A FEW APPLICATIONS OF A PHYSICAL THEOREM TO MEMBRANES IN THE HUMAN BODY IN A STATE OF TENSION.[1]

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IT will first be necessary to state the theorem which I intend to apply.

On a section of a cylinder of radius r containing fluid under a pressure of P lbs. per square inch let us take two diametrically opposite points A B and find the tensions per inch run of the cylinder at these points.

The total pressure acting on the semi-circumference of a ring an inch wide will be $P\pi r$. But the stress or tension at A and B is not represented by this total pressure, but by the sum of the resolved parts of its components in a direction at right angles to the diameter AB. The sum of these components is equal to the pressure on an area formed by the diameter of the cylinder multiplied by unity or $= 2Pr$, but since an equal share is borne by A and B, the tension or stress at either point is:

$$T \propto Pr \text{ or } P \propto \frac{T}{r}$$

This is for the simple case of a surface curved circularly in one direction. If the surface be curved in a direction at right angles to this, the second radius of curvature being r_1, we have :—

$$P \propto T\left(\frac{1}{r} + \frac{1}{r_1}\right) \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad (a)$$

From whence it is seen that the tension of a membrane enclosing a fluid under a given pressure is not a fixed quantity, but is greater the greater the radii of curvature of the membrane. The truth of this may be put to a practical test by distending a large and a small bag of the same material with a fluid, the pressure of which is gradually raised. It will always

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be found that the larger of the two will be the first to burst, while the smaller will remain intact under a very much higher pressure.

If, now, we apply this to the heart, we see that the muscle of the heart in the distended state of the organ must, on account of the larger size of its cavity, and necessarily greater radii of its walls, make a much greater effort at contraction, in order, by means of the tension of the walls, to raise the contained blood to the pressure of that in the aorta, than when the ventricle is more contracted, as *e.g.* at the middle or end of systole.

Again, the thinner the heart-wall is,—that is to say, the fewer muscle fibres there are on cross section,—the more will each fibre have to exert itself to bring about a given tension; and when it is considered that the wall of the heart is thinnest when the organ is most dilated, it will be seen that this must form another and not unimportant factor in contributing to the difficulty of commencing systole.

Let us now go a little more accurately into these points.

If we apply the fact that the surface area of a sphere varies as the square of its radius, and bear in mind that the total number of muscle-fibres is a constant quantity, we see that the number of muscle-fibres per unit of area on the heart-wall varies inversely as the square of the radius,—that is, supposing for a moment the heart to be a sphere. Hence, for a given exertion of muscle-fibre the tension will vary inversely, as the square of the radius, or

$$T \propto \frac{1}{r^2}.$$

Still regarding the heart as a sphere, the formula (a) above,

$$P \propto T\left(\frac{1}{r}+\frac{1}{r_1}\right)$$

now becomes

$$P \propto \frac{2T}{r}$$

But $T \propto \frac{1}{r_2}$ as just shown, therefore

$$P \propto \frac{1}{r^3}.$$

Thus we arrive at the somewhat startling result that the pressure varies inversely as the cube of the radius when the exertion of each individual fibre is taken as constant, or if we regard the pressure as constant, the exertion or pull of each muscle fibre must vary as the cube of the radius of curvature. So that if we suppose the heart to be in two conditions, the first condition having a certain diameter, and the second a diameter twice as great, the heart will have to exert itself eight times as much in the second position as in the first, in order that the same pressure may be exerted on the contained blood.

This enormous disadvantage under which the heart in the beginning of systole labours, is surely more than sufficient to demand extra mechanism to overcome it; and it now remains to be considered how it had best be done.

The difficulty could easily be disposed of by heaping on layers of muscular fibres outside those which were barely sufficient in the contracted condition; but this would be attended by the disadvantage of more required room, and of having the extra fibres acting at a time when they were wholly unnecessary, *i.e.*, towards the middle and end of systole, which would not be in accordance with the principle of least action enunciated by Dr Haughton in his great work on animal mechanics, and shown by him to be universal in the animal economy. Some cheaper way must, then, be sought. It is found in the columnæ carneæ, and, as I hope to show, in the musculi papillares also. These muscles, which stretch across the cavity of the heart from wall to wall, exert their influence more immediately on the blood by pulling more directly on the ventricular wall, and consequently are more efficient than if they lay on the outside of the wall itself. On account also of the rapidity with which their origins and insertions approximate the one to the other, their force must diminish towards the middle, and fade away towards the end of systole; thus compensating in a singularly beautiful way for the disadvantage at which the heart-wall is placed at the commencement of its contraction.

