical or spindle-shaped aneurism it may, however, very well occur that the pressure is higher there than in the vessel which conveys the blood to it.

It is not clear to me what sort of idea Cohnheim forms of the blood-current, when he says[1]: "denn es ist ja unzweifelhaft richtig, dass besonders bei den umschriebenen aneurysmatischen Erweiterungen des ganzen Aortenumfangs ein Theil der Triebkraft, mit der das Blut vorwärts bewegt wird, dazu verwendet werden muss, die Bluttheilchen von einander zu reissen, d. i. die innere Cohäsion der Blutflüssigkeit zu überwinden, was bekanntlich bei der Fortbewegung in parallelen Linien, also in einer Röhre von unverändertem Lumen, mit dem geringsten Kraftaufwand geschieht." The elastic wall of the blood-vessels is of great importance to the blood-current, because by this means the shock which the fluid in a non-elastic tube, and thus the wall itself, would experience, is very much diminished. Owing to this the blood-current really acquires more the character of a constant stream. Moreover the lumen of the tube becomes wider under the influence of the hydrodynamic pressure, and a wider tube offers less resistance. The

[1] Cohnheim. *Vorlesungen über allgemeine Pathologie.* 2te Aufl. Th. 1. p. 94.

elastic wall in itself, however, has no bearing on this resistance nor, disregarding the shock, is it important to the cardiac action. If we imagine the tube to possess a non-elastic wall and yet to be made permanently as wide as, when elastic, it becomes under the influence of the hydrodynamic pressure, the heart would have to do the same amount of work in order to pump the same quantity of fluid through it, as through the elastic distended tube. The tube being of the same width nQ fluid will flow through every minute in both cases; and in order to effect this the same amount of energy is required. The elastic wall acts with respect to each contraction of the heart in the same way as does an elastic band between a horse and a waggon. Here too the shocks are diminished, but the horse must expend as much labour to draw the waggon along the same distance, with an elastic band as without one.

When Cohnheim, at page 95 of the work just mentioned, discusses the influence on the blood-current of the loss of elasticity in the wall he says: "Was aber dieser Elasticitätsverlust für die Circulation bedeutet, liegt auf der Hand: es kommt der ganze Antheil mehr oder weniger in Wegfall, welchen die Elasticität der Arterienwand an der Vorwärtsbewegung des Blutes hat."

This "ganze Antheil" is certainly not great, for in the state of equilibrium of the blood-current it is nothing. The "Vorwärtsbewegung" of the blood is caused entirely and solely by the action of the heart; and whether a certain amount of energy is detained for a moment in the wall during the systole of the heart, in order to be afterwards used, or whether the same amount of energy is exerted immediately on the fluid, is quite a matter of indifference to the "Vorwärtsbewegung". If, owing to morbid states, the elasticity of the coats of the vessel is diminished, the resistance becomes greater, because the lumen of the vessel at every systole of the heart cannot increase so much and the mean diameter of the vessel thus becomes smaller. In addition to this the lining generally becomes much rougher, which will give rise to still greater resistance, because there arise all kinds of irregular collisions of the particles of the fluid against these roughnesses.

With respect to the blood-current another factor comes into consideration. The heart works, not merely as a forcing-pump during its systole, but also as a suction-pump during its diastole[1]; and just as the effects of the action of forcing are to be best observed in the

[1] S. de Jager. *Pflüger's Arch.* Bd. xxx. p. 491.

arteries, so are those of the action of suction in the veins. Here we may apply the observations previously made at the close of paragraph *e*. The suction may be considered as negative pressure on the current, and the hydrodynamic pressure in the tube can thereby become negative. If then we consider the pressure in the veins, especially in those which lie nearest to the heart, we find it to be the algebraic sum of the hydrodynamic pressure resulting from the energy imparted to the fluid by the systole of the heart, and of that resulting from the energy caused by the diastolic aspiration. The latter will be negative, (with respect to the atmospheric pressure), and it therefore depends on the respective magnitude of each whether the sum will be positive or negative.

The blood, it is true, makes a complete circuit, and the atmospheric pressure is nowhere directly exerted upon it; but, as I have already observed, it must really be taken into account with respect to the blood-pressure, especially since the heart during its diastole must overcome the pressure working on its outer surface. When the thorax is closed this pressure is the atmospheric pressure minus the elastic action of the lungs: when the thorax is open it is the full atmospheric pressure. Hence if it were the blood coming from the veins which had to bring the heart into a diastolic state, it would have to overcome this pressure. But as I have already said, the heart also works as a suction-pump, and we can really show negative pressure during the diastole, at the end of the venous system, viz. in the auricles, even when the thorax is open.

That it is so difficult to indicate this negative pressure in the veins themselves, at a certain distance from the heart, is sufficiently obvious from what has been said above. In the article I have referred to above, I discussed these subjects at greater length, and I shall presently return to them.

While speaking of this action of the heart, it may not be superfluous to make an observation on the manner in which Hermann in his text-book[1], which in other respects is excellent and therefore generally used, tries to represent this action. At page 48 we find given as a scheme, for the action of the heart, a force- and suction-pump with a double piston. By the working of this instrument a constant current may be set up in the tube conveying the fluid, but the objection to this instrument is, that it is not possible to have a pause corresponding to the pause of the cardiac action. In the heart-pause every compartment of the heart

[1] L. Hermann. *Kurzes Lehrbuch der Physiologie.* 1882. 7te Aufl.

is in a diastolic state; with this pumping-apparatus it is not possible for both pistons to stand at the maximum height at the same time. The current in the veins near the heart is then in reality not constant. During every heart-pause an accumulation of blood in the veins really occurs, hence during this period a storage takes place. We also perceive variations in the pressure in the jugular vein synchronous with the heart-beats, when we register that pressure by means of a manometer.

Disturbances in the above mentioned state of equilibrium of the blood-current may arise under different circumstances: viz. if any change occurs, 1st in Q, 2nd in n, 3rd in the force of the systole, or 4th in the resistance somewhere in the vascular system. We will first discuss these effects on the arterial blood-pressure, considering the veins simply as conducting tubes.

1st. If Q increases nQ will become greater, and thus v_x too. If a greater quantity of blood flows through per minute, the height of pressure which we have taken as H_1, (in the heart) must be considered as greater, and thus P_x will also increase with v_x. This is possible if the force of the contraction of the heart increases at the same time; and in this case the energy imparted to the fluid mass m will be greater, and with it v_x and P_x. It is also possible if the time t, necessary for the blood to flow out into the origin of the arteries, increases with the increase of Q. If n remains the same, then the pause between two contractions of the heart will become smaller, and thus there will be less time for $N_n = M_n$ to escape from the arteries. The number of terms of the series $(M + M_1 + M_2 + \ldots\ldots) - (N + N_1 + N_2 + \ldots\ldots)$ will thus increase, i.e. the capacity of the arteries will increase, and thus also the tension in the wall. As follows from what has been said in paragraph g, the height of pressure H_1, and with it v_x and P_x, will become greater when t becomes greater, the force of the systole remaining the same. Thus with the increase of the quantity of blood which the heart transmits at each systole, velocity, hydrodynamic pressure, tension in the wall, and capacity of the arteries become greater.

2nd. If n, the number of heart-beats per minute, becomes greater, nQ will also become greater and with it the height of pressure H_1. The result is thus the same as in 1.

3rd. If the force of the contraction of the heart increases, this is the same as saying that the real height of pressure in the reservoir, and therefore also the 'reduced' height H_1, increases. This will be of effect only when Q at the same time increases with it, and in this case what has been said in 1 is applicable.

4th. If anywhere in the vascular system the resistance becomes greater, then, with the same action of the heart, less fluid will flow through the point of increased resistance. Seeing that the quantity of fluid flowing through every section in the unit of time must always be the same, this quantity will decrease in every other point of the vascular system, nQ will everywhere become smaller. In order that the state of equilibrium may be reestablished nQ must decrease, the action of the heart, and thus also H_1 remaining the same; or, in order that nQ may flow through the point of increased resistance, n or Q or the force of the systole must increase. Since the heart does not work, in normal circumstances, with its maximum force, it is possible for the last-mentioned change to take place. The effects for v_x and P_x in this case are like those described in 1, 2 and 3.

If the equilibrium is reestablished by a decrease of nQ, the action of the heart remaining the same, then neither n nor t must decrease, for H_1 would thereby decrease. But n and t and the force of the systole remaining the same, then Q must decrease. If nQ becomes smaller, v_x also becomes smaller, and since H_1, and thus the quantity of energy imparted to the mass m remains the same, more will be left for $P_x = T_x - \frac{1}{2} mv_x^2$. The hydrodynamic pressure at a point between the heart and the point of increased resistance will thus increase, while the force of the systole remains the same. I wish especially to point out that this increased hydrodynamic pressure does not all contribute to restore the state of equilibrium. In order clearly to prove this let us take a reservoir with a constant height from which fluid flows constantly through an elastic tube of uniform width. If A fluid flows through in the unit of time, then if by compression we narrow the tube at any place, the resistance at that place will become greater, and the quantity of fluid now flowing through will become smaller than A. With the decrease of A, v_x also decreases. Seeing that the height in the reservoir remains the same, the fluid mass m will flow into the tube with the same energy, whether there is any narrowing or not; but since, owing to the part of the tube having been narrowed, v_x and thus $\frac{1}{2} mv_x^2$, has become smaller, more will remain for the potential energy $P_x = T_x - \frac{1}{2} mv_x^2$ between the reservoir and the part narrowed. Everywhere between the reservoir and the part narrowed, m will possess a greater potential energy, and thus the hydrodynamic pressure will be greater.

Beyond the place narrowed the state of things is different. The mass m will approach the narrowing with greater energy than it would

possess, if there were no narrowing, but on its passing through the narrowed part, much energy will be lost. T will hence largely diminish; and, though at a point y beyond the narrowing the velocity v_y, and with it also $\frac{1}{2} mv_y^2$, will be smaller than when there was no narrowing, yet there will remain less for P_y, since T_y is so much smaller. If we call the point where we have effected a narrowing B, the hydrodynamic pressure in front of B will have increased, and that beyond B decreased in comparison with the pressures, which existed at the same points, before B had been narrowed. We might here say: the difference of pressure between the points in front of and beyond B has been increased. Yet the quantity of fluid flowing through has decreased. The greater difference of pressure is thus merely the consequence of the narrowing. If now there is a cock at the origin of the tube, then, on the cock being opened n times a minute, the state of things remains the same, only with this difference that the height in the reservoir must be supposed to be H_1. Upon B being narrowed not nQ but nQ_1 will flow through the cock, nQ_1 being $< nQ$. To make nQ_1 again equal to nQ, the height in the reservoir must be raised, or n or t must increase.

Hitherto we have spoken of the effects of the four conditions which give rise to a disturbance of equilibrium only with respect to the arterial blood-pressure. Owing to the complete circuit which the blood makes, these conditions must also exercise an influence on the venous blood-current. When variations occur in nQ they must occur everywhere in the vascular system, and thus in the veins too. If the quantity nQ which flows from the heart into the arteries is increased, then a state of equilibrium is possible only when the quantity which flows through the veins into the heart is equally increased. We can directly prove this from the relations which appear in the case of the reservoir.

If in this case by any circumstance nQ increases, more fluid will have to be carried into the reservoir, in order to keep it at a constant height. In the same way the heart must receive more blood from the veins, if it is to pump more into the arteries.

If, owing to any of the conditions mentioned in 1, 2 and 3, nQ increases, the hydrodynamic pressure and the velocity of the blood-flow will increase. Let us, for convenience sake, suppose the increase of nQ to be occasioned by an increase of n. With n heart-beats, the blood still comes with a certain energy through the veins into the heart. This energy is still so great that not only the actual energy

of the blood-mass m is still present, but that $T_x - \frac{1}{2}mv_x^2$ has not even yet become nil. P_x still possesses a certain magnitude even in the veins close to the heart. The cause of this relation must be sought in the fact, that from time to time the blood ceases to flow freely into the heart, viz. during the systole. This condition may be compared to one in which a cock is supposed to be placed at the end of the venous system, and to be opened at every diastole. The quantity which can flow through the cock will depend on the number of times the cock is opened per minute, and on the quantity which can flow through during each opening.

If the cock is now opened more than n times a minute, then more fluid will flow out in a minute. We can compare this condition to one in which the mean resistance at the end of the tube decreases. Thus the effect will be that the fluid flows out with greater velocity, and that P_x will consequently become smaller. If the number of heart-beats per minute becomes greater, the number of diastoles will become as many times greater. Hence the mean resistance at the end of the venous system decreases; the velocity in the veins will thus increase, and the hydrodynamic pressure decrease. Seeing that the decrease of P_x upon the increase of the number of diastoles will be most perceptible in the veins, and the increase of P_x upon the increase of the number of systoles will be most perceptible in the arteries, the hydrodynamic pressure will under these conditions increase in the arteries and decrease in the veins. Thus, the simultaneous increase of the number of diastoles co-operates with the increase of that of the systoles.

The same effect will ensue if nQ increases by an increase of Q. The fall of the pressure in the veins cannot of itself be considered as a proof of the suction-force of the heart; but, since it has been proved in other ways that the heart during its diastole really possesses a suction-force, the natural consequence of the increase of the number of diastoles, or of the quantity of blood which is drawn in at each diastole, will be increase of velocity and decrease of hydrodynamic pressure in the veins, as may be inferred from what has already been said.

In case 4, the resistance had increased somewhere in the vascular system. In whichever part of the vascular system this may have occurred, the hydrodynamic pressure will have increased in front of the point of increased resistance, i.e. between it and the heart. Beyond this point, it will have decreased. If equilibrium has been reestablished, in consequence of nQ, owing to increased cardiac action, flowing through the place of increased resistance (as has been discussed in 4), we may

then apply to the veins (beyond the point of increased resistance), what we have just said. The hydrodynamic pressure in the veins will thus decrease. If the cardiac action remains the same, and thus less than nQ actually flows through because Q decreases, then less will flow out of the veins, and an elevation in the hydrodynamic pressure in the veins will arise, (viz. of that part of the pressure which is determined by the diastole). The last-mentioned condition will arise in the organism only when the heart is already performing its maximum labour before the moment at which the increased resistance is developed.

From all this there will follow: 1st, upon an increase of the number of heart-beats (n) a rise in the arterial, a fall in the venous blood-pressure; 2nd, upon an increase of the quantity of blood (Q) which is pumped out at each systole, a rise in the arterial, a fall in the venous blood-pressure; 3rd, upon an increase of the force of the systole (taking effect only when Q is increased), a rise in the arterial, and a fall in the venous blood-pressure; 4th, upon an increase of resistance[1] anywhere in the vascular system, a rise in the pressure in front of this point and a fall in the pressure beyond it.

