SOME OBSERVATIONS ON THE SIGNIFICANCE OF BLOOD-PRESSURE READINGS IN MAN.

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DIFFERENT opinions are held as regards the practical value of blood-pressure estimations in elucidating the actual conditions of the circulation. It may be stated with confidence that a single estimation of systolic or obliteration pressure, using the first reading obtained, is in some cases open to serious fallacy—apart altogether from psychical disturbances, etc.—even as an estimate of the maximum (systolic) pressure obtaining at that time in the artery under examination.

In former communications we urged the importance of using *continued* local pressure as an aid to correct estimation of the systolic pressure in certain abnormal conditions of the arterial system, an addition to the usual method.^{4 5}

But the systolic pressure, even if accurately gauged, gives very insufficient evidence as to what is going on in the circulation. For example, in cases of complete heart-block, where very high systolic readings may be obtained, the mean blood pressure is low and the blood flow is very defective, as shown in various ways, including manifestations of cerebral disturbance from cerebral anaemia, etc. Here the systolic estimation would fallaciously rate the "blood pressure" as being above normal. Again, in cases of slow cardiac failure the systolic pressure may be about the normal level, while the circulation is very inadequately carried on, as evidenced by obvious signs (cyanosis, dyspnoea, etc.).

Diastolic Pressure.

The importance of estimating the minimal or diastolic pressure is now becoming more generally recognized, and with the easy applicability of the auditory method there is no difficulty in the way of its routine use.

Apart from the influence of aortic diastolic pressure in determining (a) the strain upon the closed aortic valves, and (b) the resistance to the opening of these valves by the ventricular systole, the distending force of load which it imposes on the arterial tube is of prime importance. The properties of the arterial wall are such as to show relatively great effects from long-continued tension as compared with the transient application of distending force, as in the brief rises of pressure that constitute the systolic waves. Experiments upon excised surviving arteries demonstrate very strikingly the different degrees of yielding of the vascular wall in the two cases, especially in a tonically contracted artery.³

As long as the diastolic pressure remains low, even with very high systolic readings, the tendency to the giving way of vessels (cerebral haemorrhage, etc.) seems to be relatively slight—for example, in aortic regurgitation.

The greater proneness of the leg arteries to degeneration is especially associated, in all probability, with the high diastolic pressure in the erect position, rather than with the concomitant increase in the systolic level, which is also induced by the influence of gravity.

The evidence available at present is insufficient to show in what measure the injurious effects of an excessive diastolic pressure are to be attributed (a) to the influence of mechanical strain upon the tissue elements of the arterial wall, and (b) to the influence of a high distending pressure on the flow of blood and lymph in the walls of the arterial tube.

Much requires to be done to elucidate the relations of increased or diminished expansion, depending either on alterations in the tone of the arterial muscle or in variations in the intra-arterial pressure, on the blood flow in the capillaries of the vasa vasorum and on the lymph flow; also the relations of hypermyotrophy and atrophy of the media and hypertrophy of the intima to the intra-arterial

pressure, as well as to the various toxic influences that are known to affect the nutrition of the vessels.

Is hypermyotrophy a response to increased internal pressure, comparable with some forms of cardiac hypertrophy developed in certain conditions? Is the diastolic pressure the one that is of primary importance in this connexion?

In regard to various conditions which may exercise a special influence on the diastolic pressure, variations in peripheral resistance are most important, a low diastolic pressure naturally resulting from defective peripheral resistance, and vice versa, unless the heart action is much altered. Apart from alteration in peripheral resistance, quickening of the heart rate tends to raise the diastolic pressure by diminishing the time for outflow through the periphery between the pulse beats, and the converse holds with a slowed heart; that is, the diastolic pressure is lowered, with an increase in the range between the systolic and diastolic levels (pulse pressure). It must not, however, be assumed that a slow pulse rate is necessarily associated with a low diastolic pressure. Compensating adjustment in the vascular mechanism often keeps the diastolic pressure quite up to a normal or even heightened level with a slow heart beat, in spite of the lowering tendency of the latter. And the diastolic pressure may be very low with a very quick heart beat, for example, with very defective peripheral resistance, lessened inflow into the heart, etc.

