Studies in blood-pressure, physiological and clinical / by George Oliver.

Prof. Halliburton F.R.S.

With the Writer's kind

regards.

Jan 1st 1908

STUDIES
IN
BLOOD-PRESSURE

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IN

BLOOD-PRESSURE:

PHYSIOLOGICAL AND CLINICAL

BY

GEORGE OLIVER

M.D. Lond., F.R.C.P.

SECOND EDITION, ENLARGED

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PREFACE TO THE SECOND EDITION.

THIS brochure in its second edition has undergone considerable enlargement by the incorporation of new material, the result of further work and experience on the part of other observers as well as myself in the field of clinical sphygmomanometry. I trust that this additional matter will render this little manual more efficient in fulfilling the aims of the original edition—namely, to encourage the practice of blood-pressure measurement, and to furnish some hints and guidance to those who may embark on it.

Having not infrequently obtained some unaccountably irregular results, many

of which were clinically inconsistent, from the application of the armlet to the arm and forearm on one or both sides of the same subject, I began to doubt whether this method is in certain cases as satisfactory in practice as the observations with it on normal subjects had led us to expect. I therefore tried to obtain some light on this matter; and to this end I applied the armlet in a large number of my cases to the arm and forearm of each patient, and employed other modes of reading the arterial pressure as well as the armlet method — noting in each case the agreements, disagreements, and their relation to the collateral clinical evidence (cardiac). The reader will find in the following pages the results of these numerous clinical observations, which seem to me to indicate that different degrees of thickening of the arterial wall suffice to account for the irregularities in the armlet readings which I had

observed.[1] The want of uniformity in reading the systolic pressure by the armlet method in subjects of arterio-sclerosis has also been recorded by other observers.[2]

As a practical outcome of this work, I am led to suggest the use of a supplementary method, founded on a different principle from that of the armlet, and, according to my observation, unaffected by arterio-sclerosis. And I must say, that since adopting the conjoint use of both methods, my clinical observations on the circulation have been more in accord with each other; and have been more instructive in affording a clearer conception of the state of the arterial wall and of the blood-pressure.

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[1] That thickening of the arterial wall may vitiate the reading of the blood-pressure obtained by counter pressure outside the vessel, was forcibly suggested by the demonstration of Dr. William Russell at the first meeting of the Association of Physicians of Great Britain and Ireland, held in London last May.

[2] See O. K. Williamson, M.D., etc., in *The Lancet*, vol. ii., 1907, p. 1516.

It may seem to be an elementary precaution to suggest—though it is, I think, quite a necessary one—that in studying the blood-pressure clinically, the observer should, above all things, take a comprehensive view of the whole circulation; and in particular he should compare the condition of the heart with his record of pressure. Moreover, when that record is furnished by a method the determinations of which are not disturbed by arteriosclerosis, the observer should try to educate his tactile sense of pulse-resistance (I fear a vanishing art) by aid of the pressure measurements. My long experience in pulse-feeling and pulse-pressure measuring has shown me the clinical advantages to be derived from combining both methods; and the observer who adopts the instrumental method of measuring the arterial pressure, will do well to be on his guard lest he should allow his tactile skill

in examining the pulse to become impaired by neglect.

As the measurement of blood-pressure is becoming a somewhat leading method of clinical observation, it is to be hoped that its rapid recognition may not engender an overestimation of its importance. It is true, blood-pressure plays its part—and that an important one—in the pathological drama; but it is not the only actor. Therefore the observer, however zealous he may be in prosecuting the study and observation of blood-pressure, should endeavour to strictly estimate the *rôle* it plays and accept its teaching with well-balanced appreciation of its limits.

It will doubtless interest my readers to know that Case E, in the series of senile pressure readings recorded on pp. 119–20, is that of Sir Henry Pitman, Emeritus Registrar of the Royal College of Physicians of London, the *doyen* of our profession. To Sir Henry I owe

my grateful thanks for the permission accorded me to measure his arterial pressure, and for the kindly interest he showed in the observations.

HARROGATE.

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CHAPTER I.

BLOOD-PRESSURE MEASUREMENT: APPARATUS AND METHODS.

During the past few years physiologists as well as clinicians have displayed much activity in devising methods of sphygmometric observation in man, which, in trustworthiness, may be regarded as approximating to the manometric procedure of the laboratory. And it may, indeed, be said that they have in a large measure succeeded not only in attaining this most important end, but in making the suggested methods easy of application and as free as possible from errors of technique.

It is needless to enlarge on the importance of the accurate observation of the blood-pressure in man, especially in our inquiries into living pathology, or (as preliminary or supplementary to the hæmomanometric record in animals) in

building up our knowledge of therapeutics, or in applying that knowledge; for that will be obvious to all. But before dealing with the practical aspects of hæmomanometry we should study the physiology of blood-pressure and try to solve some problems of a purely elementary nature which await further inquiry, for our knowledge of blood-pressure in normal man is far from complete, notwithstanding the good work which has been done towards its elucidation by such able investigators as Marey, Roy and Adami, Mosso, Leonard Hill, von Basch, Gärtner, von Recklinghausen, Erlanger, Potain, Riva-Rocci, Gumprecht, Hensen, C. Martin, A. Martin, Stanton, Crile, O. E. Stephens, T. C. Janeway, Cook, Briggs, Pal, Bernd, and others.

Various attempts have been made to measure the blood-pressure in man since the year 1834, when Hérisson[1] intro-

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The principle of measuring Blood-pressure by encircling a limb by a rigid cover enclosing fluid in a rubber bag, the compression of which is varied by mechanism, was introduced by Roy and Adami in 1890.[2] Their work, constituting the first serious step in the study of sphygmomanometry in man, was founded on the following mechanical combination: namely, (1) *a*

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Though this ingenious apparatus was not sufficiently convenient for general clinical use, it embodied the mechanical combination which has recently obtained general acceptance in the form of an armlet and a recorder. This more practical method was almost simultaneously conceived by Riva-Rocci[1] and Leonard Hill,[2] who substituted air for water as the medium for varying compression, and

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A Compressed-air and Spirit Manometer.

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aneroid can be trusted unless it is standardised frequently by the mercurial manometer. I have therefore been led to devise a manometer which is a sensitive and practical indicator of the arterial pressure, and one, moreover, which does not sacrifice accuracy for mere convenience.

The **recorder** (Fig. 3, A) is constructed on the well-established principle of the "compressed-air manometer" for many years employed in measuring the hydrostatic pressure of gases; the pressure of the gas being equilibrated by the compression of the air contained in a closed tube beyond the indicator, which is thus manometrically balanced between the two compressions. The adoption of this principle in sphygmometry is not new; for it was followed by W. R. Pond in 1875 (his sphygmoscope consisting of a glass tube empirically graduated with a closed bulb above and a bulb below covered by a membrane for the reception of a fluid index), and by Barnard, Hill, and Hicks in 1898.

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The **glass tube,** 8 or more inches in length and having a capillary bore, has a bulb at its upper end and a smaller bulb at its lower end (Figs. 1 and 2) where it is bent up to receive a rubber tube. Just above the lower bulb the bore is contracted (Figs. 1 and 2). The tube is mounted on a narrow strip of thin wood, through the lower end of which a pin, fixed to the sides of the box, passes, so that the tube may be raised from the horizontal position in the box to a vertical one before and after an observation.

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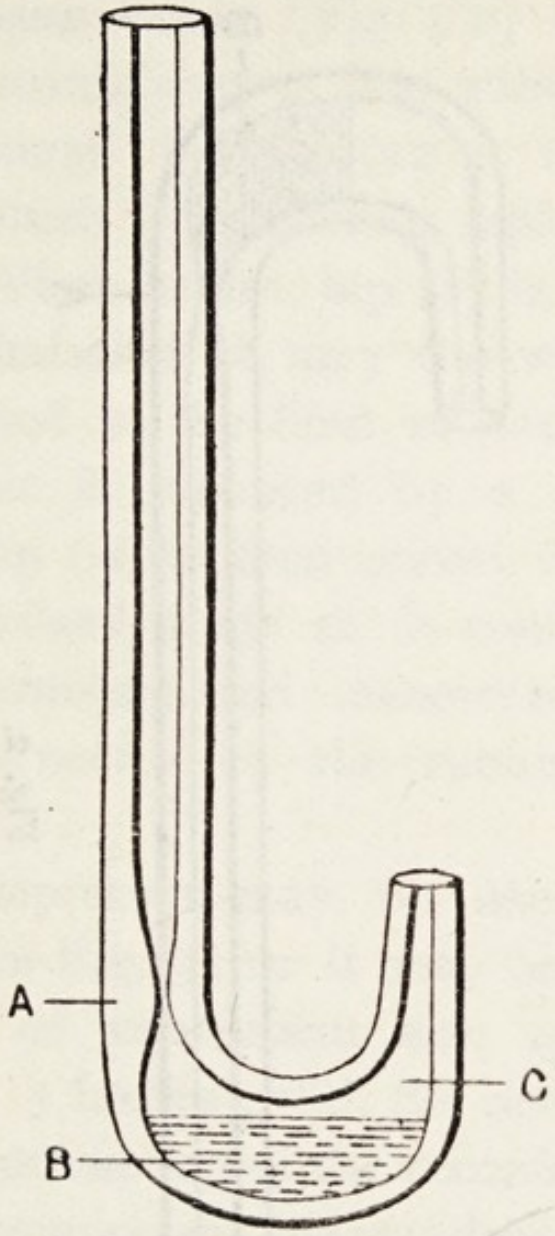

Fig. 1.

Lower end of the glass tube of the indicator raised to the perpendicular position: showing A, the contraction of the bore; B, the position of the meniscus fluid; and C, the continuity of the air inside the bore with that outside.

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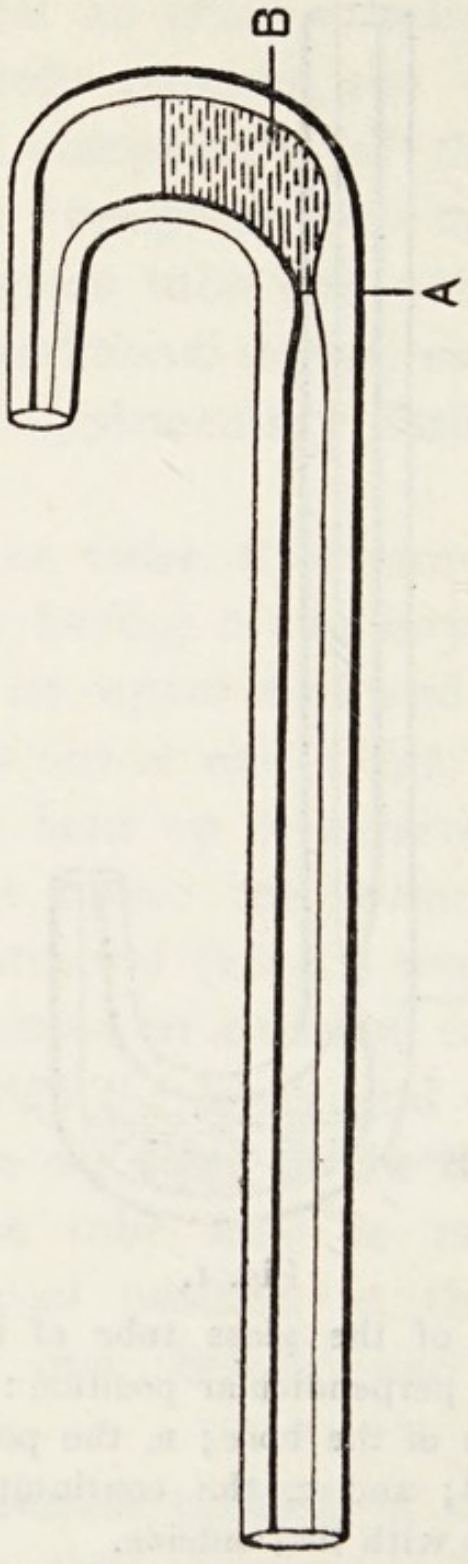

Fig. 2.

Lower end of the glass tube of the indicator placed in the horizontal position ready for an observation: showing A, the zero point; and B, the position of the meniscus fluid.

is quickly dissolved again by adding a few drops of spirit.

The compressor (Fig. 3 B) consists of (1) a strong canvas bag adherent to (2) two small boards (12 × 18 cm.), through which (3) a strong rapid screw passes. When in use, tap (*a*) is opened for the admission of air; the wheel (*e*) is unscrewed to the limit of the screw, which limit is indicated by a clicking sound; tap (*a*) is then closed, and the wheel is turned, when air is compressed into the arm-bag and manometer, previously connected by the rubber tubes (*c* and *a*).[1]

The compressor may be used separately, as in Fig. 3; or it may be set up in a box of convenient size (namely, 9 × 2¼ × 5 inches), with the rest of the apparatus consisting of the recorder fixed on the air-compressor, the standard armlet, and the phalangeal bag.

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[1] The air capacity of the compressor is so adjusted to that of the armlet as to secure compression to 300 mm. when the armlet is closely adjusted. Should the easy turning of the wheel of the compressor fail, it will be restored by letting a drop of oil fall on the screw when the wheel is unturned to its fullest extent. Those who prefer the ball-filler can, of course, use it with my recorder and armlet.

The armlet (Fig. 3 c) is more portable and adaptable to the shape of the limb than those now in use; and it is equally available for the forearm as for the arm. It consists of a canvas bag of the full standard width (12 × 16 cm.), to which are attached three straps (covering the width of the bag) with friction buckles. The outer rigid cover, consisting of the straps, is therefore in three sections, which enable the observer to adjust the bag perfectly to the fusiform shape of the forearm.

The practical advantages of this manometric arrangement are:

(1) *Reading the index in the horizontal position*, which contributes to ease and accuracy in work.

(2) *The avoidance of leakage* and its consequence—a falling index—from a defective air-valve, which cannot be rectified without loss of time and trouble. This not infrequent defect of the ball or pump filling, which is always a source of annoyance and inaccuracy in observation, is entirely obviated by this valveless arrangement.

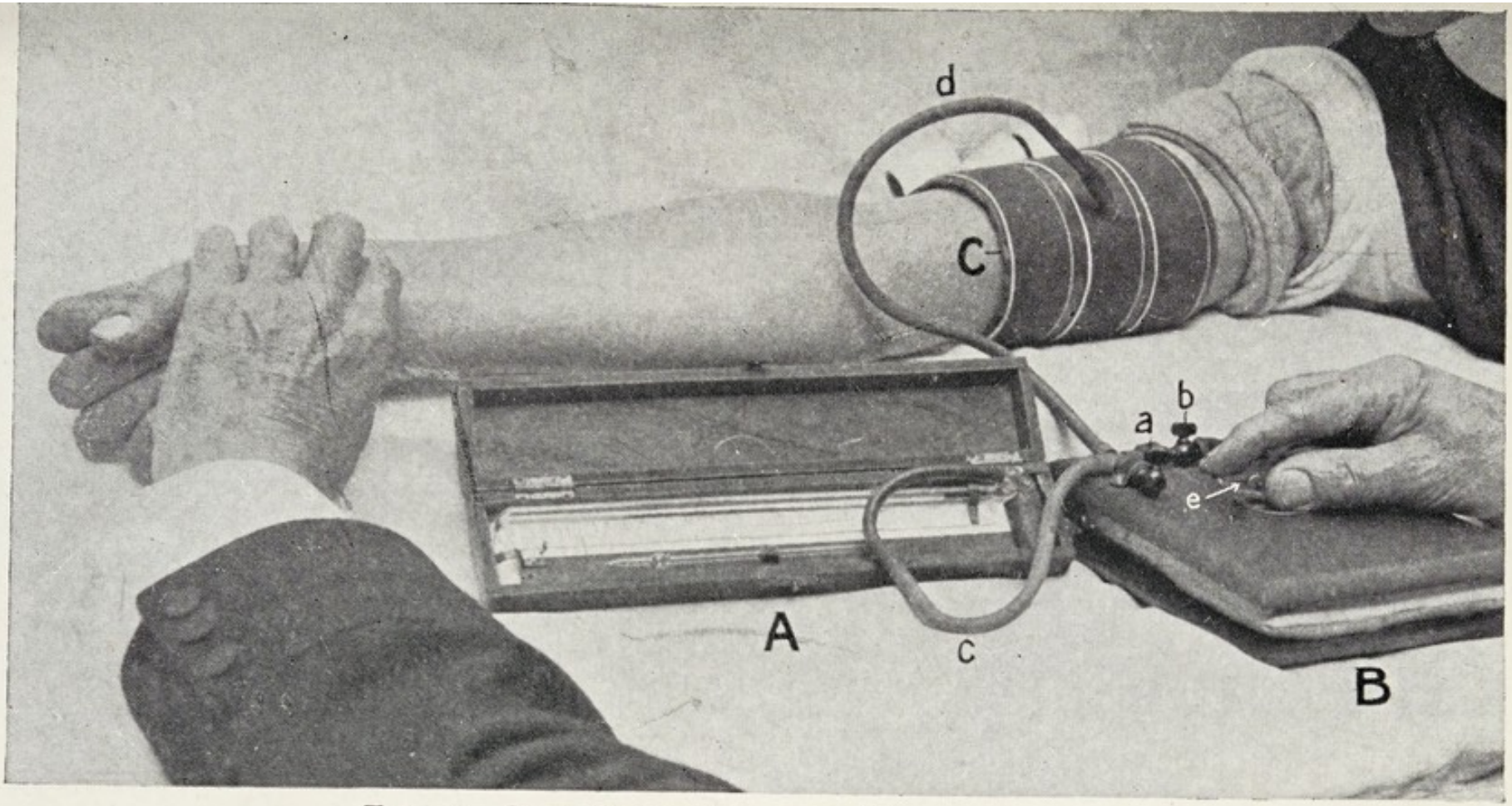

FIG. 3.—THE COMPRESSED-AIR HÆMOMANOMETER.

A, the recorder. B, the compressor: *a*, tap for occluding and releasing the air; *b*, tap for augmenting the oscillations of the meniscus; *c* and *d*, rubber tube connectors. C, the armlet.

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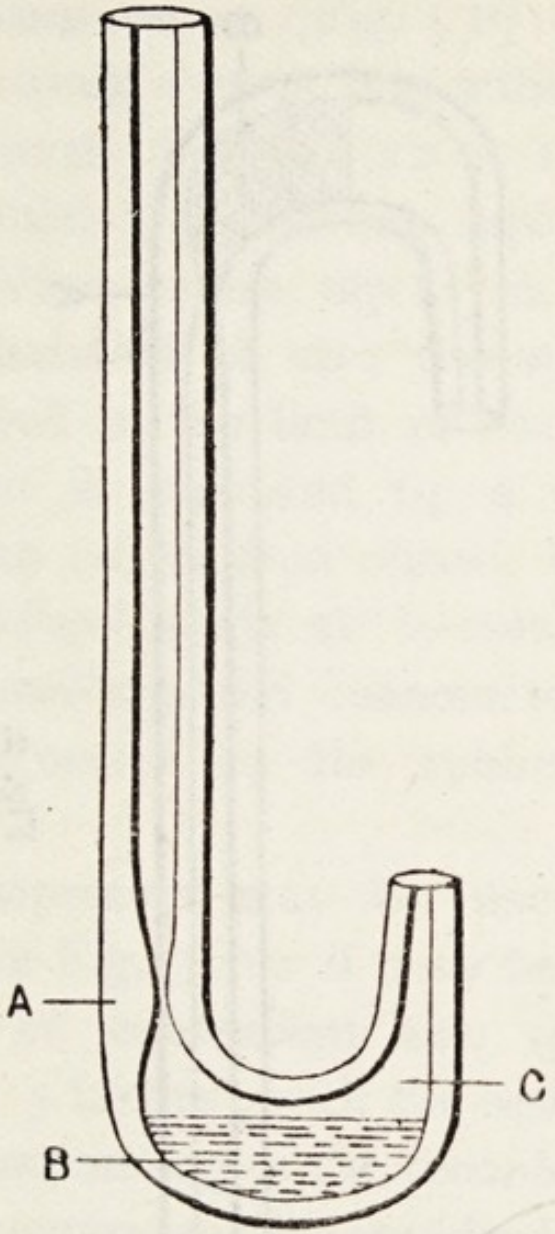

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Lower end of the glass tube of the indicator raised to the perpendicular position: showing A, the contraction of the bore; B, the position of the meniscus fluid; and C, the continuity of the air inside the bore with that outside.

indicator be allowed to dry up (as after prolonged disuse of the instrument), it

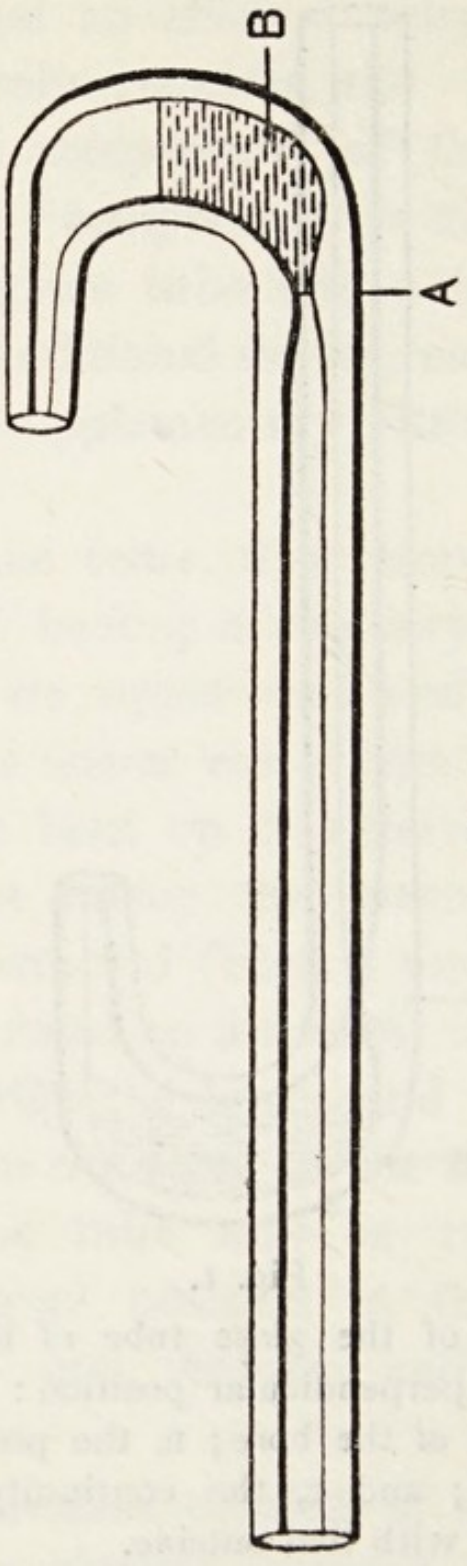

Fig. 2.

Lower end of the glass tube of the indicator placed in the horizontal position ready for an observation: showing A, the zero point; and B, the position of the meniscus fluid.

is quickly dissolved again by adding a few drops of spirit.

The compressor (Fig. 3 B) consists of (1) a strong canvas bag adherent to (2) two small boards (12 × 18 cm.), through which (3) a strong rapid screw passes. When in use, tap (*a*) is opened for the admission of air; the wheel (*e*) is unscrewed to the limit of the screw, which limit is indicated by a clicking sound; tap (*a*) is then closed, and the wheel is turned, when air is compressed into the arm-bag and manometer, previously connected by the rubber tubes (*c* and *a*).[1]

The compressor may be used separately, as in Fig. 3; or it may be set up in a box of convenient size (namely, 9 × 2¼ × 5 inches), with the rest of the apparatus consisting of the recorder fixed on the air-compressor, the standard armlet, and the phalangeal bag.

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[1] The air capacity of the compressor is so adjusted to that of the armlet as to secure compression to 300 mm. when the armlet is closely adjusted. Should the easy turning of the wheel of the compressor fail, it will be restored by letting a drop of oil fall on the screw when the wheel is unturned to its fullest extent. Those who prefer the ball-filler can, of course, use it with my recorder and armlet.

The armlet (Fig. 3 c) is more portable and adaptable to the shape of the limb than those now in use; and it is equally available for the forearm as for the arm. It consists of a canvas bag of the full standard width (12 × 16 cm.), to which are attached three straps (covering the width of the bag) with friction buckles. The outer rigid cover, consisting of the straps, is therefore in three sections, which enable the observer to adjust the bag perfectly to the fusiform shape of the forearm.

The practical advantages of this manometric arrangement are:

(1) *Reading the index in the horizontal position*, which contributes to ease and accuracy in work.

(2) *The avoidance of leakage* and its consequence—a falling index—from a defective air-valve, which cannot be rectified without loss of time and trouble. This not infrequent defect of the ball or pump filling, which is always a source of annoyance and inaccuracy in observation, is entirely obviated by this valveless arrangement.

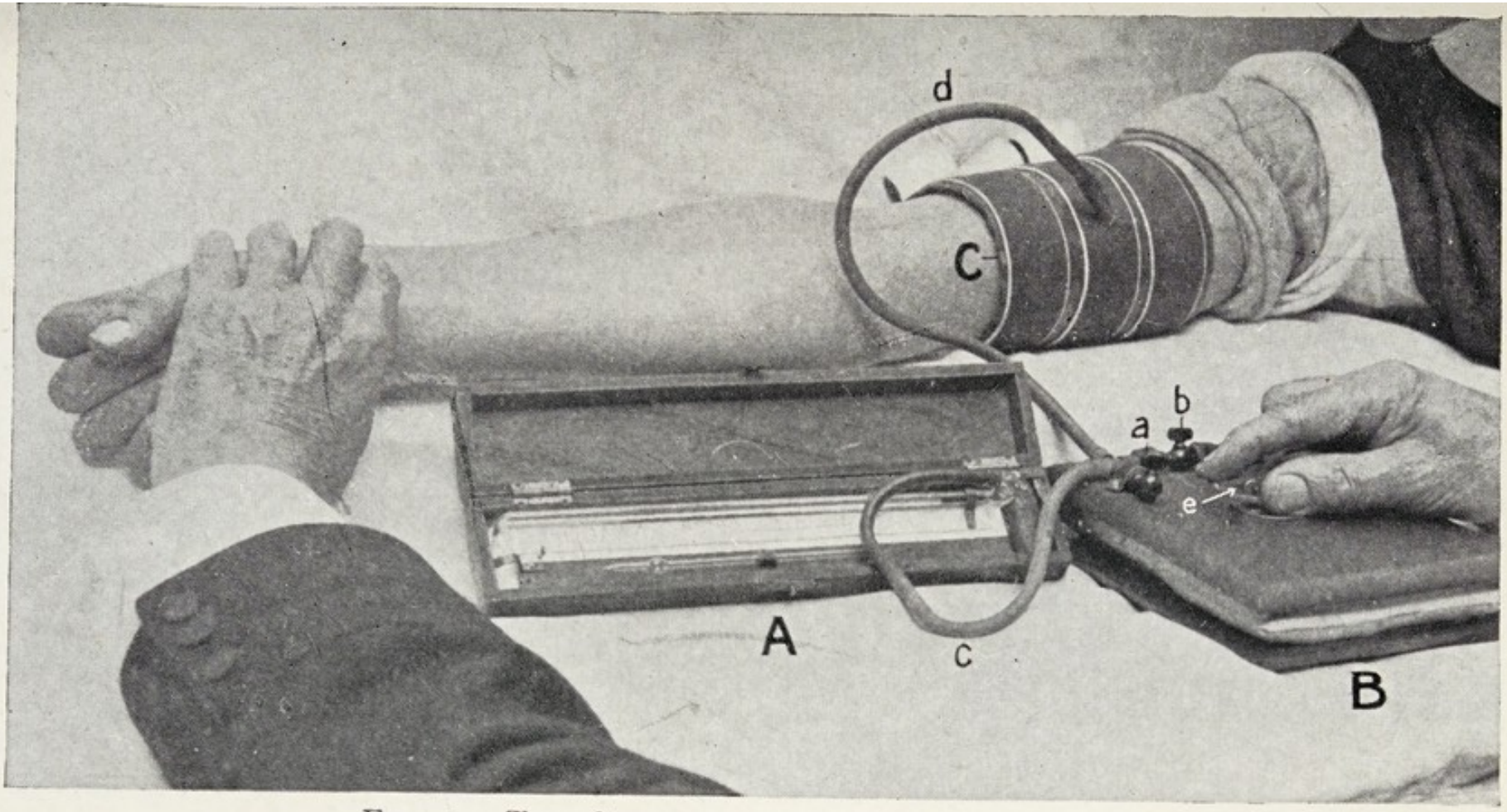

FIG. 3.—THE COMPRESSED-AIR HÆMOMANOMETER.