Upon a decrease of any of these factors, all that has been said of them will be reversed.

It further follows, from the elasticity of the wall of the blood-vessels, that where the hydrodynamic pressure varies, the capacity of the tube will also always vary with it. Where the capacity increases a certain quantity of blood is detained, where it decreases, a certain quantity is expelled. Hence in order to bring about a state of equilibrium, it is necessary that the capacity should again become constant, that M_n should become equal to N_n. The circuit of the blood, in connection with the peculiar action of the heart, just makes this possible with the limited quantity of blood present in the vascular system. We have seen, that just when in the arteries the hydrodynamic pressure, and with it the capacity, increases, both of them decrease in the veins. Owing to the complete circuit and the limited quantity of blood, a simultaneous rise of hydrodynamic pressure, and thus also of capacity in both arteries and veins is impossible. Upon a disturbance of the state of equilibrium, and upon its being restored, or upon a new one arising, a certain quantity of blood is shifted from one part of the vascular system to another.

[1] I need hardly observe that I speak only of the increase of resistance in the total section of any area of the vascular system. Increase in any part of this section may be compensated by a decrease in another part.

Hence, any change of pressure which we observe in the manometer must, if at the place where we determine this pressure E_x, R_x, and Z_x remain constant, be occasioned by one or more of the above mentioned circumstances. I shall speak presently concerning the influence on the blood-pressure of variations in the capacity of the blood-vessels due to circumstances operating externally on the blood-vessels.

A series of experiments has led me to the above mentioned views. I will not maintain that these views are in every respect quite new. What is new on this earth of ours? Yet I thought it might be useful to embody them in a clear and concise form.

We may with ease experimentally change nQ, and we shall then see that what has been discussed under 1, 2 and 3 does actually take place. It is easy to change n; there are greater difficulties in changing Q, or the force of the systole.

In such experiments I always determined simultaneously the pressure D_x in an artery (the carotid) and in a vein (the jugular). For this purpose I attached to Ludwig's Kymographion a double manometer. To the carotid was attached, in the usual way, one of the manometers containing mercury. The other manometer was filled with a saturated solution of bicarbonate of sodium. In each manometer a float armed with a pen rested on the surface of the fluid. At first, however, it was not easy to construct for the soda-manometer a float, which should respond accurately to every oscillation of the level of the manometer. The float must, of course, be light, and has then the drawback of dancing up and down at every change in the level of the fluid; but in addition to this the great drawback is, that it constantly keeps adhering to the wall of the tube made wet by the soda. After a long trial I was most satisfied with a small ebonite bell placed on the soda-level. On the top of this bell is affixed a thin aluminium rod, and to this the registering pen. This little bell, which floats by reason of the air contained in it, must correspond in diameter exactly with that of the tube of the manometer, and, that it may nowhere be jammed, the latter must be everywhere precisely of the same bore. Seeing that the wall of the tube is always moist, no air, but only a small layer of fluid is found between the wall of the tube and the edge of the bell; on this account the friction is extremely small, and the dancing up and down on the level of the fluid is effectually prevented. Upon trial it appeared to me that this little bell responded to even very slight oscillations still more accurately than the usual float of the mercury-manometer does to sudden oscillations of the level of the mercury.

On the "papier sans fins" I thus obtained the curves of the pressures in the carotid and in the jugular, the heights of which above the zero line were expressed in the carotid in millimeters of mercury (Hg), in the jugular in millimeters of the solution of bicarbonate of sodium (CS).

In a dog of $4\frac{1}{2}$ kgrm. the blood-pressure in both vessels was registered in this way in morphia and chloroform narcosis.

The pressure in the right carotid was ± 86 mm. Hg, in the left jugular ± 134 mm. CS;

After this both the vagosympathetic nerves were cut through, owing to which *n* very much increased.

The pressure in the carotid rose to ± 134 mm. Hg.

The pressure in the jugular fell to ± 9 mm. CS.

In experiments like these it is necessary to observe whether upon the section of the nerves the respiration is greatly modified. If strong convulsive respirations arise, these greatly influence the blood-pressure in both kinds of blood-vessels (I shall afterwards return to this subject), and the direct effect of the section of the nerves on the blood-pressure cannot be ascertained. The above experiment shows that the great increase in the number of heart-beats which follows upon section of both the vagosympathetic nerves occasions, as has been discussed above, a rise in the arterial pressure, and a fall in the venous.

Another instance: A dog of $13\frac{1}{4}$ kgrm. in morphia and chloroform narcosis. The pressure in the right carotid is ± 150 mm. Hg, in the left jugular ± 79 mm. CS. After section of both the vagosympathetic nerves the pressure in the carotid is ± 174 mm. Hg, in the jugular ± 45 mm. CS.

Still clearer is the result of a decrease of the number of heart-beats, or of an actual stoppage of the heart owing to stimulation of the vagus. We then see upon a fall in the carotid-pressure a rise in that of the jugular. I have many experiments showing this. If we stop the stimulation so that the heart-rate increases again, we then see the pressure in the carotid rise to its normal height, while the jugular pressure again falls to its normal height.

Instances: A dog of 30 kgrm. in morphia and chloroform narcosis. Both the vagosympathetic nerves are cut. The pressure in the right carotid is ± 98 mm. Hg, in the left jugular ± 46 mm. CS. The left vagus is stimulated, in consequence of which the heart-rate is greatly reduced. The pressure in the carotid falls to ± 48 mm. Hg, that in the jugular rises to ± 88 mm. CS.

After the stimulation has ceased both return to their normal height.

A dog of 10½ kgrm. in morphia and chloroform narcosis. Both the vagosympathetic nerves are cut. The pressure in the right carotid is ± 144 mm. Hg, in the left jugular ± 100 mm. CS. The right vagus is stimulated. The heart ceases to beat. The carotid pressure falls to ± 18 mm. Hg, the jugular pressure rises to ± 140 mm. CS. After the stimulation has ceased both return to their normal height.

The way in which the rise and fall occur in the carotid pressure is a very peculiar one. If the stimulation is so strong that the heart suddenly ceases to beat, the carotid pressure falls at first very rapidly, but the inclination of the fall soon changes. At the moment that, in the last mentioned case, the carotid pressure was 18 mm. Hg, the curve was almost horizontal. The reason of this is, that, after the cardiac action has ceased, the blood keeps flowing under the influence of the energy existing in it, but it is not this energy alone which keeps up the flow. It is now especially the energy accumulated in the wall of the arteries which comes into action, and keeps up the flow. Seeing, however, that no more fresh blood is supplied by the heart, this energy present in the wall is continually diminished by consumption, and the tension in the wall continually decreases. The force with which the blood is propelled becomes in consequence smaller and smaller. Hence the velocity also continually decreases. The current will last until the tension in the vascular system has everywhere become the same. It appears from the above instance, that, if equilibrium is established with a pressure of 18 mm. Hg in the carotid, there is in the jugular a pressure of 140 mm. CS only, that is to say, the tension in the arterial system is still greater than in the venous, notwithstanding that the heart has ceased to beat for 15 seconds. Rollett[1] asserts that even after cardiac action has ceased half-an-hour, or longer, the stream in the capillaries still continues. Thus it is a very long time before the tension in the vascular system has become everywhere the same.

This question is of importance, because it follows hence that, if in order to trace the waves in the blood we trace the waves in a tube attached to a reservoir, we must not compare the artery to a tube having a large and free opening to the atmosphere. For in such a tube upon a sudden interruption to the flow of fluid, as by closing a cock, the pressure in the tube will not only rapidly decline to zero, but will even become negative. Nothing of the kind is seen in the arteries upon a sudden suspension of the cardiac action; and this is due to the

[1] Rollett. *Handbuch der Physiologie, herausgegeben von* L. Hermann, Bd. IV. p. 317.

enormous resistance the blood meets with at the end of the arterial system in the numerous and very small capillaries. I have previously[1] shown by means of a minimum manometer that a negative pressure is never observed in the aorta.

Since in the above mentioned experiment I had inserted a T piece in the jugular, I could compress the jugular either on the cardiac or capillary side of the attachment of the manometer. In the latter case the jugular pressure largely diminished, often even to below zero.

In a dog of 21 kgrm. in morphia and chloroform narcosis, the vago-sympathetic nerves of which were cut, stimulation of the vagus gave the same effect as that which has been described above. Without stimulation the pressure in the jugular was ± 21 mm. CS. The jugular was then closed on the capillary side of the T piece, and the manometer fell to ± 2 mm. CS. The pressure thus measured was however really that of the vena cava sup. If the vagus was now stimulated the carotid-pressure as usual again fell, while the pressure in the vena cava rose to ± 10 mm. CS.

When in this dog the jugular was clamped on the cardiac side of the T piece the manometer of the jugular rose to ± 247 mm. CS. The pressure in the carotid at this moment was ± 100 mm. Hg. On the vagus being now stimulated the pressure fell in both the blood-vessels, in the carotid to ± 16 mm. Hg, in the jugular to ± 165 mms. CS.

This simultaneous fall in both the blood-vessels upon stimulation of the vagus, when the jugular is closed on the cardiac side of the T piece, does not always appear with equal clearness; sometimes the fall in the jugular all but fails. The cause of this lies in the valves of the veins. Let us for a moment consider this matter a little more closely. When the jugular is closed on the cardiac side of the T piece, the manometer no longer indicates the usual venous pressure; the vein must now be considered rather as a simple continuation of the artery. If it were not possible for the blood to flow away anywhere else than through the part in the vein which has now been closed, the pressure in the vein would then really become equal to that in the artery from which the vein receives the blood. Since however collateral paths are usually present, this seldom will be attained. Yet the venous pressure approximates to the arterial pressure, and if, owing to a reduced heart-rate, the pressure in the artery declines, that in the vein will fall with it. If this fall in the vein does not occur it is because the flow of the blood out of the vein is prevented by the valves.

[1] S. de Jager. *Pflüger's Arch.* Bd. xxx.

I had first frequently observed the simultaneous fall in both blood-pressures, and I was surprised, when, sometimes under circumstances in other respects the same, the fall in the jugular did not occur. I accordingly so arranged an experiment that, while the cock between the manometer and the jugular was closed, I reduced the manometer of the jugular to zero. If, after this, this cock is opened, the jugular manometer begins and continues to rise. During this rising the vagus is stimulated. The regular rise of the manometer then instantly ceases. The manometer cannot fall as I have already said, owing to the valves in the veins, but the absence of a further rise proves that the pressure in the jugular has virtually diminished. As soon as the stimulation ceases, and the carotid pressure thereby rises, the regular rise in the jugular manometer again occurs until upon a new stimulation it again ceases. This may be repeated till the jugular manometer has risen to the pressure which prevails in the jugular without vagus stimulation.

I might add to the above instances many more, all of which show the same results. It is sufficiently obvious from them, that really upon the increase of n, the pressure rises in the carotid and falls in the jugular, and that upon the decrease of n the pressure falls in the carotid and rises in the jugular; but that there may exist circumstances which modify this effect. In making the experiment we must take care that no powerful irregular respiration modifies the effect; to prevent forced respiration with a closed glottis, I first in every experiment performed tracheotomy. Moreover we must bear in mind that owing to an accidental coagulation in the jugular, on the cardiac side of the T piece, or to an accidental compression, or a marked narrowing at this part, the reverse effect may take place.

As I have already observed, the pressure in the jugular sometimes falls below zero, if the jugular is closed on the capillary side of the T piece. Since in this case, it is the pressure in the vena cava sup. which is really measured, it is thereby proved that the pressure in this vein is at that time negative. Other investigators have also found the pressure in the vena cava sup. negative[1]. This proves nothing as to the suction force of the heart. When the thorax is closed, there is a negative pressure in the cavum thoracis, which is exerted on the outer surface of the heart, and is able by itself to give rise to a negative hydrodynamic pressure in the veins close to the heart. When the thorax is open I have never seen the pressure fall below zero. We shall see, in discussing the influence of respiration on the venous

[1] Volkmann. *Haemodynamik*, and Jacobson, *Arch. f. path. Anat.* XXXVI.

blood-pressure, why the suction force of the heart, when the thorax is opened, cannot give rise to negative hydrodynamic pressure. I have already previously spoken of this matter. I have tried, however, to show in other ways that the heart really sucks the blood out of the veins.

I have several times repeated the experiments I am now about to discuss. A dog was completely curarised. The mercury-manometer was attached to a carotid; the thorax was opened, and the respiration kept up by means of a pair of bellows. The soda-manometer was attached to a glass tube, and the latter was inserted, through the jugular, into the vena cava sup. while the other jugular was closed by means of a ligature. When the pressure in the two blood-vessels had been accurately registered, the thoracic aorta was suddenly severed, after the animal had been brought into apnoea by means of *O*, and the artificial respiration had ceased.

I hoped and thought that, after section of the aorta, the heart would still keep working on with sufficient force to suck out the blood from the veins, and to pump it into the arteries. Seeing that no new blood is now conveyed from the arteries, and that therefore no hydrodynamic pressure, due to the systolic action of the heart, any longer arises in the veins, the hydrodynamic pressure resulting from the diastolic action of the heart, i.e. a negative pressure, ought to become more distinct.

The result did not answer the expectation, though I made some observations of great importance. Goltz and Gaule[1] have already shown that even in the ventricles a negative pressure can be indicated only when the heart is contracting powerfully. If the heart has become exhausted, there is no trace whatever to be found of a negative pressure.

Now the conditions of our experiment are as unfavourable as they can possibly be for the heart's action. In the first place the effect of the curare is bad for the heart, secondly so is the open thorax, thirdly so is the accompanying loss of blood, and fourthly, the section of the aorta is most unfavourable of all for the heart's action. The heart will then no longer contract with its normal force, and it is no wonder that no powerful relaxation then ensues. Other factors, which may contribute to the relaxation, now disappear, such as the tension in the arteriae coronariae (Brücke).

Immediately after the section of the aorta, we see the carotid pressure fall in a vertical line to zero. (The line of descent is quite

[1] Fr. Goltz and J. Gaule, *Pflüger's Arch.* Bd. xvii. p. 100.

different from that obtained when the heart ceases to beat in consequence of vagus-stimulation. In this case the conditions resemble those of a tube opening freely into the air.) The jugular or more properly speaking, the vena cava manometer also markedly falls, but more slowly than that of the carotid. We see that at every heart-beat blood is really pumped over, for the carotid manometer rises at every heart-beat in order to fall again to zero. I generally observed, that, if the heart absolutely ceases to beat, there is a certain positive pressure still present in the veins; this is owing to the fact that the blood can no longer flow into the heart, nor can it, owing to the valves, flow back to the capillaries.

