STATE OF THE ARTERY AND THE CIRCULATION UNDER THE SPHYGMOMANOMETER CUFF DURING COMPRESSION.

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It is generally assumed that the lumen of the brachial artery is completely obliterated when, in the course of a blood pressure estimation, the pulse at the wrist ceases to be palpable, and on the face of it this assumption appears almost to be self-evident. Moreover, has it not the sanction of physiologists generally? Consult here the figure in Starling's *Physiology* (1926, p. 806), also Howell's textbook (1927), and Gallavardin's figures reproduced in Halls Dally's *High Blood Pressure* (1926, p. 42)—nothing could be more categorical and plain to see. In spite of all this I have been driven to doubt the *fact*, and this doubt has grown in strength with further consideration. May I be allowed to produce my reasons? I must add that this is not an academic question for mere debate; it is a plain matter of fact or not, and it raises a definite practical issue in respect of the interpretation of the manometer readings.

Reaction of the Tissues to Compression.

To begin with, then, as we are dealing with compression and compressibility, it must be asked, Are the so-called soft parts of the arm—Gallavardin's *parties molles*—rightly thus named? They consist of adipose tissue, muscle fibre, and a fibrous framework which embeds and encapsules; further, of a system of vessels, blood- and lymph-containing, which ramify through the whole mass; we can probably omit the consideration of the last-named, the lymphatics, as negligible. Enclosing this tissue-complex is the dense exoskeletal structure of the skin, more or less loosely tied to the deeper parts, and, completing the list of factors which enter into the problem, there is the central core of bone, which presents its rigidity to serve for attachment as well as to supply a surface for counterpressure.

Now there will be no doubt in our minds as to the unyieldingness of the bony core; is there any as to the yieldingness of the "soft parts"? *Fiat experimentum*—thrust the thumb forcibly into the mass of the biceps; does it not sink in, and does this not settle the question? No, for another interpretation offers—namely, that within certain limits these parts are free to move and that the thumb has just thrust them aside; in other words, the experiment has shown displaceability—not compressibility—and it must be repeated, with displaceability ruled out. This can be effected most readily by enlarging the area of compression, and it is secured by the cuff of the sphygmomanometer, which not only covers a large area but envelops the whole circumference of the arm in its grasp. Thus conditioned, when the instrument is put into action do we as a matter of observation see any appreciable displacement of the soft parts either above or below the cuff, or does the cuff sink in, as did the thumb? Again the answer is No; a little puffiness above its upper edge and a similar slight puffiness below the lower edge, together with a moderate general tumefaction and a duskiness of the forearm, is all that is noted (the last-named appearances find their explanation in certain congestive circulatory effects). There is accordingly no evidence of a displacement of the parts from under the cuff, either distally or proximally, nor of a yielding of the soft parts to the pressure of the cuff. Gallavardin's figures have, therefore, no foundation in fact, and the term "soft" parts is a misnomer; all that the thumb and cuff experiments have established is that the tissues are more or less mobile—displaceable, but not compressible within the pressure range of the manometer.

On *a priori* grounds the same conclusion might have been reached, for of the parts in question the adipose tissue consists of a mass of cells filled with fat in the liquid state; while the muscle substance is so heavily hydrated

that in the living state it exists as a jelly, more or less fluid; and the blood is a liquid, which, though eminently displaceable, *given the outlet*, not so favoured behaves like other liquids and resists enormous pressure, in comparison with which the 100 to 300 mm. of mercury of the manometer scale are absolutely negligible.*

The position, then, at which we have arrived is that under the conditions secured by the sphygmomanometer the immobilized tissues are incompressible; further, that, regarding their liquid and semi-liquid state, we are justified on physical grounds in assuming that they will behave like other liquids; consequently that any pressure applied to the surface of the imprisoned area under the cuff will be transmitted immediately, uniformly, and without diminution throughout the substance of the cuff segment of the arm, for at each stage of the investigation, as the column of mercury halts in its ascent, the opposing forces are equilibrated, and the problem before us becomes one of hydrostatics, such as is exemplified exactly by the Bramah (hydraulic) press.†

This brings us to the subject proper of this paper—namely, the state of the artery and the behaviour of the blood stream under the rising pressure in the cuff of the instrument. It will make for clearness if we consider these effects in the following order: (1) in the arm below the cuff; (2) in the arm under the cuff; and (3) in the arm above the cuff.