A, the recorder. B, the compressor: *a*, tap for occluding and releasing the air; *b*, tap for augmenting the oscillations of the meniscus; *c* and *d*, rubber tube connectors. C, the armlet.

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(3) *The much more satisfactory control of the index* by the screw than by the pump. Another defect of ball filling is the uncertainty of regulating the compression, and thus of definitely controlling the position of the index—a certain amount of training of the co-ordination of the muscles being necessary before the observer acquires the needful dexterity. On the other hand, adaptation of the compression by means of the screw is definite and easy, even to the tyro, who on this score has indeed nothing to learn by practice, for he can thus graduate the position of the index exactly as he pleases, without unintentionally overstepping or falling short of the point at which he wishes to adjust it.

(4) *The gradual rise of compression* in place of the intermittent jarring increment of it with every stroke of the pump. Needless oscillation and disintegration of the index are thus avoided.

(5) *Increased precision in reading the arterial pressures* (diastolic and systolic). The compressor method of filling the

armlet furnishes two grades of motion in the indicator, one or other of which can be used by the observer. When the air in the compressor, as well as that in the armlet and tubing, is in circuit—as in the ordinary use of the apparatus—the motion of the indicator is reduced to a minimum; the reading of the arterial pressures (and especially that of the systolic pressure) then becomes much more exact than when the indicator is oscillating freely. When, however, the volume of the air in circuit is reduced by closing the tap (Fig. 3 *b*), which shuts off the air in the compressor, an amplified motion of the index is obtained, which the observer can thus secure whenever he desires to do so; or, by *nearly* closing the tap, a medium increase of the oscillation may be obtained, while the air in the compressor is retained in circuit and the index responds to the turning of the screw.[1]

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[1] When using the mercurial manometer I prefer the compressor and armlet to the ball-filler and the ordinary armlet. The latter cannot, however, be used with the compressor, because its ratio of air-capacity is too great.

The objections are: (1) the lodgment of spirit on the bore after an observation, which lodgment slightly diminishes the volume of air within the recorder subjected to compression; and (2) disparity of temperature of the air inside and outside the bore.

The first objection is met by raising the recorder to the vertical position for two or three minutes, when all traces of spirit will drain away from the bore before another observation is made.

The second objection is met by establishing the continuity of the air inside and outside the bore before each observation, as presently to be described.

The observer should bear in mind that compressed-air manometers are easily affected by variations of the external temperature; he will therefore be on his guard, while making an observation, to avoid the play of a draught of cool air on the instrument (*e.g.* from a door or window), or the heating of it by the direct rays of the sun, or by a fire, or by the contact of the hand.

When an observation is to be made the observer

(1) adjusts the armlet comfortably and *closely*;

(2) raises the recorder to the vertical position, and withdraws the plug in the rubber tube attached to the nozzle.

(3) He then quite easily disperses into the lower bulb any portion of the index, which commonly lodges in some part of the bore—and especially in the lower portion of it—after disuse of the instrument, by adopting the following simple procedure: he takes the rubber tube near its attachment to the nozzle of the recorder between his *left* thumb and finger, and nips it so as to obliterate the bore; at the same time, with his *right* thumb and finger he takes hold of the tube close to his left hand, nips it firmly, and draws his right finger and thumb (still nipping) along the tube to its open end; and in doing so, exhausts the air from the tube. On then withdrawing his left thumb and finger (which all the time have been nipping the tube), *while he still*

keeps a firm grip of the end of the tube with his right hand, the spirit is quickly dislodged, and drains into the lower bulb. Should, however, any portion of the index still remain in the bore, it is readily withdrawn by a repetition of the process. The procedure can be further simplified by merely grasping the tube between the finger and thumb, and drawing the former *firmly* along the tube resting on the thumb, so as to exhaust the air.

(4) He restores the recorder to the horizontal position, when the indicator automatically stops at the zero point (Fig. 2). Should, however, the index go slightly above the zero line, a few drops of the index fluid or spirit will restore the automatic stopping at zero.[1]

(5) He expands the compressor by unturning the wheel, closing tap *a* (Fig. 3), and makes the connections with the armlet and the indicator.

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[1] When there is quite an excess of index fluid the meniscus stops slightly short of the zero line. But this defect can be easily rectified by abstracting a little of the fluid by a strip of blotting paper.

(6) Finally he compresses the air in circuit; and after the completion of the observation he may use the air over again by unturning the wheel of the compressor, or he may release the compression by undoing tap *a*.

The Reading of the Blood-pressures (Arterial and Venous).

As nervous perturbation vitiates trustworthy observation of the arterial pressure, the practitioner should wait until the patient has become free from apprehension and is quite accustomed to the medical examination of his case, before he attempts to measure the pressure; and this precaution is all the more necessary should the pulse be hurried. It is as well also not to surprise the patient by raising the air-pressure in the armlet before telling him what he is about to do.

The arterial pressure may be taken either in the sitting or recumbent position. In the sitting posture, which is more convenient and expeditious for clinical

work generally, the arm should rest comfortably, with all its muscles relaxed, on a table of convenient height, so as to bring the armlet on a level with the heart. In the recumbent position the arm should be extended on a table by the side of the bed, and on the level of the body.

The arm should be bared, though a shirt or other thin covering does not alter the reading of the pressure. The patient should rest for some little time after any physical exertion. The first pressure reading should not be accepted unless it is corroborated by the second or third reading. The last is the most trustworthy, as a rule; and it is best to take this quite casually, after the patient's attention has been directed to some matter of general rather than personal interest.

I.—*The Brachial Arterial Pressure.*

(A) **The Diastolic Pressure** is denoted by the point at which the maximum oscillations of the index are produced,

beyond which they are lessened by further air-compression. It has been observed that, after reducing the oscillations by increasing the compression, there may in exceptional cases appear a second development of maximum oscillation just below the reading of the systolic pressure. The observer should not in such cases accept this higher increment of pulsatile movement as indicating the diastolic pressure, but should in all cases carefully look out for the first diminution of the oscillations (disregarding any second development), and take the lowest maximum pulsation of the index as denoting the diastolic pressure.

The finger generally assists the eye in determining the diastolic reading; for, on placing it over the radial while the air-compression rises in the bag, the character of the pulse changes, approximating to that of aortic regurgitation, and when the diastolic pressure is exactly balanced by the compression, the pulsation felt in the artery and the oscillation of the index simultaneously become greatest, and on

further increasing the compression, even to a slight degree, the pulse-volume and the index oscillation rapidly diminish together. This tactile effect induced by balancing the minimum arterial pressure by compression is, I think, more pronounced in cases of *plus* diastolic pressure than in those of normal or *minus* pressure. Observers vary greatly in their ability to recognise the point of maximum oscillation; and those who cannot quite trust the eye alone for this purpose will find it useful to call in the assistance of the finger.

A further aid to the reading is the maximum subjective throb developed by balancing the diastolic pressure by the compression.

(B) **The Systolic Pressure** is read (Fig. 3) by the finger,[1] which is placed

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[1] In the first edition of this brochure I described a *visual* method of reading the systolic pressure, as well as the *tactile* one, which is usually adopted. The visual method consisted in (1) strapping the digital bag around the wrist in connection with the armlet *in situ*; (2) nipping the rubber tube connecting the two bags after successive additions of air, until the index, deriving its pulsation from the radial artery, becomes quite motionless. I always

on the radial artery (the armlet *C* encircling the arm or the upper part of the forearm), and noting the degree of compression required to extinguish totally the pulsation. The reading may be made either by gradually pressing the index up until the point of extinction is reached, or by employing at once an excess of pressure, which is gradually reduced by slowly liberating the air-valve until the beat reappears. The latter method gives a slightly lower reading; and most observers agree that it should be accepted as the standard method.

II.—*The Forearm Arterial Pressure.*

The Arm is anatomically adapted to Hæmomanometric Observation.—The arm, from its cylindrical form, even compressibility, and its single bone and artery, is anatomically well adapted to hæmomanometric observation; but these

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found that the readings thus obtained were higher (5 to 10 mm.) than those afforded by the finger placed over the artery; and the greater sensitiveness of this method, thus shown, has been verified by others.—See *Berl. clinich. Wochenschrift*, June 1907.

structural advantages are unfortunately counterbalanced by practical drawbacks, arising from the complete obstruction of a large section of the circulation and the general vaso-motor effect of the constriction throughout the arterial system (*see* p. 36).

Hæmomanometric Observation on the Forearm. — Hence the practical importance of reducing these physiological disadvantages of this mode of observing the arterial pressure without impairing its accuracy. I have therefore frequently made observations on the forearm, which are much less objected to by patients than those on the arm, and the readings so far have not differed in the two areas except in arterio-sclerosis (*see* p. 112).

III.—*The Phalangeal, or Distal Arterial Pressure.*

Apply the small bag (2·5 × 9 cm.) around the third phalanx of the middle or ring finger or the second phalanx of the thumb, and buckle.

The diastolic distal pressure is read, as usual, from the maximum excursion. In reading it, the observer requires to be specially careful to recognise the *lowest* maximum, which is followed by diminished oscillation; for he may easily overlook the latter, and be misled by the development of a *higher* maximum excursion which does not denote the diastolic pressure.

The systolic distal pressure may be taken from the first and second phalanges in the following manner[1]: (1) After adjusting the bag, the finger is rendered bloodless by squeezing a stout rubber ring (the compressor) over it as far as the lower edge of the bag. (2) The pressure is raised to 150 millimetres of mercury. (3) The compressor is removed, when the finger should remain bloodless. (4) The pressure is

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[1] The method here described is that of Gärtner, the only modification being the substitution of the phalangeal bag for Gärtner's pneumatic rubber ring, because it furnishes more uniform results and is adaptable to fingers of all sizes.

very gradually released until the finger-tip (or nail-bed) suddenly flushes, when the height of the indicator is read. At the same time a slight throb is felt, which increases to a maximal degree when the pressure falls to the diastolic point, and then decreases. Several consecutive observations on the same finger furnish higher readings from the development of a vaso-paresis, caused by repeated compression.

But I think it is highly probable that the phalangeal systolic pressure may frequently be read in a simpler and more expeditious way. I have observed when the digital circulation is sufficiently active (the finger being quite warm) that the development of the lowest maximum oscillation, indicative of the diastolic pressure, is followed by diminished oscillations, which are succeeded by a further development of maximum excursions, the position of which on the scale corresponds with the reading of the systolic pressure *à la* Gärtner. My impression is that this second development of maximum

oscillation is caused by the end (or systolic) pressure of the unoccluded and probably dilated portion of the digital artery pulsating on the upper margin of the bag; and this explanation of the appearance of the higher maximum oscillation is supported by the fact that it only takes place when the digital vessels are somewhat relaxed or dilated and are not contracted.

The principal objection to digital observation of the arterial pressure is the great liability of the fingers to vaso-motor disturbances from variations of temperature and from repeated compression in a prolonged *séance.* Should the fingers be in the least cold no response is obtainable until they are warmed, *e.g.* by placing the hand on a hot-water bag, etc., when of course the readings of arterial pressure are somewhat raised by the vaso-dilation necessary to secure the required response.

IV.—*The Venous and Capillary Pressures.*

The Venous Pressure may be determined by ascertaining the Pressure required to permit Refilling of a Collapsed Vein.—Select a vein free from branches on the dorsum of the hand, the wrist, or the forearm. It should possess an efficient valve—a fact proved by placing a finger on its distal end and emptying the vein with another finger drawn along it, when the piece of vein as far as the valve will remain collapsed. Place the phalangeal pad across the distal end of the vein, and adjust the strap around the hand, wrist, or forearm without compression, and buckle. Inflate the bag to the pressure of 40 mm. and empty the vein. Then *very gradually* release the pressure, when at a certain point in the fall, which denotes the venous pressure, the vein suddenly refills.[1]

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[1] This mode of reading the venous pressure can also be made by the hæmodynamometer (p. 46).

The Venous Pressure may also be determined by Gravity.—I have observed that the venous pressure may be accurately measured without the aid of a blood-pressure instrument; a foot-rule or measuring-tape being all that is necessary. If the veins on the dorsum of the hand are sufficiently visible, it is found that when the hand is held in the vertical position, with the fingers extended, and is very gradually raised, the veins, at a certain height above the level of the apex of the heart, are seen to collapse quite suddenly. The completion of the observation is definite—for the transition from a partial to a complete collapse takes place within the space of half an inch. It is observed that the veins nearest the fingers collapse somewhat sooner than those nearest the wrist; but the difference is so slight that I prefer to take the collapse of all the veins of the dorsum as the indication for measurement.

I conclude that at the moment of collapse of the veins the blood-pressure within them is practically nil—being

balanced, as it were, by the force of gravity; and that we may express this point of annulling the hydrostatic rise of the blood in mm. Hg. pressure. This may be done by making a very simple calculation. If we take the average specific gravity of the blood as 1·060, and that of mercury as 13·570, the 25·5 mm. contained in one inch will represent 1·985 mm. Hg. (or approximately 2 mm. Hg.). Therefore, if we multiply by two the number of inches above the level of the apex of the heart at which the veins collapse, we ascertain in mm. Hg the venous pressure.

This simple method of measuring the venous pressure has afforded me more uniform results than other methods; and, as it enables one to discriminate between differences of 1 mm., it is definite and delicate. In applying it, it is important to see that the pressure is not artificially raised. This may occur through nervous perturbation, or obstruction of the venous flow by tight clothing, or even by the observer placing his hand round the forearm.

The lowering effect of cold on the venous pressure should also be kept in mind.

The Capillary Pressure (subungual). —I am disposed to think that this gravity method yields an approximate reading of the capillary pressure; for, as a rule, just when the vein collapses there is observed a slight blanching of the nail—especially its central portion—which is made somewhat more apparent when the nails of the opposite hand, with the fingers flexed downwards—so as to retain their blood in the elevated position—are compared with the uplifted nails side by side. The measurement from the apex of the heart to the nails gives, as a rule, a reading for the capillary pressure 8 or 10 mm. Hg. higher than that of the venous pressure—*e.g.* capillary pressure, 25–30; venous pressure, 15–20 mm. Hg.

CHAPTER II.

BLOOD-PRESSURE MEASUREMENT (*continued*): READY CLINICAL APPARATUS AND METHODS.

I AM persuaded that if blood-pressure measurement is to be adopted *quite generally* as a clinical method, it must be made thoroughly practical; in a word, it must be simple, compact, easy, and quick. But mere portability and simplicity in execution should not be allowed to override or to spoil the essentially important quality of fundamental accuracy.

READY METHODS OF MEASURING THE ARTERIAL PRESSURE.

Objections to the Armlet Method.—The armlet method, described in the last chapter, is by no means ideal in practice, and, like all other instrumental aids to observation, it has its defects—for no instrument is, or ever will be, free

from some fault or other. For example, errors in reading the arterial pressure may arise from faulty adjustment of the armlet; and these may be considerable (*e.g.* 20 or more mm.) when the armlet is adapted loosely. But this is a defect in technique, not in the method itself; the observer, therefore, merely requires to be reminded of it as a precaution. But as a method it is open to three objections: namely, (1) That it profoundly disturbs the circulation of the limb—arresting the venous flow and creating the greatest possible peripheral resistance in a moderately large fraction of the body; (2) that it produces a widespread effect on the vaso-motor nervous system; and (3) that in arterio-sclerosis it may afford too high a systolic reading, because the arterial wall does not collapse evenly (*see* p. 113). The objections 1 and 2 (local arrest of the circulation and the reflected effect of the discomfort produced by the compression) are said to raise the general arterial blood-pressure 5 or 10 mm. Hg, according to the duration

of the compression and the sensitiveness of the subject. My own impression is that these objections are of little or no practical importance; for by another method, to be presently mentioned, which is free from compression or discomfort, identical readings are obtained *in all cases in which there is no suspicion of arterio-sclerosis.* But the third objection has some weight (*see* pp. 111–123). From the patient's point of view this method is also sometimes objectionable; for sensitive nervous subjects now and then resent the circular compression of the arm—and more particularly the higher compression of over 200 mm. Hg.—and may sometimes be so disconcerted by it as to refuse to submit again to the ordeal. This objection to brachial hæmomanometry, though comparatively insignificant and practically non-existent in male patients generally, is a substantial one with some women, especially of the upper classes. The practical convenience of having an alternative method, which is in no way objectionable, is, then, very

considerable. But besides this, the busy practitioner requires for *routine* blood-pressure observation, and for the purpose of picking out cases for more particular study, a method which is time-saving, unobtrusive, easy to apply, and, moreover, free from discomfort to the patient and from the drawback of inducing local or general disturbance of the circulation, and also sufficiently accurate to indicate without doubt any important alteration in the arterial pressure and to ensure the avoidance of error.

Ready Clinical Methods. — Many such methods have been suggested from time to time during the past seventy-five years or so; and all have depended on deriving the reading of the arterial pressure from individual arteries.

Methods founded on the Actual Closing of an Artery.—The most important survivals of these methods are the instruments of V. Basch [1] and Potain. [2]

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[1] "Der Sphygmomanometer und seine Verwirthung in der Praxis." *Berl. klin. Wochenschr.*, 1887, vol. xxiv.

[2] "La Pression artérielle de l'Homme à l'état normal et pathologique." Paris, 1902.

The method of both is identical; it is founded on the application of an air-bulb to the radial artery—the bulb being in connection with a small aneroid, which indicates in cm. of mercury the degree of compression required to *close* the artery, *as denoted by the cessation of pulsation* on the distal side of the bulb. This method is ideally practical. But, unfortunately, one's trust in it is considerably impaired by finding, as I have frequently done, that it affords *marked differences* (varying from 20 to 50 mm. Hg.) between the readings of the arterial pressure made on the same hydrostatic level, and at the same moment in the normal radial, ulnar, temporal, and other accessible arteries—the corresponding arteries likewise frequently affording quite different readings. Similar evidence has also been furnished by V. Basch[1] and Potain[1] themselves. These discrepant readings, which contradict our knowledge of the equality of the arterial pressure in all the accessible

[1] *Op. cit.*

arteries (*see* p. 56), therefore reveal a fundamental defect in the method of reading the systolic arterial pressure by small air cushions or bulbs pressed over the individual artery (*e.g.* radial) so as to *close* it. They are not caused by the pad itself—whether its wall is too thick, or its capacity too small; for I have experimented with pads having the thinnest of walls, and varying in diameter from three-quarters to over two inches, with exactly the same results. Nor are they caused by differences of resistance in the arterial wall—for observers who have investigated this point are agreed that the pressure required to close an empty normal artery is practically nominal (*e.g.* from 1 to 2 mm. Hg.—V. Basch).

The divergent readings are obviously due to (1) varying degrees of resistance in the tissues overlying the vessels, and (2) the different anatomical relation of each artery to the underlying resisting surface or bed on which it is compressed. When the artery is *closed* by the pressure brought to bear on the air-bulb, these

varying degrees of resistance, as well as the resistance of the blood-pressure, are estimated together, and we read this composite resistance as arterial pressure.

Therefore the principle of ascertaining the arterial pressure from an individual artery by *closing* it by compression is fundamentally vitiated by variable tissue-resistance. In practice I have observed that the readings thus obtained from the radial agree with those furnished by the armlet method in the majority of cases; but every now and then I have encountered discrepancies, and in one or two cases these have been as large as 30 or 40 mm. Hg. These results show that, though the bulb method may give comparable results in the general run of cases, it is untrustworthy, for any case may prove to be an exception. On the other hand, the armlet method does not yield discrepant readings from normal individual arteries: for example, the ulnar affords the same systolic reading as the radial—a fact which proves that the method is fundamentally accurate.

A Method founded on the Balancing of the Pulse-pressure Wave.—Can the arterial pressure, from its minimum to its maximum, be determined accurately in a single artery without actually closing it? If this be possible, then the vitiating influence of varying tissue resistance may either be eliminated, or at any rate be greatly modified.

Writers on sphygmometry have always grouped together all the instruments which derive their readings of the arterial pressure from a single artery—whether they actually close it or not—as if they followed the same principle. For example, my hæmodynamometer (Fig. 4), which was designed to balance the blood-pressure in an artery and not to close it, has been so classified. It is true, when I introduced the instrument some years ago, I did not realise its full capacity, which has been only recently disclosed by comparing the results it furnishes with those obtained by the armlet method. I brought forward the instrument as an indicator of the maximum oscillation,

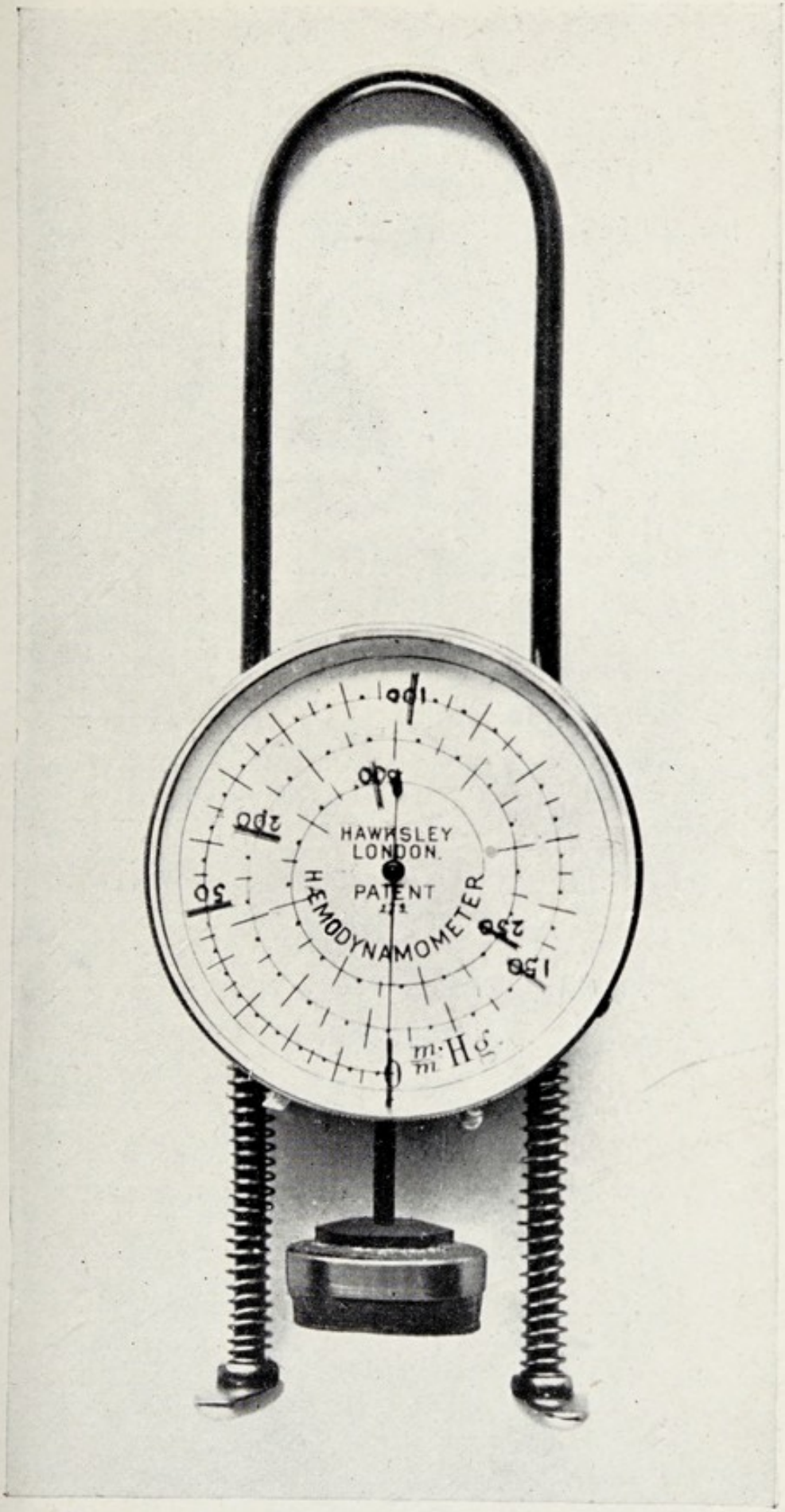

Fig. 4.—The Hæmodynamometer. ($\frac{3}{4}$-size.)

To face p. 42.

which is now regarded as the criterion of the diastolic pressure. Recent observation has, however, shown that the instrument not only indicates this pressure, but furnishes likewise the systolic pressure.

For many years I have been conversant with the fact that the instrument provides two maximum oscillations of the indicator, one lower, which denotes the diastolic pressure, and the other higher on the scale—the two maximum oscillations being separated by somewhat diminished excursions of the indicator. The full significance of the higher maximum oscillation, which has caused some uncertainty in past observation, has now been made apparent by comparing it with the readings of the systolic arterial pressure furnished by the armlet method. It has been found to denote the systolic pressure, just as the lower maximum oscillation indicates the diastolic pressure. Therefore each of these divergent points in the cycle of the pulse pressure produces in the hæmodynamometer its own visible

indication in the form of a maximum oscillation of the indicator; and the area of amplest oscillation thus bounded is a measure of the height of the pulse-pressure wave, which is of variable length in different cases (*see* pp. 85–6).

Between the two maximal excursions of the indicator the oscillations diminish quite appreciably in the majority of cases. But in others, as when the two maximal oscillations are closer together (*e.g.* 10 or 15 mm.), this diminution is less apparent and requires rather close observation for its detection; but should they seem to be nearly uniform, they still mark out the limits of the pulse-pressure wave—for the lower maximum excursion is easily defined and the upper one is clearly shown by the diminution, which is not succeeded by a group of increased oscillations higher on the scale. The primary diminution of the oscillations and their subsequent development to the second maximum point, beyond which they finally decline, doubtless depend on several causes, such as: (1) the disc-like

shape of the pad; (2) the fluid in the pad being fixed in quantity and small in volume; and (3) the virtual closing of the artery, which causes a distension and increased pulsation of the vessel on the proximal side of the pad—hence the second maximum oscillation is properly developed only when the pad projects well (about a ¼-inch) beyond the metal ring, but disappears (the lower maximum oscillation alone remaining) when the side of the projecting portion of pad is quite inclosed within a rigid ring—this excluding lateral pulsation on the pad.[1] But whatever the explanation may be as to how the two maximum oscillations are produced—and I am satisfied that they

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[1] The pad is made to project fully ¼-inch beyond the metal ring which surrounds it, and is divested of its back spring (as thus modified four years ago); and the instrument is mounted on a slide, which greatly facilitates its use (Fig. 4). Set up in this way the sensitiveness and accuracy of the instrument are increased. The only important objection to this portable little instrument is the advisability of having the standardisation tested by the instrument-maker periodically (say once in 12 months or so). When the armlet method is also used, the need for restandardisation of the hæmodynamometer is shown when a disparity is apparent between the readings furnished in normal subjects by the two methods (*see* p. 49).

solely depend on mechanical conditions—their existence is apparent enough.

The hæmodynamometer yields identical readings of the arterial pressure (diastolic and systolic) in all the accessible arteries (radial, ulnar, superficial temporal, posterior tibial, dorsalis pedis, superficial palmar arch)—a result which is in accord with our physiological knowledge, and demonstrates that this mode of measuring the arterial pressure in individual arteries by merely *balancing* the pulse-pressure wave through a fluid medium of limited volume is not vitiated by variations in tissue resistance, which is, as I have shown, the fundamental defect of the method of compression, which secures continuous *closure* of the artery.

How to observe the Arterial Pressure by the Hæmodynamometer.—The right arm on its ulnar side is placed on a table (this being on a level with the ensiform cartilage in the sitting posture and with the back in the recumbent position), and the wrist is kept bent to the angle of 45° by the observer's left

hand holding back that of the patient.[1] The feet of the slide are so placed as to elude the track of the artery, and the pad of the instrument (Fig. 4) is placed over the vessel *at right angles to its bed.* Pressure is then made by gently and gradually pushing forward the body of the instrument by the thumb of the observer's right hand holding the slide.[2] The indicator will then rise on the dial and show the degree of pressure brought to bear on the pad and the vessel, and will begin to pulsate when measuring 50, 60, 70, or 80 mm. or so. The pulsations gradually increase in size as the indicator is made to rise farther on the scale, until the lower maximum point is reached (diastolic reading), beyond which they gradually diminish, and again increase under a higher pressure until a second maximum oscillation is produced (the systolic read-

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[1] The observer should never neglect to follow this important direction, as it is essential to accurate observation that the artery should be rendered somewhat taut by thus bending the wrist.