I once saw in a dog, after the cessation of the cardiac action upon the aorta being divided, that the pressure in the carotid was zero and in the jugular still 30 mm. CS. After a few seconds the heart began to beat again. At each systole the rise in the carotid manometer was again visible. The jugular manometer had again begun to fall, and on its curve the influence of each heart-beat could be distinctly observed. As the elevations in the carotid curve at each heart-beat became lower, the jugular manometer, at the moment when there were no longer any elevations visible in the carotid-curve, though the heart continued to beat, had fallen to zero.

I have occasionally observed actual suction, and saw the manometer of the vein fall to a few mm. CS. below zero.

A slight change in the experiment afforded me another important result. The experiment was made in the same way, but instead of dividing the aorta the vena cava inf. in the thorax was closed in order to cut off the flow of blood to the vena cava sup. as much as possible. The pressure in the vena cava sup. fell to zero. The carotid pressure also fell considerably (we shall presently return to this phenomenon), but it still remained 60 mm. Hg. With this zero pressure in the vena cava sup., the heart was still able to pump blood from the veins, which open into the vena cava sup. (except the two jugulars which were ligatured), and to keep the pressure in the carotid at 60 mm. Hg.

How much, however, the heart suffers under this imperfect flow of blood to it appears immediately from this, that if we again release the vena cava inf. and the two manometers again begin to rise, these do not again return to their previous height. The carotid pressure remains lower. But the fact is especially important, that if we for the second time compress the vena cava inf. the pressure in the vena cava sup. no longer falls to zero, but even remains $\pm$ 40 mm. CS. above zero. We

have here the proof that, while the heart has now suffered, it is no longer capable of such powerful diastoles, and thus can no longer pump out the vena cava sup. by suction so effectually as it did the first time.

I have already observed that there are objections to changing Q artificially. The influence of this change I have occasionally traced by obstructing the diastole of the heart in a curarised dog, with open thorax and artificial respiration. I laid the whole flat of my hand around the heart and then compressed the heart a little with a force as constant as possible. In this way the diastole is obstructed, and we may assume that at each diastole less blood is taken into the ventricle, and thus less pumped into the artery at the next systole, in other words that Q really decreases. In this case too we see the pressure fall in the carotid and rise in the jugular. The experiment is not fully convincing as to the influence of Q, for n also varies under this operation, and we do not know how it fares with the force of the systole.

Cohnheim in the first edition of his *Vorlesungen über allgemeine Pathologie*, p. 36, to show the influence of obstructed diastoles on arterial and venous blood-pressure, describes two experiments, which, in my opinion, prove nothing on this point. In one experiment he compresses the thorax of a rabbit. In this case so many conditions in the thorax which may influence both blood-pressures are changed, that the effect thence arising can by no means be regarded as simply the effect of obstructed diastole. We may say the same of the second experiment, in which he inserts into one of the pleural cavities of a dog a caoutchouc-balloon, and then inflates the balloon. In the second edition of the book, these two experiments are no longer mentioned in treating of obstructed diastole, but Cohnheim takes an experiment of François-Frank[1], which proves much more directly what we have said above. In this experiment the pressure on the outer surface of the heart is raised in the pericardial cavity by means of air or fluid. The effect here also is a fall in the arterial and a rise in the venous pressure. The only objection I have to this experiment is that the possibility exists that the great veins which carry the blood to the heart will be narrowed by the higher pericardial pressure, and that the effect which is observed cannot with certainty be attributed solely to the obstructed diastole, but may also in part be due to the increased resistance at the end of the great veins.

[1] Fr. Frank, *Gazette hebdomad.*, 1877. No. 29.

With this I will now leave the influence of variations in cardiac action on the arterial and venous blood-pressures.

Variations in resistance somewhere in the vascular system frequently occur both in physiological and pathological conditions. We have only for instance to remember the varying tonus of the smaller arteries. I shall not discuss the influence of all these resistances; they may of themselves, as it were, be inferred from what has been previously said. Nor have I extended my experiments over all these possibilities. Moreover, I wish at greater length, in Part III. of this article, to treat of certain of the principal causes of ever-varying resistances in the vascular system, viz. those which accompany respiration. For the moment, I wish to confine myself to the influence of changed resistance in one of the great veins or arteries, especially because we shall then see that, though the principles we have laid down remain true, there may be circumstances which may produce a great change in the effect.

In the venous system a great resistance may be occasioned by compressing the vena cava inf. either immediately below or above the diaphragm. By this means a great part of the venous system is placed *hors de combat.*

The blood-pressure was again registered in a carotid and in a jugular. The thorax and abdomen were opened. Tracheotomy was performed and artificial respiration carried on. Both the vago-sympathetic nerves were cut. The experiment may be made under morphia and chloroform narcosis, or with curare. The last method has this advantage that all muscular contractions are thereby prevented, and that, by giving *O*, we may easily bring the animal into a state of apnoea and then stop the artificial respiration. Yet there is no great objection in continuing the respiration, if the respiratory movements always occur at the same rate and to the same extent. With a pair of bellows moved by a motor with uniform rapidity and uniform force this may be easily attained.

The result I arrived at from both methods of experiment was the same: on compressing the vena cava inf. the pressure falls both in the carotid and the jugular; on again releasing the vena cava inf. the pressure rises in both blood-vessels.

This is apparently in conflict with the conclusions which we formerly arrived at, but the cause of this apparent deviation lies in the limited quantity of blood present in the vascular system, in connection with the difference in the coefficient of elasticity of the different parts of this system.

When the vena cava inf. is closed, the resistance in the venous system greatly increases, for the blood can now flow back from the aorta to the heart only through the remaining veins. On the side of the obstruction away from the heart, the vena cava inf. with all its ramifications towards the capillaries, the capillaries themselves, and the arteries from which these arise, are all excluded from the blood-current. If we attached a manometer to the vein near the place compressed, on the side towards the capillaries, we should see that the pressure here was very high, for it would be pretty nearly the same as that in the artery. Supposing that there are nowhere small collateral communications, that all the blood passing through the aorta abdominalis under ordinary circumstances is carried off to the vena cava inf., then this part of the vascular system is, as it were, quite excluded from the rest, and the quantity of blood accumulated in this part and thus excluded from the circulation is not small, for, owing to the higher pressure, which arises on the capillary side of the obstruction, the capacity of the veins is greatly increased. That part of the whole quantity of blood which still participates in the circulation has thus become much smaller, and not only so, but the quantity of blood, which now flows through the remaining part of the vascular system, is also much smaller than the quantity which flowed through the same part before the compression. The condition of the vessels through which the blood still circulates resembles that, which we should obtain, if we had removed from them a certain quantity of blood, and the mean tension in them had thereby been decreased, without any decrease of cardiac action.

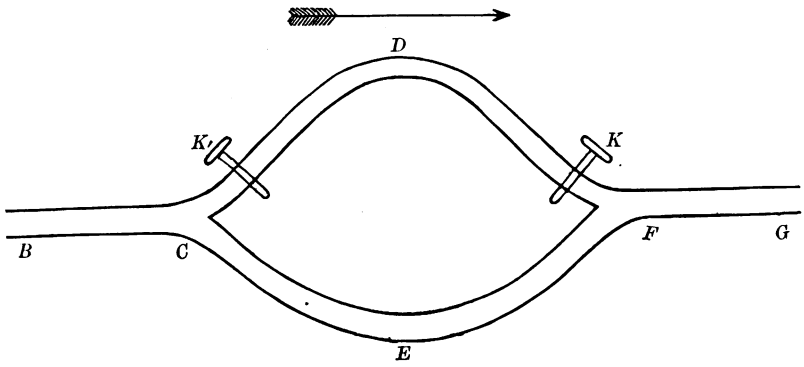
Thus a totally new state of equilibrium arises with a smaller vascular system, but, moreover, with a quantity of blood which is smaller than that properly belonging to the part of the vascular system left for the circulation. If the coats of the veins had the same coefficient of elasticity as those of the arteries we should then see, on compressing the vena cava inf. not a fall, but a rise, in the carotid pressure. This is a fact we must attend to in every increase of resistance in the venous system.

We must not suppose that closing the vena cava inf. simply cuts off part of the supply of blood to the heart. The supply to the heart is, indeed, actually in part thus cut off, but the fall in the carotid pressure is owing to a very great diminution in the quantity of blood which the heart receives in consequence of the enlargement of the capacity of the veins on the capillary side of the place compressed. If this capacity were not enlarged, i.e. if the veins had a great coefficient of elasticity,

then the heart would still receive less blood on compressing the vena cava inf., but the carotid pressure would rise.

To represent this matter clearly, I will imagine a reservoir attached to a non-elastic tube *BC* (figure 5) which tube divides into two parts *CDF* and *CEF*, these two parts again uniting into one tube *FG*. The

FIG. 5.



fluid in the reservoir has a constant height. If now the fluid flows through, there will be a certain hydrodynamic pressure in *BC* and *FG*. In the unit of time *A* fluid flows out at *G*. At *K* there is a cock. If this is closed the fluid can pass only through *CEF*. If the height of pressure in the reservoir remains the same, less fluid, *A*,' will flow out at *G*, *A'* being $< A$. The pressure in *BC* and thus also in *CDK* will increase, while that in *FG* will decrease. Supposing then that the height in the reservoir is kept constant by the fluid flowing out at *G* being continually brought back into the reservoir, we shall then have to bring into the reservoir, in the unit of time, *A* fluid if *K* be open and *A'* if *K* be closed.

Let us now assume that the part of the tube, *CDF*, is very long and elastic, with a small coefficient of elasticity. On the cock *K* being open, a certain quantity of fluid flows through; let us again suppose *A*. Now let *K* be closed; then since, as in the non-elastic tube, the pressure increases in *CDK*, the capacity of *CDK* increases, and to supply this increased capacity more fluid is necessary, which can be drawn only from the reservoir. If this capacity has again become constant, a constant quantity of fluid flows out at *G*, which must again be brought back to the reservoir; but the reservoir cannot thereby be raised to the

height which it had before K was closed. The constant height in the reservoir has decreased, viz. by just so much as answers to the quantity of fluid withdrawn from it to supply the increased capacity of *CDK*. If the height in the reservoir has decreased, the hydrodynamic pressure in *BC* will not be so high as would result simply from the increase of resistance due to K being closed. It will thus depend only on the increase of capacity of *CDK*, how much the height of pressure in the reservoir will decrease. If this increase of capacity be very great, the height of pressure in the reservoir may fall so low that the pressure in *BC* falls after the closing of the cock K.

This is just what occurs on compressing the vena cava inf. It appears from what has been said, that the smaller quantity of fluid which the reservoir receives when K is closed, is no reason for the fall of pressure in *BC*. When *CDK* was not elastic only $A' < A$ flowed out upon K being closed, and thus $A' < A$ was brought back every time into the reservoir; but yet the pressure in *BC* rose. In the same way the carotid pressure also would rise on closing the vena cava inf. if the veins had a greater coefficient of elasticity. Hence it follows that the fall, which occurs in the carotid pressure, cannot be immediately ascribed to the smaller quantity of blood which the heart receives.

In this experimental example, viz. the closing of the vena cava inf., the heightened pressure in the venous system arises in consequence of a part of the section of this system having been entirely removed; but it is plain that everything which has been said is similarly applicable when the increased resistance is equally distributed over the whole of the venous system.

Upon increased resistance in any part of the venous system we have thus to consider two factors: 1st, the increased resistance which occasions a rise in the arterial pressure, and a fall in the venous pressure beyond the place of increased resistance; 2nd, the withdrawal of a certain quantity of blood from the circulation, which quantity supplies the greater capacity of the veins in front of the place of greater resistance, and thereby brings about a decrease in the mean tension over the whole vascular system.

These two factors co-operate with respect to the jugular pressure, and counteract each other with respect to that of the carotid. Owing to the great increase of the capacity of the veins caused by the pressure being raised in front of the place of increased resistance the second factor in most cases predominates. The closer to the capillaries the place of increased resistance, and hence the smaller the part of the

venous system which lies in front of this point, and thus the smaller the whole increase of capacity, so much the less will the second factor be felt, and it will be all the more probable that a fall in the jugular pressure will be accompanied by a rise in that of the carotid.

I am convinced, though I have not made the experiment, that if the splanchnic nerves be cut, and the carotid pressure thereby falls, the jugular pressure will rise, and if the splanchnics are stimulated, a rise in the carotid pressure will be accompanied by a fall in that of the jugular. In this last case it is only the arterial system which lies in front of the place of increased resistance arising from the augmented tonus of the smaller arteries caused by stimulation of the splanchnics. Here too upon a rise of pressure the capacity will increase, but the quantity of blood by this means accumulated there, and thus, as it were, withdrawn from circulation, is small, because the increase of capacity is small, owing to the large coefficient of elasticity of the arterial coats.

But here too there is a limit to the rise in carotid pressure with the simultaneous fall in that of the jugular. Let us put the place of increased resistance still farther up in the arterial system, closer to the heart, viz., in the aorta itself. I made such an experiment in the same dogs that were used for the compression of the vena cava inf. The thoracic aorta was compressed just above the diaphragm. Both carotid and jugular pressure rose. Again another deviation from what has been previously said in 4; but only an apparent deviation. For what occurs under these circumstances?

As soon as the thoracic aorta is compressed, a great part of the vascular system is again placed *hors de combat;* but a certain quantity of blood, instead of being withdrawn from, is driven into the remainder of the system. Beyond the place of compression no fresh blood is conveyed. The energy accumulated in the coats of the arteries in that region now comes into action, and propels the blood through the capillaries to the veins, until the tension in the veins which carry off the blood becomes equal to that in the aorta beyond the place compressed. By this a certain quantity of blood is conveyed to the veins.

Suppose that the thoracic aorta carries off all its blood to the vena cava inf., and all the blood which flows through the branches of the aortic arch passes into the vena cava sup., then the part of the vascular system from the place where the aorta is compressed to the vena cava inf. inclusive, will no longer take part in the circulation. The circulation in fact is confined to the remaining part of the vascular system.

The tension in the vena cava inf. now increases, owing to the greater quantity of blood which comes into it, and seeing that this vein communicates through the auricle with the vena cava sup., the blood will flow from the vena cava inf. into the vena cava sup., which will also become fuller.

The figure, just given, again affords us the example of this, if we suppose the cock to be at $K_{,}$ instead of at K while CDF is again elastic. Let us again suppose, that A fluid flows through if $K_{,}$ be open. A certain hydrodynamic pressure will obtain in the tube, and the wall of CDF will thereby be stretched. As has been previously shown, a certain quantity of energy is thus here accumulated. To keep the reservoir at a constant height, A fluid must be brought into it in every unit of time. Now let $K_{,}$ be closed. Owing to this the fluid must now flow through CEF only and the resistance will therefore be increased. Less fluid will consequently flow through; the hydrodynamic pressure must rise in BC and fall in FG. But, immediately after the closing of the cock, the energy accumulated in the wall of KDF comes into action and will expel fluid thence, until the tension in KDF establishes an equilibrium with the hydrodynamic pressure at F. During this time more fluid will thus flow out at G, and if all the fluid from G be again brought into the reservoir, the height of pressure in the reservoir will rise, because more fluid is brought into it than flows out of it. If this increased quantity, brought into the reservoir, be only great enough, in other words, if the decrease of capacity of KDF is great enough, the height of pressure in the reservoir may become so high, that after the closing of $K_{,}$, the pressure in FG rises.