In doing blood-pressure estimations we find it desirable to determine the diastolic index while the armlet pressure is being raised; in some (rare) cases where arm compression leads to a marked rise of blood pressure, the diastolic reading taken with a rising armlet pressure may differ from that obtained during the lowering of the armlet pressure (after determining the systolic index) by as much as 20 mm. This was especially the case when simultaneous estimations were made in the two arms. But a decided effect was sometimes observed when only one arm was tested. Comparison of the diastolic values with rising and falling armlet pressures may thus give important indications of the liability of the arterial pressure to vary in an abnormal way, when a large artery is closed, as in the process of systolic estimation.

As regards the impressions derived from *digital* examination of the pulse, we have found the impression gained as to (a) the tension in the vessel within the beats (diastolic pressure), and (b) the extent of the change at systole (pulse pressure) to be specially fallacious in some cases—apparently more deceptive than the impression as to the compressibility of the pulse (systolic pressure). An enormous pulse pressure (for example, 120 mm. in a case of aortic regurgitation) may be present, as shown by measurement of the systolic and diastolic pressures, when the finger fails to give any adequate conception of such a pressure wave along the artery. And the measured diastolic pressure may be very high when the impression given by digital examination is that it is rather low.

Some Points in Relation to the Auditory Method.

In former papers we urged the importance of the auditory (auscultatory) method as a simple and accurate means of estimating the diastolic or minimum pressure as well as the systolic or maximum pressure, and our further experience of this method has entirely borne out all that we have said in this connexion.⁵ It is clear that if the study of blood pressure is to be successfully applied to elucidate the circulatory conditions in man in health and disease, the examination must include routine estimation of systolic and diastolic pressures with computation of the pulse pressure; such estimation by the auditory method is easy and expeditious, and must replace the very inadequate procedure of systolic estimation alone, as well as the use of the oscillatory method for diastolic estimation.

The term "diastolic" pressure is used in the same sense as "minimum" pressure, to indicate the pressure at the deepest part of the trough between the systolic waves. Of course the arterial pressure varies much at different phases of the period of cardiac diastole, so that there are different levels of pressure at different points in the diastolic interval. The same applies to the period of systole. But, as by common consent the term "systolic pressure" is applied to the highest level resulting from

the cardiac systole, so the term "diastolic" pressure designates the lowest point to which the pressure falls between the pulse-beats.

In regard to the *rationale* of the auditory method to the recognition of systolic pressure, no difficulty arises. But in regard to the use of this method for diastolic pressure estimation, the question may be raised as to how the sound produced in the systolic phase and depending upon the systole can serve as an index of diastolic pressure. The answer is that, though the sound is actually elicited by the systolic wave, its occurrence or non-occurrence and its character when present, are determined by the intra-arterial pressure existing during the diastolic phase and its relation to the external or armlet pressure. If the intra-arterial diastolic pressure stands in such a relation to the armlet pressure that some distortion of the arterial tube from the circular form—that is, some flattening—occurs during diastole, then the loud clear tone is heard. On the other hand, when the relation is such that the intra-arterial diastolic pressure is not sufficiently below the level of the armlet pressure to ensure some flattening between the pulse beats, the sound, if audible at all, is dull in character. The change (during the lowering of the armlet pressure) from the clear to the dull and weaker sound, occurring as soon as the external and internal pressures are approximately equal, constitutes the index of diastolic pressure. In addition to the lines of evidence (as to the correctness of this index) stated in our former papers (study of circulatory schema, comparison with oscillatory method, etc.), we may add the direct experimental test carried out by us in conjunction with Dr. J. R. Murray.⁶ Using anaesthetized large animals (sheep), we measured the systolic and diastolic pressures in one carotid artery by means of valved manometers (maximum and minimum) connected with a cannula inserted into the artery at the root of the neck on the proximal (cardiac) side of an armlet which was placed round the neck. The armlet being inflated in the usual way, the actual intra-arterial pressures were read off by one observer on the valved manometers, while simultaneous estimations were made by the auditory method by another observer; the auditory tambour (phonendoscope) was placed over the other carotid on the distal side of the armlet. These simultaneous readings by different observers (changing places from time to time) showed remarkably close correspondence; the auditory indications proved a strikingly accurate guide to the actual intra-arterial pressures as measured by the valved manometers. Such observations were carried out while the levels of blood pressure (systolic and diastolic) were made to vary widely—raised by compression of the abdomen, lowered by amyl nitrite, excess of chloroform, etc., also at very different rates of heart beat, etc. In all such conditions the accuracy of the auditory indications remained unimpaired.