[2] It is most important to preserve the line of pressure exactly at right angles to the bed of the artery

ing), after which they finally decrease. The readings are made at the midway point of the maximum excursions (diastolic and systolic). When the oscillations of the indicator are comparatively small in range, it is much easier to read accurately the points when they attain their maximum development than when they are large. The radial very frequently furnishes a response which is too voluminous ; then the observer should select another superficial artery which gives a smaller reaction, such as the ulnar artery or superficialis volæ ; and for this purpose the dorsal extremity of the radial just before dipping down to form the palmar arch is most suitable for blood-pressure observation with the hæmodynamometer. In all cases in which the pressure has been derived from the radial the observation should be corroborated, if possible, on the ulnar artery, which should afford the same reading.

Those who prefer to read the arterial pressure by taking the systolic pressure

only will find the hæmodynamometer a convenient instrument for quick and accurate observation—for they have merely to develop the highest maximum oscillation of the indicator.

The readings of the hæmodynamometer have been compared with those of the armlet method in some hundreds of cases, with the following results. They have been found to be identical in all, except in several cases in which sclerosis of the brachial artery was demonstrable. In these the armlet method invariably afforded higher readings of the *systolic* pressure—the excess varying from 10 to 50 and even in exceptional cases to 100 mm. Hg. (*see* p. 115); while as a rule it yielded *diastolic* readings which were either practically identical with or were not very much higher than those of the hæmodynamometer. It was therefore the systolic pressure of the armlet which was more particularly raised in the sclerotic cases.

The practical advantages derived from the conjoint use of these two modes of

measuring the arterial pressure have been considerable. It has shown, (1) that the armlet method is apt to over-estimate—and sometimes to considerably overestimate—the systolic pressure and to exaggerate the height of the pulse-pressure wave in subjects suffering from arterio-sclerosis (*see* p. 113); (2) that the hæmodynamometer is useful in indicating when this overstepping of the systolic pressure reading takes place and the existence of arterio-sclerosis may be inferred;[1] and (3) that the use of the combined methods therefore yields more comprehensive results than either singly—giving, in fact, a measure of the sclerosis as well as that of the arterial pressure.

The two maximum oscillations (diastolic and systolic) may also be demonstrated by the compressed air

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[1] The liability of the armlet method to yield in arterio-sclerosis too high readings of the systolic pressure as defined by the finger is still greater in the graphic records of the pressure—for these not infrequently show ample oscillations, 20, 30, or even 40 mm. above the point at which the pulse ceases to be felt.

manometer.—This fact has already been referred to in connection with the application of the digital pad (*see* pp. 29, 30). It may likewise be shown by the use of a circular rubber bulb, fixed in a metal cover and connected with the manometer by a rubber tube. For example, it is apparent when the bulb is applied to the superficial palmar arch in the following simple manner: (1) Place the flat end of the bulb on the table; (2) bring the *left*[1] palm (just above its centre) over the bulb; and (3) press downwards the back of the patient's hand the required degrees; when the index will rise and afford first the diastolic maximum oscillation, and next, after some diminished excursions, the systolic maximum oscillation.[2] The bulb, applied to the radial and ulnar arteries, does not however always yield both maximum oscillations—for the systolic one often fails

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[1] The left palm affords a better response than the right, because its epidermis is less thickened by use.

[2] Though this method is generally successful when the hand is warm, it may fail when it is cold or when the palm is horny.

to appear. The best way to apply the bulb to the arteries of the wrist is to have it mounted on a slide with springs (Fig. 5), and to place it *in situ*, when the forearm of the patient is placed on the table, with the palm upturned, the wrist being well extended and resting on a book. The feet of the slide are planted, one on the radius and the other on the flexor tendons ; and pressure, which can be graduated with great nicety, when applied to the brass cover, raises the index, which is under perfect control —the observer stopping it at any point, or lowering it and again raising it, as he may wish. The observation should be made deliberately—the index being raised or let down somewhat slowly. The diastolic pressure is read, as usual, by the lowest maximum oscillation. The systolic pressure may be taken when the distinct throb of the index ceases, or is reduced to a mere quiver ; or when the index, pushed up well beyond the pulsating area, begins to throb again on being gradually lowered.

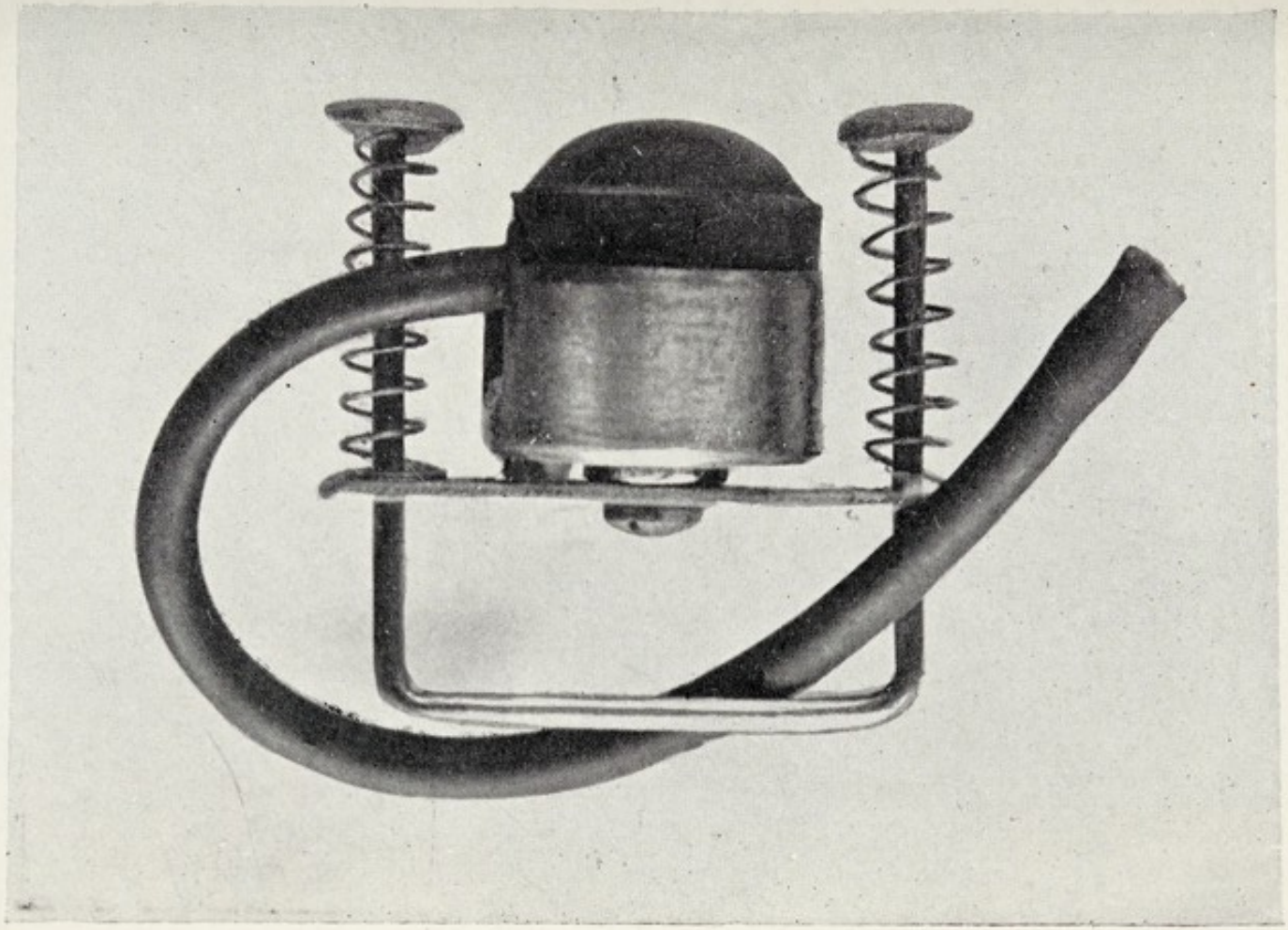

Fig. 5.—The Manometer-Bulb Mounted.

To face p. 52.

The systolic reading thus made is generally a few millimeters (5 or so) too high ; but it has the advantage of having a more definite finish than the reading obtained from the area in which the oscillations rapidly diminish.

This mode of taking the arterial pressure by balancing the pulse-pressure wave is quick (not requiring more than half a minute), and the readings it affords have so far agreed with those furnished by the armlet method in normal subjects ; and in arterio-sclerosis they have corresponded with those of the hæmodynamometer.

Should the observer care to read the systolic pressure by closing the artery with the bulb, he will perhaps find that the application of it by the aid of the slide will afford more uniform results than the digital use of the detached bulb *à la* Potain ; at the same time he should be reminded that, according to my observation, the data afforded by this method are not always to be trusted (*see* p. 41).

CHAPTER III.

THE PHYSIOLOGY OF BLOOD-PRESSURE.

The Blood-Pressure in Different Parts of the Arterial System (the Arm, Forearm, and Phalanges).

As our knowledge of the blood-pressure in the different districts of the arterial system in man is not so complete as one could wish, I will, as a contribution to it, adduce a few data showing the relation between the different pressures in the arm and forearm (upper and lower), and in the finger.

Absence of Physiological Stimulation of the Circulation necessary for the Comparative Observation of the Different Pressures.—But for the purpose of this comparison it is necessary to obtain the condition of physiological rest in the circulation—as complete rest as

possible; for, as will be subsequently shown, a state of circulatory activity, such as that which follows exercise or the ingestion of food, disturbs the inter-relation of the different pressures. I have observed in every normal subject that there are periods during the waking hours—recurring with remarkable persistency, in which we may say that there is a fair degree of uniformity in the activities of the circulation—when the arterial pressure becomes practically constant. It undoubtedly varies within certain limits in different individuals, and it is true these limits are not so close as those of the temperature of the body; but for each individual of normal tone it is fairly constant in the different areas. These periods recur, as a rule, quite regularly during the hour before each meal when the body is in a state of rest.

The Brachio-radial and Phalangeal Pressures.—I have not found any appreciable difference between the arterial pressure (systolic and diastolic) taken from the arm and that taken from the

forearm (upper and lower). If the pressure is actually less at the wrist than it is in the arm, the reduction is not, as a rule, detectable with certainty. In the arm the first appreciable fall in the pressure is found at the first phalanx, where it may be found to range from 10 to 25 mm. of mercury. The reduction increases in the second and third phalanges; and in the latter it may even fall to 50 mm. of mercury—the pressure then being, roughly, a little less than half that of the brachio-radial region.

Rapid Fall in the Finest Divisions of the Arterial System.—These data indicate that the fall in blood-pressure which takes place between arteries of intermediate size and the capillaries becomes quite appreciable only in the somewhat finer divisions of the arterial system, beyond which it is extremely rapid.[1] It

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[1] I have elsewhere shown that the arterial pressure obtained from the accessible individual arteries of the hand and elsewhere by the hæmodynamometer is not appreciably different (see *Blood and Blood-pressure*, 1901); and this conclusion is confirmed by observations made with the compressed-air manometer.

is observed that the systolic pressure diminishes somewhat more rapidly than the diastolic—in a word, the two pressures approximate more and more as the terminal arteries and arterioles are approached. The low reading obtained from the last phalanx suggests that in this area we are probably approaching the arteriolar or precapillary pressure. Pressure readings from this distal part of the circulation are not, however, always equally available—as, for example, when the fingers are cold the pulsations are abolished or become greatly enfeebled. Dr. A. Waller, applying his digital sphygmograph to the finger-nail, with an air-tight connection with a mercurial manometer, obtained an approximate pressure of only 20 mm. of mercury, and found that the pulsation was abolished by cold.[1]

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[1] *Brit. Med. Journ.*, vol. ii., 1900, p. 840.

The Relation of the Brachio-radial Pressure to the Central-arterial Pressure.

In the normal state of the circulation it may be inferred that the brachio-radial pressure bears a certain definite and fairly uniform relation to the pressure in the central arteries; and this is indeed the accepted basis of our clinical reliance on the guidance of the brachio-radial pressure. But may it not follow, from our knowledge of the physics of the circulation, that a reduction of the calibre of the arteries of a limb will disturb this relation—the brachio-radial pressure diminishing in proportion to the contraction of the brachial arteries, with a consequent rise in the central pressure? This anticipation is not, however, verified by observation; for the arteriometer and the manometer have clearly shown that with reduction of the radial calibre the brachio-radial pressure generally rises (*see* pp. 62, 69, 160, 238). Whether or not there is at the same time a dispro-

portionate rise in the central pressure it is impossible to say. That there should be a fall of the arterial pressure in a limb with moderate or even decided contraction of its arteries, does not, however, seem probable, when we bear in mind, as we have just seen (p. 56), that we do not encounter a decided diminution of the *normal* pressure until we reach the arteries of very small calibre—such as the digital. To ensure a fall in the brachio-radial pressure, we must suppose that the arteries of the limb are capable of being diminished to such small dimensions as those of the distal arteries; and that is not probable. We may therefore conclude that our knowledge of the normal arterial pressure (proximal and distal) accords with the results of clinical observation, that the reading of the brachio-radial arterial pressure is a trustworthy criterion of the central pressure and of the pressure throughout the proximal arterial system.

The Physiological Variations of the Blood-pressure in the Intermediate and Distal Parts of the Arterial System.

The Pressure Variations produced by Stimulation of the Circulation.—The common feature of all forms of stimulation of the circulation (such as that induced by digestion, exercise, warmth, nervous excitation) is a rise in the blood-pressure in the distal area—the last phalanx of the finger or thumb. The rise is generally large relatively to the constant pressure. In normal conditions it is associated with either a fall or a *moderate* rise in the brachio-radial pressure.

The Distal Rise with Fall in the Intermediate Arterial Pressure is due to Vaso-dilatation.—It is quite conceivable how this event in the distal vessels is brought about when the blood-pressure falls in the intermediate arteries, for vaso-dilatation, unfolding the distal circulation, will cause the pressure to

rise in the peripheral arteries, arterioles, capillaries, and veins. This increment of distal pressure, produced by vaso-dilatation, will be all the more pronounced and sustained should the maximum arterial pressure, due to the ventricular contraction, be well maintained.

The Distal Rise when Part of a General Rise in the Arterial Pressure is due to Cardiac Stimulation, which may overbalance the Lowering Effect of Vaso-constriction.—But when the pressure in the peripheral vessels participates in a general rise of arterial blood-pressure, some other factor will come into play if vaso-contraction takes place, for that will tend to lower the pressure in the distal vessels. Observation points to cardiac stimulation as that factor; for I have always observed that when the pressure rises throughout the arterial system (in the digital as well as in the brachio-radial area) the systolic pressure rises still more. It is therefore obvious that the lowering effect of *moderate* or *physiological* vaso-contraction

on the distal blood-pressure is more than compensated by augmented ventricular contraction. The two types of normal peripheral increment of blood-pressure —viz. from (1) vaso-dilatation only, and (2) moderate vaso-contraction *plus* cardiac stimulation—have been followed by the arteriometer (*see* p. 238), which has afforded evidence of radial dilatation in the former case and contraction in the latter.

Is there a Vaso-cardiac Reflex?—The relatively increased rise of the systolic over the diastolic pressure reading when vaso-contraction comes into play, suggests that there is a vaso-cardiac reflex; the ventricular action being stimulated, with probably a more effective and increased output, by vaso-motor contraction, and becoming less energetic as the peripheral vessels dilate. A strictly nervous reflex may not, however, be necessary to produce these changes in ventricular activity; for may they not be induced by variations in the aortic and intra-ventricular pressures which may directly stimulate the ventricle? How-

ever produced—whether by way of reflex or by fluid conduction—they show us the physiological method by which the activity of the ventricle is influenced by states of the peripheral vessels—the method, indeed, which we follow in our therapeutic efforts to alter and control the arterial pressure.

The Pressure Variations produced by Digestion.—There is much discrepancy apparent in the results obtained by different observers as to the effects of digestion upon the systemic blood-pressure, some indicating a constant fall, others a constant rise, and still others showing an absence of regular variation. This apparent want of consistency in the results of observation certainly does not afford a promising outlook for the discovery of a constant alteration in the blood-pressure induced by digestion. My observations, founded on a close study of this subject extending over many years, however, dispose me to think that these apparently discordant conclusions are susceptible of reconciliation.

Observation has shown that the ingestion of food invariably raises the arterial pressure in the distal area (last phalanx of finger or thumb). The rise begins, as a rule, within five or ten minutes after the inception of food, and attains its maximum development of from 15 to 20 mm. of mercury in an hour; then it declines, and in the course of from one and a half to two and a half hours it gradually settles down to its minimum level, until the next meal, or until exercise is taken. It has been observed that when the distal pressure is high before a meal (*e.g.* $\frac{80—90\ \text{S}}{60—70\ \text{D}}$ mm. Hg.), the digestive increment of that pressure is less than when it is lower at that time (*e.g.* $\frac{65—70\ \text{S}}{45—50\ \text{D}}$ mm. Hg.).

The **hypotonic** effect of digestion on the arterial system (indicated by a fall in the brachio-radial and a rise in the phalangeal or distal pressure) is much more common than the hypertonic effect, and is quite usual in the earlier rather

than in the latter half of life. In all cases it supervenes during sleep, and as a rule during recumbency after meals. It is, however, not infrequently met with even in the erect or sitting posture in subjects who frequently present the hypertonic digestive effect. The brachio-radial systolic pressure may likewise fall along with the diastolic, or it may remain stationary, or may slightly rise. It is obvious that in this condition of the circulation the arterio-cardiac reflex (*see* p. 62) is in abeyance, and is indeed unnecessary for securing an adequately increased supply of blood to the capillaries.

The **hypertonic** effect of digestion on the arteries, which secures a uniform rise of blood-pressure throughout the whole arterial system, is secured by vaso-contraction, which incites the ventricle to increased action. Hence after meals the rise in the systolic pressure somewhat exceeds that of the diastolic, and the pulse-rate increases, and this rise in the contractile energy and in the output of the ventricle suffices to overcome the

augmented tone of the arteries and to raise the distal pressure. This form of the digestive stimulation of the circulation is more frequently met with in the latter half of life.

It is therefore apparent that the primary aim of both these forms of the digestive disturbance of the circulation is to raise the capillary blood-pressure, and that this end is secured either by merely relaxing the arterial and arteriolar muscular tissue or by increasing the activity of the cardiac muscle.

The Pressure Variations of Exercise.—The following is an epitome of the results of my observations:

(1) *During* all forms of exercise the arterial pressures (minimum and maximum) are raised throughout the whole systemic area of the circulation; and it seems probable that the blood required to produce and sustain this rise of blood-pressure is derived from the splanchnic area.[1] The rise extends to the terminal portion of the arterial system; and during

[1] *Blood and Blood-pressure*, pp. 162–4.

the continuance of the exercise it tends to give way in the intermediate and central, while it continues high in the distal, portions of the circulation.

(2) *Immediately on ceasing* the exercise the arterial pressure may be found to be either supernormal, normal, or even subnormal; the duration and degree of the exercise, the staying power of the subject (his training, etc.), and the temperature of the air all being important factors in producing this varying result. If the arterial pressure is raised, it soon begins to fall, the diastolic generally falling somewhat before the systolic; and first of all the fall is apparent in the brachio-radial area, while it still remains high in the third phalanx, where it will continue to be raised for some time, if the subject is heated by the exercise, or the atmosphere is warm. Eventually the distal pressure settles down, and the brachio-radial pressure is restored to normal. These variations of the arterial pressure after moderate exercise generally require for their subsidence the lapse of half an

hour. As I have observed a secondary rise of pressure during rest following this primary restoration, it is better to wait an hour after active or prolonged exercise is over before making a clinical observation. These pressure variations of exercise are more pronounced and of longer duration in warm than in cold weather.

The pressure variations of temperature are very considerable. These are specially apparent when the body generally is exposed to wide differences of temperature, as in hot or cold weather, baths, etc. Warmth approximates the normal co-relation between the intermediate and distal arterial pressures (these becoming even uniform), while cold exerts the contrary influence.

A considerable range of variation of the distal-arterial pressure therefore characterises the normal state of the peripheral arteries, their response to physiological stimuli being large and their pressure, when free from the effect of such stimulation, having a certain margin of variability consistent with good health.

The Vaso-motor Control of the Arterial Pressure.

Tonicity is an Important Factor in determining the Arterial Pressure.—While the body is in a state of rest the normal arterial pressure is mainly controlled by tonicity of the arteries, and especially that of the arterioles, whether splanchnic or systemic. Tonicity exerts not only a direct effect on the pressure, but probably also an indirect one reflected through the cardiac centre (*see* p. 62). When tone diminishes, the volume of the blood increases, first in the peripheral vessels and then in the veins (splanchnic and systemic), and the arterial pressure falls. When, on the other hand, tonicity increases, blood is withdrawn from the veins (and especially the splanchnic veins), and the arterial pressure rises.

Storage of Blood in the Veins is a Provision for Physiological Work and Rest.—The large calibre of the veins (systemic and abdominal), far in

excess of that required for the transit of the average volume of blood circulating through them, serves an important physiological purpose—namely, to provide for the temporary storage of blood. During exercise the systemic veins become loaded with blood, and their reserve capacity enables them temporarily to retain such portions of the blood as the heart may not for the moment be able to discharge into the arteries. In the intervals of rest the blood employed in systemic work reverts to the capacious splanchnic reservoir, whence it is again withdrawn by the physiological activities of the system. In this fact we find the *raison d'être* of the large capacity of the splanchnic veins, which form an adjunct to the systemic circulation, serving as a reservoir for the supply of blood needed for the performance of work—whether cerebral or muscular—and as an overflow chamber in times of rest.

Clinical Disturbances of the Normal Interchange between the Splanchnic and the Systemic Areas

of the Circulation.—In health a certain degree of functional interchange is maintained between the two systems (splanchnic and systemic), so that the "give" and "take" of each fall within certain limits; but in disease this normal balance is apt to be greatly disturbed. In one group of cases (the hypotonic) the splanchnic reservoir is continuously surcharged, while the systemic "draw" is lessened and the arterial pressure is lowered. In another group (the hypertonic) the splanchnic reserve is greatly reduced, and the arterial pressure is raised.

The Blood-pressure in the Systemic Veins.

The methods of observing the venous pressure on the dorsum of the hand (*see* p. 32) have yielded the following results:

Posture.—The pressure in normal subjects is lower in the recumbent than in the erect position, and intermediate in the sitting posture: *e.g.* recumbency 10-15,

sitting without support 15-20, and standing 20-25 mm. Hg.

Muscular Action has a pronounced effect in raising the venous pressure. This is even observed in the postures: for example, it may be 10–15 mm. Hg. while sitting with back supported and all the muscles relaxed, and on sitting erect it may rise to 15-20 mm. Hg. But the increment is much more decided in active exercise of all kinds; even in cycling on a level road (and therefore with only slight effort) the venous pressure may be doubled—*e.g.* 15–20, rising to over 30–40 mm. Hg.; and in cycling on a rising gradient it may exceed 50 mm. Hg.

Contraction of the muscles, by compressing the veins, raises the pressure: this fact, which was utilised in the days of venesection, is well shown by raising the hand until the veins collapse, when muscular contraction of the arm refills them.

Mental Exercise and Psychological Excitement and Perturbation likewise increase the pressure. Hence the

importance of quiescence of mind and emotion during the observation.

Digestion also raises the venous pressure. Its augmenting influence is, however, counteracted by the reducing effect of the splanchnic drain which takes place after a meal—especially during the first hour. But, notwithstanding this fact, the systemic venous pressure does rise as a rule even in the erect postures during digestion.

Temperature.—Warmth raises the venous pressure and cold lowers it.

Respiration influences the pressure quickly and powerfully. All forms of effort which increase the intra-thoracic air-pressure raise the venous pressure markedly ; such as coughing, straining at stool, lifting a heavy weight with the glottis closed, Valsalva's experiment (forcible expiration with closed mouth and nostrils).

The effects of deep inspiration and expiration on the venous pressure are readily studied by gradually raising the extended hand until the veins on the

dorsum *just* collapse (*see* p. 32), maintaining it at that level and taking a series of long-drawn, deep inspirations and prolonged expirations, when the veins will be seen to fill with every inspiration and to empty with every expiration. These respiratory variations in the collapsed veins show the far-reaching effect of deep breathing on the peripheral circulation—the rise and fall in the venous pressure being indicative of the same variations in the capillary and tissue lymph circulation (*see* pp. 76–8). These observations are not, however, quite in keeping with what we know of the dilating effect of inspiration and the contracting effect of expiration on the intra-pulmonary vessels, which should induce a fall in the venous pressure during inspiration and a rise of it during expiration. But this discrepancy may be accounted for by (1) the fact that the variations in blood-pressure produced by inspiration and expiration are actually not synchronous with the respiratory movements—the maximum effect of each

being intermediate between inspiration and expiration [1]; and by (2) the muscular action on which deep and prolonged inspiration entirely depends—expiration being quite independent of it.

Sleep and drowsiness, by reason of the vaso-motor relaxation induced, favours a rise in venous pressure.

How is the venous pressure modified?—The following are the leading factors: in *reduction*—(1) diminution of the ventricular output and contractility, (2) contraction of the arterioles and (3) splanchnic drain; in *increase*—(1) augmentation of the ventricular output and contractility, (2) vaso-dilatation, (3) increased intra-thoracic air-pressure, and (4) compression of veins during muscular contraction.

The Venous Pressure as an Indicator of the State of the Peripheral Circulation.—This physiological study of the venous pressure shows that a rise of the venous pressure is an external and

[1] *A Manual of Physiology*, by G. N. Stewart, M.D., etc. London, 1899, p. 249.

visible sign of increased activity in the peripheral circulation—indicating the passage of a larger volume of blood through the capillary area; and a fall in that pressure shows either diversion of blood to the splanchnic area or a contraction of the peripheral vessels.

The Relation between the Venous and the Arterial Pressures.—The arterial as well as the venous pressure is raised during active exercise and during muscular contraction and effort; but in quiescent conditions of the body, when the venous pressure is raised by either warmth, sleep, or digestion, the proximal arterial pressure falls, while the distal arterial pressure rises as part of the increment of peripheral blood-pressure.

The Capillary Blood-pressure.

The Agreement between the Variations of the Venous and Capillary Blood-pressure. — Observation has shown me that variations of the capillary pressure are more definitely afforded by

the venous pressure than by the methods of direct measurement which have been proposed. I have found that the venous pressure taken by the gravity method is the best indicator of the capillary pressure, when the hand is normally warm; this is an important proviso, for when the hand is cold vaso-contraction of the venules takes place, which favours a rise in the capillary pressure, while it determines a fall in the venous pressure.

The Amount of Tissue-fluid indicates the Capillary Pressure.—But observation has further demonstrated, that the amount of tissue-fluid present in the last phalanx is the best and most trustworthy guide to the variations of the capillary pressure. The method of observation of the tissue-fluid is elsewhere described (*see* Appendix).

The Relation between the Venous Pressure and the Tissue-fluid.—As the venous pressure is a good gauge of the capillary pressure, there should be a similar relationship between its variations and those of the volume of lymph which

circulates through the interstitial tissue. Observation shows that this is so. I have found that (1) whenever the veins on the dorsum of the hand collapse at the level of the cardiac area (from apex to base) the reading of the tissue-fluid falls to zero (*i.e.* the lymphometer reading indicating either an absence of tissue-fluid or the presence of merely a point or two of lymph); and (2) the amount of lymph rises with the increment of venous pressure, as shown by the veins having a higher collapsing point than the cardiac area.

These and other observations have shown (1) that the tissue-lymph circulation in the systemic area is but an extension of the capillary circulation, and (2) that it is tidal, or to and fro in character—a circulatory movement which is the outcome of the rise and fall in the capillary blood-pressure.

The Normal Gradient of Blood-pressure.