1. *Effects in the Segment of the Arm Distal to the Cuff.*

These will be just those of a ligature round the arm. The ligature is very broad and its constrictive force is developed gradually, but on neither of these counts does its mode of action differ from that of any other ligature, in that it impedes both the entry of blood into the segment in question and still more markedly its escape therefrom; for the thin-walled low-pressured veins, in particular those of the surface, will be the first vessels to feel and yield to the pressure of the cuff at its lower border, and the first squeeze of the ball should suffice to close them. The deeper veins would follow suit quickly, for the cuff pressure effectually prevents any displacement of the tissues of the forearm, and these, becoming at once incompressible, transmit rapidly the surface pressure to the deeper parts. The primary effect therefore is venous obstruction along the line of the lower border of the cuff, with consequent gathering of the blood behind the obstruction, a movement which must work backwards towards the capillary fields and through these to the arterioles, leading ultimately to more or less arterial overfilling—in brief, to a choked state of all the vessels. It will, however, be noted that this general congestion is predominantly venous, and further that the accumulation of blood will again be most marked in the superficial veins, because the loose skin offers less resistance than the deeper parts.

These effects will continue throughout the duration of the experiment, and the surcharged vessels, therefore, will oppose an increasing resistance to the entry of blood from the cuff segment of the arm.

2. *Effect in the Cuff Segment of the Arm.*

In contradistinction to the line of pressure (that of the lower edge of the cuff) which is responsible for the above-described effects, we must now regard the consequences of a broad circular area of pressure which the cuff lays upon the whole of this segment. The skin is now immobilized along with all the other tissues; everything outside the walls of the vessels is, therefore, incompressible, and so the full force of the cuff pressure is brought to bear upon the blood, which, though *per se* incompressible, is not only displaceable, but is in act of displacement along a given line—that of least resistance.

* Professor A. W. Porter of University College informs me that there are no data available as to the compressibility of bodies in the colloidal or jelly state, but he holds it as almost certain that they will come close to liquids in their incompressibility.

† Exactly, in so far as the extravascular tissues of the arm are concerned, but through the blood vessels which permeate the tissues a flow of blood is all the while being maintained, and this complicates the physical problem, introducing a dynamic factor. However, this disturbing factor diminishes progressively as the experiment proceeds, and in the end, when the blood pressure is determined, it vanishes wholly with the arrest of the pulse.

Let us apply this to the state of things at the very commencement of the experiment. The blood is in actual movement through the vessels under a pressure which falls progressively from the arteries to the capillaries and from these to the veins, the ultimate destination of which is the right ventricle. Accordingly the internal blood pressure will be at its lowest, in this segment, at the mouths of the veins as they emerge from the upper edge of the cuff. Above this point the fall continues, and it is accentuated where the veins enter the chest by an actual *minus* pressure; there is, therefore, no impediment along this route; indeed, the *minus* intrathoracic pressure *favours* actively the centripetal flow. What, then, must follow when the encircling cuff begins to exert its grasp? The emptying of the veins must be accelerated, and in this process the superficial veins, which first feel the pressure, will take precedence. But very rapidly the surface pressure will be transmitted to the deep veins, and these will suffer a like emptying. An end to this depletion, however, must come as the walls of the veins flatten out and come into contact under the rapidly rising external pressure: thence onwards they are effectively sealed. How far back towards the capillary fields, or perhaps even into them, the emptying proceeds can only be determined by direct observation, for it is impossible to gauge *a priori* at what point the intra-arterial pressure which is striving to fill the veins will counterbalance the opposing pressure on the veins, which after the completion of the *forward* emptying process will now be directed *backwards*. Thus the primary effect of loading the surface of the cuff segment will be an emptying of the veins more or less, probably to a considerable extent—an effect exactly the opposite of that witnessed in the distal segment.