Relation of Systolic and Diastolic Values to Efficiency of Circulation.

When systolic and diastolic pressures have been estimated and found to be at normal or approximately normal levels, with a normal rate of heart-beat, is this sufficient to prove that the circulation is proceeding in normal fashion? In the great majority of cases presenting the normal features referred to, it is the case that the blood flow is proceeding normally. But there are exceptional conditions where, with normal systolic and diastolic pressures, the mass movement of the blood is gravely defective, and the results are evident in the patent signs of circulatory defect—dyspnoea, pallor, or lividity, etc. Though the pressures may be normal, the volume of the arterial stream may be abnormally small, giving rise to circulatory defect. A small output of the heart with a suitable adjustment of the peripheral resistance by constriction of arterioles (necessary to maintain the aortic pressure in presence of the small output of the left ventricle) may establish a condition in which normal or not far from normal pressure readings may be accompanied by very deficient mass movement of the blood. The circulation has in this case been reduced to a smaller size than is compatible with the needs of the animal. The arterial stream may be seriously lacking in volume while showing readings of systolic and diastolic pressure that are not far from normal. With such defective volume the capillary flow is seriously reduced on account of the deficient mass

movement of the blood, the small cardiac output being associated with narrowing of the peripheral outlet from the excessive contraction of arterioles which is necessary to maintain the aortic pressure in presence of the small output of the left ventricle.

In children and in small animals the volume of the arterial stream is, of course, very small as compared with the adult man, while the normal relation of peripheral resistance is related to the cardiac output in such a way that the aortic pressures may be pretty similar. The latter pressures in the aorta being maintained without any undue constriction of arterioles, the capillary circulation is a perfectly efficient one, and the mass movement of the blood is rapid. The smaller volume of the arterial stream is adapted to the smaller capillary field to be supplied. With the enormously large capillary field of the adult person a great reduction in the volume of the arterial stream, even with pressures up to the normal, means a serious defect in the capillary stream and the mass movement of the blood in the circulation—hence, signs of circulatory inadequacy. On the other hand, the volume of the arterial stream may be normal, while the circulation is still inadequate; in this case a pressure defect will be evident—abnormally low systolic or diastolic readings or a small pulse pressure. With deficient pressure values a reduced volume of arterial stream would of course imply a still more serious deficiency in the circulation.

Common Readings in Defective Circulations.

The more common conditions, when the circulation is defective, are: (a) Low systolic and diastolic readings (as in haemorrhage, shock, etc.), or (b) a systolic pressure which may be normal or even higher, attended by an abnormally high level of diastolic pressure, as in some cases of slow cardiac failure. Here the peripheral resistance is increased, keeping up the systolic pressure, notwithstanding a poor cardiac output, but this involves a high diastolic pressure—that is, a small pulse pressure and associated defective movement of the blood. The rate of the heart beat has, of course, to be taken into account in connexion with the pulse pressure. The defective pulse pressure is here of great significance and at once shows the true state of the circulation, whereas the systolic reading taken alone would give an entirely false indication.

It is clear that estimation of the systolic and diastolic pressures with the concomitant determination of the pulse pressure yields data of great importance and value, the precise significance of which in different conditions will, no doubt, become much greater with increasing knowledge. But while this is the case, such estimations must on no account be regarded as superseding or rendering unnecessary the use of other sources of information regarding the condition of the circulation—for example, obvious signs such as pallor, lividity, defective volume of arterial pulse, venous turgescence, dyspnoea, etc. Error from misuse of the results of blood pressure estimation, by blind adherence to mechanical standards, is easily avoided by common-sense application of other obvious signs taken in correlation with the blood pressure data.

Discrepancies in Systolic Readings from Different Limbs.