These studies have shown that observation on all the accessible arteries has

demonstrated that they contain a store of high and fairly uniform pressure, which does not appreciably diminish until the blood reaches the finer divisions of the arterial tubing—such as the digital arteries. From the physiological standpoint we may regard the distal arterial region as consisting of the smaller arteries (or arteriettes as they may be termed), in which the pressure falls rapidly, and the arterioles; for observation shows that the stopcock action of the arterioles is really not confined to these terminal vessels, but extends to the whole of this area, which is throughout richly endowed with muscular tissue. Indeed, the arteriometer has demonstrated that the musculature of the proximal arteries also participates in vaso-motor contraction; but to a less extent than the vessels of the distal arterial region—the contraction of the arteries being perhaps more of a defensive character, so as to counteract the dilating effect of the increment of pressure induced by the stopcock function of the distal vessels.

The arterial pressure is a reservoir, or head of pressure, held in reserve by the vaso-motor system for the supply and distribution of the blood down the steep gradient of the peripheral vessels—in a word, for the extra blood supplies required by the various forms of physiological activity. During vaso-contraction the steepness of this gradient increases—providing that the augmented ventricular action does not diminish it by overbalancing the increased peripheral resistance—and during vaso-dilatation the capillary pressure rises and the areolar tissue becomes more freely irrigated by the plasmatic circulation. The best general external sign of these alterations of increase and decrease of the pressure-gradient is afforded by the venous pressure; a rise in that pressure indicating a more voluminous peripheral circulation, and a fall of it showing a reduction of that circulation.

The Normal Arterial Pressure.

The Average or Mean Normal Pressure.—What is the normal arterial blood-pressure in the average healthy adult? Observing all the elementary precautions to secure uniformity of observation—such as avoiding the effects of exercise and of mental perturbation, and selecting the same area of the circulation (*e.g.* the brachio-radial), the proper time of day (if possible an hour or so before meals), the same posture, and the level of the heart—it will be found that the normal arterial pressure in adults is somewhat less variable than is usually believed, a diastolic pressure of from 95 to 100 and a systolic pressure of from 105 to 130 mm. of mercury being the figures commonly met with. A good average normal radio-brachial reading in a young or middle-aged adult (*æt.* 35 or 45) may be taken as $\frac{115\text{-}25\ \text{S}}{95\text{-}100\ \text{D}}$, individual variations being included in the range just indicated.

The Range of Physiological Variation. — When, however, the causes of physiological disturbance are disregarded, a wider margin must be given for the normal variant—*e.g.* $\frac{90\text{-}145\ \text{S}}{80\text{-}115\ \text{D}}$.

In women the pressures are generally from 5 to 10 per cent. less than in men; in children and adolescents they, as a rule, approach the lower $\left(\frac{90\ \text{S}}{80\ \text{D}}\right)$, and in subjects over 45 and in the elderly the upper $\left(\frac{135\text{-}145\ \text{S}}{105\text{-}115\ \text{D}}\right)$ normal limit. The arterial pressure does not, however, *necessarily* rise with age (*see* pp. 118–20).

The Influence of Temperament and Mode of Life.—Nervous, anxious temperaments, and occupations which involve much anxiety, worry, and nerve-strain, tend to produce somewhat higher levels of arterial pressure—especially in the latter half of life. On the other hand, placid temperaments and routine occupations—especially of the physical order—

dispose to the lower degrees of pressure. I have observed that, as a rule, in subjects of good condition and training—such as athletes—the arterial pressure is certainly not raised, and is indeed very often somewhat below the average normal pressure—a fact which points to the importance of maintaining the functional activity of the peripheral circulation by exercise. And this conclusion is supported by the observation that in those normal subjects who follow sedentary occupations for many hours daily indoors, the pressure, though generally normal, is more frequently above and less frequently below the mean normal pressure line.

The Influence of Body Weight and Build.—It seems to me that small and light subjects have generally somewhat lower arterial pressures; so that a rise above normal in such subjects which may appear to be comparatively slight, should be regarded as more significant than in subjects of the average build. Broad, large-built subjects usually have a somewhat higher arterial pressure.

The Influence of Hot and Cold Weather.—The modifying influence of unusually hot or cold weather (the hypotonic effect of heat and the hypertonic effect of cold) on the blood-pressure should also be kept in mind. According to my observation, warm weather, especially when decided and continued, may let down the brachial pressure (systolic and diastolic) from 10 to even 20 mm., and raise the phalangeal pressures to a corresponding degree; and cold weather may raise the former and lower the latter to a similar extent. Hence during spells of hot weather an arterial pressure may appear to be quite normal, whereas when the temperature is ordinary and temperate it may exceed the upper physiological limit; and in very cold weather the pressure may seem to be higher than it is at other times.

There is some evidence to show that in tropical countries (such as India) the arterial pressure is lower than in colder and more temperate countries. McCay, of Calcutta, thus writes: "The pressure

[taken *à la* Riva-Rocci] over a very large number of observations varied between 83 mm. Hg. and 118 mm. Hg., the average being slightly over 100 mm. Hg. (sitting position, arm level with heart). This is much lower than the blood-pressure laid down for Europeans in European (colder) countries." [1]

The Physiological Relation between the Systolic and Diastolic Pressures.

The Range of Pulse Pressure.— The difference between the minimum and maximum pressures is the complete cycle of the pulsation pressure. At the pressure-constant periods I have found that it varies in different individuals from 10 to 15, 20, 25, 30, 35, or 40 mm. (the average being about 25 mm.), the smaller differences being present in subjects below middle age and especially in children and in women, and the larger after middle age and especially in elderly subjects (over

[1] *The Lancet*, vol. i., 1907, p. 1484.

60 years). The difference is remarkably uniform in the same individual, but it is much less so during pressure-variable periods. As a rule, with vaso-contraction the difference widens; and with vaso-dilatation, while the two pressures usually fall together, they tend to approximate somewhat. According to my observation, as a rule in normal subjects, a rise of 5 mm. over 100 in the diastolic pressure is associated with a somewhat greater rise in the systolic; but the increment of the systolic over that of the diastolic pressure within the physiological limits diminishes as the pressure rises.

CHAPTER IV.

ON CLINICAL HÆMOMANOMETRY.

Most clinical observers are now much alive to the value of blood-pressure measurement as an aid in diagnosis, prognosis, and treatment. It is therefore no longer necessary, in commencing the study of clinical hæmomanometry, to insist on the shortcomings of the digital observation of the pulse and on the advantages to be derived from reading the blood-pressure definitely. This procedure is now accepted as a useful addition to other methods of clinical observation by many physicians in Germany, Italy, France, America, and in this country; and is winning its way into the body of the profession, for the general practitioner is realising its practical value.

It therefore follows that now a whole

army of observers is at work on the measurement of clinical blood-pressure, instead of, as formerly, a devoted observer here and there ; and we may consequently hope that the many gaps in our knowledge of the subject will soon be filled up, and that hæmomanometry as a clinical method will become firmly established.

The following illustrations of the clinical value of blood-pressure measurement in medical practice, derived partly from my own field of observation and partly from that of others, will form as it were a bird's-eye view of the results of manometric observation and a nucleus for further inquiry.[1]

On Subnormal and Supernormal Arterial Pressure.

Subnormal Arterial Pressure.—We may employ the term "subnormal" in

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[1] The reader should consult *The Clinical Study of Blood-Pressure*, by C. Janeway, M.D., which, besides containing the author's observations and conclusions, furnishes a good *résumé* of the results of blood-pressure measurement scattered through different publications and journals.

association with the arterial pressure in much the same sense as when we associate it with temperature. We then signify a pressure which is habitually, or for varying periods, below the mean normal level of pressure furnished by the average of subjects of similar age, etc. Nature, however, by her adaptive mechanisms, prevents the arterial pressure from falling unduly, or to such an extent as to threaten life; and this fact may suggest a doubt as to whether the term subnormal arterial pressure is admissible. It is believed that the corrective mechanism is brought into play through the reduction of the normal cerebral blood-pressure in the erect posture setting up vaso-constriction, which stimulates the ventricle, when the arterial pressure is restored to a higher level. In this way the area of subnormal pressure is kept within very narrow limits, which are indeed quite insignificant when compared with those of supernormal pressure.

Undoubtedly in certain diseases, such

as Addison's disease, there is subnormal pressure; and when the vaso-motor tone is very low—from whatever cause—the arterial pressure may remain for some time in the lowest area of physiological variation.

Supernormal Pressure.—When the systolic pressure is found to range persistently *over* 150 mm. Hg., it may be said to be supernormal. But the essential feature of such a pressure is its *persistency*; for in health the systolic pressure may quite *temporarily* rise as high as 180 or even 200 mm. Hg.—as under conditions of nervous excitement or perturbation. Therefore, before an observer can be fairly well assured as to whether the arterial pressure is raised or not, he should ascertain whether the alteration in pressure is persistent, or is only temporary, and should sedulously exclude nervous excitement in the subject. The latter precaution is of prime importance; and on account of it nothing in medical practice requires more tact and circumspection than the taking of the

arterial pressure. High readings—*e.g.* $\frac{220—260\ S}{150—180\ D}$—should be accepted at first with some hesitancy, especially when the pulse-rate is raised; and the observer should always remember that when the peripheral resistance is increased, the influence of nervous excitement, however slight, becomes much more pronounced than when the distal onflow is normal or subnormal. Hence the pressure is, as a rule, more variable in the elderly than in the young. High apparently normal pressures, such as a persistent systolic pressure of over 145–55 mm. taken within an hour before a meal, should, however, be looked upon as somewhat suspicious and requiring watching, especially when the diastolic pressure is raised from 10 to 20 per cent. In subjects of over 50 or 55 years of age such pressures should not be passed by in a routine way as normal. They should be subjected to treatment; and then, should a lower level of pressure be attained, with an improvement in the general health and

well-being, it may be inferred that such moderate increments of pressure were not normal to the individual, and indicated pathological departures in the direction of supernormal pressure. It is important to detect such departures in their initiatory stages, for they are then readily impressed by treatment; whereas, when left undetected, they are apt to increase slowly in a latent way, month by month, year by year, and, without inducing any important warning symptom, may suddenly induce cerebral hæmorrhage, or they may gradually develop secondary cardiac disease, etc. (*see* p. 135).

Some Chronic Ailments in which the Sectional Area of the Peripheral Circulation is apt to become Reduced.

I will take my first illustrations of the value of clinical blood-pressure measurement from a group of chronic diseases, in which the common pathological features, variously combined in the different members of the group, may be said to be

(1) some bio-chemical alteration of the plasma or lymph, (2) degenerative changes in the peripheral vessels, and (3) hyperplasia and contraction of the interstitial tissue, with obliteration of many of its small vessels and capillaries. So far as the circulation is concerned, the common outcome of these pathological changes is a less or more pronounced tendency to a rise in the arterial pressure, which is to be ascribed chiefly to a reduction of the sectional area of the arteries, and more particularly of the peripheral vessels (small arteries, arterioles, and capillaries) —a reduction which is effected first by narrowing of the arteries and arterioles by muscular contraction, then probably by shrinking of the large capillary bed, partly from obliteration of vessels and partly from contraction of the interstitial tissue, and finally by degenerative involvement of all the peripheral vessels. In this group we may include senility and pre-senility, the various phases of chronic rheumatism and chronic goutiness, the forms of arteritis and of

arterio-sclerosis, and chronic interstitial nephritis.

Senility.—From 20 to 45 or 50 the arterial pressure does not, as a rule, undergo any appreciable variation; but after middle age (50) the radio-brachial arterial pressure, as a rule, shows a slight rise, the systolic pressure creeping up from, say, 120-5 to 130-5, and even 140; and if at 60 to 65 it does not exceed 135-40, it may be regarded as fairly normal for that age. Meanwhile, the diastolic pressure will probably show a smaller increment, or may remain, as it often does, at 95 to 100 until quite late in life.

The rise of the systolic pressure as age advances is due to the extra call on the ventricle from (*a*) a rise in the peripheral resistance from shrinking and obliteration of distal vessels (arterial, capillary, and venous), and (*b*) diminished elasticity in the central arteries. The heart thus maintains the normal capillary pressure required for carrying on the physiological work of the circulation. According to

my observation, so long as the arterial pressure in the third phalanx does not fall below $\frac{65\ S}{45\ D}$ mm. Hg., or the venous pressure below 10 mm. H.g., it may be inferred that the digital capillary pressure is normal. The peripheral degenerative effects of age manifest themselves in limitation of the normal variations in the distal blood-pressure, when the increments of tissue fluid associated with the physiological work of digestion, exercise, etc., become somewhat reduced. The scalpel reveals a marked difference in cutting through the cartilages of the elderly and the old, as compared with those of earlier life; the cartilaginous tissue being more rigid and showing evidence of the deposition of lime salts. The fibrous tissues are also more resistant—and this is exemplified in the toughness of the flesh of old animals. These and other similar facts point to the thickening and shrinking of the interstitial tissue, in which lies the meshwork of the capillary circulation, with the

probable deposition of early salts (calcium and magnesium carbonate and phosphate) in the areolar tissue spaces. Such tissue-changes will doubtless increase peripheral resistance, and will tend to raise the arterial pressure in the aged. But it must not be supposed that an increment in that pressure is a necessary result of age, even in the moderately old; for quite old subjects are not infrequently met with in whom it is not apparent. There are probably two reasons which may explain this fact. It may be due to delayed senility, which is far from uncommon; and observation shows that, as a rule, a postponement of the normal senile increment of pressure is favourable to longevity (*see* pp. 118–20). But there may be another cause why the arterial pressure does not rise with age—namely, an equable shrinking of the whole circulation—the heart, the volume of the blood, as well as the periphery, all being reduced in the general involution.

The following systolic pressures, when persistent, may be regarded as verging

on the pathological: 140 mm. at 40–5; 150 mm. at 50–5; 160 mm. at 60–70, and require watching and treatment. Atheroma of the accessible arteries, even though pervading them rather extensively, may have comparatively little effect on the arterial pressure, unless it also invades the thoracic aorta, or the terminal arteries and arterioles; so that we may infer that a rise of the arterial pressure in the senile is not due so much to atheromatous thickening of the arteries of intermediate size as to the involvement of the centric and peripheral parts of the arterial system.[1]

It frequently happens that the rise of arterial pressure in the senile and pre-senile depends, *in the first instance*, largely on hypertonia of the peripheral arteries, and not so much on organic changes in the vessels, though these may eventually become predominant (senile arterio-sclerosis). In the hypertonic stage

[1] The reader will bear in mind that when the brachial arteries are thickened in the aged, the armlet furnishes a higher reading of the arterial pressure than the hæmodynamometer (*see* pp. 114–122).

it is probable that we can do something by way of treatment to prolong life and to ward off future possibilities (*see* p. 170 *et seq.*). The great aim is to raise the capillary pressure and to restore the normal working range of that pressure.

I have obtained evidence to show that in the elderly the arterial pressure may be persistently high in the morning (*e.g.* 8 a.m.), whereas it may be quite normal or even low in the evening. Just lately a patient (*æt.* 76), who is able to read his own pressure accurately, has furnished me with an interesting night and morning record, from which I learn that his average range of variation in the systolic pressure extends from 130 in the evening to 160 mm. Hg. in the morning, and that this type of variation is constant. In such a case it is obvious that no reliance can be placed on an observation made during the course of the day, and especially during the latter half of the day.

Chronic Rheumatic and Gouty Ailments.—Some three years ago Sir William Gowers, in an instructive lecture

on "Lumbago: its Lessons and Analogues,"[1] grouped together the various forms of chronic rheumatism and gout under the common denomination of "fibrositis," or inflammation of the systemic fibrous tissue. This conception includes many painful systemic ailments, such as the varied group of muscular rheumatism (lumbago, pleurodynia, torticollis, etc.), periarthritis, unilateral or interstitial neuritis, and other stromatic affections of the systemic tissues, which are commonly ascribed to a gouty state, either generated by the habits of life of the individual, or inherited. The seat of these troubles is the cellular or areolar tissue, which forms the bed of the tissue-lymph circulation. In this large group of cases the peripheral circulatory mechanism is very frequently involved; but the alterations of blood-pressure met with are often comparatively slight, and may easily pass unnoticed. It is frequently difficult,

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[1] *Brit. Med. Journ.*, 1904, vol. i., p. 117.

especially when the subjects are over fifty years of age, to estimate how much of chronic rheumatism and chronic goutiness is due to senile or pre-senile fibrosis, and how much of the circulatory disturbance may arise from the reflex effect of pain on the arterial wall, or may be associated with the early stage of arteriosclerosis. All these pathological conditions appear to me to be so closely allied and related to one another that it is futile to attempt to differentiate between their effects on the periphery of the circulation. But notwithstanding this fact, blood-pressure observation is often not only interesting but valuable in the study of this group of cognate ailments; frequently, indeed, affording a hint in regard to treatment and management. In some of these fibrositic ailments the proximal blood-pressure may not be appreciably disturbed. As a rule, however, there is certainly distinct evidence of hypertonia of the arteries, with moderate increments of arterial pressure; but now and then we meet with a hypotonic

condition, with lowering of the brachial pressure. Thus the manometer will often afford a useful hint as to treatment; for in the hypotonic form of goutiness we can afford to be somewhat more generous in regard to diet.

In the hypertonic forms of goutiness there are generally observed indications of a lowering of the peripheral circulation—reduction of capillary pressure, of the exudation of tissue-fluid, and of the venous pressure—with a moderate rise in the proximal arterial pressure; but in some cases the evidence of restricted variation in the capillary pressure with subnormal irrigation of the interstitial spaces is not associated with an appreciable increment of the arterial pressure. On the other hand, cases are not infrequently met with in which the capillary pressure is moderately high, and the exudation of tissue-fluid is large; but the pressure and the exudation do not undergo much alteration under different physiological conditions. In both classes of cases the normal to-and-fro variations

of the capillary pressure and of the flow of tissue-fluid are greatly diminished; in the one case there is a lowered capillary pressure, with reduction or absence of tissue-fluid, and in the other there is a less or more continuously raised capillary pressure with excess of that fluid. In both there is therefore one common feature—namely, diminished alternation; and this is a condition which will restrict interchange between the blood and the tissues, and will favour the deposition of insoluble residua in the interstitial spaces. We know that such deposition does take place in the gouty, for that fact is patent in the gross and obvious form of concretions of urate of soda in and about the joints, in the fibrous tissues, such as peri-cartilaginous fibrous tissues and cartilages (external ear, alæ nasi, tarsal cartilages). These tophaceous aggregations of uratic deposit are instructive in showing us that the precipitation takes place mainly in the fibrous tissue, which is the bed of the tissue-lymph circulation —an irrigation system, which is the most

languid portion of the circulatory apparatus; and these obvious collections of sodic urate suggest that the same process of deposition, to which they bear witness, may take place in a diffused form throughout the interstitial tissue of the gouty, in a form so minimal and so evenly incorporated with the tissue as to escape recognition. The tophi may merely differ physically from such a generalised deposition, just as uric acid calculi differ from the delicate microscopic crystals of uric acid. The material deposited may be in a colloidal form and of neutral reaction.[1] When in that condition it may

[1] We should remember, in regard to the retention or deposition of residuary products in the lymph spaces, that the blood strives for uniformity and purity. For example, when the kidneys fail in eliminating these materials, their passage into the lymph spaces takes place, and the chemical constitution of the blood is thus preserved; this fact is proved in the case of sodium chloride (*see* p. 179), and it may be that there is a tissue retention of urates when the kidney is inadequate for their elimination. It is true that urates do not figure in the published analysis of lymph; but if they do not exist in the tissue-fluid, how can we account for the uratic deposits in the fibrous tissue? It may be that they have not been detected in the lymph drawn from the thoracic duct, though they are present in the tissue-fluid, which cannot be analysed.

not cause local pain, or discomfort, or outward sign of its existence. But should the flow and ebb of lymph to and from the interstitial spaces become more active from any cause, this dormant, painless deposit may either become disturbed, or undergo chemical dissociation, with liberation of free uric acid or some irritant product. Subacute or acute attacks of gout may thus arise. The exciting causes of such attacks are numerous: according to my observation, the following, among others, play a leading rôle: mental emotion and excitement, physical and mental over-exertion and fatigue, an unusually heavy meal, the increased ingestion of chloride of sodium (*see* p. 179 *et seq.*) or of alcoholic stimulants (especially in the form of beer or wines), the partaking of certain articles of food, the toxæmias, an injury (however trivial, such as sprains, etc., in some cases), warm baths, a common cold, or influenza.[1] Now, it is

[1] I have frequently found that deep sleep may be followed by headache in gouty subjects. In sleep I have found the capillary and venous pressure high, and the

obvious that these causes are all varied in their character; yet they present one feature in common. They are all powerful stimulants of the peripheral circulation; they all raise the capillary pressure and increase tissue irrigation. In the non-gouty they are harmless; but in the gouty they are risky, and may excite something more than their physiological effects. I am disposed to think that this difference depends on the character of the chemical residua retained or deposited in the tissue spaces. Urates are not poisonous or hurtful; for they may remain in the fibrous tissue as harmless, painless tophi for indefinite periods. And I think it may be safely inferred that a generalised deposition of urates in the tissue spaces will be equally painless, unrecognisable, and dormant during many years, until it is disturbed by a rapid rise

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tissue-lymph circulation most copious; and on awaking I have observed that the distal congestion at once subsides and the ebb-tide of lymph is rapid. Should any toxins be formed in the interstitial spaces, they will, if soluble, be swept into the blood on awaking.

in peripheral irrigation, or is chemically broken up at the same time. May not some physiological product liberate free uric acid crystals from the deposited urate ? and I have found in the test tube that lactic acid, which is generated in muscular action, has this property. So it is not improbable that a chemical change in the plasma may be a precursor of acute gout, as well as the more active peripheral circulation.

I have entered thus fully into the probable pathological change which takes place when the patient passes from a state of goutiness into that of an acute attack of gout, because it helps to explain the alteration I have sometimes observed in the peripheral circulation, either just before or during the attack—especially when this induced a fair amount of symptomatic fever : namely, a much more active state of that circulation and of its addendum, the lymph circulation, with, as a rule, a fall in the proximal arterial pressure. But this alteration in the distal and proximal arterial pressure

readings in gouty subjects can take place only so long as the arteriettes and arterioles retain their vaso-motor activity, or at any rate a fair measure of it. This fact may explain the rarity of acute gout in well-developed arterio-capillary fibrosis; in fact, this condition of peripheral degeneration prevents some other ailments connected with vaso-motor variation from manifesting themselves—such as migraine and angina pectoris vaso-motoria. And I have sometimes thought that spasmodic asthma may clear up with the development of arterio-capillary fibrosis. Thus it would seem that nature may apparently cure an ailment by setting up another morbid condition.

Arterio-sclerosis.—Clinically we meet with two leading forms of arterio-sclerosis: one in which the thickening of the arterial wall is less or more local, or is unequally distributed (nodular atheroma), and the other in which it is quite generally diffused throughout the arterial system (diffuse or generalised arterio-sclerosis).

The inequalities of **nodular atheroma** are well recognised. They may be so local as not to appreciably raise the general arterial pressure, or to raise it but slightly, even though many of the accessible arteries (such as the temporal, radial, etc.) may be thickened. Hence we sometimes meet with cases in which there is *apparently* rather extensive arterial disease, and yet the subjects of it may live into good old age without developing the sequelæ of arterial degeneration. But as a rule extensive atheroma is associated with a readily appreciable rise in the arterial pressure; and I think it must be rare to meet with a case in which the brachial artery is felt by the finger to be distinctly thickened and cord-like without an increment in the reading of the systolic pressure being revealed by the armlet, and even in some cases by the hæmodynamometer, which yields a lower reading (*see* pp. 50, 114, *et seq.*). According to my observation, atheroma is apparently often more advanced in the brachial than in the other arteries of the

limbs; for the armlet readings, identical in normal subjects in the arm and fore-arm, are as a rule higher in the arm. But this fact is open to another interpretation (*see* p. 123).

In diffuse or generalised arterio-sclerosis or arterio-capillary fibrosis the accessible arteries may not be appreciably thickened, especially in the earlier stages of the disease, and yet the arterial pressure may be raised persistently and definitely. It would seem as if the disease begins more particularly in the terminal divisions of the arterial system—splanchnic and systemic, and especially splanchnic. In the initial stage of the disease the armlet and the hæmodynamometer afford the same systolic readings. In this stage the peripheral resistance is apparently due mainly to muscular contraction in the arterioles and arteriettes; for these readily respond to vaso-dilator remedies, and the increment of arterial pressure, which is not so high as it subsequently becomes, quickly subsides after each dose. In this hypertonic

stage, the stage of pre-sclerosis of Huchard, the diastolic pressure rarely rises above 120 mm. Hg. or so, and is often only 110 or 115; and the systolic pressure does not, as a rule, exceed 160 mm. Hg., and is frequently only 145 or 150; and the arteriometer demonstrates the contraction of the radial calibre and the favourable effects of treatment in dilating it (*see* pp. 238, 239). But as the disease advances organic changes in the arterial wall develop; when vaso-dilators only partially relieve the pressure, and ultimately may fail to lower it. In this stage the accessible arteries—such as the brachial—as a rule become thickened, and the arterial pressure increases considerably; the systolic armlet readings advancing to such high figures as from 200 to 260, while those of the hæmodynamometer[1] rarely exceed 200 mm. Hg., and are frequently somewhat less.

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[1] When referring to the readings of the hæmodynamometer I include those of the compressed-air manometer furnished by the bulb mounted on a slide, for they are identical (*see* p. 52).

Does thickening of the arterial wall vitiate the armlet reading? My observations have demonstrated to me that the thickened arterial wall in arterio-sclerosis has a greater effect in disturbing the accuracy of the readings of the arterial pressure obtained by the armlet method than is usually supposed; the general impression being that it has but little effect. It is true that V. Basch [1] and C. Martin [2] have experimentally shown that the excised sclerosed radial—even though sclerosed to a high degree—is closed by a comparatively low fluid pressure, such as 5 to 7 mm. Hg., whereas a normal radial is collapsed by a still lower pressure—in fact, a negligible pressure of 1 or 2 mm. But in practice I have obtained evidence which suffices to show that the difference between the closing pressures of a normal and of a sclerosed arterial wall must be considerably greater than these figures suggest.

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[1] *Op. cit.*

[2] *Brit. Med. Journ.*, 1905, vol. i., p. 866.

I have already pointed out that when the arterial walls are normal there is no appreciable difference between the readings of the systolic or occluding pressure in the arm and the forearm (*see* p. 55); but in cases of arterio-sclerosis I have frequently found that discrepancies of 10, 20, or 30 mm. may be met with. Moreover, the nominal differences (if any) in the readings obtained from the corresponding areas, when the arteries are normal, are apt to become pronounced when the vessels are sclerosed.

Can we estimate thickening of the arterial wall?—But this is not the only evidence available to show that the armlet method may furnish in arterio-sclerosis an incorrect estimate of the actual arterial pressure. I have found the use of the hæmodynamometer by the side of the armlet method very instructive in studying the blood-pressure in arterio-sclerosis. I have shown (p. 49) that when the arteries are normal the systolic reading of the hæmodynamometer is identical with that of the armlet. But

I have observed that this agreement fails very frequently, if not invariably, when there is arterio-sclerosis of the brachial arteries—when, in fact, the arteries are felt to be thickened. In such cases the systolic reading of the armlet may exceed considerably that of the hæmo-dynamometer ; and the pulse diminishes much more slowly during the last 30, 40, or 50 mm. Hg. of compression than when the arterial wall is normal.

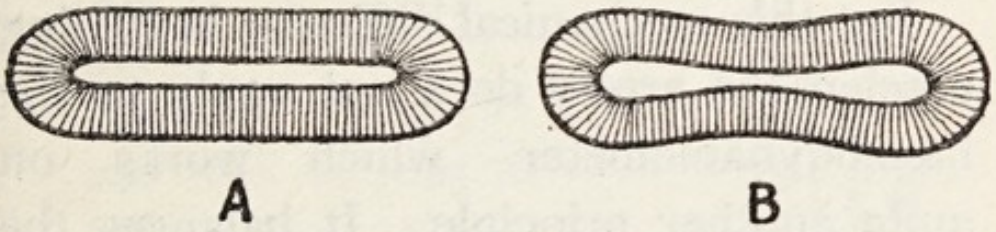

Fig. 6.—(After V. Ricklinghausen).