From the moment of the sealing of the veins, at this or that point, the circulation is interrupted up to the point of emergence of the veins from the upper edge of the cuff; but blood is still being squeezed into the arteries of the segment, though under increasing difficulties, so long as the intra-arterial blood pressure exceeds that in the extravascular tissues; the arteries, therefore, at this stage will perforce be all open.

The next step brings us to equality of the two opposing forces—that of the column of mercury in the manometer and that of the intra-arterial blood pressure; let us assume this to be 150 mm. The following diagram will represent the situation.

Stasis is now complete, for no blood can enter the artery; at the same time the artery maintains its patency, for there is no surplus of force to compress its lumen, and we have before us a given volume of blood imprisoned between the orifice of the artery and the sealed veins. The problem has thus become one of hydrostatics pure and simple, which may be indefinitely prolonged, *ceteris paribus*. We must further note that along with the cessation of all movement of the blood the pressure throughout this fluid is of necessity equal in all the vessels remaining patent.

Let us now suppose that the next squeeze of the ball raises the manometer pressure to 160 mm.; what will happen? By an excess of 10 mm. the equilibrium will have become unstable, and this surplus is brought to bear upon the whole volume of blood; what will follow? The imprisoned fluid will seek an outlet, pressing equally in all directions, both peripherally towards the veins and

centrally towards the heart *by way of the arteries*. But there is no thoroughfare peripherally, for there the sealed veins are held by the same excess of pressure as that which seeks relief. In like manner, at any given point along the vessels up to the brachial trunk itself, any attempt to shift the blood towards the veins will be countered—thus, up to the orifice O in the diagram, where an excess of 10 mm. finds itself unopposed. What then must follow, theoretically, is that the fluid will begin to dribble out backwards, reversing the normal circulation; there is no help for it.

In actuality a surplus force of 10 mm. in the face of the inertia of the mass of blood to be moved, against the surface resistance opposed by the multiple branching channels which contain it, would, without doubt, be imperceptible, certainly negligible; but, even so, we must note that a surplus of 10 mm. Hg (the apparent stasis still persisting) holds open the orifice at O. And actually, if we should push the experiment to a really appreciable extent—say to readings of 300 mm. or more—then an appreciable expression of the blood would be inevitable. But again what follows?—the orifice will still be kept open by the expulsive force until the last *quantulum* of blood has been driven out. Of course, the patency of the lumen of the vessels will, in that case, be gradually diminished as the volume of the blood to be expressed diminishes.

Within the time limits of a blood estimation, however, in which we should raise the manometer reading to 20 or 30 mm. above the pressure necessary for the extinction of the pulse, there will be no chance for these small surpluses of such short duration to effect any perceptible reduction of the volume of the imprisoned blood—the containing arteries therefore would remain fully patent.

On these grounds I am compelled to conclude that the theory of an obliteration of a section of the brachial artery in explanation of the extinction of the pulse is wholly untenable. I would, moreover, point out that any argument which could be brought forward in favour of the obliteration of the brachial artery by the outside pressure on its walls would hold equally for every other artery in the cuff segment of the arm, since its internal blood pressure could not exceed that in the brachial artery. But what force could conceivably shift all this blood, and where could room be found for it? Per contra if this is so, if the arteries keep their patency throughout, how do we explain the extinction of the pulse at the wrist at a given moment, which is an indisputable fact?

A reasonable answer to this question is, I think, not far to seek, but it involves certain preliminary considerations. The first is that the blood flow results from variations in blood pressure along the course of the vessels, and that this blood pressure is caused by the elastic stretch of the entire arterial system, but mainly, perhaps, by the elastic stretch of the aorta, which acts as a transformer between the power chamber of the ventricle, where all the energy is generated, and the capillaries. This action resembles exactly that of the elastic bag of the Spray apparatus, which, intervening between the ball and the nozzle, converts the intermittent grasp of the hand into a continuous efflux from the nozzle.