In a former paper we described the occasional presence of large discrepancies between the systolic readings obtained from different limbs, apparently due to abnormal local conditions.* The higher readings may be greatly reduced by the application of continued local compression, as already described, and in this way the divergent readings may be harmonized, the same readings being then obtained from all the limbs; further local compression may then cause no further lowering of the systolic values. As already stated, it is only in some abnormal arteries that such results are obtained; in the majority of thickened arteries practically correct readings are obtained without the preliminary use of continued local compression. The effects of local compression are not dependent on the production of an ischaemic condition of the limb. The closure of the brachial artery—by digital pressure as employed—leads, of course, to

* In this connexion we specifically exclude the cases of arm-leg differences associated with aortic regurgitation.

more or less pronounced stasis in the limb, but not to ischaemia. The latter condition, induced by the application of an Esmarch bandage, was found by Hill¹ and his collaborators to lead to reduced systolic readings for some time after the obstruction to the arterial flow was removed. This holds good in a normal limb as well as under abnormal conditions.

This observation deals with very different conditions from those present in the use of local compression. Local compression, as we employ it, leads to no alteration in the subsequent systolic reading either in normal persons or in the majority of pathological conditions. We have repeated this observation very frequently and with constant results. A pressure reading was first taken from the two arms. One arm was then subjected to repeated compression (for example, twenty times), or, as is preferable, closure of the brachial artery by digital pressure was maintained for three minutes in the area of application of the armlet, the latter being loosened and slipped down on to the forearm or removed altogether. At the end of the period of closure the armlet, reapplied to the upper arm, gave, in the normal subject, a reading which, when compared with one made from the other arm, showed no discrepancy. Comparison with a (second) reading from the other arm which has not been subjected to continued or frequently repeated compression is, of course, essential, as the actual pressure in the arterial system might have undergone some alteration during the period occupied in the observations. The procedure of local compression clearly induces no change in systolic readings in ordinary conditions of the vascular system; this is also true of the great majority of abnormal conditions.

It is only in a small minority of cases with thickened and contracted arteries that a notable reduction of the systolic reading is induced by local compression as described. We have not observed such reduction in any case where there was not evidence of abnormality in the arteries. In cases where divergent readings have been obtained from different limbs, local compression has an equalizing effect. Repetition of the local compression does not induce an indefinite reduction of the readings—not below the level found in a normal or soft artery.

Parallelism between Results from Excised Arteries and Human Limbs.

A long series of experiments (conducted in association with Dr. J. E. Kesson⁴) on excised arteries tested in different conditions in a circulatory schema showed a remarkable parallelism as regards the results of continued compression with those described above. With relaxed arteries (normal or thickened) the "systolic" reading was practically the same before and after continued compression, whereas with powerfully contracted and thickened arteries the subsequent reading was greatly reduced—down to the level of that obtained with relaxed vessels. The continued compression induced relaxation at the compressed part, as was evident on direct examination of the artery, which was soft and easily distensible at that part, in marked contrast to the firm and resistant adjoining parts which had not been subjected to compression. This reduction in the resistance of the arterial wall was temporary; after a time, varying according to circumstances, recovery occurred more or less completely; the contraction and resistance to compression returned, causing the external (obliteration) pressure to be again an over-estimate of the systolic pressure within the artery by the amount of the resistance to compression offered by the arterial wall. Similarly, in the human subject, when the systolic reading had been reduced by continued local compression of the thickened artery, recovery occurred after a time, and the original systolic value was restored; in some cases this restoration was watched and found to occur with remarkable precision in observations repeated from time to time on the same patient over a period of many months. But in most cases the extra resistance, as indicated by the result of local compression, varied considerably from time to time in the same person.

The striking resemblance in the results got from resistant excised arteries and those sometimes obtained from human limbs naturally suggested the possibility or probability of a similar explanation being applicable in the two cases. But it is to be noted that while the parallelism between the results from excised arteries and from the

living subject is a perfectly definite and very striking one as regards the nature of the changes in pressure readings, the actual amounts of the alterations in the readings differ widely in the two classes of observations; in other words, there is a very close correspondence in the *kind* of change which occurs in each case, but a great difference in the *degree* of such change.