A. The manner in which the *normal* artery is closed by compression. B. The manner in which the *sclerosed* artery is closed by compression.

V. Ricklinghausen has suggested that a thickened or calcified artery may yield too high a systolic reading when determined by obliteration of the pulsation beyond the armlet, because the vessel fails to collapse evenly as a normal artery

does—the centre of the vessel collapsing readily enough, while a chink is apt to be left at each side, which is only closed under a much higher degree of compression (Fig. 6). This suggestion affords an explanation of my observations; and these have shown that this want of uniformity in the closure of the central and lateral portions of a sclerosed artery is not only found in a few exceptional cases, but is not at all uncommon in arterio-sclerosis.

But this mechanical difficulty in closing a sclerosed artery does not apply to the hæmodynamometer, which works on quite another principle. It balances the blood-pressure wave; and to do this it is merely necessary to overcome the resistance of the central portion of the sclerosed vessel, which has been shown to be nominal.

The following are a few typical selections from the discrepant figures furnished by the two methods in a considerable number of cases of arterio-sclerosis which I have lately observed. So far I cannot

say that I have met with quite identical readings in any case in which thickening of the brachial arteries was demonstrable by the finger; though in some such cases it is true the differences were small, *e.g.* from 10 to 20 mm., but they were indubitable.

The systolic pressure in mm. Hg. furnished by:

Armlet.	Hæmodynamometer.	Difference.	
270	215	55	Average difference over 50 mm. Hg.
255	195	40	
245	185	40	
240	165	75	
240	175	65	
230	185	45	
230	175	55	
220	165	55	
220	160	60	
215	175	40	

It will be observed that in this group of figures, though all the readings furnished by the armlet exceed 200 mm. Hg., only one of those yielded by the pulse-pressure gauge oversteps that figure, the

majority being much below it. I have omitted from these selections two observations which afforded the difference of 105 (Armlet 260, Hæmodynamometer 155; A. 255, H. 150), because such a large difference is doubtless exceptional in arterio-sclerosis. My observations indicate that in commencing or slight forms of arterio-sclerosis the differences are small—from 10 to 20 mm.—but that in the fully developed or advanced stages they are much higher, reaching 40, 50, 60, or 70, and even 100 mm. I have found that the comparative readings of the diastolic pressure are, as a rule, much less discrepant in arterio-sclerosis than those of the systolic pressure. Hence in this disease the armlet commonly furnishes a larger pulse-pressure range than that afforded by the hæmodynamometer.

The following systolic readings show that the armlet may afford supernormal figures in arterio-sclerosis, whereas those of the hæmodynamometer may be normal.

Armlet.	Hæmodynamometer.	Difference.	
215	140	75	Average difference over 47 mm. Hg.
190	145	45	
180	135	45	
180	125	55	
175	140	35	
175	125	50	
170	125	45	
165	135	40	
160	125	35	

The systolic pressure may be comparatively low in arterio-sclerosis.

Armlet.	Hæmodynamometer.	Difference.
150	115	35
140	115	25
130	115	15
120	105	15
115	105	10

Therefore, even when the armlet readings are low the thickened arterial wall may still be a disturbing factor (though less pronounced) in their production.

The conjoint use of these two methods may reveal the beginnings of arterio-sclerosis, as in the following case, in which the earlier readings were identical, whereas the later ones (after several months) became divergent.

Armlet.		
Arm.	Forearm.	Hæmodynamometer.
130	130	130
170	160	145
155	145	135
150	140	130

The differential readings in syphilitic arterio-sclerosis.—Just lately I have found the conjoint application of these methods also useful in following the effects of treatment in a case of syphilis of four years' standing; the differential reading of 35 mm. being reduced by 24 mercurial inunctions and sulphur baths to 10 mm.

Discrepant readings in the elderly (over 60) are quite common; and are indeed practically the rule after 70. They are generally associated with some rise of the arterial pressure, as determined by the method of balancing the pulse-wave by the hæmodynamometer or the manometer-bulb; and in most cases this rise is quite moderate or even slight, such as $\frac{145\text{-}160\ \text{S}}{115\text{-}120\ \text{D}}$. But when this mode of observation shows that the pressure is

not actually raised, or remains low, notwithstanding the detection of a moderately high reading by the armlet, I have observed that life may be prolonged much beyond the allotted span, with the retention of an exceptional degree of bodily and mental vigour. This fact is illustrated by the following readings afforded by five subjects, the youngest in her ninetieth and the oldest in his hundreth year.

	Hæmodynamometer and manometer-bulb.		Armlet.		
A. (Recumbent, in her 90th year.)					
Right side	$\frac{110\ S}{95\ D}$	Forearm	$\frac{130\ S}{95\ D}$	Arm	$\frac{140\ S}{95\ D}$
Left side .	$\frac{110\ S}{95\ D}$	,,	$\frac{145\ S}{95\ D}$	,,	$\frac{180\ S}{95\ D}$
B. (Recumbent, in her 93rd year.)					
Right side	$\frac{135\ S}{105\ D}$	Forearm	$\frac{175\ S}{125\ D}$	Arm	$\frac{190\ S}{130\ D}$
Left side .	$\frac{135\ S}{105\ D}$	,,	$\frac{155\ S}{115\ D}$	,,	$\frac{180\ S}{127\ D}$
C. (Sitting, in her 96th year.)					
Right side	$\frac{115\ S}{95\ D}$			Arm	$\frac{185\ S}{115\ D}$
Left side .	$\frac{115\ S}{95\ D}$			,,	$\frac{190\ S}{115\ D}$

	Hæmodynamometer and manometer-bulb.	Armlet.	

D. (Recumbent, in his 98th year.)

Right side	$\frac{125\ S}{100\ D}$	Forearm $\frac{170\ S}{120\ D}$	Arm	$\frac{180\ S}{120\ D}$
Left side .	$\frac{125\ S}{100\ D}$	" $\frac{160\ S}{115\ D}$	"	$\frac{190\ S}{120\ D}$

E. (Recumbent, in his 100th year.)

Right side	$\frac{105\ S}{82\ D}$	Forearm $\frac{135\ S}{95\ D}$	Arm	$\frac{135\ S}{95\ D}$
Left side .	$\frac{105\ S}{82\ D}$	" $\frac{125\ S}{92\ D}$	"	$\frac{135\ S}{95\ D}$

In all the subjects the accessible arteries were thickened in variable degrees; and in each case some arteries were decidedly more sclerosed than others. In all the subjects the brachial arteries were felt to be sclerosed, with a considerable diminution of their normal elasticity. In case E, in whom the arterial pressure was lowest, the indications of thickening and loss of elasticity were very much less evident. In none of the cases was the pulse hard to the finger; in A, C, D, and E it was indeed quite as soft

as is usual in normal subjects in the middle period of life (40–60), and in E this feature was even still more pronounced, suggesting the soft pulse of 20 or 25. In B the tactile resistance of the pulse resembled that usually met with about 60. In A, C, and D it was easy to define the heart's apex beat, which was well inside the nipple line; in B it was just within it; but in E it was not palpable, though the stethoscope indicated by the maximum apex sounds that it occupied its normal site. The second aortic sounds were not accentuated in any of the cases. Notwithstanding the development of the senile arterio-sclerosis to a fairly advanced stage and the high readings of the armlet in subjects A, B, C, and D, there was no evidence whatever of cardiac strain in any of these cases. I therefore hold that the low readings of the hæmodynamometer and the manometer bulb in all the subjects — such readings being also uniform in all the accessible arteries—are consistent with the long life and well-being

of the patient, the soft and compressible state of the pulse, and the normal condition of the heart; and that the higher readings of the armlet are quite incompatible with these facts and are accounted for by the sclerosed state of the arteries of the arm.

Identical readings in the aged are, however, now and then met with, even in subjects over 70 years of age, as in the following examples, all well preserved for their years :—

Age.	Identical readings of the systolic pressure with the armlet and hæmodynamometer.[1]
81 . .	135
80 . .	145, 135
78 . .	150, 180, 135
77 . .	140
76 . .	145
75 . .	125
74 . .	150, 135
73 . .	150, 145
71 . .	145, 145, 140
70 . .	165, 135

In nearly all these cases the armlet was applied to the forearm only, which

[1] The figures refer to different cases.

has as a rule yielded lower readings in atheroma than the arm. Probably some disparity would have been detected by readings derived from the arm. Perhaps a small artery, like the radial, when thickened may close more uniformly under pressure than a larger one, such as the brachial, which may be more likely to leave lateral chinks (*see* p. 113).

These clinical studies on the conjoint use of the manometer and the hæmodynamometer have led me to conclude:

(1) That in arterio-sclerosis and other pathological conditions of the circulation the use of the hæmodynamometer suggests that the systolic pressure does not really often exceed 200 or 220, and probably does not overstep 250 mm. Hg. In a word, it would seem the normal arterial pressure is practically never more than doubled in the clinical field.

(2) That the armlet method in arterio-sclerosis is of diagnostic value when it affords variable readings in the arms

and forearms, and higher readings in these areas than those furnished by the manometer-bulb and the pulse pressure gauge. Further observation may show that atheroma yields more variable readings in the different areas than diffuse or generalised arterio-sclerosis.

(3) That the lower reading furnished by the hæmodynamometer in arterio-sclerosis is frequently of practical value. It will be useful if it subdues the unnecessary anxiety which is apt to be engendered by some of the high figures yielded by the armlet. It may likewise exert a salutary influence over treatment; for it may, for example, indicate a normal or comparatively normal pressure underlying an apparently supernormal one, or it may suggest the use of cardiac tonics, though the pressure may seem high enough according to the armlet. Moreover, the lower reading may explain some clinical incongruities, such as the absence of the signs ot cardiac stress and the maintenance of well-being in spite of a seemingly high pressure, or the super-

vention of circulatory failure inconsistent with such an apparent pressure.

(4) That when the arterial wall is normal the readings of the pressure furnished by the armlet are trustworthy enough; but when it is much thickened, their value as determinations of blood-pressure are somewhat impaired.

Testing of the Arterial Wall.—Inspection and palpation of the accessible arteries may throw light on the pathological condition (thickening), and for this purpose digital examination of the brachial arteries should not be neglected in all cases of suspected arterio-sclerosis. On extending the elbow, the artery, when sclerosed, is felt to be thickened, can be rolled under the fingers against the humerus; and the vessel may sometimes curl up on flexing the elbow—this curling indicating a diminution or loss of its rubber-like elasticity. When the wall of the artery is in an advanced state of thickening or is calcified, the fingers of course easily define the contour of the vessel; but when, in the early

stage of the disease, the thickening is slight, the detection of it may require some skill and delicacy of touch, which are only acquired by somewhat prolonged practice in this mode of observation. The observer will best acquire the necessary tactile ability by first of all learning to define the wall of the normal brachial artery; and then he will gradually acquire the skill to detect the early stages of thickening.

The physiological play of the arterial walls is best tested by administering a dose of a vaso-relaxant (such as a tabloid containing $\frac{1}{100}$ gr. of trinitrine, or $2\frac{1}{2}$ gr. of sodium nitrite), and observing the effects on the arterial and venous pressures, which develop in the course of ten minutes or so. It is easy, as a rule, to observe the effect on the pressure derived from the last phalanx of the finger or thumb. In a normal state of the distal arteries, the pressure rises, when the fingers are warm, from 30 to 40 mm. Hg. (*e.g.* from $\frac{65\text{-}70}{45}$

to $\frac{100\text{-}105\,S}{80\quad D}$). Should the reaction be greatly reduced (*e.g.* to half, or to a third, or even less), we may conclude that the peripheral vessels are invaded by organic disease. In some cases I have failed to obtain any response.

In case the phalangeal reading is not available, the reaction may be studied in the brachio-radial area. Should the pressure there fall 20–30 mm. Hg., and the venous pressure rise, we may, I think, infer that the distal vessels (and especially the splanchnic) are as yet not seriously invaded; but should it fail to fall, or should it fall to a less degree, their organic involvement may be suspected.

In this way, as it seems to me, the manometer may enable us to trace the transition from the responsive to the less responsive or even the non-responsive stage of the disease; and to gauge the nature of the peripheral resistance—whether muscular or organic, or both.

Arteriometric Observation of the

Radial Artery in Arterio-sclerosis.—For over twelve years past I have found the arteriometer (*see* pp. 130 and 138) of some service in the study of atheroma and arterio-sclerosis, especially in affording evidence of organic changes in the walls of the middle-sized arteries; the radial calibre being, as a rule, reduced, and the postural variation of it either failing to occur, or being greatly restricted in range, in proportion to the rigidity or loss of elasticity in the vessel wall. The arteriometric study of the radial shows that the arterial wall may pass through three stages in diffuse arterio-capillary fibrosis—namely: (1) muscular contraction of the calibre, (2) reduced arterial calibre from organic changes (chiefly), and (3) increased arterial calibre from passive dilatation—the distending effect of the high arterial pressure on the arterial wall being no longer resisted by adequate muscular contraction.

Chronic interstitial nephritis provides us generally with higher readings than are usually met with in most other

diseases; ranging from $\frac{220}{140}$ to $\frac{260\text{-}280\ S}{160\ \ D}$.

In this disease the hæmodynamometer will, however, afford systolic readings from 50 to 70 mm. lower than those of the armlet. This fact is well illustrated by the following readings afforded by an extreme case:

Hæmodynamometer and manometer bulb.	Armlet.
$\frac{210\ S}{155\ D}$	$\frac{300 + S}{175\ \ D}$

In this case the brachial arteries were much thickened, and the difference between the systolic readings of the two methods furnishes a measure of the arteriosclerosis; and the reading of the hæmodynamometer and the manometer bulb indicates the high arterial pressure. Blood-pressure measurement is most valuable in routine clinical work in enabling one to pick out cases of contracting kidney, which otherwise are apt to pass unsuspected. It must not, however, be inferred from this remark that excessive pressures —though observed in the majority of

cases—are to be met with in all cases, or throughout the course of chronic interstitial nephritis; a failing ventricle may, for example, let down the arterial pressure.

Some of the very highest readings are found in **uræmia** (systolic armlet pressures of from 270 to 300); and observers are mostly agreed that there is a direct relation between uræmic headaches, vertigo, amaurosis, vomiting, coma, etc., and the arterial pressure. Cook and Briggs[1] and Pal[2] have shown this, and the former have pointed out that a fall of pressure in uræmic coma may take place a few hours before death.

In **chronic parenchymatous nephritis** also high readings of the arterial pressure are met with, though less frequently so than in chronic interstitial nephritis; and in **acute nephritis** (*e.g.* scarlatinal) the readings are, as a rule, moderately high; but in the **amyloid kidney** and in **cyclical albuminuria**

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[1] "Clinical Observations on Blood-pressure." *Johns Hopkins Hosp. Report*, 1903.

[2] *Centralbl. f. innere Med.*, 1903.

they are generally normal or even subnormal.

Diseases of the Heart.

Blood-pressure measurement as a clinical method is not so useful in the study of **uncomplicated** heart diseases as in that of diseases which implicate the periphery of the circulation. There is only one valvular disease—namely, **aortic insufficiency**—in which we can at present say (in the absence of arterio-sclerosis when the armlet is used) that such measurement affords distinctive aid in diagnosis and in estimating the extent of the disease; the arterial pressures in all other forms of *uncomplicated primary* cardiac disease are generally normal and never subnormal (Starling).[1] I emphasise the words "uncomplicated" and "primary" because the complications of primary heart disease, such as arterial disease, renal complications, the excito-vaso-motory effects of dyspnœa and of mental anxiety,

[1] "Observations on the Arterial Blood-pressure in Heart Disease," by Hubert J. Starling, M.D. Lond., etc. *The Lancet*, vol. ii., 1906.

comprise the various pressure conditions of **secondary heart disease,** and these include the factor of increased peripheral resistance, which is, indeed, the primary pathological factor. We are, however, in need of more extended information than we possess concerning the arterial pressures in simple valvular diseases produced by acute endocarditis.

In **aortic insufficiency** all observers are agreed that the distinctive features of the arterial pressures are (1) a low diastolic and (2) a relatively high systolic reading, an example of an average formula being $\frac{140\,S}{80\,D}$. The range of pulse-pressure (*i.e.* the difference between the minimum and maximum pressure of each pulsation) is always much above the normal; for example, whereas the range in health varies from 25 to 40 mm. Hg., in aortic insufficiency it is rarely less than 60 or 70, and is often as high as 80 or 90 mm. Hg. I have myself observed 95, or over 125 per cent. of the diastolic pressure in a case presenting a very pronounced lesion.

In double aortic disease, and in aortic disease conjoined with mitral, it is often difficult to estimate the part played by the aortic insufficiency, or the degree of it. In such cases the readings of the pressures, which afford the pulse-pressure range, become a manometric measure of the aortic leakage. Hensen[1] and Janeway[2] have observed that the systolic pressure in aortic disease is very fluctuating without obvious cause, and my observations confirm this teaching.

Competency and failing competency. — In *competency* we have a fairly normal pressure gradient from the arteries to the capillaries and the veins; that is, from the high arterial pressure to the reduced capillary and the still further reduced venous pressure. In *failing compensation* this gradient becomes less steep, for the venous and capillary pressures rise to relatively higher levels, until, in fact, the capillary pressure

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[1] *Deutsch. Arch. f. klin. Med.*, 1900.

[2] *The Clinical Study of Blood-pressure.* New York and London, 1904, p. 198.

may relieve itself in exudation (ascites or œdema).[1] Starling[2] believes that vaso-constriction is the first effort made to correct the cardiac inefficiency by thus inciting the ventricle to restore the out-put to its previous amount, and that the constriction in the splanchnic area leads to the increased uptake of fluid from the alimentary canal, and thus induces plethora, which with increasing failure of the heart-muscle overloads the veins and capillaries.

An early sign of failing competency is not only an irregularity in the ventricular rhythm, but a marked irregularity of the systolic pressure of the individual pulsations; so that in measuring the systolic arterial pressure, successive additions to the pneumatic pressure in the armlet will filter out varying proportions of the pulsations, until at last there remain but a few scattered pulsations,

[1] In the production of œdema there is a not improbable factor which we may lose sight of, namely, the rifting effect of extra pressure on the cement between the endothelial cells of the capillary wall, which may permit of filtration under a comparatively low pressure.

[2] *Op. cit.*

to balance which a fairly high pressure is needed. If, therefore, in such cases we take the mean between the pressure required to obliterate some of the feebler beats and that necessary to annul the strongest, we may obtain a reading of the systolic pressure somewhat lower than that which was furnished by the ventricle when it was competent; and the figure thus obtained is a measure of the incompetence.

The relation between the arterial pressure and **dilatation of the ventricle** requires further study. The common explanation is that dilatation is the mastering effect of intra-ventricular pressure over a weakened state of the ventricular wall. If that were so, the dilatation results from the residuum of unexpelled blood, which grows larger in proportion to the diminished contractility. Dr. James Mackenzie[1] contends that we may have a thin-walled ventricle contracting and expelling its contents well, until it may

[1] "A Preliminary Inquiry into the Tonicity of the Muscle Fibres of the Heart." *Brit. Med. Journ.*, 1905, vol. ii.

actually rupture, without dilating; or we may have dilatation of the left ventricle with low intra-ventricular pressure, as in mitral stenosis. He traces dilatation to a depression of the function of tonicity—one of Gaskell's five cardinal properties of the myocardium (rhythmicity, excitability, conductivity, contractility, and tonicity). Probably this view is correct in primary cardiac disease. Tonicity may be so low that dilatation may take place when the intra-ventricular pressure may be presumed from the arterial pressure to be normal or even subnormal, as in acute infectious fevers and in the anæmias. But apart from such cases and from primary cardiac disease, my observations of the arterial pressure show as a rule that there exists a relation between the peripheral resistance and the dilatation.

A ventricle of normal tonicity resists dilatation from increasing peripheral resistance, and gradually becomes hypertrophied; later on it may, however, dilate in proportion to its failing tonicity. We have then developed the **secondary**

dilatation of peripheral origin, as in the later stages of Bright's disease and arterio-sclerosis. At the same time it should be borne in mind that the systolic arterial pressure afforded by the armlet will very frequently remain high, though the ventricle is dilating. I have also met with several cases of **primary** dilatation of peripheral origin, in toneless and anæmic subjects, and especially in women of middle or late middle life, in whom the arterial pressure was only moderately raised. The early detection of such cases in their recoverable stage is important; and then the manometer is most useful in following out the treatment directed to relieve the embarrassed peripheral circulation, and at the same time to tone up the myocardium.

Diseases which induce Deficient Oxygenation (Cyanosis) and Overloading of the Venous System.

When the venous pressure is raised by an embarrassed pulmonary circulation, as in attacks of **bronchial asthma,** in

the dyspnœa produced by **emphysema, cardiac disease,** etc., the capillary pressure is also raised; and in consequence of the increased peripheral resistance thus set up, the arterial pressure becomes as a rule markedly supernormal (yielding systolic readings of from 180 to over 200 mm.). When the cause is temporary, as in uncomplicated bronchial asthma, the supernormal pressure generally falls, sometimes becoming even normal, in the inter-paroxysmal periods; but when the cause is persistent, as in emphysematous, cardiac, and cardio-renal cases, the high arterial pressure remains, is not much modified by treatment, and is necessary for well-being (*see* p. 221).

Some Nervous Diseases.

Melancholia and Mania.—It is now generally agreed among alienist observers that in melancholia the arterial pressure is high, the rise being proportionate to the mental anguish, though high in some cases with stupor; and that in acute

mania the pressure is low, and especially so after the subsidence of an outbreak.

Insomnia. — The manometer often affords a useful hint as to the treatment of insomnia. It shows that in certain cases there is a hypertensive state of the circulation, such as obtains in hypertonic goutiness, when vaso-dilators are indicated, and are often alone most useful in favouring sleep; and that in other cases hypotonia with splanchnic stasis obtain, when treatment by agents which tone the arterioles (systemic and splanchnic) will frequently restore the sleeping power.

Neurasthenia.—According to my observation, the prominent features of the circulation in neurasthenic subjects are a subnormal reading of the systolic and diastolic pressures, with evidence of splanchnic stasis. There are, however, neurasthenics with vascular changes, and in them the arterial pressures, especially those afforded by the armlet, are raised.

In **epilepsy** the pressure is, I believe, as a rule, normal or rather subnormal,

and is certainly not high in the inter-paroxysmal periods, or even just after a paroxysm; and this fact may be occasionally of importance in diagnosing between epilepsy and uræmic coma, in which the arterial pressure is persistently very high. It is a mistake to suppose that nervous affections necessarily produce high arterial pressure.

Raynaud's Disease, notwithstanding its vaso-constrictive nature, does not, as a rule, produce a rise in the arterial pressure. This negative effect on the blood-pressure is doubtless explained by the limitation of the peripheral arterial constriction to a finger or a few fingers or all the fingers, and by the absence of splanchnic vaso-constriction, which is an important factor in maintaining super-normal arterial pressure.

Painful affections (*e.g.* neuralgia, migraine, angina pectoris, colic) all tend to raise the arterial pressure by inducing vaso-constriction. In **angina pectoris** the arterial pressure during the inter-paroxysmal periods will of course be

lower than that which obtains in the paroxysms. My observations, made in the quiescent intervals, dispose me to think that the relation between a permanent rise in the arterial pressure and the severity of the angina is less definite than is usually supposed. In angina vaso-motoria the pressure may be normal or only slightly in excess of normal; but in angina gravior, in which we have the cardio-vascular innervation of angina less or more complicated by cardio-vascular organic changes (*e.g.* coronary atheroma and its secondary cardiac degenerations—fatty, fibrous—aortic disease), the rise in the arterial pressures is generally quite moderate—*e.g.* from 15 to 25 per cent.—though it may reach the upper ranges, such as $\frac{230\ S}{155\ D}$, which are about the highest I have observed in this type of the disease. The manometer is generally much more useful than the finger in helping us to definitely differentiate between angina vaso-motoria and angina gravior. James Mackenzie has shown that the pulsus

alternans, or alternating action of the heart—which, according to F. B. Hoffmann and Wenckebach, indicates exhausted contractility of the cardiac muscle—is always associated with the symptom-complex of angina pectoris. He therefore regards angina pectoris as evidence of the impairment of the function of contractility. He does not think that arterial pressure is a guide to the condition of contractility.[1]

In **glaucoma** there is sometimes hypertension of the arterial system as well as of the eyeballs.

In **tinnitus** the arterial pressure may be raised, when the vaso-dilators may prove useful remedies in relieving this troublesome symptom.

HÆMORRHAGE AND ANEURYSM.

Cerebral Hæmorrhage (Apoplexy).[2]

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[1] *Brit. Med. Journ.*, 1905, vol. ii., pp. 846-7.

[2] In estimating the prognostic value of the arterial pressure in regard to the possible occurrence of apoplexy, the observer should check the armlet reading by that of the hæmodynamometer or manometer bulb; the latter reading being frequently, according to my observation, a closer rendering of the actual arterial pressure than the former. It is the pressure underlying the arterio-sclerosis, which in such a case as this is of prime importance.

—Large cerebral hæmorrhages which produce symptoms of compression of the medulla (coma, slow pulse, and stertorous or Cheyne-Stokes breathing) set up extremely high armlet readings of the systolic arterial pressure (280-300 mm. Hg.), even higher than those which have been observed in uræmic coma. In such cases the manometer enables the observer to recognise the rising pressure of the early stage, the fully developed pressure of the highest degree of compression of the medulla, and the falling pressure of the terminal stage of apoplexy; it helps him to estimate the degree of that compression, and affords him an idea of the extent and site of the lesion (a clot in the anterior having a smaller compressing effect than one in the posterior fossa).[1] Cook and Briggs have published a striking case in which the surgical evacuation of a large clot reduced a pressure of 360 mm. Hg. to 130 in twenty minutes, the pressure gradually settling to its normal (165 mm.

[1] Janeway, *op. cit.*, p. 249.

in an arterio-sclerotic patient) within two hours after the operation.[1]

Recurrent Epistaxis in subjects of over fifty years of age is, of course, of greater clinical significance when the arterial pressure is raised than when it is normal. Hence manometric observation is useful in such cases, either in suggesting early treatment for the hypertonic condition, or in allaying unnecessary anxiety as to future eventualities when the pressure is normal.

Pulmonary Hæmorrhage.—There is some uncertainty as to whether hæmoptysis is preceded by a rise of arterial pressure; and we are in need of observations on this point in phthisis. There is, however, one thing quite clear—that it is inadvisable to prescribe remedies in hæmoptysis which contract the splanchnic arterioles, such as adrenalin, ergot, digitalis, veratrine, barium, lead; remedies like subcutaneous morphia, subcutaneous

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[1] "Clinical Observations on Blood-pressure," by H. W. Cook and J. B. Briggs. *Johns Hopkins Hosp. Rep.*, 1903.

calcium chloride (gr. i–ii), and the nitrites (amyl nitrite, trinitrine) being preferable.[1]

Hæmatemisis. — Hæmorrhoidal Hæmorrhages.—A hypotonic state of the vessels will favour hæmorrhage from the abdomen; so that when the manometer indicates low arterial pressures, remedies which contract the arterioles (splanchnic and systemic) are indicated, such as adrenalin, ergot, lead, etc.

Blood-pressure measurement is also useful in estimating the hypotonic effects of large hæmorrhages and the response to treatment.

Aneurysm.—The production of aneurysm is less dependent on the general arterial pressure than on some lesion in the arterial wall, causing local weakness. Williamson finds the pressure in most cases of aortic or innominate aueurysm is either normal, or but slightly above

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[1] "Some Considerations on the Treatment of Internal Hæmorrhage by the Use of Drugs," by N. E. Dixon, M.D., *Lancet*, 1906, p. 826; see also "Vaso-dilatation in Hæmoptysis," by E. Temple Smith, M.B., etc., *Brit. Med. Journ.*, 1906, vol. i., p. 917.

normal; whereas in mere dilatation of the aorta it is much higher.[1]

Perforation in Gastric and Duodenal Ulcer.

Manometric Observation in the Diagnosis of Perforation.—It is, of course, well known that when perforation takes place, the observer relies on the general and local symptoms for guidance in view of surgical interference; but it has not infrequently happened that such evidence was insufficient, for on exploring the abdomen perforation has not been found.[2] The manometer in several cases of actual perforation having revealed a rapid and decisive rise in the arterial pressure, it becomes now a question of some practical importance to resort to manometric observation in cases of apparent perforation, before deciding on operation; and it is to be hoped that future experience will demonstrate the usefulness of this additional aid to the diagnosis.