The elastic stretch of the arterial system—in which how many ventricular systoles are summed up?—absorbs the whole of the cardiac energy, the intermittent generation of which disappears quite by the time the capillaries are reached, and in the form of the elastic recoil it figures as the blood pressure, which measures the strength of the stretch. This is the sole cause of the blood stream. The fingers on the artery take no cognizance of it except as to the volume of the stream and the intravascular tension.

The pulse, on the other hand, is a manifestation of an entirely different order; it plays no part in forwarding the blood; it is an epiphenomenon, something superadded, and, seemingly, both unproductive and undesirable; for while it carries force in its progress along the arteries, the force is not available for circulatory purposes, yet it subjects the arterial wall to an additional strain, which may and does become a danger in disease. In a certain sense it runs counter to the purpose of the elasticity of the arterial wall, for it makes to reappear an intermittence which the elasticity has been at pains to suppress. In brief, it is

a percussion wave, which, arising at the aortic orifice, *quodam modo*, courses through the arteries at the rate of about 7 metres per second, whereas the blood stream is never faster than 0.5 metre per second.*

It must be obvious that these two velocities cannot be effectively harnessed to the end of one co-operative act. That which the finger on the artery takes note of, then, is the passage of a wave, and the impression gained is that of a lift, which is confirmed by physiologists, who teach that in the act of passing the artery is *expanded and lengthened*. Evidently the wave carries force.

Now let us take the stage of equipoise between the intra-arterial pressure—that is, the pressure caused by the arterial elastic recoil—and the opposing manometer pressure, when stasis supervenes; the pulse wave will, of course, present itself as usual at the mouths of the arteries, which are all open. Will it enter as usual? Why not?—it carries force; what is there to forbid? Well, there is no current to carry it. The answer to this is, it needs no current; it is not conveyed; it rides the blood stream and forces its way. We know that the percussion wave started by the cast of a stone travels as freely along the surface of a standing pool as over the smooth surface of a river, and in all directions, even against the stream. It will, therefore, enter and must reach the wrist, as usual, since the elasticity of the containing walls is not exhausted.

And now let us give another squeeze of the ball; the manometer rises, say, 10–15–20 mm., but the pulse is suddenly gone—why, if the arteries are still patent? For

* Compare Starling, op. cit., fourth edition, p. 813; also Wiggers, *Circulation in Health and Disease*, 1923, p. 190.

ILEO-CAECAI VOLVULUS FROM ADHESIONS FORMED BY LEFT-SIDED PELVIC APPENDICITIS.

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THE following case is of interest in regard to diagnosis and development. Moreover, it is a commentary on the dangers of incomplete anchorage or fixation of a viscus that has been hitherto free.

The patient, a spinster aged 33, was admitted to hospital as an acute abdominal emergency. Twelve hours previously she had been seized by violent griping pains in the left lower abdomen, accompanied by vomiting. No flatus had been passed since the onset of the attack, and the bowels had moved on the day before the attack. There had been no menstrual irregularity. There was a history of chronic constipation and occasional attacks of indigestion, confined to the region of the left iliac fossa.

The patient did not look ill. The temperature was normal, and the pulse rate 96 per minute. The tongue was furred. The abdomen was scaphoid and characteristic of the slender congenital viscerotopic type of anatomic structure. It moved freely on respiration, and some distension was present. The patient complained of pain in the left iliac fossa. There was deep-seated discomfort on pressure in this situation, but no tenderness or muscular rigidity. Pelvic examination was negative, as also was the obturator test for pelvic appendicitis. Succussion was present about the umbilicus, a sign of the greatest importance in the early stages of obstruction of the small intestine.