The alterations induced by continued compression in the human limb are, in some cases in which they are very extensive, very much greater in amount than could be predicated from the study of excised arteries. We have always borne this consideration in mind; in the first paper on the subject from this laboratory we adverted to this, and pointed out that it was impossible to account for the extensive variations in the systolic readings from different limbs sometimes met with, unless one assumed the existence of such enormous resistances as are to be met with in the powerfully contracted thick-walled arteries like the carotid of the ox or the metacarpal or metatarsal of the horse, examined under abnormal conditions calculated to give the maximum resistance, at room temperature, or under the influence of barium chloride, etc. Palpation of arteries in the living subjects under examination has not in our experience given evidence of such extremely resistant characters as were present in the excised arteries above referred to; we have never believed that the great differences in pressure readings in different limbs depended simply on the extra pressure required to distort the circular tube and bring it into a flattened position. There is, we believe, another factor which we have not so far described—resistance, due, as far as is known, to contraction of the arterial muscle acting in another way than simply by opposing the flattening of the circular tube, but in effect constituting a local resistance to the obliteration of the pulse in the artery compressed by the armlet; this depends on certain special features in the form of closure of the vessel when compressed by external pressure.

Resistance to Compression in Excised Arteries.

In an artery like the sheep's carotid, comparable in size and thickness of wall with the human brachial, contraction of moderate amount made very little difference to the pressure readings; this accords with evidence derived from other sources in showing that the normal tone of the brachial artery, and even a markedly increased tone, have no appreciable disturbing effect on blood pressure estimation. Even with extreme contraction, induced by mechanical stimulation at room temperature, use of barium chloride, etc., the resistance offered by the carotid of the sheep in these abnormal conditions, widely removed from those present in the human body—for example, 35 mm.—has not reached anything like the values sometimes observed in the differences between the human limbs in certain conditions (cases of aortic regurgitation are excluded). We conclude, then, that variations in the amount of contraction, such as occur in the normal brachial artery of man, are of no serious import in regard to blood-pressure estimation.

Janeway and Park² conclude that resistance of the arterial wall due to muscular contraction may be a source of error in blood-pressure estimations, so far supporting the view long held by Russell³ to this effect. Janeway and Park think that such error should not be more than 30 mm. in the human brachial artery.

Higher resistance values were naturally obtained from such large thick-walled tubes as the carotid of the ox with its powerful muscular coat. The values obtained from a vessel so different in dimensions from the human brachial could not be applied without qualification to the latter. And even with the ox carotid, differences (apparently due to local causes) as large as those sometimes found in the human limbs were observed only under extremely abnormal conditions; application of these results without qualification to the human vascular system would be wholly unwarrantable.

But these observations refer to normal arteries, and, as has been already stated, the differences in man have been noticed by us only in the presence of abnormal conditions of the arterial system.

In pathological arteries from the legs of old horses we recorded very high degrees of resistance to compression, the resistance depending on the presence of enormously

powerful contraction of the thick muscular coat. But the characters of such arteries on palpation are very different from anything we have felt in human limbs, and it is obviously not permissible to assume the presence of any such amounts of contraction in man.

Mode of Closure of Artery under External Pressure.

A good many years ago von Recklinghausen⁷ suggested a different form of closure under external pressure in sclerosed arteries as compared with normal ones, the latter being evenly flattened by external pressure and completely occluded (see the figure c), while in the former the walls of the tube become apposed at the middle part, while chinks still remained open at the sides (B). The fact

A indicates the circular form of the uncompressed artery; B represents the peculiar form of closure (with chinks at the sides) described in the text; C shows the ordinary form of closure.

that different kinds of tubes may undergo one or other form of closure is a fact that is very easily demonstrable. This hypothesis has, however, not gained currency as accounting for differences in pressure readings. It does not explain why *all* thick sclerosed arteries do not give different readings as compared with others in the same individual that are relatively normal. And it would not account for the effects of continued compression already described as occurring in some sclerosed arteries while absent in the great majority.