What was just now said of the vein is also applicable here: the same condition will arise, if no one part of the section of the arterial system has been completely removed, but the increased resistance is distributed over the whole section.

Also in this increased resistance we have two factors to take into consideration: 1st, the increased resistance which occasions a rise in the arterial pressure in front of the place of increased resistance, and a fall in the venous pressure; 2nd, the addition of a certain quantity of blood to the circulation, proceeding from the diminution in the capacity of the arteries beyond the place of greater resistance, and thence an increase in the mean tension over the whole vascular system.

These two factors assist each other with respect to the carotid pressure, and counteract each other with respect to the jugular pressure. Since the diminution of the capacity of the arteries will never be great,

the second factor will not manifest itself so strongly relatively to the first factor as in the case of increased resistance in the venous system. Here too the influence of the second factor will be all the slighter, in proportion as the place of increased resistance lies closer to the capillaries, because the diminution of capacity beyond this place of increased resistance is then smaller.

These important facts have never, in my opinion, been accurately set forth.

With respect to the closing of the jugular on the cardiac side of the T piece, we obtain peculiar relations, when we compare the results just discussed with those previously obtained from vagus-stimulation. We have seen that upon vagus-stimulation the jugular pressure rose, if the jugular on the cardiac side of the T piece was open, and fell if it was closed. On compressing the vena cava inf., the pressure in the jugular always falls, whether the jugular on the cardiac side of the T piece be open or closed; the jugular pressure rises in both cases if the thoracic aorta be compressed. After all that has preceded, these phenomena need no farther explanation.

We have now sufficiently made clear what influence changes in the capacity of the blood-vessels can exercise on the arterial and venous pressure. And this influence is not confined to the above mentioned instances; changes in capacity must always be taken into account. If they are small, their effect is not perceptible. As soon as they reach a certain magnitude, every diminution of capacity anywhere in the vascular system will lead to greater tension all over this system, every increase of capacity will lead to reduced tension. Hence, when upon section of the spinal cord between the atlas and occipital bone the tonus of almost all the smaller arteries is suspended, and the resistance by this means greatly reduced, the fall of the arterial pressure which results is not necessarily accompanied by a proportionate rise in the venous pressure. By the suspension of the tonus, the capacity of the vascular system increases enormously, and the mean tension through the whole of the system is greatly diminished.

Changes in capacity do not always arise from changes in hydrodynamic pressure. The example of the suspension of tonus is one not included in this rubric. Hitherto we have always considered the pressure on the external wall of the blood-vessels as constant. We have previously seen that the lumen in an elastic tube is dependent on the pressure on the internal wall, on the coefficient of elasticity, and on the pressure on the outer wall. As soon as the last changes the lumen

will also change, caeteris paribus. Now there are a number of places in the body where forces work externally on the blood-vessels and cause their capacity to change. We may give as instances the variations of pressure on the blood-vessels in the thorax and abdomen during respiration; the variations of pressure on the vessels of the alveoli during respiration, owing to the variations of atmospheric pressure in the alveoli; dilatations of the blood-vessels due to expansion of the lungs; variations of pressure on the blood-vessels of the trunk and limbs on account of muscular contractions.

Every external force which narrows the lumen of a blood-vessel increases the resistance there, every force which widens this lumen reduces the resistance. If this force remains constant, the wall will come into a new state of equilibrium with a permanent new lumen. A force exerting a constant action on a blood-vessel has thus an influence on the resistance which the blood there meets with.

There is, however, another matter which comes into account. So long as the lumen varies, the quantity of blood in that place also varies. Upon diminution of the lumen, and consequent decrease in capacity, a quantity of blood must be expelled from that place; upon enlargement of the lumen, and consequent increase of capacity, a certain quantity of blood must be drawn to that place. This action takes place only while the blood-vessel is becoming wider or narrower, and hence, when under the action of this force, the lumen has again become constant, though the force continues to act, the forcing or suction effect will entirely cease. We may thus consider every enlargement of the lumen of a blood-vessel by a force acting on it from without as a suction force in respect to the blood, every diminution of lumen as an expelling force. When the lumen has again become constant this forcing or suction ceases.

The thinner the wall, and the smaller the coefficient of elasticity, the greater will be the influence of a force acting on the blood from without. Such a force will, therefore, always exercise a greater influence on a vein than on an artery. The effect will further depend on the state of dilatation in which the blood-vessel is at the moment this external force begins to act, because the magnitude of the coefficient of elasticity depends on the degree of dilatation. Suppose that the internal pressure in the blood-vessel is slight and thus the dilatation of the wall small, then, if an external suction force begins to act, the blood-vessel will be enlarged until a new state of equilibrium ensues. If, however, the internal pressure is previously strong, and the wall therefore in a highly dilated state, then the same external suction

force will effect a smaller dilatation. An external force will thus, in general, cause the lumen of a blood-vessel to vary all the more in proportion as the internal pressure D_x is smaller[1].

So far we have not yet examined at all closely the influence of Z_x which is occasioned by the force of gravity. This Z_x always forms a part of the pressure D_x which we determine by a manometer; and in dealing with this we drew conclusions concerning a variation of P_x only when Z_x remains constant. What part Z_x contributed to D_x did not then concern us, because it was not our aim to ascertain the absolute magnitude of P_x. When however we attempt to determine the latter, Z_x must be closely investigated, and it will then appear that the part Z_x plays is sometimes very great.

In determining the blood pressure in the carotid and jugular, of which we have treated, in an animal lying on its back, these two vessels lie in all but the same horizontal plane, and the pressure in both may be at once compared with each other in respect to P_x; and in general if in a dog lying on its back we determine the pressures in two places anywhere in the vascular system, the comparative influence of Z_x, on these two pressures, will not be very different, because the vertical distance between these two places can never be very great; this, for instance, is the case, when we simultaneously determine the blood pressure in the carotid and crural arteries.

If, however, we place the animal in a vertical position, the influence of Z_x, upon the pressures in the carotid and crural, becomes very different.

Hermann[2] recently made a preliminary communication in which he states, that he has tried to ascertain what influence different positions of the body exercise on the hydrodynamic pressure. This influence may be really in part owing to a change in the cardiac action which accompanies the change in the position of the body, or, to changes in arterial tonus due to an alteration in the flow of blood to the vasomotor centre. The changes which Z_x undergoes when the position of the body is changed affect the results of the determination of D_x in different positions of the body; but Hermann has hit on a very ingenious method which enables him to keep the influence of Z_x on the level of the manometer constant, and hence, from the changes of D_x to draw conclusions concerning the changes of P_x. We have previously seen,

[1] With respect to the influence of respiration on arteries and veins in the thorax and abdomen, consult my articles in *Pflüger's Arch.* Bd. xx. and xxxiii.

[2] L. Hermann. *Pflüger's Arch.* Bd. xxxvii.

that it is an essential condition in determining blood-pressure, that the aperture of the cannula of the manometer, and of course the manometer itself, should always remain at the same height, because the zero-point of the manometer is thereby determined. Hermann so arranges his experiment that any change in the position of the animal is accompanied by a corresponding shifting of the aperture of the cannula, which shifting exercises as great an influence on the level of the manometer as does Z_x, but in a reverse sense. By this means the influence on the manometer of a change in Z_x is always neutralized, and hence a change in the level of the manometer can be directly attributed to a change in P_x. The axis round which the body must be moved, in order that this condition may arise, Hermann determines in a dead animal by filling the vascular system with fluid; in that case only Z_x affects the manometer, since P_x does not exist. He calls this the axis of the "Statische Indifferentie-Punkt" of the vascular system.

During life the hydrostatic pressure Z_x will in every animal be most strongly felt in the blood-vessels of the extremities. The hydrostatic pressure is of importance for the labour required of the heart in so far as the lumen of the blood-vessels is influenced by it. We may imagine ourselves the blood-vessels in an extremity in the form of figure 3.

The tube *ABC*, conveying the fluid, represents the arteries, the underpart *C* represents the capillary system, the tube *CDE* carrying off the fluid represents the veins. If the coats of the blood-vessels were not elastic, the position of the extremity would have no influence on the flow. Since the blood-vessels are elastic, they will become wider in places of higher internal pressure, narrower in those of lower. Z_x will be greater as x lies lower with respect to the reservoir (the heart). The artery with its large coefficient of elasticity will not be greatly influenced by Z_x; but the vein on the other hand will. The latter, when Z_x grows greater, will become much wider, and its capacity will thus increase. Hence, on the whole, all veins situated low in the body, will be widened under the influence of Z_x. This can be no detriment to the blood-current. The resistance indeed will have been reduced by the widening, and this is advantageous to the action of the heart. However, since the capacity of the veins placed low is greater than that of the veins placed high, at any given moment the quantity of blood present for the moment will be greater in the former than in the latter.

As soon as the blood-vessels are placed higher than the heart the case alters, and we may then compare them to the tube in figure 4. In

the parts lying high Z_x will work in a sense reverse to what it did before. Z_x will now become smaller than the atmospheric pressure, in other words it will become negative, and, since $D_x = Z_x + P_x$, D_x will thus become $< P_x$. In proportion as the blood-vessel is placed higher D_x will thus become smaller, and where D_x becomes smaller, the lumen also becomes smaller. Owing to this smaller lumen the blood will there experience more resistance. It is even possible that Z_x may become $> P_x$ (Z_x always being negative) and in this case D_x will also be negative. Outside the blood-vessel the atmospheric pressure will prevail, inside the blood-vessel a pressure smaller than that of the atmosphere; and if the wall of the blood-vessel be thin enough the lumen may thereby totally disappear. This is the only reason why the hand becomes empty of blood, if we place it as high as possible above the heart. The heart would in this case be so far of influence, if we suppose that owing to stronger cardiac action P_x is increased, and consequently does not become negative; but we must remember, that in this case P_x would also have to increase proportionately in every part of the vascular system. Here too again the elevation of an extremity would have no influence on the heart and on the blood-current, if the blood-vessels were not elastic.

Z_x is only of value to the heart and to the blood-current by reason of the changes which it effects in R_x (the radius of the blood-vessel). The question raised by Hermann I leave here undiscussed.

Nor is the statement perfectly accurate which Rollett makes at page 333 of the Manual already often mentioned: "Sie (die Schwere) kann zwar zunächst auf die Bewegung des Blutes keine Wirkung ausüben, wohl aber auf die Vertheilung und dadurch mittelbar auf die Bewegung. Sie begünstigt die Entleerung absteigend und beeinträchtigt die Entleerung aufsteigend verlaufenden Venen." There can, however, be no question of an assistance or obstruction to the emptying of the veins. If the state of equilibrium is present in the blood-current, the blood-vessel, whatever its direction, is nowhere either emptied or filled. A current is flowing through the blood-vessel; and, the cardiac action remaining the same, it is only the lumen which exercises an influence on this current. At the moment that the extremity is moved, for instance, upwards, we may speak of an emptying of the blood-vessels, because at this moment on D_x becoming smaller, the capacity of these blood-vessels is diminished; and, conversely, at the moment the extremity is placed lower, we may speak of an obstruction to the stream, because the capacity is then enlarged, and thus a certain

quantity of blood is detained. But this continues only as long as the lumen changes, in consequence of changes in Z_x, that is to say, so long as the moving of the extremity lasts.

Only during the change in lumen does such a forcing or suction of blood take place as we have discussed above. If the extremity be kept low, this will, as I have already said, be even favourable for the blood-current, because the vessels become wider.

Such a widened state of the blood-vessels will exercise an influence on the blood-current, disregarding changes in resistance, only when the increase of capacity is so great, that thereby such a large quantity of blood is withdrawn from the remaining part of the vascular system that the mean tension over the whole of this system visibly decreases (see above). Hence it may be explained that, for instance, under some circumstances, anaemia cerebri arises, when the body is suddenly changed from a horizontal into a vertical position.

Cohnheim expressed himself very inaccurately when he said on p. 141 of his "Vorlesungen über allgemeine Pathologie, 2te Aufl.: Dass der Name der Senkungshyperaemie falsch ist, liegt auf der Hand: wenn die betreffenden Theile mehr Blut enthalten, als in der Norm, so kommt das nicht daher, dass dasselbe in besonders reichlicher Menge in sie hineingesunken ist, sondern davon, dass es nicht, der Schwere entgegen, in gehöriger Menge hinausbefördert wird." In my opinion "Senkungshyperaemie" is a very correct name, which most accurately indicates the condition. Just before, p. 140, Cohnheim has said: "Welche Mittel der Organismus dazu verwendet, den Widerstand der Schwere zu überwinden, ist Ihnen bekannt." What Cohnheim meant by "Widerstand der Schwere" is rather obscure.

We have hitherto always spoken of one circuit of the blood. The blood, however, makes two such circuits, one through the body, and one through the lungs. From the anatomical relations, and the simultaneous action of the left and the right heart, it follows that what we have said of one circuit is also applicable to the other. The principle remains true with respect to both circuits, that through every section of the vascular system in both circuits just the same quantity of blood must flow in the unit of time, if the state of equilibrium is to be preserved. Hence it follows, that changes in the blood-current in one circuit must react on that in the other, in other words that the current in one depends on that in the other.

If the rate of the left heart changes, the rate of the right heart

is similarly changed. The description which we have given above of the effects in one circuit will thus be the same for both. The same is applicable when Q, or the force of the systole changes. Whatever change may arise, the circulation, in the long run, is only possible when, if nQ blood per minute is transmitted by the left heart, the same quantity is also transmitted by the right heart.

The dependence of one circulation on the other appears most distinctly upon a change of resistance in one of the two. As soon as, the cardiac action remaining the same, the quantity of blood which flows through a section of the vascular system changes in consequence of changed resistance, this must influence not only the flow in the circuit in which this changed resistance occurs, but also that in the other circuit.

Under physiological circumstances varied resistances constantly occur in the blood-vessels of the lungs during respiration. These changes must react on the systemic circulation. I will, in conclusion, dwell on these matters.

III. On the influence of respiration on blood-pressure.

I have just said that the resistance which the blood meets with in its circulation through the lungs constantly changes during respiration. As soon as the resistance in the pulmonary vessels increases, the hydrodynamic pressure, as follows from what has been said, will increase in the vessels which convey the blood to the lungs, the arteriae pulmonales, and decrease in the vessels which carry off the blood, the venae pulmonales. Owing to this increased resistance less blood will flow through the lungs, and nQ will thus become smaller. If nQ becomes smaller in the pulmonary vessels, this must also be the case in the systemic vessels.