The case was regarded as one of acute intestinal obstruction. The abdomen was opened by a mid-line infra-umbilical incision. Coils of ileum, moderately distended, were packed off. The pelvic colon was empty. The caecum and appendix were not in the right iliac fossa. The ascending colon, longer and much more mobile than the normal, being provided with a mesentery, was traced into the pelvis. The caecum was lying in the true pelvis, distended and completely filling it. Attempts to extract it from the pelvis were resisted until adhesions binding down the appendix to the left side of the true pelvis had been divided. In delivering the incarcerated caecum a volvulus of the ileo-caecal segment of the bowel was discovered. Rotation in a clockwise direction had taken place through more than half a circle, round the axis of the appendix, adherent to the left side of the pelvis. The bowel was quite viable. After correction of the volvulus the appendix was removed and caecal plication performed. The patient made a good recovery.

the simple reason that this new rise of extravascular pressure, caused by the squeeze of the ball, will then have sufficed to meet and cancel the expanding force value of the pulse wave; the arterial wall will thus be prevented from yielding and the wave cannot pass, though the channels are all open. This is the reasoned explanation now proffered to show that it is not necessary to invoke obliteration of the artery, to account for the extinction of the pulse at the wrist, in the process of taking the blood pressure; and in the foregoing I have endeavoured to demonstrate that on physical grounds the obliteration hypothesis is untenable. It remains that the reasons advanced should be tested.

Bacon has said: "*Visio oculorum melior est quam progressio animi*," which I suppose may be rendered "the sight of the eyes is better than the mental process"—that is, ratiocination—and one must agree that a simple act of perception would save much excogitation and debate. But in the absence of sight by the external eye (for who has seen the closed artery?) what remains but the mind's eye to probe the validity of a hypothesis which is, as it stands, a pure assumption?

It has been said that the subject is not merely academic in that it raises a definite practical issue—namely, that from the beginning of the use of the sphygmomanometer—on the assumption of arterial obliteration, it has been a moot point what proportion of the pressure readings is to be allotted to the intra-arterial tension, and what to the resistance of the arterial wall itself (this latter a very variable quantity, when in disease the artery becomes thickened and hardened). Persistent patency of the arteries would eliminate this question once for all.

The caecum and ascending colon were extremely mobile in this case, being provided with a long mesentery. Such a state of affairs is not uncommon in the slender congenital viscerotopic type of individual. The caecum was thin-walled, atonic, and dilated, thus showing commencing failure of compensation, from long-standing difficulty of emptying. The appendix was cicatrized in its terminal portion, and the lumen in the distal half nearly obliterated. Obviously it had been affected by former inflammation. The previous attacks of left-sided abdominal pain supported this finding. Ultimately the appendix became fixed by adhesions to the left side of the pelvis. The hitherto mobile caecum became thereby insecurely tethered, and volvulus of the ileo-caecal segment took place.

Interest in atypical cases of appendicitis is sometimes associated with abnormal positions of the caecum and large intestine due to errors of rotation and fixation. The errors of mal-rotation and non-rotation of the midgut loop are those most likely to undergo volvulus change, and are usually, though not always, met with in the childhood period. In the adult it is the errors of the third stage of rotation, in which the viscera find their ultimate position of fixation to the posterior abdominal wall, that occur. Premature or high fixation of the caecum or failure in elongation of the colon results in an undescended caecum. Appendicitis in such a case may be difficult or impossible to distinguish from cholecystitis. Deficient fixation or abnormal elongation of the colon results in the low pelvic type of caecum. In the present case the attacks of appendicitis were atypical, being pelvic and left-sided. Intussusception in childhood and visceroptosis in the adult are intimately associated with this type of anatomical structure. Ileo-caecal volvulus is also associated with a low pelvic type of mobile caecum. To the incomplete fixation of the caecum produced by the adhesions around the appendix is to be ascribed one of the exciting factors concerned in its production in the present instance.

The case is an interesting example of an anatomical structure varying quite definitely from the normal, but which functioned fairly well until the introduction of another factor led to serious risk to function and to life. Nature's incomplete efforts at "caecopexy" were certainly disastrous in this case. May it not be argued, therefore, that fixation operations are essentially unphysiological in congenital visceroptosis? It seems to me that they are based upon an incomplete understanding of the altered anatomical structure and function of the individual as a whole. General methods of physical education and support would seem to meet the need in the majority of cases.