consequence of an elaborate series of developmental or growth processes, parts of the abdominal cavity containing the testes are extruded and come to occupy a position in the scrotum. In pronograde monkeys the peritoneal passage, although very narrow, remains open; in orthograde primates it usually becomes closed; but even in man, in whom the process of closure is most complete, the canal remains unclosed in 30 per cent. of newly born children. In all primates, then, there is a breach in the musculature of the groin caused by the descent of the testes or of the round ligament. In all there is a second breach in the groin caused by the passage of the great blood vessels from the abdominal cavity to the thigh. The vascular aperture of the groin must vary in size; at times the vessels—particularly the vein—are greatly distended; at other times they are partially collapsed, thus giving a potential space for the escape of abdominal contents. In man the femoral vessels are of exceptional dimensions in consequence of the massive development of his thighs; hence in him the vascular apertures of his groin are large. It is not the presence of an inguinal canal or femoral ring which makes the human groin exceptionally liable to become the site of hernia, for these apertures are a common possession of the whole order of primates.

The Groin of Apes.

The human groin is one of the most distinctive features of man's anatomy; it is in a class by itself; in only one animal do we find a tendency towards the human form, and that is in the gorilla. If we did not possess some of the intermediate stages it would be difficult to believe that the human groin had been evolved from a pronograde form, such as is seen in the commoner monkeys. In monkeys the infraumbilical part of the belly has a narrow keel-like form terminating posteriorly at the sharp projecting symphysis pubis; on each side is the long narrow furrow or fold of the groin, separating the abdomen from the flat inner surface of the thigh. The two groin furrows form a V-shaped arrangement, the limbs of the V ending above at the anterior superior iliac spines, which are buried in the flanks. In the fold of the monkey's groin can be felt the particularly long anterior edge of the ilium, offering a firm line of attachment for the musculature which controls the apertures of the groin—the lower parts of the internal oblique and transversalis muscles. The groin is further strengthened by the fact that the external oblique muscle has no insertion to the iliac crest, but expends its strength on rendering tense the structures round the apertures of the groin. The whole arrangement makes up a most competent sphincteric mechanism; although the peritoneal passage to the scrotum remains open, escape of the abdominal contents is very rare. Further, these guardian muscles are under a reflex control.

The Orthograde Groin.

With the evolution of the simian orthograde posture the form and structure of the groin underwent surprisingly little change. In the gorilla we find a premonition of the changes which have transformed the human groin. The first of these is the extension of the crest of the ilium in an outward and also in a slightly forward direction. The fibres of the external oblique muscle, which rise from the lower ribs, no longer sweep to the groin but are inserted to the anterior end of the extended iliac crest; there is also the first appearance of the inguinal (Poupart's) ligament, formed out of the tendinous fibres of that part of the external oblique which is inserted to the crest. Further, in the gorilla the anterior border of the ilium, lying along the fold of the groin, has become relatively short. In man these changes have reached a climax; the crest of the ilium has become extensive and has undergone a marked bend forwards; the external oblique has gained a powerful insertion to the crest, and Poupart's ligament has become a strong and differentiated fibrous bridge between the anterior superior iliac spine and pubic spine. Lastly, the anterior border of the ilium has been reduced to a little more than a third of the primitive length seen in great anthropoids; the long iliac border which gave so secure an origin for the guardian muscles of the groin has gone. The lower parts of the internal oblique and transversalis muscles have now to seek a less advantageous origin from a new base—namely, Poupart's ligament. The folds of the human groin have been shortened and opened out, and thus assumed a wide U-shaped form. The lower abdominal region has become flattened in front.

All these changes have weakened the defensive mechanism of the human groin. To understand the meaning of these changes one has to remember that the pelvis is made up of a series of levers, which serve in balancing the body on the thigh. The climbing habits of the great anthropoids demand long and powerful pelvic levers; to suit man's plantigrade needs, to balance the body quickly and easily as he walks, short pelvic levers with short-fibred but powerful gluteal muscles become necessary; hence the transformation of his pelvis and shortening of his groin. It is not the orthograde posture which weakened the human groin but the transformation of his pelvis to suit the needs of plantigrade progression. As we have already seen, plantigrade progression is necessarily attended by high degrees of intra-abdominal tension. Modern methods of living—exactly how we do not know—can damage the reflex postural mechanism of the belly wall. Man's liability to hernia must be sought for in all these factors.