There is evidently another factor concerned, and we find reason to believe that muscular contraction constitutes such; that is, that the determination of the peculiar form of closure above referred to depends on an influence exercised by contraction of the muscular coat of a sclerosed artery under certain conditions. Powerful muscular contraction is not at all necessary. An amount of contraction that is soon overcome by external pressure of an extent sufficient to flatten the tube and bring its walls in apposition is able to determine the persistence of chinks at the sides through which the pulse can still be transmitted; a more or less considerable amount of additional external pressure is needed to close the chinks sufficiently to obliterate the pulse.

The occurrence of different pressure readings in different limbs and variations in the amount of such differences at different times are capable of explanation as being due to variations in the contraction present. The effects of continued or repeated compression—reduction of the readings with subsequent recovery, etc.—are similarly explicable as depending on the demonstrated influence of compression in temporarily removing the tonic contraction, which is again restored after a time, with concomitant restoration of the pressure values. The same explanation applies to the facts that local compression tends to equalize the diverse values which may be obtained from different limbs at the first test, and that local compression (even if repeated) does not reduce the reading below the level obtained with a soft artery.

The view here stated differs from that of von Recklinghausen in postulating activity of the arterial muscle as an essential factor, in addition to the presence of certain structural alterations in the sclerosed artery; it differs from that of Russell (who has long urged the importance of hypertonus in normal and sclerosed arteries), inasmuch as the latter seems to regard the muscular resistance as simply opposing flattening, not as determining a special form of closure.

An Illustrative Case.

If the pressure readings from two limbs differ markedly on account of the occurrence of a special form of closure in one artery as compared with the other, the pulse persisting much longer in one than in the other, it might be possible to elicit special evidence bearing upon the different

conditions. Such we have sometimes observed definitely; indeed, it was the encountering of certain facts in comparative and simultaneous estimations on different limbs, in some cases giving widely different systolic readings, that led us to regard the above-described mechanism as an operative one; the way in which it harmonized with other facts was sufficiently obvious.

In the following case, examined with Dr. J. R. Murray, these phenomena were very striking. The patient had no aortic regurgitation, but the arm-leg difference in the systolic readings was very marked. At the first test the (simultaneous) obliteration values were 115 for the upper arm and 198 for the leg. But a very marked change was recognizable in the leg artery at 116 mm.—that is, at the obliteration level in the arm. The pulse in the leg, though not abolished, was very notably cut down in volume; it persisted as a pulse of small volume with very little alteration till it became obliterated—at a much higher level.

Repeated compression was tried and was found to give marked lowering in the leg readings at successive tests—namely, 198, 186, 168, 149, 148 mm. A similar change occurred in the relation of the points of disappearance and reappearance of the pulse in successive observations. At first, when the armlet pressure was raised sufficiently to obliterate the pulse, kept at this level for some time and then lowered, the pulse did not reappear until a very much lower level had been reached—154 mm.—as compared with 198. The armlet pressure, run up again, gave disappearance of the pulse at 182 and reappearance at 152. The armlet pressure was then lowered to zero for some time; the next compression gave disappearance at 186 and reappearance at 155. At a later compression the pulse disappeared at 168 and reappeared at 166. There was thus a progressive diminution of both the actual obliteration level and of the difference between the points of disappearance and reappearance.

The arm readings behaved in a strikingly different way. There was no appreciable difference (a very few millimetres) between the points of disappearance and reappearance of the pulse, and the readings remained steady at about 115 to 116 throughout the series of observations, while the leg readings were showing extensive changes; this indicates that the aortic systolic pressure remained unchanged. It was easy in repeated observations to predicate the arm values by noting the very definite change in volume in the leg artery, as already described.

In this case diastolic pressure estimation by the auditory and oscillatory (Pachon) methods gave similar values—75 to 85 mm.—in arm and leg, varying little at the beginning and end of the series of observations.

It is noteworthy that the systolic readings were much higher with the armlet on the calf of the leg than when on the thigh; the latter often nearly agreed with the arm.