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[1] *The Lancet*, vol. ii., 1907, p. 1516.

[2] Several cases of this kind are referred to by Dr. A. Mantle in *The Lancet*, vol. i., 1906, p. 1032.

Some Chronic Wasting Diseases.

Tuberculosis, as a rule, provides us with low readings of the arterial pressures, indicative of toxæmia and malnutrition.

Diabetes, *per se,* does not appear to me to materially affect the blood-pressure, except, perhaps, when there is much emaciation or acid intoxication, the pressures then being subnormal, as a rule. Of course, when diabetes is associated with chronic nephritis or arterio-sclerosis, the arterial pressures are supernormal.

Anæmias, whether primary or secondary, are associated with subnormal pressures, except, of course, when anæmia is secondary to kidney disease.

Addison's Disease.—Since adopting the fluid manometric method, I have not had an opportunity of observing the systolic pressure in Addison's disease. The general experience of observers testifies to the existence of low systolic readings in this disease. In some reported cases the pressure records have

been extremely low : for example, Rendle Short observed in one case from 75 to 85 mm. Hg.—the lowest systolic pressure he had encountered in any disease persisting for any length of time. This observer found the sphygmometer as important a register of progress in Addison's disease as the thermometer is in enteric fever.[1]

Acute Febrile Diseases.

Any clinical method should be welcome which will give us early warning of the approach of collapse in the course of acute febrile diseases, such as typhoid fever, diphtheria, influenza, pneumonia, etc. Manometric observation has certainly afforded some promise in this direction, providing we record the readings of arterial pressure regularly, just as we do those of temperature, pulse-rate, etc.[2] After an initial drop of

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[1] *The Lancet*, vol. ii., 1906, p. 285.

[2] For this purpose, H. K. Lewis, 136, Gower Street, W.C., has arranged an excellent chart in which may be entered the pulse-rate in black, the systolic pressure in red, and the diastolic pressure in blue.

15–20 mm. Hg. of the systolic pressure, a progressive fall to a similar extent should warn the observer of increasing cardio-vascular asthenia, and suggest the adoption of increased stimulation.

In **diphtheria** the study of the systolic pressure is important, especially in the second or third week and during convalescence, when there is frequently a drop in the frequency of the pulse, and there is also other evidence of dilatation due to granular and fatty degeneration of the myocardium. Low-pressure readings are the rule in diphtheria; and a marked fall of pressure—due to the absorption of toxins—suggests a bad prognosis. When albuminuria supervenes the pressure rises.

In **typhoid fever,** Crile,[1] Cook and Briggs,[2] and others, have shown that the arterial pressure becomes subnormal in the first or second week, and gradually more so in the third and fourth weeks: 115–20 mm. Hg. systolic pressure falling

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[1] *Journ. of Amer. Med. Ass.*, 1903.
[2] *Op. cit.*

to 100-5 at the end of the febrile period; that hæmorrhage produces a rapid and decisive fall, though it is preceded for several days by a rise (Carrière and Dancourt); and that perforation or an inflammatory complication (peritonitis, pneumonia, etc.) causes an equally sharp rise, especially in the systolic pressure.[1] Myocarditis with increased arterial tension is common in typhoid fever; and the manometer is valuable in putting the observer on the track of this and other intercurrent complications of the disease. It is important to watch the restoration of the arterial pressure during convalescence.

In **pneumonia,** Gibson[2] has observed that a sudden rise of arterial pressure before the crisis means some complication; and acute delirium is often the immediate sequel to a rapid rise of

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[1] In four cases Huchard and Amblard (*Rev. de Méd.*, July 10, 1907) observed that intestinal hæmorrhage and perforation were preceded by a transient sudden rise of arterial tension, followed by low tension and quickened pulse with a bruit in the first cardiac sound.

[2] *Brit. Med. Journ.*, vol. ii., 1906, p. 999.

pressure; while a sudden fall of pressure should warn us of the immediate risk of cardiac failure.

Intermittent or Variable Arterial Pressure.

Periodical Increments of Arterial Pressure.—Now and then certain individuals are met with, generally unstable in the vaso-motor system, or gouty in type, of fifty or over fifty years of age, in whom a supernormal radio-brachial pressure will prevail for a time—may be for a few days or weeks or more—after which it will subside to a normal, or may even fall to a somewhat subnormal pressure, again rising and falling for similar though varying periods. Such periodical increments and subsidences of arterial pressure seem to depend on recurrent tightening up followed by slackening of the splanchnic and systemic arterioles and small arteries, due to some biochemical cause. They are very common about the climacteric period, and for some years

afterwards ; and are apt gradually to merge into the progressive supernormal type. I have observed that in the years immediately following the menopause, chronic gout and arterio-sclerosis are apt to develop ; and this happens more particularly in those subjects in whom flushings and perspirations were in abeyance, or are less troublesome than usual, during the climacteric period.

Accessions of Arterial Pressure in the Senile.—Occasionally similar recurring periods of heightened arterial pressure are met with in the elderly, more especially when the middle-sized arteries are atheromatous. Such accessions of arterial pressure in the aged show that the peripheral mechanism for varying the arterial pressure is still responsive, and perhaps all the more effective in raising that pressure because of the rigidity of the arteries ; and they are generally associated with mental dulness, slowness and heaviness after meals, are menacing to life, and require prompt evacuant treatment, reduction in the bulk of the meals,

restriction of the nitrogenous foods, and withdrawal of alcohol.

I have also observed in the senile, with a decidedly diminishing and delicately balanced excretory output, a great liability to instability of the arterial pressure. In such cases the pressure for varying periods may be quite moderate or even low; and then, without very obvious cause, it may run for several days or weeks on a much higher level.

A low arterial pressure is a contributing factor in certain pathological conditions: as in **cerebral thrombosis** (arterial and venous) and **hepatic inadequacy.** In one case (*æt.* 76) in which I suspected the existence of arterial thrombosis in a non-motor area of the brain, the blood-pressure afforded by the hæmodynamometer was low, whereas that yielded by the armlet verged on the supernormal; the divergent readings and the symptoms suggesting atheroma, which may have been more advanced in the cerebral arteries than elsewhere. In this case, in which there was no renal disease

or paralysis, the mental obfuscation was at first intermittent—there being clarified intervals ; then it became remittent ; and finally it merged into coma, in which the patient died. Such a case, though comparatively rare, is now and then met with in subjects well advanced in years. We know also that a low arterial pressure is not consistent with functional activity ; hence a lowered pressure in the hepatic artery—*e.g.* from undue storage of blood in the veins (especially splanchnic), or from diminished tone and contractility of the heart muscle—may favour inadequate action of the liver.

The Modifying Influence of Composite Pathological Conditions on the Arterial Pressure.

Hypotensive Conditions modifying Supernormal Arterial Pressure.—The qualifying effect of different combinations of pathological conditions on the arterial pressure should be kept in

mind. For example, a disease like chronic interstitial nephritis, though furnishing us, as a rule, with high readings—indeed, some of the highest met with in the ordinary course of work—may now and then present us with pressures considerably lower than that which characterises the average case. Some condition which tends to diminish arterial pressure, such as anæmia, debility, a failing ventricle, the presence of bile in the blood, may modify the supernormal pressure of nephritis. Hence the lower arterial pressure sometimes met with when the cirrhotic kidney is associated with a cirrhotic liver. Phthisis, too, tends to produce a lower range of pressure in otherwise hypertensive conditions of the circulation ; and it is, as a rule, a favourable indication in phthisis when the arterial pressure becomes somewhat supernormal. Intercurrent fibrile attacks are also, as a rule, hypotensive, and conduce to a temporary lowering of the arterial pressure when it is supernormal at ordinary times.

The Influence of Pulse-rate.—The pulse-rate, too, may modify considerably the arterial pressure in disease; though its influence in this respect is, as a rule, less marked in the normal subject. May not **a slow pulse-rate** or **brady-cardia** in cardio-vascular cases be frequently corrective in its tendency, preventing the higher level of arterial pressure with its increased strain on the arterial wall, which the normal pulse-rate might induce? In that curious condition, **paroxysmal tachycardia,** the pathology of which is so obscure, it is, I believe, generally supposed that a low arterial pressure prevails. But I have met with more than one case in which I have observed distinctly high diastolic and systolic pressures, *e.g.* $\frac{160\ S}{125\ D}$ (in a light subject with normal readings of $\frac{110\ S}{90\ D}$ between attacks), and a low distal pressure; pointing to a high degree of temporary peripheral resistance and an increased ventricular output. In

tachycardia it is not improbable that the aggregate output of the ventricle is increased, though that of each systole is subnormal. Hence the rise of arterial pressure which even slight peripheral contraction may induce during the paroxysms.

Accessions of pulse-rate will doubtless have a greater effect in raising the arterial pressure when the arteries are tightened, or atheromatous, and especially atheromatous with retention of normal arteriolar contractility.

The Hypotensive Effect of Rest.— The modifying effect of recumbent rest on the arterial pressure is very considerable; hence it is that the manometer will often demonstrate how valuable periods of absolute rest are in the treatment of hypertensive states of the circulation. Hence also the somewhat modified records of arterial pressure obtained from resting and recumbent cases, as in hospital, compared with those furnished by similar patients following their ordinary course of life.

The Etiological Factors of the Clinical Variations in the Arterial Pressure.

The less or more persistent pathological deviations from the normal arterial pressure are either subnormal or supernormal.

The Causes of Subnormal Arterial Pressure (*see* pp. 88–90).—The leading factors are (1) diminution of vaso-motor tone, generally associated with splanchnic drain; and (2) a reduced ventricular output. These conditions are brought about in Addison's disease by the diminution or absence of the suprarenal secretion; and in other morbid states by various causes. For example, the increased temperature of the blood and the toxins in fever produce not only a rise in the frequency of the pulse, but as a rule a fall in the arterial pressure (from the predominance of vaso-motor relaxation); and when the system is impoverished or debilitated the pressure frequently diminishes (from reduction of cardiac output and vaso-motor tone).

The Causes of Supernormal Arterial Pressure are much more complex and varied than those which induce subnormal pressure; and in the present imperfect state of our pathological knowledge on this subject it is difficult to formulate them with any approach to certainty. I think it is probable that in most cases a persistent high pressure is the result of a chain of pathological causes; that the prominent cause or causes are supplemented by co-operating causes of minor degrees of potency. For example, it may be questioned if any of the metabolic products, so far as we know, even though they accumulate in the body, are capable of sustaining a persistently raised arterial pressure by virtue of their direct biochemical action on the heart and blood-vessels; but some of them *may* be contributory, and *may* form the initial links in the chain of more potent etiological factors.

Deposition of the less soluble residua, such as the salts of lime, magnesia, and uric acid (*see* pp. 101–6), in the areolar

tissue spaces of the body—such as those of the arterial walls, sheaths of muscles, and other fibrous structures, etc.—may excite proliferation, induration, and contraction (diffuse fibroid degeneration). The calibre of the bed of the peripheral portion of the circulation may thus become reduced, with the inevitable result of a persistent rise of the arterial pressure.

Loss of elasticity—wearing out or impairment of the rubber of the central arteries—may also be an important factor of the higher pressure of the aged, of the syphilitic and gouty; the work of the supplementary heart—the elastic recoil of the aorta—being thrown on the ventricle, which reacts beyond requirement.

But none of these causes affords a satisfactory explanation of the stage of increased vaso-motor tone which clinical observation has distinctly shown to usher in, as it were, the higher and less controllable ranges of hypernormal pressure; unless we infer that an excess of some of the normal excretory metabolic products, or the generation of some abnormal

products (such as toxins from aberrant chemical changes in the abdominal viscera) takes place, and will account for this precursory hypertonia. But the nature and effects of these substances on the arterial system and the blood-pressure have not been demonstrated. The recent work on internal secretion has, however, shown that the suprarenal glands more particularly, and the kidneys, yield products which do raise the arterial pressure.

Jusué concludes from his observations on the effects (increment of arterial pressure and aortic atheroma) of injecting adrenalin into rabbits, that hypertension and atheroma are due to overaction of the suprarenal glands; and the production of aortic atheroma (whether it be true atheroma or not) from adrenalin injections has been confirmed by other observers. Then as to the renal causation of hypertension we have the experiments of Batty Shaw,[1] which support the view held by Tigerstedt and Bergman,

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[1] *Lancet*, 1906, vol. i.

that the kidneys yield to the blood a pressor substance (renin) which raises the arterial pressure, and may account for the high ranges of that pressure which we observe in chronic Bright's disease. Experiments in the laboratory having shown that intravenous injections of extracts of nervous tissue, muscle, liver, spleen, pancreas, mucous membrane of the stomach and intestines, bone-marrow, testes, mammary gland and lung produce a fall in the arterial pressure, it does not seem improbable that the pressor effect of adrenalin and renin may, in the normal condition of the body, be kept under balanced control by the depressor products derived from some of these organs and tissues.[1] It is known that adrenalin is oxidised and destroyed; specially by the liver (Longlois) and the muscles (Schäfer and Oliver). It is held by some (*e.g.* Delaunay[2]) that the liver under the

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[1] The reader perhaps should be reminded that we have no *actual proof* that these organs yield depressor substances to the blood as specific internal secretions resembling those of the thyroid, adrenal, and pituitary glands.

[2] *Gaz. hebdom. des sci. méd.*, July 21, 1907.

influence of the nervous system regulates the amount of adrenalin permitted to pass into the general circulation. According to this view, it therefore follows that a heightened arterial pressure may be maintained, not necessarily by an over-production of adrenalin derived from a hypertrophied suprarenal medulla, but by a failure in the liver to intercept and neutralise the normal amount of suprarenal secretion, which consequently accumulates in the blood.

Apart from exogenous causation (*e.g.* by lead, alcohol, tobacco, syphilis, etc.), we might formulate a hypothesis, according to which we have (1) an endogenous biochemical cause (whether metabolic, toxinic, suprarenal, or renal) inducing hypertonia, which, being proportionate to the amount of muscular tissue, is more active in the region of the arterioles; (2) initiation of organic changes in the walls of distal blood-vessels, either from the toxic action of the product on them, or from the limitation of the capillary and lymph circulation within the walls

caused by the persistent contraction; and (3) gradual obliteration or obsolescence of capillary vessels by contraction of the interstitial fibrous tissue which supports them (diffuse fibroid degeneration).

It seems to me that the manometer is corroborating the pathology of "fibroid degeneration" brought forward by Jenner, Handfield Jones, Gull, and Sutton. My old teacher, Sir William Jenner, for many years insistently pointed out that besides the fatty and amyloid degenerations there is another, "a general fibroid degeneration." This teaching was also advocated by Handfield Jones, and it was supported by Mahomed.[1] Later, Gull and Sutton[2] demonstrated the existence of arterio-capillary fibrosis, which may be regarded as only a part, though a most important part, of the larger question of "general or diffused fibroid degeneration." At the time (1872 to 1877) the significance of this work was for the moment somewhat obscured by the comparatively less

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[1] *Lancet*, 1877, vol. ii., p. 232.
[2] *Trans. Pathological Soc., London*, vol. xxviii., 1877.

important question as to whether the undoubted thickening of the distal arteries should be referred to muscular hypertrophy or to fibro-hyaline change, Sir George Johnson upholding the former explanation.[1] We now see that Mahomed was right when he remarked, "Both probably are present"; and that our chemico-physiological conceptions suggest how the "stopcock" action of the arterioles may be accounted for in the pre-fibroid stage of arterio-capillary fibrosis.

Then it is not improbable that, in the manner of a vicious circle, a persistent rise of the arterial pressure may perpetuate itself; and Sir Lauder Brunton suggests that continued increased pressure may be a factor in producing degeneration by prolonging the diastole of the artery—the diastole limiting the supply of blood and lymph in the capillaries and tissue-spaces of the arterial wall.

There are two other possible factors

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[1] "On the changes in the blood-vessels and in the kidney, in connection with the small red granular kidney." *Trans. Pathological Soc., London*, vol. xxviii., 1877.

in supernormal arterial pressure: namely, viscosity and plethora.

As the viscosity-coefficient of normal human blood is estimated to be about four times that of distilled water,[1] and as in disease it will, like other qualities of the blood, vary very considerably, it is reasonable to anticipate that it should be a factor in determining the blood-pressure. Sir Clifford Allbutt in particular has laid some stress on the importance of this factor.[2] Not having employed a method of directly estimating viscosity, I have not been able to ascertain with certainty the influence of it on the arterial pressure. Variations in concentration of the blood ought, however, to correspond roughly with different degrees of viscosity; and these I have been estimating for many years with the hæmocytometer tube by the side of arterial-pressure measurement. Moreover, my method of separating the tissue fluid of the finger by compression

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[1] See Sherrington in *Allbutt's System of Medicine*, vol. v., p. 476.

[2] See *The Lane Lectures*.

has enabled me to obtain samples of blood as it exists within the vessels, and therefore free from that fluid. The results of these observations show that in disease there is a wide range of variation in the concentration of the blood; that when the arterial pressure is persistently high, there is generally a rise in the percentage of corpuscles and in the specific gravity of the blood; that when the pressure is low, the concentration of the blood, as a rule, also falls somewhat. I have, however, met with a good many exceptions to these general conclusions. The obvious explanation is, that the output of water by the kidneys being greater when the arterial pressure is higher, the blood is apt to become more concentrated; and that, the urinary water being lessened when the pressure is lower, the blood then tends to be somewhat more diluted than normal. So that in the ordinary run of cases the variations in the concentration of the blood may be regarded as results rather than as causes of the alterations in the arterial pressure; the

increased viscosity pertaining to concentration of the blood thus set up may, however, slightly raise that pressure. I have not had opportunities of studying the relation of viscosity to hypertonia in the interesting group of cases of splenic polycythæmia with cyanosis described by Osler. Parkes Weber has, however, demonstrated that in two such cases the arterial pressure was raised.[1]

It is also reasonable to suppose that a relative increase of the blood-volume to the capacity of the vessels should raise the blood-pressure throughout the whole circulation. There is ground for believing that such is the case. Trunecek, of Prague, divides his cases of arterial hypertension into two great classes—spastic (or contractile) and plethoric ; and the latter group he further divides into polycythæmic plethora and serous plethora. He differentiates plethoric hypertension from the spastic variety by the pulse being strong and full, but not hard,

[1] *Trans. Clinical Soc., London*, 1904, p. 115 ; *Trans. Roy Med. Chir. Soc., London*, 1905.

in all the arteries (great and small), which, however, do not become tortuous; and by distension of the capillaries and veins. The blood-volume is also increased in chlorosis, according to Lorrain Smith; but in this example of serous plethora the arterial pressure is not, as a rule, raised—a fact which may perhaps be accounted for by the low viscosity of the blood in chlorosis.

CHAPTER V.

THE TREATMENT OF SUPERNORMAL ARTERIAL PRESSURE.

In attempting to control or modify a rise in the arterial pressure we should keep in mind that, though variations of that pressure may produce certain pathological effects, they themselves are but results. When, for example, the arterial pressure is raised but **temporarily**, the cause may be, and, in fact, usually is, of nervous origin—either psychical or reflected; but when the pressure rise is **persistent** it may be regarded as the outcome generally of some alteration in the chemical composition of the plasma or of organic changes in the circulatory mechanism, which may or may not have themselves resulted from some biochemical cause. It follows, therefore, that in our efforts to control a persistent rise

in the arterial pressure we should, if possible, learn what are the etiological factors at work. Unfortunately, as our knowledge in this direction is extremely limited, we must often content ourselves with symptomatic treatment, or treatment on general lines, with the application of such practical suggestions as may be derived from physiology, which is, after all, the mother of medicine.

The Psychical Management of the Patient.

We are sometimes apt to forget that when a patient of a nervous, sensitive type realises that his arterial pressure is raised, he is apt to exaggerate the import of this knowledge, and may worry—often quite unnecessarily worry—himself about it. I have met with several such cases. We all know that our nervous patients are apt to nurse their pet worries; but when the arterial pressure is raised, perturbation is more particularly injurious, for it tends to perpetuate the evil. We

should therefore do our best to dispel apprehension, and to instil into our patient as much hope and encouragement as may be possible.

It is a good rule, when treating nervous subjects, to abstain from quoting the actual figures of an observation, especially when they are high (*e.g.* 200 mm. Hg. or more).

Dietary Treatment.

Reduction of Gastric Cardio-vascular Stimulation.—The principal aim in our dietetic directions should be to select a diet which has the least stimulating effect on the circulatory organs. Hence we should reduce as much as possible not only the bulk of each meal, but also the proportion of those constituents of the foodstuffs which more particularly stimulate the cardio-vascular apparatus, such as various salts, soluble extractives, active principles and other bodies (*e.g.* alcohol), which are directly absorbed without change, and may be

regarded as exogenous hormones between the digestive tract and the circulatory apparatus, stimulating the heart on the one hand and the capillary circulation on the other.

In the normal state of the circulation the vaso-motor play of the peripheral parts is free, and responds to the variations in the heart's action; but this harmony becomes disturbed in cases of supernormal arterial pressure, in which the stress of any stimulation of the circulation falls more and more on the heart muscle, with increment of the arterial pressure. Hence we should in such cases try to modify as much as possible the cardio-vascular stimulation produced by the ingestion of food.[1]

The next important point is to correct digestive irregularities and discomforts, and to encourage extra mastication and insalivation, especially of the amylaceous foodstuffs, which are generally so soft

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[1] See some good illustrations of the value of this teaching in a recent paper by William Russell, M.D, *Brit. Med. Journ.*, vol. i., 1906.

as not to seem to need any mastication at all, and are consequently bolted, whereas it is more important thoroughly to masticate and insalivate them than the proteid foods.

The supply of meats of all kinds should be lessened below the proportion ordinarily taken, and some are satisfied merely to reduce or to disallow the red meats (beef and mutton), while they permit the taking of white and young meats (fish, poultry, veal, etc.) in ordinary quantities. Though the practitioner nearly always is inclined to select what are called lighter meats, I cannot say that I have observed any difference of effect on the arterial pressure of the various forms of roast or fried meats—whether as beef, mutton, fish, poultry, etc. A more important difference in this respect seems to me connected with the way in which the meats are cooked—whether roasted and taken with the gravy, or boiled and served without the broth. I therefore make it a rule in these cases to substitute as much as possible boiled

for the roasted or fried forms of fish, fowl, beef, mutton, etc.; and to exclude from the dietary meat extractives, such as meat soups, gravies, etc. The proportion of green vegetables and fruits should be increased, and carbohydrate foods may be taken in moderation. As a rule I think the cases do best on the reduction of an ordinary diet as a whole, with a diminution of the animal and an increase of the vegetable derived foodstuffs. The reduction of the ingesta to physiological needs and expenditure is particularly important in the management of cases of high arterial pressure; for some of these patients are inactive and, feeling weak and run down, crave for the stimulant effects of tonics, a full meat diet, an excess of salt and other condiments, and alcoholic drinks.

The Ingestion of Fluids—their nature and quantity—is not a matter of indifference. The bulk of the meals should be kept down by reducing the quantity of fluid taken with them. Fluid, of which the best is water, should be taken more

freely when the stomach is empty—*e.g.* a tumbler an hour before meals, or night and morning. Preference should always be given to soft or distilled and non-aerated water, or to mildly alkaline waters, as potash and soda salts improve the peripheral circulation; and it is best generally to advise the water to be taken warm. Hard water—*i.e.* water containing calcium salts in such proportion as to raise the hardness above five degrees—is best avoided, especially in the elderly with a premature rise in the arterial pressure, in whom there may be an inadequate elimination of calcium salts by the bowels and kidneys. Though calcification is generally believed to follow and not to precede degeneration or involution, it may turn out that inadequate elimination may lead to the deposition of calcium salts in the arteries, kidneys, and other tissues of the aged.[1] As the combination of

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An interesting communication, "The Part played by the Calcium Salts in the Blood and Tissues," appears in *The British Medical Journal* of April 20, 1907, in which W. Blair Bell, M.D., refers to three calcium physiological

calcium with an organic acid (*e.g.* citric) deprives the calcium ion of its specific physiological effect on the blood,[1] the addition of lemon juice to hard water may be a useful corrective in the elderly and gouty.

The Beverages, such as tea and coffee, are thought by some observers to be particularly noxious. Long-continued indulgence in strong tea and coffee is undoubtedly injurious, but I have seen no objection to these beverages in strict moderation, and when their ingestion is not followed by disturbing results, such as palpitation, flushing, giddiness, or wakefulness. Cocoa is the least objectionable of beverages. As to **alcoholic beverages** my impression is that high-pressure cases as a class are better without them, but that many cases in which we

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periods. "The third period is that of late life, when no calcium salts are required for the building up processes or for those of reproduction. These salts then accumulate in the tissues, especially in the vessels . . . atrophy occurs, and the individual gradually declines."

[1] See J. Coleman, *The Bio-Chemical Journal*, April, 1907.

have moderate or slight increments of pressure may be allowed small quantities without injurious effects, especially if, as a rule, we exclude wines and malt liquors. There is no doubt that when the vaso-motor system is intact, alcohol (as in the form of whisky or gin) is vaso-dilatory and has but a transitory effect in raising the systolic pressure. The latest experimental work on alcohol (that of W. E. Dixon of Cambridge)[1] shows that in moderate doses alcohol in normal animals and also in man may raise the systolic pressure a little (and especially in animals showing signs of circulatory failure), though it may not do so, while it always diminishes the diastolic pressure ("in other words the difference between the systolic and diastolic pressure tends to increase"), and that it increases the activity and output of the heart, an effect which is ascribed to the fact that alcohol is a readily assimilable food substance.

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[1] "The Action of Alcohol on the Circulation," by W. E. Dixon, M.A., M.D. Lond.—*Journal of Physiology*, March, 1907.

Chloride of Sodium as a constituent of foodstuffs and as a condiment deserves some notice in the treatment of supernormal arterial pressure. The work of Widal and Javal[1] and other Continental observers has shown that superchloridation and superhydration of the tissues take place when the kidneys become impaired in their permeability to the passage of chlorides, as in chronic nephritis and probably in other phases of renal inadequacy not recognised as organic; and that some of the manifestations of Bright's disease and heart disease (*e.g.* œdema, albuminuria, etc.) are closely associated with this tissue retention of chlorides and water and are relieved by a dechloriding diet. At present I cannot say from my own observation what is the rôle, if any, which the retention of chlorides plays in the production of the high ranges of arterial pressure which we meet with in chronic interstitial nephritis.

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[1] La Cure de Déchlorisation, par le Dr. Fernand Widal et le Dr. Adolphe Javal: Paris, 1906.

Probably it is insignificant; but I think it advisable to test the matter by observation. To apply efficiently a dechloriding diet the mere omission of salt as a condiment is insufficient. We must besides this select those articles of food which in the uncooked state contain the least percentage of salt, and omit the addition of salt in their preparation for the table. The diet thus obtained is practically, though not absolutely, chloride-free. Such a saltless diet may consist of vegetable soups (made without meat stock), fresh green vegetables, fruits, nuts, fats (fresh butter, cream), salt-free bread, farinaceous vegetables (potatoes, rice, peas), and sugar. Dufour (in 1905), Guinon and Pater (in 1906), and Déléarde (1907) obtained excellent results from a dietary like this in scarlet fever and scarlatinal nephritis. But in chronic nephritis and in other cases of high arterial pressure such a strict chloride-free diet will scarcely be necessary, though it may perhaps be adopted with advantage for a while now and then,

when accessions of pressure arise. In fact, a saltless diet soon becomes so distasteful to the majority of patients that it is difficult for them to continue it for more than a week or two at a time.