We have seen above (Part II.) what is the effect in a simple circuit, (the systemic circuit) when nQ becomes smaller without the resistance in the vascular system of the body being anywhere increased. We have seen that the pressure in the arteries will fall, while that in the veins will rise. Hence it follows that the resistance in the pulmonary vessels being increased the arterial pressure in the body will fall, and the venous will rise. The resistance in the pulmonary vessels being diminished the arterial pressure will on the other hand rise, and the venous fall. Since upon increase of resistance in the pulmonary vessels

nQ decreases, either n or Q must become smaller. Since n under these circumstances may remain the same, e.g. after section of the vago-sympathetic nerves Q is the factor that must change. Hence, upon increase of resistance in the pulmonary vessels, less blood will be transmitted at every systole, and upon decrease of resistance more. These variations in Q continue only until a new equilibrium has arisen, upon which nQ, either $< \text{or} > nQ$, again flows through constant.

Another matter, which must be noticed during respiration, is the accompanying change in the capacity of the pulmonary vessels. We have already seen that, if the capacity of any area of the vascular system becomes smaller, a quantity of blood is expelled from that area, and if its capacity becomes larger a quantity of blood is drawn into it. Hence, if the capacity of the pulmonary vessels becomes smaller, blood will be expelled from them into the systemic vessels and the tension in the latter will thus increase; on the other hand if the capacity of the pulmonary vessels is enlarged, the tension in the systemic vessels will decrease.

Seeing now that during respiration periodical variations occur in the resistance and capacity of the pulmonary vessels, the systemic circuit will periodically feel their influence. The periodical variations perceptible in the blood-pressure in the systemic vessels are called the respiratory oscillations of the blood-pressure.

From these periodical variations in the resistance and capacity of the blood-vessels, we may of course deduce a mean resistance and a mean capacity. The quantity of blood which in consequence of this mean resistance flows through the pulmonary vessels, will, caeteris paribus, determine the mean hydrodynamic pressure in the systemic arteries and veins. The mean capacity will determine the quantity of blood which the pulmonary vessels on an average contain, and thus the quantity which is present in the systemic vessels; seeing that the mean tension in the vascular system depends on the quantity of blood in the systemic vessels, the mean capacity of the pulmonary vessels will thus determine the mean tension in the vascular system.

Now, since, in the different modes and forms of the respiratory movements, the mean resistance and capacity are not always the same, it follows that the modes and forms of the respiratory movements exercise an influence on the mean hydrodynamic pressure, and on the mean tension of the vascular system.

We shall, therefore, successively consider: (*a*) the respiratory oscillations of the arterial blood-pressure, (*b*) the respiratory oscillations of

the venous blood-pressure, (*c*) the influence of respiration on the mean blood-pressure.

(*a*) *On the respiratory oscillations of the arterial blood-pressure.*— With respect to these oscillations I can be very brief, since I have previously published numerous experiments on this subject and the conclusions which may be drawn from them[1]. I will now content myself by briefly enumerating the factors, which have been given as causes of these oscillations, and indicate which of them are really of influence. As causes of these oscillations we find indicated:

1st. Variations in the blood-current in the lungs;

2nd. Variations in the lumen of the thoracic aorta under the influence of the variations of intra-thoracic pressure;

3rd. Variations in the lumen of the abdominal vessels under the influence of the variations of intra-abdominal pressure;

4th. Variations in the magnitude of the cardiac systole (therefore in Q) under the influence of the variations of intra-thoracic pressure;

5th. Variations in the frequency and magnitude of the cardiac systole (therefore in n and Q) brought about by nervous influences;

6th. Variations in the lumen of the smaller arteries owing to variations of tonus brought about by nervous influences.

Factor 6, which Schiff deemed of the utmost importance for the respiratory oscillations of the arterial blood-pressure, Kuhn[2] has not only disputed but totally refuted. In my treatise "Welchen Einfluss hat die Abdominal-Respiration auf den arteriellen Blutdruck?"[3] I have fully treated of factor 3, and shown that, with a normal mean blood-pressure, this factor exercises no perceptible direct influence on the arterial blood-pressure. The variations of intra-abdominal pressure during respiration exert an influence only on the lumen of the veins in the abdomen, and thus act in an indirect manner only on the arterial blood-pressure. We shall presently return to this subject. Factor 2 has no more influence than factor 3, as appears from what is stated in the same memoir. Factor 5 has unquestionably an influence. As I have observed in the memoir cited, we need only to compare with each other the curves of the arterial blood-pressure when the vago-

[1] On this subject consult my articles in *Pflüger's Arch.* Bd. xx, xxvii, xxxiii, and xxxvi; *Arch. Neerl.* T. xix, and xx. I also beg to refer the reader to these articles for the literature on this subject.

[2] Kuhn. "Over de respiratie schommelingen der slagaderlijke bloedsdrukking." Amsterdam. 1875.

[3] *Pflüger's Arch.* Bd. xxxiii.

sympathetic nerves are intact and when they are cut, to be directly convinced of this; but it also immediately appears from this comparison that variations of cardiac action cannot be the fundamental cause of the oscillations, but merely an accessory factor. It is precisely when the cardiac action after section of the vago-sympathetic nerves remains the same during the different phases of respiration, that the oscillations in the arterial blood-pressure, with respect to these phases, are most distinctly indicated. Factor 4 I have also previously discussed. This may not be altogether void of influence, yet under normal circumstances it is unquestionably not more than one of the accessory causes. The variations of pressure on the outer surface of the heart are in normal respiration slight, only a few mm. Hg. Now, if we consider with what a great force the systole is effected, viz. how great is the pressure which is thereby exercised on the blood in the ventricles[1], it follows of itself that variations in the pressure on the outer surface of the heart can have but a very slight influence. The same statement, though to a less extent, is also applicable to the diastole. The negative pressure which, during this period, we observe in the ventricles, proves the great force with which the diastole is effected. The negative pressure in the thorax forms thus but a small subordinate part of the cause which effects this aspiration in the heart. This is also proved by the fact, that, when the thorax is open, a great negative pressure may still, under favourable circumstances, be indicated in the ventricle during the diastole.

I have refrained from making experiments to ascertain whether this factor has any perceptible share in effecting the oscillations; and I have done so for the reason, that the pressure in the right ventricle would have to be registered at the same time with the carotid pressure, and these would have to be compared with each other during the different phases of respiration. To these experiments themselves there can be no objection; but we should then get on the recording surface a certain mean of the ventricular pressure, which mean depends on the magnitude of the maximum and minimum pressures occurring in the ventricle, and on the time during which each of these pressures obtains there. It seems to me that it is not possible to trace to what extent each of these factors operates in this matter, and without this we cannot explain the variations in the mean pressure in the ventricle, which are indicated by the manometer.

We may assume that there is an influence exercised on the auricles

[1] S. de Jager. "Ueber die Saugkraft des Herzens." *Pflüger's Arch.* Bd. xxx.

by the variations in intra-thoracic pressure. The auricles may in fact be considered as terminal expansions of the great veins. Dilatation of the left auricle, for instance, in consequence of a variation in intra-thoracic pressure will add to the dilatation of the pulmonary veins, and thus act as increase of the capacity of the pulmonary vessels. Hence, with respect to the arterial blood-pressure, we have not to consider the influence of the auricles separately, but may suppose them to be parts of the veins.

Thus, there is left as the real cause of the origin of the respiratory oscillations in the arterial blood-pressure:

The variations in the blood-current in the lungs during respiration.— There may be accessory, or occasional circumstances, which cause slight modifications in this influence, but the above-mentioned variations always remain as the fundamental cause.

I have previously shown that the curves of the arterial blood-pressure is the resultant of the curves of capacity and of rapidity of flow. (Capacitäts und Stromgeschwindigkeits curven). A rise or fall in the arterial blood-pressure in consequence of variations in the capacity of, or in the resistance in the pulmonary vessels, must therefore be considered in the way I have explained at the beginning of this chapter.

I will now merely state, as a very short résumé, how the two curves mentioned are related in the different modes of respiration, and enumerate the circumstances which may influence their occurrence.

1. Every increase in the capacity of the pulmonary vessels causes a fall in the arterial blood-pressure.

Every decrease in the capacity of the pulmonary vessels causes a rise in the arterial blood-pressure.

Every increase of resistance in the pulmonary vessels causes a fall in the arterial blood-pressure.

Every decrease of resistance in the pulmonary vessels causes a rise in the arterial blood-pressure.

2. The different modes of respiration are:
   - α. Normal respiration,
   - β. Artificial respiration (by means of a pair of bellows),
   - γ. Respiration in rarefied or compressed air.

3. During these respirations:

	During normal respiration		During artificial respiration	
	The capacity becomes	The resistance becomes	The capacity becomes	The resistance becomes
during inspiration	larger	less.	smaller	greater.
during expiration	smaller	greater.	larger	less.

The variations in the blood-current in the lungs in γ are composed of those in α and β, because, as I have previously shown[1], the factors active in α and β both occur simultaneously in γ.

4th. The longer the suspense (the pause) between two phases of respiration lasts, the more forcibly do the effects of the changed resistance (the curves of rapidity of flow) occur in the arterial blood-pressure. The more rapidly each respiratory movement takes place, the more forcibly will occur the effects of changes in the capacity (the curves of capacity) during this phase.

I need not now more fully describe the different forms of the curves of blood-pressure, as resultants of the curves of capacity and rapidity of flow. It would be merely a repetition of facts previously published. It already appears from the above statement that the variations, which may occur in the forms of the oscillations of blood-pressure, are very numerous, because there are so many circumstances which can exercise an effect on the circulation in the lungs.

I need not now consider what influence the opening of the abdomen may exercise on the oscillations; what oscillations may occur in the arterial blood-pressure as the result of variations in pressure artificially caused in the abdomen; what difference exists between the form of the curves of the blood-pressure during artificial respiration according as the thorax is closed or open; what influence the mean tension in the vascular system exercises on the form of the oscillations; what we are to understand by the "retard" etc.

b. On the respiratory oscillations in the venous blood-pressure.—To ascertain these I determined the pressure in a jugular at the same time with that in a carotid, in the manner described above, and examined the oscillations under the different circumstances under which I had previously examined the oscillations in the arterial blood-pressure only.

As I have already said above, an increased resistance in the pulmonary vessels which occasions a fall in the arterial blood-pressure, must cause a rise in the venous pressure, and *vice versâ*. With respect to the influence of the variations of resistance in the pulmonary vessels, the oscillations in the arterial and venous pressure will thus run in opposite directions.

Variations in capacity of the pulmonary vessels will, however, exercise the same influence on arterial and venous blood-pressure. Increase in capacity of the pulmonary vessels causes the tension to decrease all over the vascular system, decrease in capacity causes this

[1] *Pflüger's Archiv*, Bd. xxxvi.

tension to increase. Increase in capacity of the pulmonary vessels will thus effect a fall, and decrease a rise in the arterial as well as in the venous pressure.

The greater the variations in the pulmonary circulation, the more extensive will be the oscillations in the venous pressure. Only during artificial respiration are we able to cause these variations to occur arbitrarily in different degrees.

Other circumstances besides variations of the circulation in the lungs may influence the venous pressure. We will, therefore, first treat of the very simplest case, viz., that in which artificial respiration is applied to a curarised dog, after section of the vago-sympathetic nerves, and with the thorax and abdomen opened. We can here, at pleasure, regulate the rapidity and form of the respiratory movements, and introduce pauses at will. In this kind of respiration it is, of course, only the pulmonary circulation, that is of influence on the arterial and venous blood-pressure.

For these experiments I again use the same apparatus that I have already described[1]. Of the many cases Fig. 1, Pl. VII. is an example. It represents the curve of blood-pressure in the right carotid, and left jugular of a curarised dog of $6\frac{3}{4}$ kl. The vago-sympathetic nerves are cut, the thorax and abdomen opened.

From this curve it appears most clearly that the oscillations in the carotid and jugular run exactly in opposite directions. We see in the carotid during expansion first a little rise and then a fall, during the collapse first a fall and then a rise; in the jugular, during expansion, principally a rise, during collapse principally a fall.

I purposely do not mention separately the initial variation in the jugular curve at every phase, because I have convinced myself, my conclusions being based on the study of many curves, that in the jugular as in the carotid we have to do with a "retard", i.e., that the variations in the curve which are the effect of a phase of respiration, do not begin immediately but only some time after this phase has set in. Nor can we indicate in the jugular how great this "retard" is, but, just as in the carotid, it is certainly not under all circumstances equally great.

An initial variation, such as is often observed in the carotid curve and also in the curve Fig. 1, a variation which is the expression of the variation in the capacity of the pulmonary vessels, cannot be observed in the jugular curve, because the curves of capacity and rapidity of flow must there occur in the same direction. In the jugular pressure a rise

[1] *Pflüger's Arch.* Bd. xxxvi.

occurs on expansion, and a fall on collapse, and these oscillations appear most clearly, as the curve shows, when the expansion and collapse take place with a long pause between each. If we look at the first and the last part of the curve, where the phases of respiration were effected without any distinct pause, we see exactly how the "retard" causes the variation resulting from a phase to delay its appearance until the latter part of the phase, nay, even sometimes causes the reverse to appear. We see at *a* only a rise during the collapse. That this rise proceeds from the previous expansion is sufficiently shown from the other parts of the curve.

The figure is in fact the proof of the influence of the pulmonary circulation on the venous blood-pressure; it shows that the curves of the rapidity of flow in the carotid and jugular really run in opposite directions.

If we compress the jugular on the capillary side of the T piece, and thus determine the pressure in the vena cava sup., the oscillations preserve the same form, while the mean pressure is lower. We then obtain the oscillations in the pressure of the vena cava sup.

If, on the other hand, the jugular is compressed between the T piece and the heart, the oscillations in most cases disappear from the curve. Sometimes, however, oscillations remain in it, but we then see, what is very characteristic, that, with respect to the phases of respiration, they are now exactly opposed to those which were present before the compression. We see now in the curve of the jugular pressure during expansion a fall, during collapse a rise, in other words, the oscillations in the carotid and jugular now run parallel. These oscillations are never very extensive. When they were absent, I could sometimes cause them to appear by first reducing the jugular-manometer to its zero-point and then opening its cock, by which means a steady rise is occasioned in it (See Part II). If during this steady rise artificial respiration is carried on, we see that the rise becomes less steep during the expansion, while during the collapse it becomes much steeper. Hence when the jugular is closed between the T piece and the heart, and there are no oscillations visible in the curve, the cause of this must be sought in the valves of the veins, which impede the reflux of the blood and thus the fall in the manometer. The manometer in connexion with these valves and veins may in fact be considered as equivalent to a maximum manometer.