We conclude from such results that the systolic pressure was really the same in the arm and leg, despite the fact that a very much higher armlet pressure was required to obliterate the pulse in the leg, though the latter pulse was strikingly cut down—to one of very small volume, which persisted until the compressing armlet pressure had been raised much higher. We interpret this result as being due to a different form of closure in the arm and leg arteries respectively, and we regard the leg arteries as the ones in which the behaviour of the tube under compression was peculiar. There is, of course, the question as to whether the abnormality is in the arm or the leg—that is, whether complete occlusion is abnormally difficult in the leg or abnormally easy in the arm; either of these would obviously cause a discrepancy in the readings, the intra-arterial pressure being similar in both arm and leg.

There is clear evidence that the abnormality is in the leg. The arm readings remained constant, being unaffected by repeated or continued compression, while the leg readings changed very strikingly, being enormously reduced by repeated or continued compression; the leg readings could thus be brought down towards equality with the arm readings, but showed no further reduction—below the arm value—on further compression. And after a time the leg readings recovered their great predominance over the arm values.

These phenomena fit in exactly with the conception of a peculiar form of closure of the leg arteries when compressed by the "armlet," central parts being brought into

apposition a considerable time before the lateral chinks are closed; the pulse continues to be transmitted until a more or less considerable excess of external pressure has been applied, over and above what is needed to stop the transmission of the pulse in a normal artery. The effects of continued or repeated compression in reducing the readings in such an artery find an obvious explanation in their demonstrated influence in reducing the resistance of the arterial wall by temporarily diminishing or annulling the contraction present, and so removing a factor concerned in inducing the peculiar form of closure above described in a sclerosed artery.

Resistance to Compression of Excised Arteries.

In view of the foregoing results, it may be asked whether the high resistance to compression recorded in strongly contracted surviving arteries, possessing thick muscular coats, is associated with a similar peculiarity in the mode of closure under external pressure or whether it is simply due to the direct resistance presented (by the rigid muscle) against distortion of the tube from the circular form.

The excised arteries in which very high resistances were encountered were always powerfully contracted, with rigid muscular coats and a very small lumen. The use of the term "contraction" in this connexion may be ambiguous; it may mean that the action of the (shortened) muscular fibres may be such as to reduce the lumen to very small dimensions; or, on the other hand, the muscle may be "contracted" and rigid while the lumen is not very small—for example, in presence of high internal pressure, under the influence of cold, etc.; in the latter case, the muscle may be firmer and more resistant without being much shortened.

In the case of a strongly contracted artery with a very small lumen, there is no evidence that the peculiar form above described ever occurs in such a way as to allow persistent passage of the pulse wave through lateral chinks. The small lumen seems always to close by a simple apposition of the walls of the tube.

We conclude that the excess of external pressure over internal pressure which is necessary to obliterate the pulse under those conditions is due to the direct resistance to distortion of the rigid muscular walls of the tube. But, as already stated, we find no ground for assuming the existence of anything resembling this degree of muscular rigidity in the vessels of the living subjects. At the same time, the presence of contraction—that is, muscular resistance—is very important in human arteries when associated with certain abnormal structural conditions, determining the occurrence of special features in the process of closure of the vessel by external pressure, and thus leading to different readings of obliteration pressure that do not correspond with differences in the actual systolic pressure.*

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¹ Hill, Flack, and McQueen, *Proc. Roy. Soc.*, 1914, lxxvii, 255. ² Janeway and Park, *Arch. of Intern. Med.*, 1910, vi, 586. ³ MacWilliam, *Proc. Roy. Soc.*, 1902, lxx, 109. ⁴ MacWilliam and Kesson, *Heart*, 1913, iv, 279. ⁵ MacWilliam and Melvin, *Heart*, 1914, v, 153; *BRITISH MEDICAL JOURNAL*, March 28th, 1914. ⁶ MacWilliam, Melvin, and Murray, *Proc. Physiol. Soc.*, March 14th, 1914. ⁷ von Recklinghausen, *Arch. f. exper. Pathol. u. Pharmac.*, 1901, xvi, 104. ⁸ Russell, *Arterial Hypertonus, Sclerosis, and Blood Pressure*, 1908, 87; *BRITISH MEDICAL JOURNAL*, 1912, i, 659.

* By "closure" is meant such a degree of occlusion as is necessary to stop the transmission of the pulse wave as tested on the peripheral side.