I have observed that as we grow older (say over 65 years or so) there is apt to arise a tendency to indulge more and more in the use of salt. When the arterial pressure rises unduly in elderly subjects we should do well to look out for this disposition. The suggestion of the late Sir W. Roberts, that we should substitute chloride of potassium for chloride of sodium as a table salt, is supported by Bunge,[1] who has shown that when potassium is ingested in excess of sodium a loss of the latter from the system takes place. Unfortunately, chloride of potassium is by many disliked as a table salt, but it is less objectionable when combined with 50 per

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[1] G. Bunge: *Text-book of Physiology and Pathological Chemistry* (translated by F. A. Starling), 1902.

cent. of sodium chloride, or with potassium nitrate. The following formula of an apressor table salt may be found useful: ammonium hippurate, 1 part; potassium nitrate, 4 parts; potassium chloride, 25 parts [1]

A strict milk diet, sufficient in quantity to maintain the health and condition of the patient—*e.g.* six pints a day—supplies to the system from 100 to 200 grains of salt (different authorities giving the salt percentage of milk as ranging from 1·6 to 3·0), so that such a diet is by no means saltless. Nevertheless, experience shows that a milk diet or **a lacto-farinaceous diet** for a few weeks has sometimes an excellent effect on high arterial pressure and on the general health.[2] Unfortunately, milk is often

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[1] Messrs. Oppenheimer, Son & Co., prepare a portable form of this salt.

[2] See an address on Arterio-sclerosis by W. P. Herringham, M.D., *Brit. Med. Journ.*, vol. i., 1907, p. 61. According to the recent experiments of C. J. Coleman, milk as a diet does not appear to increase the coagulability of the blood, as much of its calcium content is not available as a factor in controlling the coagulation, for it is probably combined with *organic* (and not *mineral*) acids, which

badly borne in some cases, especially when the bowels are inclined to constipation and the liver is inactive; but even then it may often be made to agree by adding to it citrate of soda or potash, or when there is gastric acidity sodii bicarbonas or liquor magnesii carbonatis. But **a lacto-vegetarian diet** is more suitable in most cases; a moderate daily quantity of milk (*e.g.* two or three pints) when taken with vegetable food-stuffs (bread, rice, green vegetables, and fruits) should not seriously raise the low percentage of ingested salt and, moreover, it advantageously increases the proportions of the proteid and fat, especially when combined also with cream, cheese, and eggs. Cheese in moderation is perhaps the least objectionable form of proteid food in these cases. Meats of all kinds contain even a smaller percentage

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combination does not shorten the coagulation time. (*The Bio-Chemical Journal, op. cit.*) So it is not improbable that if calcium favours a rise of arterial pressure (*vide seq.*) milk may fail to do so, just as it fails to increase the coagulability of the blood.

of native chloride than milk; but the cardio-vascular stimulant effect of the extractives which they contain more than counterbalances any advantage they possess in their relatively low percentage of salt. Boiled meats, however, containing less salt and extractives than roast meat, may be added to the dietary without disturbing its comparatively salt-free character. Fresh-water fish (such as trout, tench, pike, and crayfish), which contain much less salt than salt-water fish, may also be taken if we do not require to adhere to a strict minimum supply of ingested salt. Caviar, pickled salt-water fish, molluscs, and shell-fish should be avoided. The condiments—pepper, mustard, vinegar, lemon, etc., when taken in moderation, are useful in relieving the insipidity of the dechloriding diet.

When the systolic arterial pressure maintains a high range this dietary should be tried, and especially when there are albuminuria and any œdema, however slight. But even when the arterial pressure is equally high and

there is not even the merest trace of albumin in a pellucid urine, we may still entertain a suspicion of kidney insufficiency—such as may diminish the permeability of the chlorides; and we may find a trial of the dechloriding diet as useful in such cases as in the albuminuric ones. When the kidneys are sound and normally permeable to sodium chloride it is not likely that the ordinary use of salt will interfere with the eliminatory work of the kidneys or will raise the arterial pressure, except quite temporarily; for normal renal elimination of the chloride is too rapid to allow of a permanent rise in the percentage of it to take place in the plasma and tissue fluids. We may therefore infer that if there is a permanent retention of chlorides it will be invariably pathological and not a physiological condition, and our aim in treatment is to restore the chloride equilibrium by reducing the amount of the ingested chloride to that which is eliminated by the kidneys. The question,

therefore, is one of renal inadequacy in the elimination of the chlorides.

Should the arterial pressure be permanently raised without there being any failure in the elimination of chlorides, urea, and the xanthin bases, the reduction of ingested salt is not likely to lower the arterial pressure—though it is well to test the matter by observation.

Tobacco smoking is sometimes looked on as a questionable habit when the arterial pressure is increased, and knowing that nicotine raises that pressure and will even produce something like arterio-sclerosis in animals we may be disposed to look on smoking with suspicion. But smoking does not necessarily imply the absorption of all the nicotine contained in tobacco—as when, for example, the weed is chewed. For combustion breaks up the nicotine into such products as pyridin, carbon monoxide,[1] etc., which predominate in

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[1] See *The Lancet*, Jan. 2, 1904, p. 43; and Feb. 6, 1904, p. 395 (J. S. Thompson, M.D.).

the smoke, though this may also contain a little nicotine. Hence the physiological effects of smoking on the arterial pressure will be due to the combined action of all the products of combustion as well as those portions of nicotine which escape destruction and which may be directly absorbed from the end of a cigarette or cigar. From the observations which I have made on myself and other normal subjects not habituated to smoking, I find that it always raises the systolic pressure (*e.g.* from 10 to 20 mm. Hg.) without producing a corresponding rise in the diastolic pressure; in fact, the latter pressure will generally either remain unaltered or may even fall, the pulse pressure range (*i.e.* the difference between the minimum and maximum pressures) being increased. The effect, as a rule, passes off quite quickly, generally in ten minutes or so; but notwithstanding this fact, it is undesirable in the management of cases of supernormal arterial pressure to permit indulgence in a habit which raises the systolic arterial pressure.

Besides, Barazzoni found that in subjects with high-tension pulses the rise of the tension produced by tobacco smoking is more exaggerated than in subjects with low or medium tension, and leads to dyspnœa, palpitation, præcordial distress, vertigo, tremors,[1] etc. In most cases, when the habit of smoking can be given up, it is best to encourage the patient to do so; and abstinence can generally be easily enforced in those who have been light or occasional smokers or who experience slight palpitation, heart hurry, anginal pain, or other discomfort after smoking. But in the habitual smoker it is not always easy even to limit the indulgence, though we should endeavour to do this whenever possible.

Rest and Exercise.

The value of **absolute rest** in the management of high-pressure cases should be emphasised; for I doubt if we all quite realise it as fully as we ought to do.

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[1] *Gazzetta Medica Italiana*, 1905.

We not infrequently see its calmative influence on the arterial pressure of bed cases (as in hospital), the pressure readings in similar pathological conditions being generally lower in them than in patients leading their ordinary lives. Absolute rest is the best sedative of exalted ventricular action, that large factor in maintaining supernormal pressure, especially in the higher ranges; and this is often apparent even when the response of the peripheral arteries to a vaso-dilator is either greatly limited or is not apparent. In many such cases increments of pressure recur from cardiac excitation induced reflexly or biochemically, when recumbency for varying periods is invaluable, even though the heart muscle may not show signs of actual failure. The periods of absolute rest required may vary from a day once or twice a week to even three or four weeks at a time [1]; and after the lapse of

[1] During the prescribed rest evacuant treatment of the liver and bowels should be more particularly maintained.

the longer interval of resting, care should be taken to see that the assumption of the accustomed exercise is gradual, or the good effects of the recumbency may be nullified.

Then, on the other hand, the prescription of **exercise** is important.[1] When the arterial pressure rises beyond a certain moderate degree the patient is apt to become somewhat lethargic and inactive. We should in such cases encourage the taking of as much regular, moderate, and equable exercise as the patient's limit will safely permit. Walking is the best of all exercises, but, unfortunately, even when indulged in quite moderately, these patients frequently complain of early "fagging." Then in suitable cases gentle cycling on roads of low gradients

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[1] We have seen that rest may lower the arterial pressure by calming an overtaxed ventricle in a state of irritable activity. On the other hand, the moderate exercise of the muscles may also diminish the pressure by: (1) oxidising and destroying the adrenalin, which maintains their tone and, by accumulation in the blood, raises the arterial pressure; (2) generating lactic acid and perhaps other depressor metabolic products; and (3) dilating the peripheral circulation.

and with a low-geared cycle is preferable, or horse riding—a form of exercise which we are apt to overlook in this mechanical age. All sudden forms of exertion should be avoided, such as straining at stool, etc. But the prescription of exercise when there is a rising pressure in the elderly requires discrimination and caution. Many men over sixty years of age, who have led sedentary lives, on retiring from business are apt to adopt the ways and games of young men, and, being unconscious of the fact that they have already used up much of their endowment of rubber in their arteries, are apt to come to grief in cardio-vascular breakdown. Such cases are an object-lesson to us to preach moderation in physical exercise to the elderly in whom we detect signs of diminished elasticity and play in the vascular walls, so that they may be induced to economise their remnant of rubber. Still, on the other hand, moderate activity in the elderly is generally beneficial, and we should always remember that after middle life involution and

degeneration are fostered by inactivity; hence a sudden retirement from active business often leads to a premature break-up. The cardio-vascular stimulation of exercise should always be avoided just after a meal. The patient should then rest recumbent or semi-recumbent for a good part of an hour; and after that interval has elapsed he should always take exercise, however limited it may be. Special exercises, such as tension exercises, and graduated resistance and slow deep breathing exercises, are often valuable in widening the peripheral vessels and in letting down the arterial pressure; and massage is useful when the heart begins to weaken and is not equal to efficient active exercise.

Balneological Treatment.

Some experience in the use of different kinds of baths in the treatment of these cases has clearly indicated that some balneological procedures are more potent than others in relieving supernormal

arterial pressure. **Immersion in warm fresh water** lowers the arterial pressure chiefly by relaxing the cutaneous and splanchnic arterioles; but this effect is quite transitory, and the cutaneous circulation is left more amenable than before the immersion to the contracting influence of a lower temperature. But when the water contains or is charged with certain chemicals (such as chlorides, sulphides, extract of pine, peat, and carbonic acid) the lowering effect on the arterial pressure is prolonged. Hence the calmative influence of **peat, sulphur, pine, saline, and carbonic acid** baths, followed by dry, warm packing on the circulatory organs and the nervous system, and their adaptability to the treatment of those cases of increased arterial pressure associated with some phase of nervous irritability, or with contraction of the cutaneous blood-supply and some inefficiency of the skin as an emunctory organ. A course of such bathing will produce good results, even when the arterial pressure is moderately increased.

But I rarely find it desirable to rely upon these still baths entirely, for they are apt to leave the muscular system relaxed and toneless. They are more valuable as intercurrent baths in a course of **massage bathing,** which exerts a more decided effect in lowering the arterial pressure while maintaining or even improving the nutrition and tone of the muscles. **Warm massage douching,** exemplified by the Aix-les-Bains douche, followed by the needle bath of alternating temperature and warm pack, is, according to my observation, one of our most valuable balneological procedures for treating cases of supernormal arterial pressure. In it we have in synergic combination the action of warmth, massage, and vibration in diverting the blood to the peripheral vessels of the richly endowed vascular area of the muscles as well as that of the skin.

Electrical Treatment.

My experience of the therapeutic value of electricity in cases of supernormal arterial pressure is somewhat variable.

It has scarcely given me sufficient confidence in its efficacy to encourage me to rely on it as the sole mode of treatment beyond the dietetic and general management. But I have not infrequently found it to be a useful adjuvant when resorted to along with other balneological procedures.

Radiant electric light baths, which secure active cutaneous elimination at a comparatively low temperature, have proved most useful in many of my cases; and I frequently advise their continued use twice or three times a week.

The D'Arsonval current for twenty minutes daily, with or without bipolar massage, or the electric breeze, or the sparkling application to the limbs and body, has occasionally afforded me encouraging results. Montier has adduced some rather striking evidence in support of the reducing effect of D'Arsonvalisation in hypertension; he having, for example, observed in some cases, even after a few *séances* only, a fall from such high readings as 220 or 230 mm. Hg.

to 150 or 160—a fall which was, moreover, remarkably persistent notwithstanding the discontinuance of further electric treatment.[1] I have not myself observed such satisfactory results.

Schnee's four-cell electric bath (galvanic or sinusoidal) is another form of electrical treatment which I have found useful in hypertension, especially in chronic gouty subjects.

Care of the Skin.

It is important in these cases to maintain the cutaneous circulation at its optimum efficiency. Warmth diverts to the skin a comparatively large volume of blood, which lowers the general arterial pressure; hence the value of **warm efficient woollen clothing** and of **warm equable climates** (such as those of Jamaica, India, or Egypt) during our winter and spring months. **Cold,** and especially **cold windy climates,**

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[1] *Journal de Physiothérapie,* 15 août, 1905.

are most injurious in high-pressure cases.[1]

Cutaneous elimination should be encouraged; a thorough perspiration should be obtained once or twice a week by means of a **lamp bath** (home Turkish bath), or an **incandescent light bath,** or a **Turkish bath,** followed by efficient massage and removal of effete epithelium. By active perspiration the plasma and tissue-fluids are relieved of an excess of sodium chloride and metabolic residua.

Venesection.

So far I have had no experience of venesection in the ordinary course of treatment of high-pressure cases, apart from the actual accidents of uræmia

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[1] High altitudes, even though the air is still, are contra-indicated in these cases; chiefly because of the strain thrown on the heart by the cold and the accelerated breathing necessitated by the altitude. Hence the injurious effects are more apparent when the heart muscle is beginning to fail and when the breathing is easily disturbed by exertion. Sheltered moderate altitudes are, however, sometimes beneficial in hypertonia and supernormal pressure when the heart is fairly equal to its work.

and cerebral hæmorrhage. Now that we have in the clinical manometer an instrument of precision to guide us in selecting the right case for venesection, should we not avail ourselves of the rapid hypotensive effect of this measure more often than we do when suspiciously threatening symptoms arise, or when in certain plethoric subjects a very high pressure does not respond sufficiently to the ordinary preventive measures, especially if the free use of vaso-dilators is not effectual? Under such conditions many years ago I used to apply Junod's boot, with apparently favourable results; but I think a limited venesection would be still more effectual than this. The apparatus acts like a magnified cupping-glass; it consists of (1) a metal boot sufficiently large to accommodate the leg up to the thigh, where the opening is rendered air-tight by a broad band of indiarubber, and (2) an air-pump attached to a nozzle communicating with the interior of the boot through which the air within is rarefied.

Medicinal Treatment.

Sometimes dietetic treatment, regulation of rest and exercise, and some balneological procedure—such as thermo-massage—will suffice to correct the increment of arterial pressure, more especially in its slighter degrees. But in most cases it will be found that some medicinal treatment or other in addition will be either useful in expediting the progress or will be necessary. That treatment may be directed into one or other of the following lines—namely (1) to secure (*a*) free evacuation and correction of the liver and bowels, (*b*) gastro-intestinal antisepsis, and (*c*) sedation of the gastro-cardiac reflex; and (2) vaso-dilatation and sedation of the vaso-motor nervous system.

Evacuant and Antiseptic Treatment.—There is no doubt that nearly all forms of supernormal arterial pressure are benefited less or more by purgatives; not so much, however, by the occasional aperient dose as by a daily increased

discharge from the bowels. The benefit thus derived may be ascribed to (1) the stimulation and correction of the liver and other abdominal glands; (2) the daily baling out through the intestinal mucous membrane of salt[1] and water from the blood and the tissues, with stimulation of the intermediary circulation; and (3) the removal of intestinal toxins. To be efficient the aperient must produce a free watery discharge. Saline purgatives should be taken in hot water in the early morning, such as sodii sulphas, or soda tartarata, or sodii sulphas with magnesii sulphas; and, in addition, there should be an occasional mercurial dose—*e.g.* once or twice a week (calomel or pilula hydrargyri) at bedtime. Just now there is a widely entertained belief that the generation of toxins in the gastro-intestinal area (chronic intestinal auto-intoxication), is the possible, if not

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[1] The normal proportion of salt in the fæces is greatly increased in diarrhœa. *Vide* "L'Elimination du Chlorure de Sodium par la Diarrhée" (*Comptes Rendus de la Société de Biologie*, 1903, p. 929).

probable, cause of several ailments of obscure origin, such as gout, arterio-sclerosis,[1] etc. Metchnikoff has shown us the importance of avoiding the ingestion of bacteria in raw fruit and vegetables by heating and cooking them. But cooking is objectionable in depriving certain raw fruits and vegetables of their delicate and appetising flavour. In the case of grapes, salads, and perhaps strawberries, we may, however, render them aseptic without impairing their natural flavour by dissolving one of Dr. Rideal's tablets of bisulphate of soda in ten ounces of water and using this as a wash water. Intestinal antiseptics may often be used

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[1] During the past summer I have noted the arterial pressure of patients suffering from colitis, who have undergone the Plombières treatment at Harrogate. In those in which the pressure was raised I cannot say that I have observed any marked or appreciable fall of it which could be ascribed to the improvement of the colon, even in the most successful cases. Moreover, I have met with several cases of pronounced chronic colitis affording a somewhat low arterial pressure, or certainly without any rise whatever over the normal pressure. From the study of these cases I cannot say that I have discovered any direct evidence showing that colitis is pathologically connected with arterio-sclerosis, gout, or a supernormal arterial pressure. I am therefore rather disposed to doubt if the colon is responsible for the production of a pressor toxin.

with advantage when the arterial pressure is persistently raised, such as benzo-naphthol, salol, salacetol, and tylmarin (acetylated ortho-coumaric acid), which tend to ease the peripheral circulation like the other members of the aromatic series. Mercurials are also not only antiseptic, but relax the arterial wall, and I have observed that parvule doses of calomel (*e.g.* tabloids of $\frac{1}{10}$th grain) twice or three times a day may be taken for a few weeks now and then with a favourable effect on the arterial pressure. The red oxide of mercury tabloids ($\frac{1}{20}$th grain) are also worthy of a trial. Small daily doses of sulphur—such as the compound sulphur or the guaiacum and sulphur tabloids—are not only useful as laxatives but in favouring a fall of the arterial pressure.

2. **Sedation of the Gastro-cardiac Reflex.**—In many cases of supernormal pressure of the paroxysmal type, as in angina pectoris, it is of some importance in the medicinal treatment to soothe the nerve-endings in the stomach. This may

be attained from the medicinal standpoint by administering before meals, when the stomach is empty, such remedies as bismuth (carbonate, trisnitrate, salicylate), cærium, hydrocyanic acid, sodium bicarbonate, magnesium carbonate, small doses of liq. morphiæ, etc., in those combinations which seem to the prescriber to be best adapted to the case in hand; and it is often advisable to continue such treatment for lengthened periods.

3. **Apressor Remedies.** — Arterial pressure may be reduced either by diminishing the output of the ventricle or by widening the bed of the whole arterial system, and especially its peripheral portion. Experience shows that it is futile, if not injurious, to attempt to obtain a depressor effect and to maintain it by drugs, which lower the action of the heart. Just lately Coley, of Philadelphia, for example, has shown that such drugs as aconite, veratrin, antimony, and chloral are unsatisfactory and scarcely justifiable remedies for the purpose of

reducing the arterial pressure.[1] Our choice of apressor remedies is therefore restricted to those which favour dilatation of the calibre of the arteries, and thus ease the flow of blood through the distal portion of the circulation. Their physiological action on the arterial pressure (distal and proximal) is easily demonstrated by the manometer in man by administering a dose of nitro-glycerine; and I have elsewhere shown how useful it is in the course of clinical observation thus to gauge the vaso-dilator response of the arteries (*see* p. 125).

Desideratum in an Apressor Remedy.—Control of a persistently high arterial pressure by drugs is less or more unsatisfactory. Should an impression be made, there is the gradual failure to maintain it, or the great liability—often, indeed, the certainty—of the recurrence of the higher pressure on discontinuing the remedy or on reducing the dose of it. The effect is, indeed, rarely curative.

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[1] *Brit. Med. Journ.*, 1906, vol. ii., p. 1454.

The desideratum in an apressor remedy seems to be the discovery of some agent which will modify arterial pressure on the lines followed by nature—some product or other which may be continued without harm so long as it may be required. We may, as experimental work proceeds, find the solution of this question in physiological chemistry, and we are already beginning to realise that arterial pressure may be the resultant of the interaction of pressor and apressor products supplied to the blood by the internal secretions of the various organs and tissues, and that the higher ranges of pressure we meet with may arise from the predominance of the pressor products, the anti-bodies being generated in insufficient quantity.[1]

The Depressor Effect of Ingested Thyroid Gland.—We know that the adrenal and the pituitary glands and the kidneys yield pressor bodies; but our

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[1] The accumulation of pressor products in the blood may also depend on insufficient destruction of them, *e.g.* adrenalin by the liver.

knowledge of apressor secretory products is much more limited—the internal secretion of the thyroid gland being about the only one with an experimentally proved depressor action. I have observed that the ingestion of the thyroid gland increases the calibre of the arteries and lowers the arterial pressure, and in many cases of increased arterial pressure it is sometimes useful, especially in subjects over forty-five or fifty years of age of a gouty or obese type; but it is not a remedy which should be prescribed in a routine way in the majority of cases. When given in guarded doses with each meal, it will often modify the pressure and improve the general health, and then it may with occasional supervision be safely continued with advantage for long periods. On the other hand, I have seen quite small doses of thyroid produce cardiac irregularity and dilatation in arterio-sclerosis. The supervention of precordial uneasiness, palpitation, or irregularity of the heart's action should at once suggest the discontinuance of

the remedy or the reduction of the dose. Thyroid is sometimes better borne when combined with a small dose of strophanthus or digitalis; and it may be advantageously associated with a little free iodine (tinct. iod.). It has been thought by some (Stockman, Rolleston)[1] that the iodides exert their beneficial effect on arterio-sclerosis and heightened arterial pressure by stimulating the thyroid; they do not, however, reduce the arterial pressure. And just lately Chalmers Watson[2] suggests that the good results obtained from a bread-and-milk diet observed by Herringham[3] in certain cases of arterio-sclerosis with increments of arterial pressure, arise from the profound influence of a diet, in which milk largely preponderates, on the structure of the thyroid gland which he has demonstrated in animals—this therapeutic effect being, according to him, "indirect,

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[1] *Clinical Journal*, 1905.
[2] *Brit. Med. Journ.*, vol. i., 1907.
[3] *Op. cit.*

the thyroid gland spontaneously recovering under the influence of lessened strain on its functional activity."

The Depressor Effect of some other Normal Constituents of the Plasma.—But I think we may question whether the internal secretion of the thyroid is the only apressor agent present in the plasma, notwithstanding its obvious importance. Is it not probable that the arterial pressure is normally modified by some of the saline constituents, some tending to raise the pressure, while others lower it in proportion to their preponderance? The quantitative relation between the various salts of the serum is probably maintained within certain limits in health; and in disease that relation may at times be disturbed either by the undue ingestion or the defective elimination of certain salts (such as calcium carbonate and sodium chloride). Somewhat cognate physiological facts point to the important influence of the salts of the plasma. Ringer has shown that the salts of calcium,

sodium, and potassium must exist in an ideal circulating fluid in the right proportion, and those of calcium are of singular importance in maintaining the contractility of the ventricle; and Waller has demonstrated that potassium depresses and calcium augments the variations of electric response in nerve. Moreover, we also know the important rôle played by calcium in the coagulation of blood and milk. May not a similar relationship subsist between the various salts of the plasma and the blood pressure? In man the depressor effects of the potassium salts, whether as carbonate or citrate, on the arterial pressure are often more striking than might be expected from the results of experimental work; and the maintenance of a lower level of pressure thus obtained may be partly due to the corrective effect of the ingested alkaline salts on the disturbed balance produced by the predominance of pressor salts. In practice we frequently combine the nitrates and nitrites with the carbonates or citrates of potassium. Sir Lauder Brunton associ-

ates nitrate of potash (20 grains) and nitrite of sodium (from a half to two grains) with this alkaline treatment (potassium bicarbonate) in a large tumbler of water in the early morning; and I have observed excellent results from a continuance of this prescription in many cases of decided supernormal pressure. Alkalies are generally well borne by such cases for long periods. But my observations in man dispose me to conclude that there may be other salts normally present in the plasma which tend to lower the arterial pressure, such as the hippurates and benzoates. I have given these salts a good clinical trial during the past two years or so, and I am satisfied that they are useful in many cases of hypertension and of moderate increments of arterial pressure. As a rule they improve the general health and well-being, and are well adapted for long-continued use in the preventive treatment in the hypertonia of goutiness and arterio-sclerosis and especially in the elderly. In many cases of hypertension, especially in women and

overworked anxious men, I have observed that the association of ammonium bromide with ammonium hippurate or benzoate is still more beneficial in providing a higher degree of sedation of the vaso-motor nervous system than is furnished by either remedy alone. There are also other remedies furnishing hippuric acid to the system which are worthy of trial in cases of supernormal arterial pressure, such as quinic acid, styracol, tylmarin, which are obtainable in the tablet form. It is interesting to find how many remedies with a benzene nucleus are useful in gout as well as in arterial hypertension.

Exogenous Apressor Remedies.— The most important of these vaso-dilator remedies are the nitrites of amyl, isobutyl, glyceryl, sodium, erythrol, and mannitose ; all of which vary in the energy and duration of their vaso-dilator activity. The most active members of the group, amyl and isobutyl nitrites and nitro-glycerine, have the shortest dilating effect, and are most useful in combating

the paroxysms of the hypertensive disease, angina pectoris ; whereas the other members, the nitrites of sodium, erythrol, and mannitose, exert a more prolonged, though a somewhat less energetic, control over persistent arterial tension—lowering the average level of supernormal pressure, and guarding against accessions of it. We should not suppose that every case of supernormal pressure should be treated by some member or other of the nitrite group. To do so would surely remind us of the crude practice of the early days of thermometry, when antipyrin, aconite, and other anti-febrile remedies were too frequently prescribed in a routine way to suppress every rise of temperature. The nitrites should, as a rule, be held somewhat in reserve at first, and should not be employed until after the failure or partial failure of the preventive or corrective treatment ; and their use and dosage, when indicated, should be the result of experimental observation in each case. The dose and its frequency having thus been properly adjusted, the

nitrites may in suitable cases be continued for lengthened periods when necessary; or they may be more particularly prescribed during periods of active increments of pressure which are apt to supervene. I am just now putting on trial a combination of all the slowly acting vaso-dilators,[1] which appears to me of some promise.

Vaso-dilators may be advantageously combined with cardiac and nerve tonics; but it is always advisable in such cases, every now and then, to watch the effects of the continued use of vaso-dilators on the heart and the nervous system—for all the nitrites tend in the course of time to diminish the tonicity of the ventricle and to impair nerve tone. Should the slightest indications of dilatation appear, they should be combined with small doses of digitalis or strophan-

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[1] The combination consists of sodium nitrite, gr. ½, erythrol tetranitrate, gr. ⅓, mannitose nitrate, gr. ¼, ammon hippurate, gr. j, made into a chocolate tabloid (B. W. & Co.), which may be called "Tabloid Sodium Nitrit. Co."

thus, or both[1]; and should the nerve tone fail, the vaso-dilators may be associated with small doses of quinine, strychnia, and phosphorus.

Iodine, as potassium and other iodides, is another apressor remedy which has for many years been relied on as our sheet-anchor in the treatment of arterio-sclerosis, whether it is associated or no with a rise in the arterial pressure. And in cases of this type the iodides are often surprisingly well borne in large doses by the mouth, without producing the symptoms of iodism or of depression; but I do not find that the manometer shows that these remedies exert a direct reducing effect on the arterial pressure at all, and therefore my observations agree with

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[1] A good formula is that of Messrs. Parke, Davis & Co. in their tablets Tinitrin co. (digitalis, strophanthus, and trinitrin). I also find the following pill after meals three times a day useful in hypertension of the arterial wall with slight or moderate supernormal arterial pressure and cardiac dilatation (apex beat displaced to the left with submammary tenderness—a reflex from the overtaxed ventricle): pulv. fol. digitalis, extr. nucis vom. *aa* gr. ½, ammon. hippurat. gr. j, pepsin porci (Fairchild) gr. iij, extr. gentian, q.s.

those of Stockman, Burnet,[1] Janeway, and some other observers. But notwithstanding this fact, I have often found a long continuance of the iodides in small doses useful in reducing supernormal arterial pressure. I cannot, however, say that I have observed a palpable reduction of the thickening of the accessible arteries from the prolonged use of these remedies, even in full doses. The preparations, iodopin and iodalbin (Parke, Davis & Co.) are perhaps worthy of prolonged trial in arterio-sclerosis.