It may be objected that placing muscles inside the heart cavity would necessitate its enlargement for the purpose of making room for them. But a little consideration will show

this is not the case. For suppose the heart a sac with smooth walls, it would be quite impossible for it to contract so as to obliterate or even almost obliterate its cavity when expelling blood under considerable pressure, owing to the difficulty of approximating the origins and insertions of the fibres, especially of the innermost layers, so that the only difference their presence makes consists in diminishing the quantity of residual blood in the heart.

The urinary bladder is no exception to this, but rather an illustration. For the last quantity of urine is got rid of not by the exertion of the bladder wall, but by the *vis a tergo* of the abdominal muscles and the pumping of the accelerator urinæ muscle, aided to some extent by gravity. Here, too, the resistance to be overcome is negligible, being nothing more than that offered by the friction of the urine against the walls of the urethra. The bladder, furthermore, as shown by frozen sections, is in the contracted condition not spherical, but somewhat T-shaped, which it could not be if its walls were in contraction at the end of the act of micturition.

The musculi papillares are usually considered as having only to do with controlling the mitral and tricuspid valves. But they must also aid in expelling the blood. For the exertion necessary to prevent the valves from flapping back into the auricles must also react on the ventricular wall and help it in its effort at contraction. They must then be looked upon as having the double function of controllers of the valves and true working muscles of the contracting heart itself.

Another and a very interesting example of similar, though less complete, prominence or ridging of muscular fibres on the inside of a contractile sac, for the purpose of exerting greater pressure on the contained fluid, is exhibited as a pathological condition in the urinary bladder in cases where the outflow of urine is gradually resisted, as in the bladder of enlarged prostate. Here the innermost fibres raise themselves up from the wall hypertrophy, and by their peculiar disposition increase their efficiency. I am inclined to regard this as an attempt on the part of the bladder to simulate the construction of the heart by providing, when the necessity arises, its own columnæ carneæ; and if this be so, it cannot fail to strike one as being an

unusually elegant example of the wonderful resources of Nature when combating disease.

In cases of valve disease the heart becomes dilated to allow for regurgitation, in order that the quantity of blood thrown into the aorta may as nearly as possible be kept normal, and so the blood-pressure kept up. There is, however, reason to believe that in spite of this the blood-pressure in most cases falls a little. Concomitant with dilatation we have hypertrophy. What is the cause of this hypertrophy, especially if the blood-pressure be lower than before? The reason is because the tension required in the ventricular wall to raise the blood, even to a lower pressure, is greater when the heart is pathologically dilated, and so its wall-curvature more gradual, than when of normal size, and the curvature sharper.

It is a well-known fact that the apex of the heart is its thinnest part. The reason is simple. It is because here the curvatures of the wall are sharpest, and, as above shown, the tension required to resist or cause a given pressure in the ventricle will be less than where the curvature is more gradual. The same reason explains why, when the heart bursts, as in buffer accidents, without any part having been specially diseased, the thinnest part is not always chosen as the seat for rupture.

This question of tension in the walls of a sac containing fluid under pressure explains the absence from fusiform aneurysms and varicose veins of any effort to heal spontaneously; for the more the vessels dilate, the higher the tension in their walls rises, and the more incapable they are of contracting to diminish their calibre.

In fact, when we take into consideration the comparatively great radii of curvature of the sac of an aneurysm, and the consequently great height of the tension in its walls which the blood-pressure in its interior must give rise to, and bear in mind the diseased condition of its walls, we cannot help wondering that they do not more frequently burst than they are known to do.

Let us now for a moment regard the uterus in labour, and I think we shall see the real reason why letting a quantity of *liquor amnii* flow away precipitates parturition. It is because

this act diminishes the capacity, and so the radii of curvature of the uterine walls, and thus enables the organ to exert a greater pressure on its contents than in its former more dilated condition.

The "atony" of the uterus, where the quantity of *liquor amnii* is excessive, is, I believe, to be explained in the same way—that is, the too great curvature of the walls of the uterus,—and is not to be attributed to the overstretching of the muscular fibres; for it is not easy to conceive that the *liquor amnii* can increase so rapidly as not to give time for the muscular fibres to adapt themselves to the change. Whether this reason is the only one I am not prepared to say, but that it is a very important one no one can deny.

It will be admitted that the thickness of the heart at any place bears a direct proportion to the relative tension at that place. Hence it follows that in the equation (*a*) above, we ought to be able to substitute t the thickness of the walls, for T the tension. The equation will then be

$$P \propto t\left(\frac{1}{r_1}+\frac{1}{r}\right).$$

or if the pressure be constant, $t\left(\frac{1}{r_1}+\frac{1}{r}\right)$ will also be constant; or, in general terms, the thickness varies as the radii of curvature.

I have experimentally put this to the test in the following way. Some hearts were obtained, and the auricles having been ligatured, the aortic and pulmonary valves were destroyed, and glass tubes tied into their orifices. The ventricles were then dilated with spirit under a head of about 12 in., and so left until they were hardened. Points on the ventricular surface were then selected and marked with labelled pins, and the curvatures in two directions at right angles to one another estimated for each point. The heart was finally cut, and the various thicknesses at the marked points measured.