ED. SERGENT and L. Nègre have reported to the Society of Biology the results of the examination of the stools of sixty-seven pilgrims returned to North Africa from Mecca; they included thirty-six returned to Algiers and thirty-one to Morocco. Thirteen (10 per cent.) had dysenteric bacilli of the Flexner type in their intestines. The examination of the stools of a certain number of natives resident in the neighbourhood of Algiers who had not been to Mecca gave a negative result. In three of the returned pilgrims (4.5 per cent.) cholera vibrios were found in the intestines, and in two instances the vibrios presented all the true characters of the cholera vibrio. The pilgrims were all in good health, and the observations show that among those returning from Mecca there are carriers who escape existing sanitary measures. An abstract of the paper is published in the *Bulletin de l'Office International d'Hygiène Publique* for August, 1914.

THE BARBER-SURGEONS' COMPANY.

AN ADDRESS DELIVERED TO THE HUNTERIAN SOCIETY OF LONDON.

By THOMAS GLOVER LYON, M.A., M.D.,

PRESIDENT; SECOND WARDEN TO THE BARBERS' COMPANY, 1914.

ARMS OF THE COMPANY.

BY the kind hospitality of the Worshipful Company of Barbers, the Hunterian Society is now privileged to hold its meetings in a place associated for centuries with the medical profession.

In the year 1461 a corporation which had been solicited by Thomas Morsted, Chirurgien to Henry IV, was granted to Jacques de Fries, his physician, and John Hobbes, his surgeon, by Edward IV, in the names of St. Cosmo and St. Damianus, brothers, physicians and martyrs. This corporation was called the Company of Barber-Surgeons of London, and received authority over all practising the same arts in and about the metropolis.

In spite of this charter, however, many unqualified persons continued to practise. There is an interesting account of how quacks were punished in the minutes of the Company. In the fifth year of the reign of Richard III one Roger Clark was prosecuted for quackery, having given a piece of parchment rolled up and said to be inscribed with the words of a charm, to be used round the neck against fever. The culprit was convicted and sentenced to the following peculiar punishment: He was "led through the middle of the city with trumpet and pipe, he riding on a horse without a saddle, the said parchment and a whetstone for his lies being hung about the neck, and an urinal being hung before him and another behind."

Up to the reign of Henry VIII barbery and surgery were practised by the same individual, but even during this period members of the Company specialized in one branch or the other. Many of those favouring surgery went to the wars either in England or on the Continent. These were often financed by the barbers, who received a share of the large fees paid by the nobles and other wealthy men for surgical attendance. One can imagine how competent men were sought after, and how great were the sufferings of those who were unable to secure their services, by the following quotation from Gale:

I remember when I was in the Wars at Muttrel (Montreuil), in the time of that most famous prince Henry VIII, there was a great rabblement that looked upon themselves to be surgeons, some were sow gelders and some horse doctors, with tinkers and cobblers. . . . But when the Duke of Norfolk, who was the General, saw how the people did die and that of small wounds, he sent for me and certain other surgeons, commanding us to make search how these men came to their deaths, whether it was by the grievousness of their wounds or by lack of knowledge of these so-called surgeons. . . . In the end this rabblement were committed to the Marshalsea, and threatened by His Grace to be hanged for their worthy deeds except they would declare the truth who they were and of what occupation, and in the end they did confess themselves to be as I have declared to you.

In the third year of Henry VIII's reign (1512), a licensing body was created, whose approval was necessary before anyone could practise as a physician or surgeon in the City of London or within seven miles of it. This consisted of the Bishop of London, or Dean of St. Paul's, aided by four doctors of physic, and for surgery other persons of discretion "experts in this faculty." In spite of the existence of this body, in the thirty-second year of Henry VIII's reign (1541), the Barber-Surgeon Company was united to another company, the Company of Surgeons, by Act of Parliament. This Act divided the members of the Company into barbers and surgeons; the barbers were not allowed to practise surgery except bleeding and drawing of teeth, and the surgeons were not to practise barbery or shaving. The art of surgery to which the charter of 1541 refers comprised the care of "bruises, hurts, and other infirmities, the letting of blood and the drawing of teeth." There has always existed an antagonism