That, if there are oscillations, these run in a direction opposite to those which occur when the jugular is not closed, is, of course, owing to the fact, that the jugular in this case is again to be considered pretty

much as a prolongation of the artery, and the jugular manometer consequently executes oscillations along with those in the arterial pressure.

During artificial respiration, when the thorax is still closed, the same oscillations occur in the carotid and jugular as when the thorax is open. I have previously discussed what is the influence exercised on the pulmonary circulation by artificial respiration when the thorax is closed, and have argued that some modifications then occur in the carotid curve in consequence of this circulation being thereby somewhat modified. These modifications are with respect to the jugular so slight, that we scarcely perceive the difference between a curve which is taken when the thorax is closed and one taken when it is open. Yet when the thorax is closed, the varying intra-thoracic pressure of the artificial respiration must exercise an influence on the lumen of the large veins and of the auricles. During expansion these must become somewhat narrower and during collapse somewhat wider. During expansion blood will thus be expelled from the veins of the thorax, and the resistance to the flow of blood will at the same time be there increased. Seeing that the jugular is situated outside this seat of narrowing, the pressure in it must under both circumstances be increased. The rise in the jugular pressure during expansion which is the effect of the change in the circulation in the lungs themselves, will thus be increased by the narrowing of the veins in the thorax, both during expansion and during the subsequent pause.

The narrowing of the veins in the thorax, while it is taking place, will be favourable to the carotid pressure, but the pause of expansion will occasion a fall. Thus here the changes in the lumen of the veins in the thorax will only contribute to increase the already existing curves of capacity and rapidity of flow. In the case both of the jugular and carotid this always remains, compared with the effects of the circulation in the lungs themselves, a secondary matter.

It is further clear that, if the inflation is effected by means of a very strong, positive tracheal pressure, the diastole of the heart will be thereby obstructed, and the fall in carotid, and the rise in the jugular, will be still further increased. In what exact way this factor exerts its influence on both blood-pressures cannot however be proved by direct experiment.

If we consider what takes place during normal respiration, we know that the changes in the pulmonary circulation are diametrically opposed to those which occur during artificial respiration (by means of

positive tracheal pressure), and I have already discussed how the oscillations in the carotid pressure are diametrically opposed to each other in both cases. The same rule is applicable to the oscillations in the jugular pressure.

Let us take an example, Fig. 2, Pl. VII. It represents the curves of the blood-pressure in the right carotid and the left jugular of a dog of 19 kgrm. in morphia and chloroform narcosis, on which tracheotomy had been performed, and the vago-sympathetic nerves had been cut.

We see here in the carotid, during inspiration, first a slight fall and then a rise (the fall is sometimes wanting), during expiration first a slight rise and then a fall; in the jugular, during inspiration a fall, during expiration a rise, preceded by a slight fall.

In the carotid curve the usual curves of capacity and rapidity of flow again occur; the curves of the rapidity of flow preponderate; sometimes, during inspiration, the curves of capacity are even wholly wanting. The respiratory movements here occurred rather rapidly. The expiration succeeded immediately to the inspiration and after every expiration a pause ensued. I therefore need not enter into further explanation of this carotid curve.

The oscillations in the jugular pressure in this case, during normal respiration, also run exactly opposite to those in the carotid pressure. As appears from the curves, there is, during inspiration, hardly any "retard"; during expiration, however there is a certain "retard[1]." The changes in the lumen of the large thoracic veins and of the auricles, under the influence of the varying intra-thoracic pressure, have also to be taken into account here during normal respiration. Both during the widening of these veins with the inspiratory movement, and during the time they are widened during an inspiratory pause, a fall in the jugular pressure will be occasioned; and conversely, during expiration, and the ensuing pause, a rise is occasioned in this pressure. The oscillations in the jugular pressure caused by the pulmonary circulation are thus somewhat enlarged by these accessory circumstances. These circumstances will also be advantageous to the curves of capacity and rapidity of flow in the carotid pressure.

I have already in a previous memoir spoken of the influence of the abdominal respiration on the venous blood-current. I have since tried to trace this influence by means of experiments. The effect of this

[1] The oscillation occurring at *a* in the carotid pressure was due to a very superficial respiratory movement, which, as appears from the curves, had no influence on the jugular pressure.

influence is obtained in all its purity, if in a curarised dog, after section of the vago-sympathetic nerves, the thorax is opened while the abdomen remains closed. The animal is then by means of *O* brought into apnoea, and while the artificial respiration is suspended, the abdomen is from time to time pressed upon. If the animal has not suffered too much under all these manipulations, so that the mean arterial pressure is still high, then comparatively slight pressure on the abdomen produces no effect either in the jugular or in the carotid. An effect is apparent only when a strong pressure is exerted on the abdomen.

With respect to this pressure I must observe, that compressing the abdomen, and so lessening the abdominal cavity, does not always produce the same changes in the amount of the intra-abdominal pressure. I have previously shown that this depends on the tension of the abdominal wall, which is in turn dependent on the intestines being more or less full.

In the jugular pressure we see a rise while the abdomen is being compressed and a fall when the pressure is removed. Figure 3 gives us an instance of this. It is taken from a dog of $13\frac{1}{2}$ kgrm. which was prepared in the manner described above. It represents the curves of pressure in the right carotid and the left jugular. In the figure we see in the jugular pressure the effect just mentioned. But what we observe in the carotid pressure is also of the greatest importance. We still really see oscillations here in the carotid pressure. We see at first a rise in the carotid pressure when the abdomen is compressed, after which the curve keeps at the same height. On the pressure being removed from the abdomen a fall in the carotid pressure takes place, after which the curve keeps the same height. How are these oscillations to be explained?

I have previously shown that even on great pressure being exerted on the abdomen the lumen of the aorta abdominalis undergoes no changes leading to perceptible variations in blood-pressure. We shall therefore not discuss this matter here. The variations we observe in both blood-pressures must originate in changes in the lumen of the small and large veins in the abdomen under the influence of the heightened intra-abdominal pressure. These veins must be thereby narrowed. The consequence of this must be that a certain quantity of blood is expelled from them to the other parts of the vascular system, thus increasing the tension both in the arteries and veins. We cannot compare this condition to that which arises when we close or narrow a limited portion of the vena cava inf. In this latter case the resist-

ance at the part narrowed increases, but the diminution in capacity which takes place is slight, on account of the portion of vein narrowed being very short, and the blood which is thereby expelled is insufficient to lead to any perceptible change in tension. On the contrary all veins up to the place narrowed will be greatly widened, and the capacity there will thus greatly increase, so that there must occur a decrease of tension in the vascular system; we therefore see, as I have shown in Part II., a fall in the pressure in the carotid and the jugular. Upon increase of intra-abdominal pressure, however, all the veins in the abdomen are narrowed, the capacity thus becomes smaller, and a rise in pressure in the arteries and veins must ensue.

That the pressure in the carotid, as appears from Fig. 3, increases more than in the jugular must be explained by the fact, that the heightened resistance arising in the abdominal veins will favour the elevation of pressure in the carotid, but hinder that in the jugular. Hence, the rise in carotid pressure due to powerful compression of the abdomen, is not followed by a fall. Sometimes indeed we observe a faint inclination to fall, for instance, at *a* and at *b* in Fig. 3. We must, however, remember that it is very difficult to exercise with the hand a pressure on the abdomen which shall be absolutely constant. We may very well assume, that the intra-abdominal pressure decreased here, because the pressure on the abdomen did not remain the same. If we suddenly press on the abdomen, so that the blood is, as it were, expelled with a shock from the veins, the steepness of the rise in the carotid pressure is much more marked, and after this a fall ensues. This is, however, a simple mechanical effect of the great blood wave which arises in the vascular system.

I must further observe, how small is the extent of the oscillations in the jugular, and what a great difference in extent arises, if we press on the abdomen when the thorax is still closed, as may be seen in Fig. 4. We shall presently return to this subject.

It follows from the above that if, during normal respiration, the intra-abdominal pressure varies, its influence on the blood-pressure is slight, because the variations which then occur in the abdominal-pressure are also slight, as I have previously shown. The influence that might be exercised would be a narrowing of the veins by means of which the tension throughout the vascular system would increase. Owing to the presence of the valves in the veins, and the alternating increase and decrease of pressure both in the abdomen and thorax, the venous blood-current will be promoted by respiration. This I previously

expressed by saying that these alternating pressures in connection with the system of valves operate as a force-pump and suction-pump on the venous blood.

If in a curarised dog the thorax and abdomen are not opened, oscillations occur in the carotid and jugular, after section of the vago-sympathetic nerves, on pressing the abdomen, even although the pressure exerted may be slight. Figure 4 represents the curves of blood-pressure in the right carotid and the left jugular of a dog of 11 kgrm. prepared in this way. From the curve of the carotid pressure it is plainly evident that the oscillations in it are occasioned by the expirations and ensuing inspirations, brought about by the pressing on the abdomen. These will lead to the usual variations in the pulmonary circulation. The oscillations in the jugular curve also owe their existence to the same variations. The rise in jugular pressure, while the abdomen is being pressed on, is the same as that which arises as the result of an expiration, because it is really the effect of an expiration, caused by the pressure. So also the fall in the jugular pressure, when the abdomen is released again, is the effect of the inspiration which then takes place.

We have only to compare the curves of Fig. 4 with those of Fig. 3, to observe the great difference between them, both in the jugular and carotid pressure. It speaks for itself that on powerful compression of the abdomen the events discussed in Fig. 3, and in Fig. 4, will all occur.

It is only by a great number of experiments, of which the cases mentioned are a few instances, that I have been led to the explanations given above. We must exercise the utmost caution in explaining what we observe in the jugular pressure, because a variation in this pressure, as has now been made clear, may depend on such an infinite number of circumstances. It is absolutely necessary that, in judging of variations in the venous pressure, we should at the same time also observe what happens in the arterial pressure. Especially so, since in the curve of the venous pressure, we do not meet with a curve of capacity and rapidity of flow, as we do in that of the carotid, where these factors act in contrary directions on the mean blood-pressure ; hence the phenomena in the venous pressure do not occur nearly so distinctly as they do in the carotid. Many other circumstances too, with which we have now made acquaintance, if they occur simultaneously, act in the same direction on the jugular pressure.

The oscillations in the jugular pressure during respiration thus run in a direction opposite to those in the carotid pressure, if they owe their existence to changes in resistance occurring during respiration,

whether these changes originate in the pulmonary vessels (the chief factor) or somewhere in the large veins between the capillaries and the place where the venous pressure is determined. Changes in capacity, however, whether in the pulmonary vessels (again the chief factor) or somewhere in the systemic vascular system, if they occur during the respiration, will influence the carotid and jugular pressure in the same way.

This same principle appeared very plainly also in an experiment in which I accidentally observed oscillations in the carotid and jugular pressures, the origin of which had no connection with respiration. In the carotid pressure of a curarised dog of 7½ kgrm. with thorax and abdomen closed, I observed splendid Traube-oscillations. The artificial respiration was shallow in character, and tolerably frequent. These Traube-oscillations embraced several respiratory oscillations. The ascending line of such a Traube-oscillation in the carotid pressure was accompanied by a descending line in the jugular-pressure, and the converse. During the rapid respiratory movements respiratory oscillations were observable in the carotid pressure, but not in that of the jugular. If the respiration was suspended, the respiratory oscillations of course disappeared from the carotid pressure, but the Traube-oscillations continued both in the carotid and jugular. It thus appears from this experiment that if Traube-oscillations occur in the carotid pressure, they also occur simultaneously in the jugular pressure, but that these oscillations run in opposite directions in the two pressures.

I also made some experiments for the purpose of ascertaining the influence of respiring condensed or rarefied air. The experiments were made in the manner I have already described[1], with this difference only, that the pressure in the jugular also was now registered. In that article I explained how the oscillations in the arterial blood-pressure may assume a great many forms, and may be composed of the curves of capacity and of the rapidity of flow, appearing during normal and artificial respiration. This is also applicable to the oscillations which, under these circumstances, we see occurring in the jugular pressure. As both during normal respiration and during that produced by inflation, the oscillations in the jugular run in an opposite direction to those of the carotid, so also we see the same thing occur during respiration of condensed or rarefied air. Just as in the carotid pressure so also in the jugular pressure the oscillations assume many different forms

[1] *Pflüger's Arch.*, Bd. xxxvi.

according as condensed or rarefied air is applied, whether during both phases of respiration, or, during one only, or, according as the condensation or rarefaction operates more or less quickly in accordance with the rapidity of the respiratory movements. Yet the forms of the oscillations in the jugular pressure are not so diverse as in the carotid pressure, because the curves of capacity and of the rapidity of flow always occur in the same direction in the jugular. It is of course unnecessary, when we once know these relations, to enumerate the different forms of the oscillations in the jugular pressure.

These experiments were of great importance in ascertaining the relations of the mean blood-pressure during these different modes of respiration. We will in conclusion occupy ourselves with this matter.

c. *On the influence of respiration on the mean blood-pressure.*— From what was said at the opening of this chapter it follows that the mean pressure in arteries and veins depends on the mean capacity and the mean resistance in the pulmonary vessels. With an increase in the mean capacity of the pulmonary vessels, the mean tension in the blood-vessels of the body will be less, and with a decrease in the capacity, greater. The influence of the mean capacity is the same on arterial and venous pressure.

With an increase of the mean resistance in the pulmonary vessels, the hydrodynamic pressure will be lower in the arteries, and higher in the veins; with a decrease of the mean resistance it will be higher in the arteries and lower in the veins. My previous experiments have taught me with respect to the capacity and resistance in the pulmonary vessels, that the capacity is the largest, the resistance the smallest in normal respiration; that the capacity is the smallest, the resistance the greatest when the lungs are artificially inflated. Let us tabulate the different states in which the lungs may be, in such an order of succession, that we begin with that state in which the capacity is the largest, and the resistance the smallest, and conclude with that in which the capacity is the smallest, and the resistance the greatest. Such table is then as follows:

1. State of normal inspiration,
2. State of normal expiration,
3. State of total collapse of the lungs, the thorax being open,
4. State of expansion of the lungs by inflation, the thorax being open.
5. State of expansion of the lungs by inflation, the thorax being closed.

(In cases 4 and 5 the lungs are supposed to be in the same degree of expansion.)

From this sequence it follows that, as far as the capacity of the pulmonary vessels are concerned, the tension in arteries and veins will be the smallest during the state of normal inspiration, and the greatest during the state of expansion of the lungs by inflation with the thorax closed.