The Liability to Femoral Hernia.

At the beginning of this lecture I pointed out that the human pelvis was the sport of contending interests. In answering the needs of locomotion it has become less efficient as a part of the abdominal wall. In the liability of women to femoral hernia we see a disharmony between the sexual and containing functions of the pelvis. At puberty a growth takes place in the pelvis of women at three sites—at the symphysis pubis and at the two ilio-sacral joints. The new bone laid down at these joints leads to an increase of all diameters of the pelvis. Exactly the same kind of growth is seen in the pelvis of men suffering from the later stages of acromegaly; the sexual transformation of the pelvis is the result of a growth mechanism controlled by hormones. In women, in whom the sexual growth is marked, the pubic part of the pelvis becomes elongated and the vascular compartment under Poupart's ligament widened.² Hence an increased liability to femoral hernia in women. A femoral sac is never the result of a developmental process; it is a structure which has never been seen at any stage in the development of human embryos or fetuses.

REFERENCES.

- ¹ *Essays and Observations*, edited by Sir Richard Owen, vol. i, p. 45.
² See a recent paper on this subject by Dr. J. Allison Pantou, *Journ. Anat.*, 1923, vol. 57, p. 106.

VENOUS PULSATIONS AND VENOUS TRACINGS IN GENERAL;

WITH SPECIAL REFERENCE TO THE "V" WAVE OF THE POLYGRAPHIC TRACING.

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In the first place it must be stated that the polygraphic tracing is a fact; it is Nature's own record. This is true, but the statement needs qualification, for an instrument intervenes between Nature and the recording surface. Nature, therefore, actuates a mechanism, and we must ask, Does the latter transmit faithfully, does it render an exact transcript? This is a primary question, and it raises an element of doubt as to the record.

Passing from the objective side of things to the subjective, we find the observer—another instrument, but one much more complex—intervening between the tracing and its real significance. The observer reads, conjectures, draws conclusions, and makes pronouncement, the judgement speaking the final word. But the judgement, Hippocrates tells us, is difficult, "*judicium difficile*" (Book I, Aphorism 1), and it is fallible we know. Again, therefore, there enters an element of doubt, this time as to the interpretation.

Proceeding now to the subject before us let us endeavour to follow, as closely as we are able, the course of the cardiovascular activities, as they repeat themselves in each cardiac cycle; then let us look at the tracings obtained by the polygraph and see in how far the two harmonize the one with the other.

We shall first note that normally the venous flow from the capillaries onwards presents a smooth current without trace of pulsation, until we come within a certain radius of the

heart. True the velocities change, increasing as we proceed centripetally, but these merge into each other insensibly: the current keeps its smoothness.

Within the certain radius mentioned the blood in the veins comes into touch with the events passing through those chambers, and theoretically should be influenced by and show these events. The events are two—namely, (1) the auricular systole, (2) the ventricular systole; juxtaposed, they follow in

FIG. 1.—The diagram measures the time relations but not the magnitudes of the two systoles. The cardiac cycle, calculated upon a pulse rate of about 72, measures 8/10 sec. The figures must vary with different pulse rates. The duration of the auricular systole is about 1/10 sec. Thus the auricle is active for 1/10 sec., and rests for 7/10; the ventricle acts during 3/10 sec., rests for 5/10. The auricular and ventricular cycles, taken separately, overlap by 1/10 sec.

this order, with an interval scarcely perceptible between the two. A pause follows, then the couple reappear. The auricular systole is a small act, difficult to determine except instrumentally; not so the ventricular, and therefore we usually reckon the cardiac cycle from the onset of one ventricular contraction to the onset of the next, as for the pulse. It would, however, be more physiological to reckon

from auricular systole to auricular systole, as this would not dissociate the two systoles.