One heart was an adult's, another a child's about twelve months old. A third, adult heart, I compressed so as, as far as possible, to imitate the contracted condition of the organ, and see if here the relationship held; but in this case the measurements of the curvature were not so reliable, as it

was difficult to make sure that the walls were uniformly compressed, and indeed I have reason to believe that I did not very well succeed.

I have tabulated the results (see below), and I think they show the theorem to be correct within the limits of experimental inaccuracy. It will readily be understood that these measurements are not very easy to take; for in the one case the curvature sometimes changes so rapidly that only a small line can be taken as a segment of a circle, and in the other case the attachments of the columnæ carneæ do not always leave it clear at exactly what point the true heart-wall stops and the columnæ themselves may be said to begin.

On the whole, however, the results more than realised my anticipations, and may, I think, be taken as proving the point.

There were two points taken on the left ventricle of the adult heart—one near the anterior, and the other near the posterior interventricular groove; but here the formula did not hold good, and the results were so much at variance with the rest that I had to omit them. This is confirmatory of the proposition; for it shows that where the conditions were disturbed by the proximity of the interventricular septum, there, as might have been anticipated, the relation between curvatures and thickness did not hold.

Another point of interest is brought about by these tables, viz., that the pressure in the left heart is about three and a half times as great as in the right in the child's case, and about six and a half times as great in the case of the adult.

This latter result, I have since found out, corresponds almost exactly with the experimental observations of Knoll on the rabbit. His exact proportion was 1:6·8. The proportion in the child's case is, I have reason to believe, not correct, as the ventricles were not in a similar condition.

There is good reason to believe that the pressure in the aorta of an adult is a little over 9 feet of water. This being so, the pressure in the pulmonary artery would be 17 inches.

This accounts for the fact that the left ventricle is more rounded on section, the mean curvature of its walls sharper, and their thickness greater than the right.

The left ventricle is the model of what a ventricle ought to

be. The right could, I think, be improved upon. The work could be more cheaply done by having another (free) heart, consisting of a simple auricle and ventricle, instead of having one ventricle tacked on to the outside of another. For this would admit of the wall-curvature being sharper, and so the required tension smaller. Whether the exigencies of space and the shape of the organs would admit of such an arrangement I can't say, for of course these elements, and perhaps others too, would enter into any plan of remodelling.

The thickness of the walls of the systemic arteries diminishes the further we go away from the heart, but more rapidly than the blood-pressure, the reason being that the bore also diminishes, and so the radius of curvature; and hence the required tension in the arterial walls diminishes proportionately.

ADULT HEART (*normal*).

Under pressure 12 *in Right Ventricle.*

Label of point chosen.	r mm.	r_1 mm.	t mm.	$t\left(\frac{1}{r}+\frac{1}{r_1}\right)$
z_1	60	60	1·5	·050
c_2	65	80	2·0	·055
d_2	32	75	1·25	·055
a_2	75	90	2·2	·054
e_2	30	45	1·0	·055
f_2	55	90	2·0	·058
	—	—	——	——
				·0545 = average.
		Left Ventricle.		
p_1	36	60	8·0	·35
z	32	80	8·5	·37
x_1	70	36	9·5	·39
r_1	30	80	8·5	·38
o_1	28	60	7·0	·36
s_1	70	40	8·5	·33
m_1	80	40	10·0	·37
w_1	32	80	8·5	·34
f_1	55	16	5·0	·40
t_1	70	24	6·0	·33
v_1	24	70	6·5	·36
n_1	60	24	6·0	·35
	—	—	——	—
				·36 = average.

CHILD'S HEART (*normal*).

Under pressure 12 *in Right Ventricle.*

Label of point chosen.	r mm.	r_1 mm.	t mm.	$t\left(\frac{1}{r}+\frac{1}{r_1}\right)$
e_1	50	22	2·2	·14
c_1	40	20	2·0	·15
h_1	18	55	2·5	·18
d_1	80	30	3·0	·14
i_1	60	20	2·0	·13
b_1	55	16	2·5	·20
a_1	80	24	3·0	·16
l_1	10	40	1·2	·15
g_1	6	18	·7	·15
	—	—	—	———
				·166 = average.

Left Ventricle.

o	18	22	6·0	·60
n	24	16	5·5	·57
v	38	22	7·5	·54
l	14	34	5·5	·56
t	50	14	6·4	·58
q	20	55	7·0	·50
s	12	36	5·25	·61
p	10	65	5·0	·58
r	12	22	4·5	·57
w	22	10	4·0	·58
y	9	18	3·0	·50
	—	—	—	———
				·56 = average.