As, however, appears from the experiments the resistance in the pulmonary vessels exercises a much greater influence than does the capacity. In every pause the curve of the rapidity of flow, except in peculiar circumstances, far surpasses in extent the curve of capacity, which has arisen during the previous respiratory movement. As to the result of this resistance, the arterial pressure will be the highest, the venous the lowest during the state of normal inspiration; the arterial pressure will be lowest, the venous highest during the state of expansion of the lungs by inflation, with the thorax closed.

The mean pressure, whether during normal or artificial respiration, however, never results from the effect of one state of the lungs, but always from the combined effect of inspiration and expiration. Let us confine our attention to the effects of the resistance in the pulmonary vessels; since these are of much greater importance to the blood-pressure, than those of the capacity, it again follows from the above table, that the pressure in the arteries will be highest and in the veins lowest during normal respiration, consisting of normal inspiration and normal expiration. If the normal respiration be superseded by an artificial one, with the thorax open, the mean pressure, if there be no other influence at work causing the pressure to vary, will be lower in the arteries, and higher in the veins, than during normal respiration. Since, however, when the thorax is laid open, other influences are always at work, such as cooling, impaired cardiac action, loss of blood etc., we had better compare the pressure during normal respiration with that during artificial respiration with a closed thorax. In this case, too, and even more so than when the thorax is open, the mean pressure in the arteries will be lower, and that in the veins higher, than during normal respiration.

To this fact I have already briefly referred[1]. I then stated that a curarised dog may be kept alive by taking a fold of the diaphragm between the fingers, when the abdominal cavity is open, and imitating

[1] *Pflüger's Arch.*, Bd. xxvii.

the respiratory movements. If, in an animal thus prepared, we keep up the respiration now by means of a pair of bellows and now again by diaphragmatic movements, we then see, as I have shown in the article alluded to, that the respiratory oscillations in the arterial blood-pressure are in these two cases diametrically opposed to each other, and also that the mean arterial pressure during respiration by means of a pair of bellows is lower than during diaphragmatic respiration.

The normal respiration is thus the most favourable to the circulation of the blood. With this mode of respiration the greatest quantity of blood will, in the unit of time, flow through every section of the vascular system.

From the above table, however, we may draw further deductions. Seeing that state 1 is the most favourable and state 5 the least favourable, the arterial pressure will be the highest, and the venous the lowest, the longer the lungs remain in a state more approximate to 1 or more remote from 5; the reverse will be the case, the longer the lungs remain in a state more approximate to 5.

Thus during normal respiration the arterial pressure will be the highest, the venous the lowest, when there are long inspiratory pauses. The longer the expiratory pauses last, the lower is the arterial, the higher the venous pressure[1]. If expiration and inspiration succeed each other without pauses, the arterial pressure will then be higher and the venous lower, according as the last part of the inspiration and the first of the expiration take place more slowly, and the first part of the inspiration and the last part of the expiration more rapidly.

In the case of artificial respiration by means of inflation, whether the thorax be open or closed, the pressure in the arteries will be higher and that in the veins lower the longer the pauses of collapse last. If the lungs each time remain long in an inflated state, the arterial pressure will then be lower and the venous higher. If inflations and collapses follow each other without pauses, the arterial pressure will be higher and the venous lower, according as the first part of the inflation and the last part of the collapse take place more slowly, and the last part of the inflation and the first part of the collapse more rapidly. With respect to the arterial pressure alone, I have already previously from time to time referred to instances of this.

[1] It must be borne in mind, that I constantly exclude varying cardiac actions caused by nervous influences, and thus suppose the vago-sympathetic nerves to be cut, or to be inactive.

In these considerations we have left unnoticed the influence of the capacity of the pulmonary vessels, because this compared to the influence of the resistance in the pulmonary vessels is of subordinate interest to the mean blood-pressure. If we take this influence into account, it will somewhat increase the effect which occurs in the venous pressure in consequence of the resistance in the pulmonary vessels, under the different circumstances; it will, on the other hand, somewhat decrease the effect in the arterial pressure.

From a practical point of view these matters are of some importance. Under circumstances in which the circulation is bad, or has nearly ceased, and in which the respiration is suspended, we ought, in order to restore the circulation, to apply such a method that the lungs will be brought each time into a state most approximate to the state of normal inspiration. Inflation of the lungs will, in all these cases, be detrimental to the circulation.

From the above we may also explain how it is that the subjectively unpleasant feelings, which occur in anaemia cerebri, for a moment decrease or disappear when a deep inspiration is taken. This will really improve the arterial blood-current, and thus the circulation in the brain.

The effect on the mean blood-pressure of respiring condensed or rarefied air becomes very evident when we register simultaneously the pressure in the artery and vein. At the conclusion of my previous article on this subject, I said that, if the degree of condensation or rarefaction applied is not too great, the mean arterial pressure hardly changes. I then felt myself obliged to attribute this to the changes in cardiac action. Since, under normal circumstances, the heart does not act with its maximum force, a certain compensation is possible. It now appears to me, however, on simultaneously registering arterial and venous blood-pressure, that this compensation is less than I formerly imagined. In the presence of such extensive respiratory oscillations as occur during the respiration of condensed or rarefied air, it is with great difficulty that we can make out whether small variations occur in the mean arterial pressure. I therefore took only the approximate mean pressure, and always placed $\pm$ in front of the numbers, which indicated the pressure. Besides, in my previous experiments, I always attended more carefully to the oscillations than to the mean pressure. When I thus determined the pressure in the veins as well as in the arteries, it appeared to me that the mean pressure changes, and that the heart is generally unable to compensate for these changes with even

comparatively low degrees of condensation or rarefaction. It is of course possible that the variations in mean pressure might be partially compensated by changes in the tonus of the smaller arteries. Whether this really occurs I cannot ascertain from the experiments.

The effect of breathing condensed or rarefied air appears most plainly, when we cause both respiratory phases to occur in it. When both inspiration and expiration are carried on in condensed air, the mean pressure falls in the arteries and rises in the veins. If we disregard the influence of the capacity of the pulmonary vessels, this phenomenon is due to the fact, that in consequence of the increased tracheal, and thus also of the intra-alveolar pressure, the lumen of the pulmonary vessels is diminished, and consequently the resistance there becomes greater, than when the animal breathes under the usual atmospheric pressure.

When both phases of respiration are carried on in rarefied air we obtain the reverse effect on the two blood-pressures, viz. a rise in the arterial and a fall in the venous pressure; but this effect, however, cannot always be distinctly observed; sometimes we do not see the least change in the mean pressure. The cause of this is to be sought in the fact, that the normal respiration is already the most favourable condition for the blood-current, and the rarefaction cannot make this condition any more favourable. When we cause the animal to breathe first condensed and then suddenly rarefied air, we see most distinctly the difference between these two modes of respiration. In this case, the mean arterial pressure will on the transition greatly rise and the mean venous pressure will greatly fall.

If during respiration the condensed air is applied in one of the phases only, the effect on the mean blood-pressure will then be less, than when this same degree of condensation is applied in both phases. Yet we then see the unfavourable influence of this factor on the blood-current; the mean arterial pressure falls and the venous rises. We see the same occur in a reverse sense, when one only of the phases takes place under the influence of rarefied air.

The degree of rarefaction, but especially that of condensation, has an influence on the magnitude of the variations in mean pressure similar to that which it exerts on the extent of the respiratory oscillations. The same statement is also applicable to the greater or less rapidity with which the rarefaction or condensation operates in accordance with the greater or less rapidity of the respiratory movements. What has been said above with respect to the respiratory oscillations is applicable here also, viz. that the duration of the different phases, and

the duration of the action of condensation or rarefaction during these phases, are of influence. The longer, for instance, during a respiratory phase the condensed air has an opportunity to act, the more disadvantageous will this be to the blood-current.

On the whole the rule may now be laid down, that, however we cause the condensed air to act during respiration, it will never be favourable, but always detrimental to the blood-current. If, with smaller degrees of the action of condensed air, we obtain no effect on the blood-pressure, because the heart compensates the influence by its increased action, then in this case greater expenditure of energy is demanded of the heart. If this does not occur, there always ensues a fall in the mean arterial and a rise in the mean venous pressure.

In all cases of weak cardiac action, or in general of bad circulation, every application of condensed air is to be disapproved of. These applications may be useful in order to improve, after an illness, the elasticity of the lungs, or to tear a slight pleuritic adhesion; we should, however, be very cautious, and never apply the condensed air otherwise than during inspiration. To weak persons—whose circulation is bad, owing to feeble cardiac action—any application of condensed air may prove a source of danger. With greater degrees of condensation, we have to fear obstruction to the diastole of the heart; especially if the cardiac action is already weak.

The application of rarefied air, on the other hand, may be favourable to the circulation. If we wish to improve bad circulation due to imperfect cardiac action, the application of rarefied air is to be recommended during expiration only. The rarefaction will, indeed, during inspiration also promote the blood-current, but the forcible exertion of the inspiratory muscles, which is required to inspire this rarefied air, is not to be recommended for weak persons. While inspiration is obstructed by the rarefied air, expiration, on the other hand, is facilitated by it.

Let us also take into consideration the fact that variation in capacity may be favourable to circulation, but only when the condensed or rarefied air is applied, not constantly, but during one only of the respiratory phases. Precisely in consequence of this alternate application the variations in intra-thoracic and intra-alveolar pressure will be greater, and will thus lead to greater changes in the capacity of the pulmonary vessels, and of the great thoracic veins. In consequence of these changes in lumen, in connection with the system of valves in the veins

(the heart itself may in this case be considered as a valve at the end of the venous system) the blood-current will be promoted. These variations will, as is already the case in ordinary respiration, act on the blood both as a force- and suction-pump. If the disadvantages of the action of condensed air, owing to the increased resistance in the pulmonary vessels, were of no importance, these changes in capacity might be favourable to the blood-current, but, as a matter of fact, their advantages are outweighed by the disadvantages of the increased resistance. As to the question during which respiratory phase these changes in capacity resulting from the application of condensed or rarefied air will be favourable to the blood-current, and during which they will be unfavourable, on this I need not, after all that has preceded, again dwell.

If in weak persons, with a feeble circulation, we desire to produce the conditions most favourable to the blood-current, we should have so to arrange matters as to cause a force operating only on the outer surface of the thorax to act on it during every inspiration, so that the normal inspiration would be effected without strong muscular exertion on the part of the subject. Upon this inspiration an expiration in rarefied air might be made to follow.

It is, of course, of no avail to place the person in a closed chamber the air of which is maintained at a constant degree of rarefaction, and then to carry on respiration through a tube connecting the mouth with the open air (the nostrils being closed). An apparatus of this kind has been described by Williams[1]. He used it in order to cause his patients to inhale medicines.

For whatever purpose this apparatus may be employed, exactly the same effect will be produced by placing the person free in the open air, and, the nostrils being closed, bringing the mouth into connection with a space in which the air is condensed. If we fill Waldenburg's apparatus with condensed air, and cause the person to breathe in it, the effect is exactly the same as that which is obtained by Williams' apparatus; and the effect will be better, if in this case we cause the person to inspire only the condensed air and to expire freely into the atmosphere. The effect in the case of Williams' apparatus will be the same as during respiration with both the respiratory phases occurring in con-

[1] Williams. *New York Medical Record*, 1885. The original article was not at my command. I had, however, the pleasure of seeing the apparatus in operation at the residence of Dr Vincent Bowditch in Boston (U. S.), who was so kind as afterwards to send me a description of it (*Boston Medical and Surgical Journal*, 1885).

densed air, and to this apparatus there are also the same objections, if not dangers, as those which I enumerated in connection with the application of condensed air, especially as the condition is one in which both phases of respiration are carried on in condensed air. The apparatus would be of some value, if, at each inspiratory movement, the air in it was rarefied, and at each expiratory movement the pressure in it was made equal to that of the atmosphere.

I shall not speak now of the influence of placing the whole person in a large apartment in which the air is condensed or rarefied. In this case quite different circumstances arise, and the mechanical influences which act on the circulation in the pulmonary vessels during the respiration of condensed or rarefied air, and therefore lead to definite effects on the systemic circulation, such as we have already discussed them, altogether disappear, and in their stead occur conditions totally different.

In conclusion, let me add a few words on Valsalva's experiment. On first inspiring deeply, and then, with closed glottis, trying forcibly to expire, the intra-alveolar and intra-thoracic pressure become strongly positive. The great veins in the thorax and the pulmonary vessels will be greatly narrowed, and a large fall in the arterial with a large rise in the venous pressure, must be the result. This narrowing may be so great that the quantity of blood which flows through is so small that the pulse can no longer be observed in the arteries. Unquestionably the obstruction to the diastole of the heart must here be taken into account. Einbrodt[1] directed attention to the fact that, by applying a strong positive tracheal pressure, the curve of the arterial blood-pressure may become a straight line, notwithstanding that a needle, stuck in the heart, shews that the heart is still beating.

In such a case $nQ_{,}$, the quantity of blood which the heart still transmits per minute, is extremely small, and $Q_{,}$ is even so slight that the elevations of the manometer due to the heart-beats can no longer be observed. In such a case the capacity of the pulmonary vessels is very small, hence a great quantity of blood must accumulate in the systemic vessels. Hence the systemic veins become very full.

That, in Valsalva's experiment, the pulmonary circulation is again of preponderating influence, may be proved by applying strongly positive tracheal pressure to a curarised dog with open thorax and by keeping up this pressure for some time. No obstruction to the diastole

[1] Einbrodt. *Sitzungsberichte d. k. Akad. d. Wissensch. zu Wien.* Bd. XL. 1860.

of the heart, or diminution of the lumen of the great thoracic veins, under these circumstances takes place, and yet I saw the arterial pressure markedly fall and the venous markedly rise, and at last the elevations, which occur in the curve of the arterial pressure in consequence of the heart-beats, totally disappear, while in the opened thorax I saw the heart still beating.

February, 1886.

DESCRIPTION OF THE FIGURES IN PLATE VII.

All these figures have been obtained by registering, with Ludwig's Kymographion, the blood-pressure in the carotid and the jugular of the dog.

In all cases the vagosympathetic nerves were cut and tracheotomy was performed. The curve *Ca* is that of the blood-pressure in the carotid, the curve *Ju* that of the blood-pressure in the jugular; the line *Z* is a horizontal one, which is situated a certain number of mm. below the zero-line of the registering manometer of the carotid, and another number of mm. below that of the registering manometer of the jugular; the curve *T* indicates the time; every horizontal division represents one second.