Each systole, being a muscular act, is more or less abrupt in its onset and ending. Each, while it lasts, interposes an obstacle to the venous afflux; each, when it ceases, withdraws that obstacle and the accumulated blood unloads itself, readjusting pressure differences. This done, the afflux is resumed under a uniform *vis a tergo*—namely, that obtaining in the tributary veins of the right auricle.

As to the *vis a tergo*, it will be remembered that in the great veins at the root of the neck the pressures in health are very small—namely, from 0 to -8 mm. Hg. Unfortunately we have very little precise information concerning the venous pressures which rule in pathological conditions, and therefore, in dealing with this aspect of things, shall be obliged to rely mainly on presumptive evidence.* Before entering upon this it will be advantageous to introduce here a diagram, which is intended to show certain points bearing directly upon the subject in hand.

FIG. 2.—The figure is based upon Henle's *Handbuch der Gefäßlehre*, second edition, p. 342, with considerable modifications, as follows: The veins are shown in thin line; the heart (as if in section and presenting the right side only) shows the auricle in thicker line, the ventricle in thickest line. These three thicknesses indicate arbitrarily three degrees of distensibility in descending order. The two cavæ with their primary branchings include the terminations of the internal jugulars above and of the renal veins below. This area of distribution corresponds to an area of no valves, for, though the internal jugulars have valves at their endings, these are rarely efficient, and the veins would therefore fill in case of reflux from the heart. The circular lines around the mouths of the cavæ are purely diagrammatic; they mark the sphincter action exerted on the mouths of these vessels during auricular contraction. I.J. = Internal jugular; Sp. = Site of sphincter fibres.

This diagram before us, let us consider the effect of the auricular systole upon the venous afflux. The circular disposition of the auricular fibres around the cavæ must tend to close them more or less, and in health the probability is that the closure is very appreciable, perhaps almost complete, otherwise the systole would be defeated in its objective, taking effect backwards into the capacious, thin-walled,

ill-filled veins as the line of less resistance, rather than forwards into the already partially filled and thick-walled ventricle. It is most probable, further, that this sphincter action is an *initial* event of the systole, for, the end in view being the raising of intra-auricular pressure, here also would be more or less frustration unless leakage backwards were checked from the outset. The part played by these sphincter fibres in auricular systole would thus be the equivalent of the action of the *a-v* valve in ventricular systole—namely, to supply a *point d'appui* for the contraction of the heart walls.

Assuming complete closure of the cavæ during auricular systole the effect would be the same as that of ligation of both vessels; no impulse would be sent back into the veins, but behind the block and proceeding backwards from it the blood would gradually accumulate. Can we see here the development of any wave (justly so named) within the veins by the smooth afflux under the low pressures of health? The period of accumulation lasts one-tenth of a second.

Assuming incomplete closure of the cavæ in health there will be more or less of actual reflux from the auricle and a wave of sorts might be registrable in the internal jugular, in spite of the comparatively small power of the auricular contraction, which would scarcely be able to generate a percussion wave. Such wave would be pre-ventricular systolic in time and in magnitude it should be quite small.

In disease, where marked congestion of the right heart and its great venous tributaries was present, there would be appreciable reflux, and it there should happen to be much hypertrophy of the right auricle (a rare occurrence) the reflux would be proportionately greater and an actual percussion wave might be present.

Whether in health or in disease, if there is reflux, it would add its volume to the accumulation by afflux, which would continue unchecked during the whole of the auricular systole. The congestion of the veins would be correspondingly accentuated, but the factor bearing the pulsation would be the reflux, not the afflux, which *ex hypothesi* contains no pulsation.

Reverting to the venous afflux, there arises an interesting question—namely, During congestion of the veins, however brought about, does the intravenous pressure begin to rise forthwith and proceed *pari passu* with the accumulation of the blood? The answer is, No; there will be no appreciable rise until the veins have reached their full capacity and their walls have begun to be distended—that is, stretched. Until stretch begins there is no resistance; until resistance begins there is no rise of pressure. But the venous system in health is never distended, in particular the great venous trunks; there is not enough blood in the body to distend arteries, capillaries, and veins. The valveless cavæ and their main branchings interpose thus a very large potential capacity between the heart and the main body of the valve-bearing veins, and until congestion has exhausted the whole of this superfluous capacity no perceptible rise in blood pressure can take place. In life the feature of the arterial system is its stretch; that of the great veins their slackness. This slackness ensures the uniform delivery of blood into the heart by preventing—or shall we say keeping down?—rise of blood pressure in the great veins during the temporary obstructions at the heart caused by the systoles of auricle and ventricle.

A complete venous block of one of the smaller or medium-sized veins may be followed by great rise of blood pressure in its branchings, but time is of the essence of this *local* manifestation; it is slow. Block at the heart itself (due to the systoles) lasts one-tenth of a second for the auricle, three-tenths of a second for the ventricle. No appreciable accumulation can take place within these short intervals.

If we put at about 4 oz. the capacity of each of the chambers of the heart, and assume that in systole the chambers empty themselves, then during each auricular systole of 1/10 sec. the amount of blood held up will be only 4/8 oz. (the cardiac cycle being 8/10 sec.)—that is, half an ounce—and during ventricular systole (3/10 sec.) it will be exactly 1½ oz. These quantities are small when compared with the size of the valveless area of the great veins, and should be easily accommodated without appreciable rise of pressure when the accumulation is by *afflux*.

If the chambers do not empty themselves the quantities of blood will be proportionately reduced.

As to the local rise of blood pressure in *reflux*, we gather that no figures are available.

Contrasting venous afflux, in its continuity (evenness) of flow, with venous reflux, in its discontinuity, we shall note that, whereas the former will tend to spread its accumulation (during arrested flow) over a large venous area, by reason of the gradual distension effected, and so will minimize rise of

* I am indebted to Dr. Charles Bolton, C.B.E., F.R.S., for information on this point.

blood pressure; the latter by its relative suddenness will limit distension to the parts nearest the heart, thus causing much greater rise of blood pressure *in situ*. This will be most observable in disease with marked backflow.

Right Ventricular Systole.

At the cessation of auricular systole the state of the ventricle should show:

1. The ventricular walls slightly on the stretch as a result of the auricular thrust.
2. The tricuspid valve in position and slightly on the stretch, owing to the elastic recoil of the ventricle. The latter involves a slight backward movement of the valve, but this is probably too small a quantity to impart any appreciable impulse to the contents of the auricle, let alone to record itself in the internal jugular vein. This minimal thrust would immediately precede ventricular systole.

At the same point of time the state of the auricle would show:

3. The auricular walls in complete relaxation (including, of course, the sphincter fibres of the cavae); the blood content everywhere in contact with the flaccid walls and in direct continuity with the blood in the great veins; the auricular walls absolutely passive* and ready to be dilated by venous afflux or venous reflux (should the valve be incompetent); the blood content ready to receive and transmit any impulse impressed upon it by the ventricle.

The ventricular systole now sets in with a force and abruptness far exceeding that of the auricular systole. In a piston-like stroke the tricuspid valve is driven towards the auricle, carrying with it the blood in the funnel-shaped space between the valve segments. The stroke takes effect upon the contents of the auricle, and a wave of propulsion or percussion is started and transmitted along the fairway of the open cavae into their main branchings. There is nothing to hinder such transmission, and the slack walls of the auricle lying passively upon their contents should favour oscillation.

Should the tricuspid valve be incompetent more or less of reflux will add itself to the wave, modifying its characters. In heart disease with overloaded right chambers the reflux may be very marked and appear in the veins at the root of the neck as a pulsation definitely ventricular systolic. No sphincter action intervenes, as in the case of the auricular systole. If, then, this latter can register itself in the internal jugular, the stronger unimpeded ventricular impulse should surely be recorded.

The time of this wave will be early ventricular systolic. As to magnitude, and as measured by the forces engaged in its production, it should greatly exceed the auricular systolic wave.

Another wave must now be considered, which is also ventricular systolic, and, according to accepted teaching, appears at the root of the neck. Its cause is referred to the left ventricle, which develops it at the origin of the aorta and propagates it thence throughout the arterial system as the pulse. Its presence in the jugular vein is explained by the close proximity of carotid and jugular, whereby the throb in the artery is communicated to the contents of the adjacent vein. This is a percussion wave pure and simple; the venous flow is in nowise affected; no question of reflux arises; no changes in the volume of the blood within the vein accompany the wave. This is the *c* wave of the venous pulse tracing.