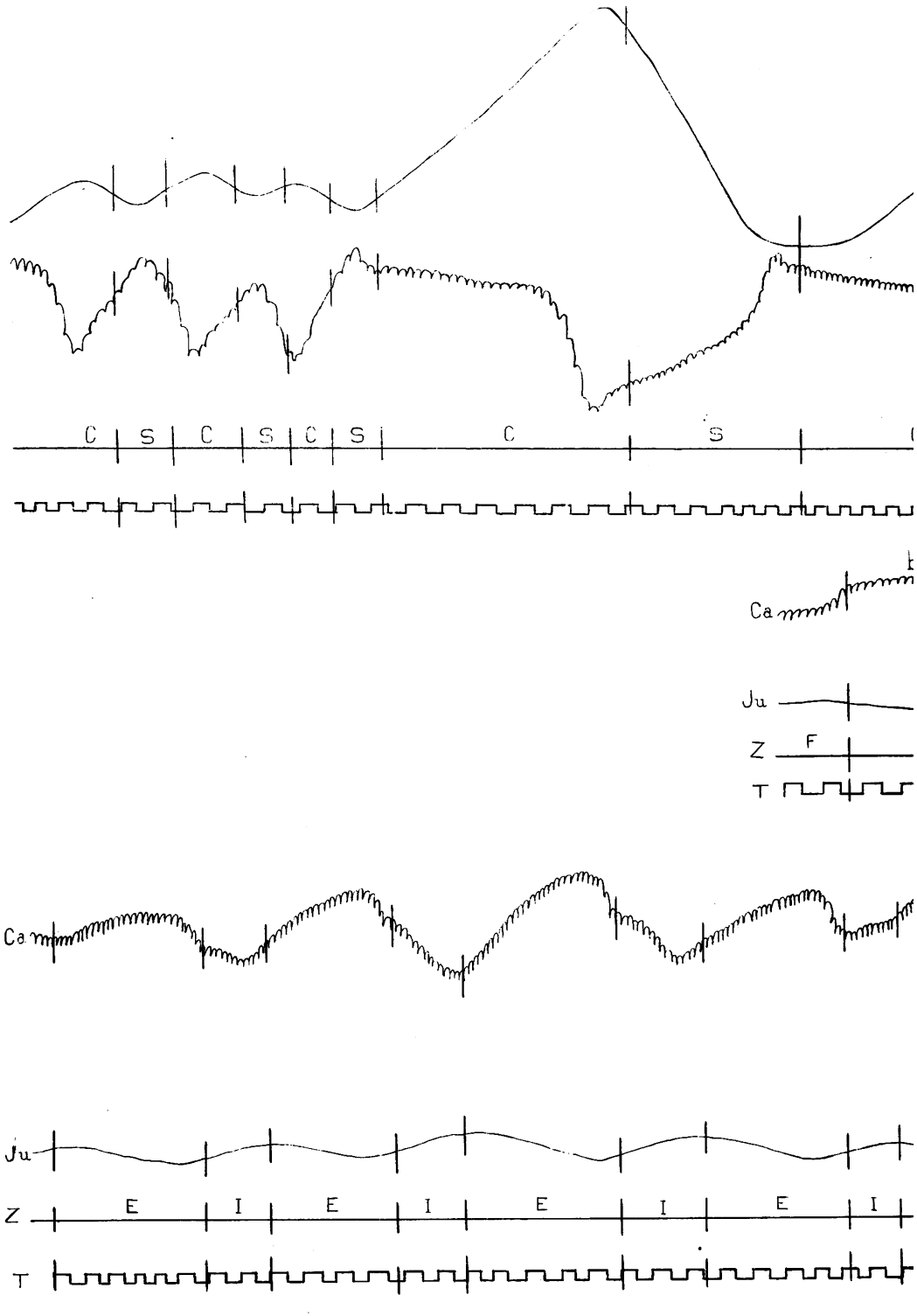
For registering the carotid pressure a mercury manometer was always used; for the jugular pressure a manometer filled with bicarbonate of sodium.

The curves all run in the direction of the arrow, which stands at the top of the Plate. Not to take up too much space, the curves indicating the respiratory movements and the compression of the abdomen have been omitted; but, in their stead, the beginning and the end of a phase is every time indicated by a small vertical line on *Ca*, *Ju*, *Z* and *T*.

S signifies inflation.
C „ collapse.
P „ compression of the abdomen.
F „ cessation of compression.
I „ inspiration.
E „ expiration.

Fig. 1. Blood-pressure in the right carotid and the left jugular of a curarised dog of $6\frac{3}{4}$ kgrm. with thorax and abdomen opened. *Z* lies 10 mm. above the zero-line of the carotid, and 14 mm. above that of the jugular. Mean pressure in the carotid ± 70 mm. Hg., in the jugular ± 54 mm. CS.

Fig. 1.



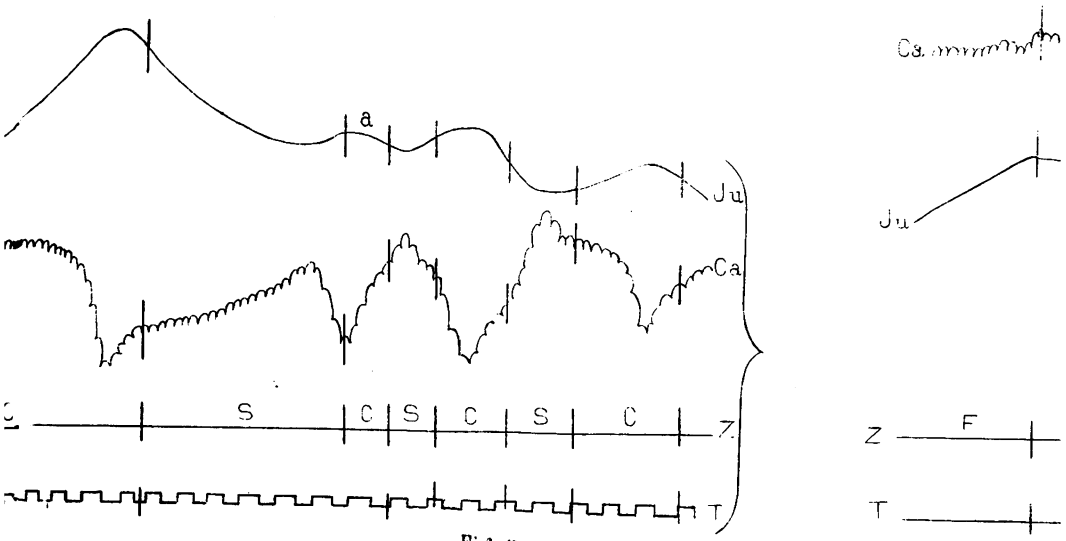


Fig. 3.

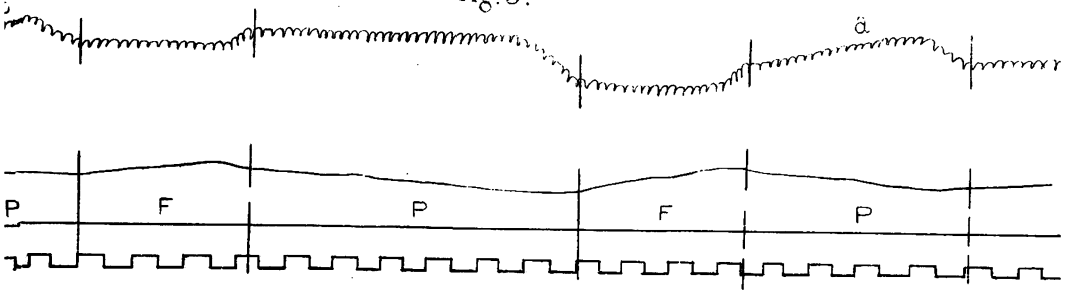


Fig. 2.

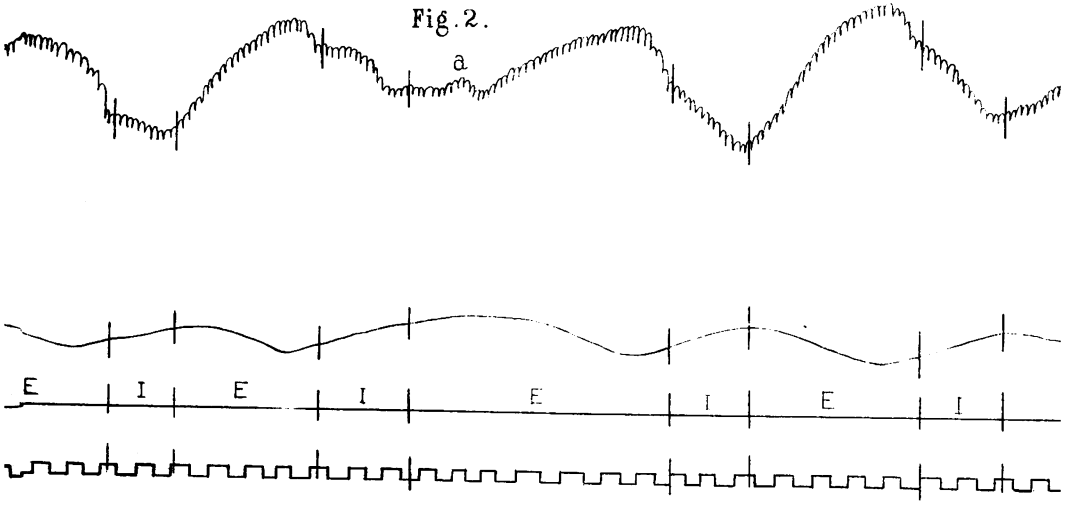


Fig. 4.

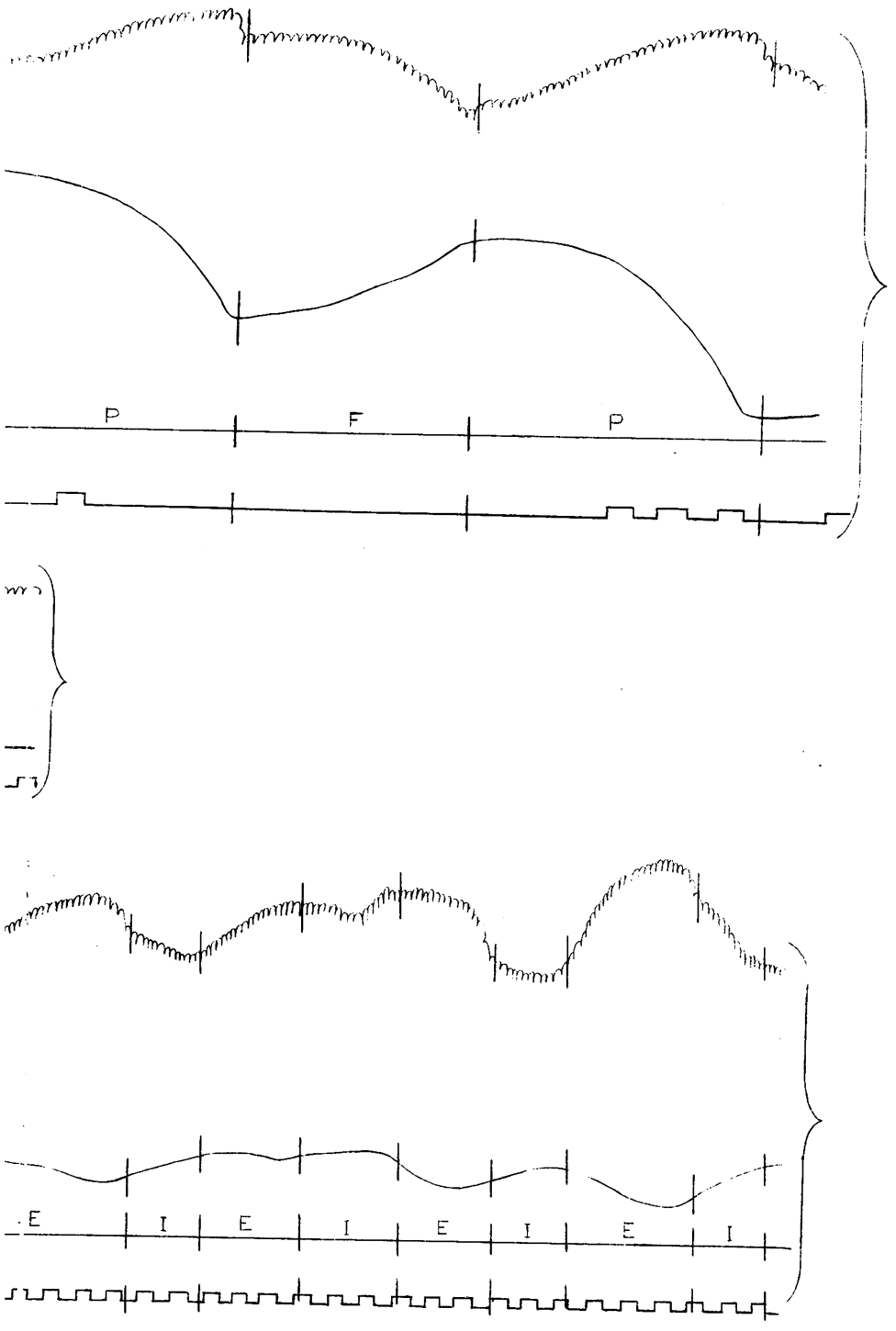


Fig. 2. Blood-pressure in the right carotid and the left jugular of a dog of 19 kgrm. in morphia and chloroform narcosis. *Z* lies 24 mm. above the zero-line of the carotid, and 47 mm. above that of the jugular. Mean pressure in the carotid ± 144 mm. Hg., in the jugular ± 56 mm. CS.

Fig. 3. Blood-pressure in the right carotid and the left jugular of a curarised dog of 13½ kgrm. with thorax opened and abdomen closed. After the animal has been brought into apnoea, while the artificial respiration is suspended, the abdomen is at intervals pressed on. *Z* lies 7 mm. above the zero-line of the carotid, and 7 mm. above that of the jugular. Mean pressure in the carotid ± 64 mm. Hg., and the jugular ± 15 mm. CS.

Fig. 4. Blood-pressure in the right carotid and the left jugular of a curarised dog of 11 kgrm. with thorax and abdomen closed. After the animal has been brought into apnoea, while the artificial respiration is suspended, the abdomen is at intervals pressed on. *Z* lies 44 mm. above the zero-line of the carotid, and 32 mm. above that of the jugular. Mean pressure in the carotid ± 180 mm. Hg., in the jugular ± 67 mm. CS.

ERRATA.

Page 138, foot note. *For* Anat. f. *read* Archiv. f.
,, 144, l. 8 from bottom, *for* Tx read Tx_1.
,, 149, in table, l. 9, *for* wall at *B read* wall at *b*; *for* wall at *C read* wall at *c*.
,, 154, l. 13, *for* the velocity at *read* the increment of.
,, 158, l. 6, *for* $2nA$ *read* $2nQ$.
,, 170, l. 5, *for* during the systole *read* during the pause and the following systole.
,, 187, l. 11 from bottom, *after* blood *read* -vessel.