The time of this wave is ventricular systolic, but it is not earliest systolic, for it does not begin till after the *a-v* valve has been fully closed, and not before the blood pressure within the left ventricle has been raised sufficiently to force the aortic valve. It therefore must follow upon the early ventricular systolic contraction.

There remains for discussion a wave marked "*v*" on the polygraphic tracing, which is said to arise during the ventricular systole, and is regarded as owning two factors—namely:

1. A rising intra-auricular and intravenous pressure due to afflux.
2. A sudden fall of the pressure upon the onset of ventricular diastole.

It has been described as a wave of storage or *stasis*. The name is unfortunate, for a wave is essentially a dynamic manifestation, and statics do not beget dynamics. The

* There is no such thing as an actively expanding auricle.

teaching amounts to this, that the gradual venous congestion of arrested afflux during the ventricular systole accounts for the up-stroke of the *v* wave; the sudden onset of ventricular diastole for the down-stroke.

But, if so, then the up-stroke should be as gradual as the venous accumulation and the down-stroke as sudden as its release—that is, as short as the click of the second heart sound. The form of such "wave" should be as shown in Fig. 3.

FIG. 3.—Intravenous pressure line of arrested afflux during auricular and ventricular systoles.

But true waves, undulations, are ridge-and-trough manifestations, each one of the series showing the descending limb as the counterpart of the ascending limb; presenting, in fact, just such a curve as the *v* wave shows (see Fig. 4).

FIG. 4.—Phlebogram and radial tracing from a normal individual. Note in particular the form of the *v* wave.

There is evident conflict here between the tracing and the process which is held to be responsible for it. Quite apart from this argument as to the form of the wave, it is difficult to conceive how a head of pressure of only 0 to -8 mm. Hg could yield a wave of magnitude even approaching that shown.

Summary.

To summarize, with brief comment, the cardiac sequence of events and the polygraphic tracings.

1. *The Auricular Systole.*—The *a* wave appears to conform with this in time, but so close-set are the two systoles that more exact timing seems desirable; in magnitude the tracing seems disproportionately great, seeing the small power of the auricle and the block interposed by the sphincter action at the mouths of the cavae.

2. *The Auricular Diastole.*—No hint of this shows on the tracing, though the ventricular diastole is credited with causing so marked a feature in the phlebogram.

3. *The Ventricular Systole.*—This is a very powerful and abrupt event; the conditions for backward transmission are very favourable, all sphincter action being withdrawn. Why is it conspicuous by its absence from the tracing?

4. *The Forcing of the Semilunar Valves.*—This event takes place in the course of the ventricular systole, at some time subsequent to the closure of the auriculo-ventricular valves. It should therefore be preceded by an early ventricular systolic wave, whereas it stands alone.

5. *The Onset of Ventricular Diastole.*—This event is supposed to correspond with the onset of the down-stroke of the *v* wave; but the want of conformity of this wave of the tracing with its theory of production has been dealt with. As shown it is exactly like waves *a* and *c* in type, yet in mode of production quite different.

In view of these apparent discrepancies I suggest a reference of the whole subject to the physiologists.

* For permission to use this figure I am indebted to the courtesy of Dr. Frederick Price and the Oxford Medical Publications. See Dr. Price's *Textbook on Diseases of the Heart*, 1918, Fig. 58, p. 101.

† Do we know what is the exact point of time when the semilunar valves are forced?

THE congress of French-speaking gynaecologists and obstetricians, which is held every two years, will take place at Geneva on August 9th, when the following subjects will be discussed: (1) The indications and technique in operations for retro-deviations of the uterus apart from pregnancy, introduced by MM. Henneberg and Proust; (2) treatment of pregnancy complicated by tuberculosis, introduced by MM. Voron and Weymeersch; (3) methods of anaesthesia in labour and gynaecological operations, introduced by MM. Rapin and Schickele.