In observing the effects of treatment by the twofold manometric method, the practitioner should be reminded that the armlet should always be applied to the same area on the same arm; for in cases of arterio-sclerosis the readings obtained from the arm and forearm on both sides are more variable than when the arterial wall is normal.

Another point to be remembered is,

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[1] "Therapeutic Action of the Iodides," *The Lancet*, Sept. 8, 1906, p. 646. Dr. Burnet advocates the subcutaneous use of iodopin.

that the observations should be made as near the same time of day as possible. This precaution is well exemplified by the exceptionally large daily physiological range observed in a patient previously referred to (p. 98). The fallacy of testing the effects of remedies in such a case by substituting an afternoon observation for previous morning ones is obvious. The conditions prior to the observation should also be uniform. The patient just mentioned can substitute for some hours the lower for the higher pressure range by playing a few rounds of tennis, or by taking an electric radiant heat bath ; so the depressor effects of prolonged exercise, such as hunting, walking, or cycling, finishing even an hour or so before an observation, may mislead the observer.

I have generally observed that the armlet and the bulb readings agree in denoting the variations of pressure which are met with in the course of treatment. Nevertheless, I have come to regard the bulb-readings as not only more convenient and time-saving, but as being more closely

in accord with the alterations in other clinical data induced by treatment, such as a reduction in the hardness or resistance of the pulse, the evidence of diminution of the ventricular load and of the strain on the aortic valve, etc.

The Amenability of Increased Arterial Pressure to Control.—Cases present decided differences in their response to treatment. This disparity is doubtless largely due to variations in the cause and in the stage of development of the pathological condition. The effects of treatment also differ considerably in the two classes of cases—namely, in the cases in which we can freely prescribe rest and regulation of exercise and life generally, and in those who must follow their avocations and their ordinary routine of life. In the former class the results are often more satisfactory, even when the pressure runs into the higher ranges. But the fact remains that the pressure is much more amenable to control in some cases than in others, in which it may be, and not infrequently is,

practically uncontrollable. My observations lead me to the general conclusions—to which, however, I have met with some exceptions—that there is an inverse ratio between the observed reaction to vaso-dilatation and the degree of persistent supernormal pressure; and that in cases following their ordinary course of life the amenability of the pressure to be controlled by treatment diminishes in a general way in proportion to the rise of pressure—the cases having persistent systolic pressures of over 200 mm. Hg. by the armlet method furnishing by far the majority of those which resist appreciable modification by treatment. But these conclusions are only such as might have been anticipated, for a high percentage (probably about 90) of the cases presenting systolic pressure of over 200 mm. are cases of chronic nephritis or of diffuse arterio-sclerosis—with or without renal implication; and in a large proportion of those in which the pressures are below 200 mm. the rise is, as a rule, the outcome of conditions less or more remediable.

The percentage of cases regarded as amenable to treatment will also be considerably influenced by the practitioner's conception as to whether the pressure is normal or supernormal in any particular case. There being no such narrow line as that of the normal temperature, each observer, though knowing the general range of the normal pressure, has gradually to build up from his experience a correction for individual instances; being mainly guided in this by the collateral signs and symptoms and by the effects of treatment in borderland cases. When, for example, he discovers that a systolic arterial pressure of 145 or 150 mm., which he regarded as doubtfully normal, may be reduced to, say, 125 or so, with an improvement all round in the patient's condition and health, he will conclude that the latter and not the higher pressure is the patient's normal reading. Such experience is likewise useful in showing that we can thus pick out cases still amenable to treatment which might otherwise be allowed to drift into the less

controllable and higher ranges of supernormal pressure. Our clinical management of blood pressure thus becomes more preventive. It seems to me, therefore, desirable to subject most cases in the doubtful area (from 145 to 160 mm.) to corrective treatment before accepting from 145 to 150 mm. in a routine way as the parting line between the physiological and pathological areas.

An Increment of the Arterial Pressure is a Result.—The fact should be emphasized that supernormal arterial pressure is not itself a primary condition to be attacked or controlled as such. It is invariably a result of some pathological cause or causes, and it is, moreover, often indeed a necessary result of conditions which we cannot remove. While realising what are the ill effects of high arterial pressure, and trying to modify them as best we may by our attempts to moderate the pressure, we are apt to forget that that pressure is not infrequently a necessary evil, and should sometimes be regarded as a compensatory

effort, or as one of the natural defences of the body. On this point Theodore C. Janeway, who has done so much to advance the clinical study of blood-pressure, has recently remarked, after referring to the futility of drugs to keep down the blood pressure: "Another common experience is that the steady downward progress with falling blood pressure, when heart weakness supervenes, is often made worse by arterial dilators. If improvement comes under digitalis, for instance, the pressure usually rises. These observations find their readiest explanation, if we believe that for such patients an average level of arterial pressure far above the normal is absolutely necessary to insure the requisite speed of capillary blood flow. For them the optimum pressure for the maintenance of a normal circulation may become 200 mm. or more, instead of 100 to 145 mm. . . . Where no symptoms of a threatening nature exist these preventive measures alone are called for. Under such circumstances a systolic blood pressure [afforded by the

armlet] of 200 mm.—yes, even 250 mm. and over—is not incompatible with a number of years of comparative comfort and activity, particularly if the response to treatment is favourable, and the patient co-operates heartily." [1]

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[1] "Some Common Misconceptions in the Pathological Physiology of the Circulation and their practical significance," by Theodore C. Janeway, M.D., *New York Medical Journal*, Feb. 2, 1907.

CHAPTER VI.

THE TREATMENT OF SUBNORMAL ARTERIAL PRESSURE.

A Low Arterial Pressure may be Normal to the Individual.—It by no means follows that the arterial pressure is abnormal in any particular case, merely because it is below the average level of pressure generally present in healthy subjects of the same age. I have met with several male adults, even in middle life, in perfect health and enjoying their life work and recreations, in whom the arterial pressure has not exceeded $\frac{95\ S}{80\ D}$ mm. Hg. Such subjects are generally slight in build. Then again there is the fact that those in training for games and athletic exercises demanding the expenditure of spurts of energy, have frequently somewhat low arterial pressures during

quiescence—the ventricle possessing the potential energy for sustaining the higher arterial pressure required during the stress of vigorous muscular action.

It would therefore seem that a subnormal pulse pressure, like a slow pulse-rate or a subnormal temperature, may be consistent with good health and the enjoyment of an active life; and is not therefore in itself significant of disease.

A Subnormal Arterial Pressure may, however, be of pathological import when it is conjoined with the signs and symptoms of disease, or impaired health—when, in a word, it is a component of a complex symptom. The pressure normal to the individual may then become actually subnormal. Such pathological depressions are observed in (*a*) some *chronic diseases*, *e.g.* Addison's disease, anæmia, hæmorrhages, neurasthenia, phthisis and other wasting diseases; and (*b*) in some *acute diseases*, such as diphtheria, typhoid, pneumonia, influenza, and other febrile ailments.

Diet.—The value of **meat** and **meat**

extractives in the treatment of an asthenic condition of the circulation has of late years been thrown somewhat into the background by our dread of uric acid and the other purin bodies, which are supposed to cause goutiness; and indeed our dietetic directions generally have thus to a considerable extent become vitiated and lop-sided. In low-pressure cases roast meats and gravies should predominate in the diet; and when meats are not admissible in the crude form, as when there is a rise of temperature, extractives of them, which are valuable cardiac stimulants and restoratives, should be given frequently in small quantities. In many cases of acute illness (such as diphtheria, pneumonia, influenza) in which the heart is apt to fail, watery extracts of muscle, when given in judicious intervals, often afford timely aid in tiding the heart over a critical period, acting, like other stimulant remedies, quickly and decisively, and enforcing the sustaining effects of food (milk, etc.) which requires time for its

elaboration and assimilation. They should not be given, as they were some years ago, as substitutes for food. **Alcohol** (especially in the form of brandy egg-flip) acts in a similar way when administered with food. **Sodium chloride** is another stimulant of the circulation which is apt to be overlooked. It should never be omitted from the liquid foods, such as milk ; and I would suggest that it should be given along with calcium and potassium chloride in the proportions present in Ringer's circulating fluid,[1] which may form the menstruum of the drinks—lemon, barley or toast water.

The due apportionment of **rest** and the regulation of **exercise** are most important in the treatment of low-pressure patients. In acute cases absolute recumbency is of course necessary. In chronic cases, and during convalescence from febrile ailments, the prescription of exercise frequently

[1] Aseptules containing the chlorides of sodium (6 parts), potassium (3 parts), and calcium (1 part) are supplied by Messrs. Oppenheimer, Son & Co. for the ready preparation of Ringer's solution—one aseptule being dissolved in 20 oz. of freshly boiled water.

requires to be well guarded, to prevent recurrence of atonia and consequent delay in recovery; and in such cases recumbency should be enjoined during the hypotonic periods following the meals and exercise, when undue draining of blood into the splanchnic veins takes place. In low-pressure cases abdominal massage and faradism of the abdomen are useful.

The **medicinal treatment** of subnormal pressure by cardiac and vasomotor tonics is so well known that more than a passing reference to it is unnecessary. Strychnine, digitalis,[1] and adrenalin are the best remedies of this class; and phosphorus, arsenic, and iron are frequently useful adjuncts. As a pressor remedy formic acid, according to Clément[2] of Lyons, is promising; this observer having recently shown that

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[1] The value of digitalin in the low arterial pressure of Addison's disease is well shown by Rendle Short, *The Lancet*, vol. ii., 1906, p. 285.

[2] "L'Acide Formique et la Force Musculaire," par le Docteur Clément de Lyon: Paris, 1905.

it is a powerful tonic of muscle fibre—whether striped or unstriped—and quickly abolishes the sense of fatigue. The tonic effects of formic acid or the formates is much appreciated by French physicians just now.

The Depressor Effect of the Toxins of Diphtheria and of other Infective Febrile Diseases may be met by the Routine Administration of Strychnia.—This fact in regard to diphtheria is thus clearly stated by James Davidson: "As practically all the cases of diphtheria in the Edinburgh City Hospital were by routine practice put upon strychnine and alcohol on admission, the fall of blood pressure due to the toxic condition was to a large extent annulled. In cases which did not receive any such stimulant a fall was in every case noted until this treatment was commenced. During the routine administration of such stimulants over long periods, the blood-pressure response to any single dose was apparently absent, or in any case was much less marked than that following the

first few doses given to the patient; but in these circumstances, if the routine order were suspended for one or two periods, the blood-pressure was noticed to be affected. This constant blood-pressure level, as maintained by the use of strychnine at regular intervals, seems to represent the ideal to be aimed at in the stimulant treatment of toxic conditions such as diphtheria." [1]

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[1] "Blood Pressure in Fevers," by James Davidson, M.D., etc., *The Lancet*. vol. ii., 1907, p. 1087.

CHAPTER VII.

ON ARTERIOMETRY.

HAVING found the calibration of the radial artery useful and instructive when made by the side of blood-pressure measurement, I will describe the little instrument (Fig. 7) which I devised some years ago for the purpose of determining the internal diameter or calibre of the artery. It is applied in the vertical position, *i.e.* at right angles to the artery and to the arm (see Fig. 8). Each observation comprises two stages. In the first the pointer and the dial travel for a certain distance, which varies in each case, from *right to left*—then the dial ceases to move. In the second stage the pointer suddenly leaves the stationary zero and travels from *left to right*—in the direction of the arrow on the dial. During this second stage the artery is being

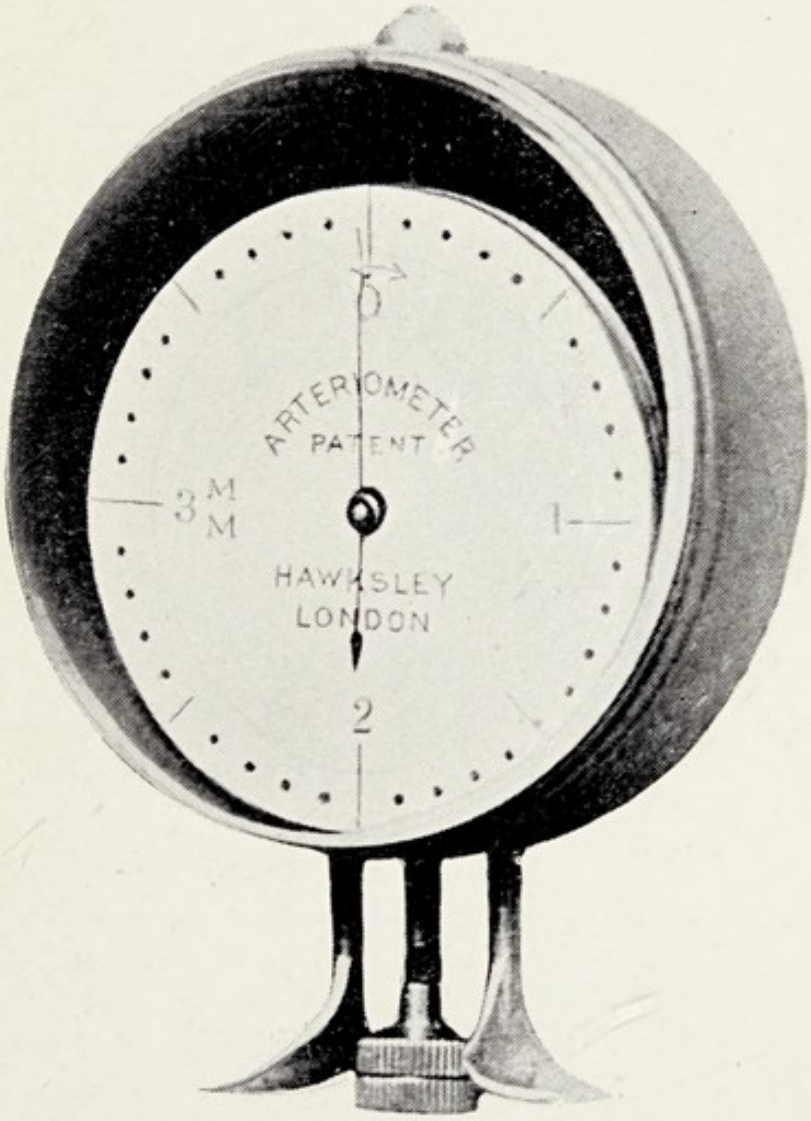

Fig. 7.—The Arteriometer. (Full-size).

The arteriometer, the hæmodynamometer, and the hæmomanometer are made by Mr. Hawksley, 357, Oxford Street, London, W.

To face page 230.

closed, and the excursion of the pointer from zero to the point at which pulsation beyond the pad completely ceases provides a measure of the calibre of the vessel.

I.—How to use the Arteriometer.

The Wrist-rest.—The box in which the instrument is carried, when opened and inverted, becomes a convenient wrist-rest. The strap which surrounds it provides three variations of extension, any one of which may be selected as the best adapted to the individual extensibility of the wrist, which varies in different cases.

Directions.—(1) Extend the wrist (preferably the right one) over the wrist-rest, and **see that the elbow leans on the table and that all the muscles are relaxed**; raise **slightly** the radial side;[1] put the left hand over the upturned palm and thumb, and thus maintain the contact of the dorsum against

[1] This direction is of special importance in taking the radial calibre in the recumbent posture; for then the patient is very apt to evert the supine forearm.

the wrist-rest and prevent the thumb from being extended; and place the pad of the left index finger over the radial artery, so as to close the vessel and exclude a reflux beat that may be present, when the tip only will perceive the direct pulsation. The artery and the adjacent structures are thus rendered slightly tense and at rest—conditions which are necessary to definite observation.

(2) Examine the bed of the artery with the finger or the blunt end of a pencil, test the closing of it, and select the best site for the observation. If this should not happen to be in a line with the prominent part of the styloid process, which forms a good general landmark for this observation, stain the site with a dermographic pencil.

(3) Hold the arteriometer between the index finger and the thumb of the right hand, and plant the foot-rests, one on the styloid process and the other on the flexor tendons, rendered tense by the extension of the wrist, and adjust the pad carefully over the site of the artery.

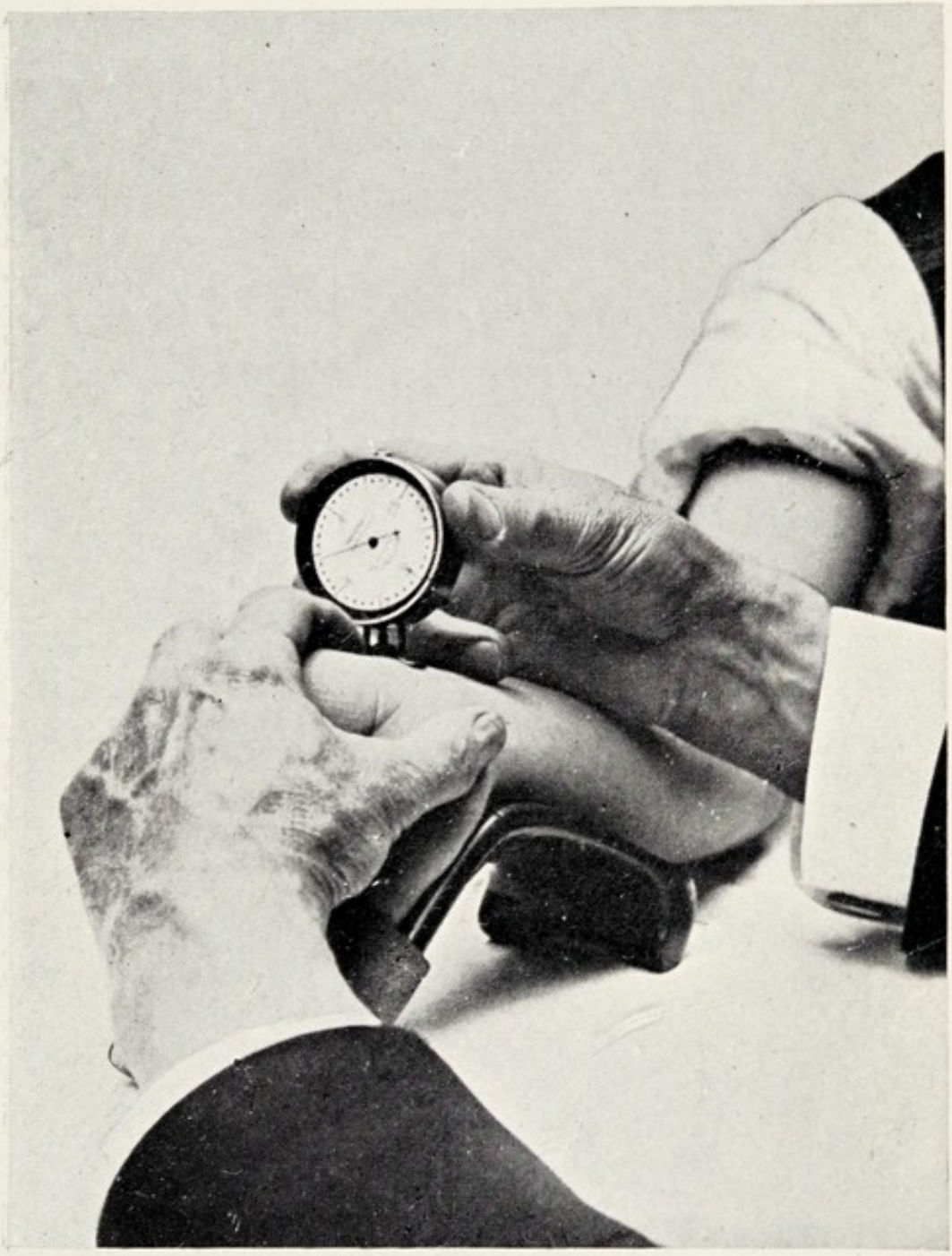

Fig. 8.—Mode of using the Arteriometer, the wrist being extended on a Wrist-Rest.

To face page 232.

It is important that the observer should see that the inner foot-rest is well supported by the tendons, and if these are not felt to be firm enough the wrist should be somewhat further extended. The instrument should be held, throughout the observation, perpendicularly to the plane of the limb ; and **should not be inclined backwards** (see Fig. 8).

(4) On applying the instrument, it will be observed that the indicator travels on the dial in the direction opposite to that of the arrow, and generally for more than one revolution, and in doing so, on reaching zero on the index, it rotates the dial. The extent of this excursion to the left, which takes place before the radial measurement can be made, depends on the relation which the site of the artery bears to the two fixed points on which the foot-rests are planted, *e.g.* whereas in the average formation it may not exceed a revolution and a half, it may, when the wrist is round and prominent, approach or complete two revolutions, and when exceptionally shallow, it may not compass more than one. The

mechanism allows of two backward revolutions. The stem may be shortened or lengthened by screwing the pad up or down. When it occupies the medium position, indicated by a line across the middle of the flat portion of the screw, on applying the instrument, the indicator settles in almost every case in some part of the second revolution.[1] The instrument should be applied somewhat gradually, so as to avoid propelling the zero point of the dial in advance of the pointer.

The observation proceeds by slowly pushing down the outer case—care being taken to bear equally on both foot-rests—when the indicator will recede to the left a few points farther, and then will **suddenly move in the opposite direction**—that of the arrow—leaving the dial stationary. This point indicates **the commencement of the radial measurement**; and, in order to make

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[1] In quite exceptional instances the pointer may barely complete one revolution before it leaves zero—when the pad should be lengthened by unscrewing it; or it may actually complete the second revolution—when the pad should be shortened by screwing it up a little.

it as definite as possible, it is necessary carefully to preserve the same line of pressure. It is advisable to repeat the turning point of the movement of the indicator a few times by raising and lowering the case, which is then pushed farther downwards, until the artery is gradually closed, when pulsation is no longer perceived by the tip of the index finger.[1] The completion of the observation should be quite definite, and this may be repeatedly tested by allowing the pulsation to return, on slightly relaxing the downward pressure on the case, so that the indicator may recede a point or two, and then to obliterate it as before.[2] **The interval traversed by the indicator, from the point when it leaves zero and moves in the direction of the arrow to that which shows the artery to be occluded, affords the**

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[1] When the "reflux" or "recurrent" beat from the palmar arch is present, it is excluded by the pad of the finger.

[2] It will be found that in most cases the well-trained finger does not require more than $\frac{1}{20}$th of a mm. for the purpose of defining the absence of pulsation.

measurement of the calibre. If, however, pulsation does not cease after the indicator has measured off as much as 3·0 mm., the observer should suspect that either the pad is incorrectly placed in regard to the artery, or the line of downward movement of the pad has not been made at right angles to the resistant plane on which the artery rests, or the spot chosen has not been sufficiently adapted to closure of the vessel from the want of counter-support, etc., or perhaps the structural conditions are altogether unsuitable for this mode of observation.

Normal Postural Variation.—The calibre follows the variations of the arterial pressure due to gravity and change of posture. In assuming the erect postures (sitting or standing) it immediately attains its maximum, and just as quickly falls to its minimum in recumbency. As a rule, in healthy subjects the postural variation extends from ·4 to ·6 mm., the average calibre in the erect postures being from 2·0 to 2·3 mm. and in recumbency 1·5 to 1·8 mm.

II.—Epitome of Clinical Observation of the Radial Calibre.

In the clinical field the arteriometer affords useful information concerning disturbances of the vaso-motor system and the state of the arterial wall. It therefore determines important facts relating to the circulatory mechanism; whereas the hæmodynamometer[1] merely measures the blood-pressure as distinct from the condition of the arteries. The two instruments are therefore complementary to each other in clinical research bearing on the circulation.

Reversed Postural Variation.—When the arterial pressure is lowered by a reduction of tone in the arterial wall, inducing splanchnic stasis, the postural measurements of the radial calibre are reversed—becoming maximum in recumbency and minimum in the erect

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[1] The manometer-bulb (p. 52) also measures the blood-pressure apart from the state of the arterial wall; but unfortunately its rendering of the systolic pressure is not always quite so definite as that of the hæmodynamometer.

postures. In this way the arteriometer enables the observer to measure the effects of fatigue on the circulatory system in whatever way induced, whether by exercise, heat, ill-health (acute or chronic), hæmorrhage, or otherwise.

The Radial Calibre is Subnormal.—This may indicate: (*a*) A congenitally small radial artery merely, when the arterial pressure and the postural variation of the calibre will be normal. (*b*) A subnormal arterial pressure, with general wasting of tissue and reduction of the volume of the blood. (*c*) Arterial constriction with raised arterial pressure and an absolute or relative reduction of the venous pressure, the postural variation of the calibre ceasing should the constriction be very persistent and decided. This condition of the arteries is observed in the early stage of arterio-sclerosis—the stage which has been termed pre-sclerotic (*see* pp. 109–10). (*d*) Endarteritis (from syphilis, lead, gout, etc.), atheroma, and arterio-sclerosis. The calibre is not only reduced, but has lost its postural variation.

The Radial Calibre is Supernormal.—This may indicate: (*a*) A normally large radial artery, when the arterial pressure and the postural variation of the calibre will be normal. (*b*) An excessive arterial blood-pressure overcoming the arterial tonus (*see* p. 128).

The Arteriometer is Useful in suggesting Vaso-dilator or Vaso-contractor Remedies and in measuring their Effects.—I have frequently found the arteriometer of considerable service in indicating the line of treatment to be followed; suggesting the adoption of measures and remedies which either reduce a hypertensive or which raise a hypotensive state of the arterial walls. The conjoint use of it and the hæmodynamometer or the manometer-bulb is a source of sustained interest in enabling one to follow the relation between the vaso-motor state of the arteries and the blood-pressure, and thus daily to exemplify in practice our physiological knowledge of that relation.

APPENDIX

THE HÆMOCYTOMETER AND LYMPHOMETER

I. The Hæmocytometer

The Principle followed.—The method is based on the following facts: (*a*) When a candle-flame is viewed through a glass tube containing water, a transverse line of bright illumination is seen, consisting of closely packed, minute images of the flame produced by the longitudinal fibrillation of the glass. In the process of drawing out, the tube becomes minutely corrugated or fibrillated in the direction of its axis. The corrugations are extremely small, but they are easily observed when a tube is held before a flame and the structure of the glass is examined by a pocket lens, when the corrugations are seen to produce alternate bright and shaded lines disposed in the vertical direction. Each corrugation acts as a lens, hence the horizontal line made up

of a congeries of images of the flame. (*b*) When some fixing fluid (such as Hayem's) is added to blood, the mixture presents a certain degree of opacity according to the amount of blood or of the fixing fluid. (*c*) The opacity thus produced completely shuts out of view the illuminated line, until, on diluting further, a definite point is reached, when it can just be detected as a streak of light *across* the tube—the dawn, as it were, of the bright minute images of the flame seen when the opacity is absent or is further reduced. Repeated observation has shown that the development of this delicate horizontal line by dilution with the fixing fluid is a very sensitive indicator of the percentage of the blood corpuscles.

The apparatus consists of (1) a graduated flattened tube; (2) an automatic blood measurer; (3) a mixing pipette; (4) a candle; and (5) a bottle of Hayem's solution[1] (*see* Fig. 9).

The determination of the scale was made by the enumeration method extended to

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[1] The formula is as follows: Hydrargyri perchloridum, gramme 0·5; sodii chlorid., gramme 1·0; sodæ sulphas, grammes 5·0; aqua distillata, 200 c.c. Messrs. Oppenheimer & Son, 179, Queen Victoria Street, London, E.C., supply aseptules of Hayem's solution.

120 squares applied to the blood of normal man. One hundred degrees presents the generally accepted standard of 5,000,000 corpuscles per mm.; therefore 80 degrees is equivalent to 4,000,000, etc.

The white corpuscles in a state of health do not affect the readings.

The Mode of Observation.—The pipette, previously dried by passing through it a needle threaded with cotton, having been accurately filled and cleared from adherent blood by the finger, the rubber nozzle of the mixer filled with Hayem's solution is then applied over its bevelled end, and the blood is thoroughly washed into the flattened tube (Fig. 10). When the blood is not suspected to be particularly poor in hæmoglobin and corpuscles, the amount of fixing fluid added in the first instance may raise the column to the 60 or 70 per cent. mark; otherwise the first charge should be less. The contents of the tube are then uniformly mixed by inverting the tube a few times with the thumb over its mouth, care being taken on removing the thumb to draw it over the lip of the tube so as to restore as much as possible the fluid adhering to it. This procedure should of course be repeated after every addition of the diluting fluid. The observation is

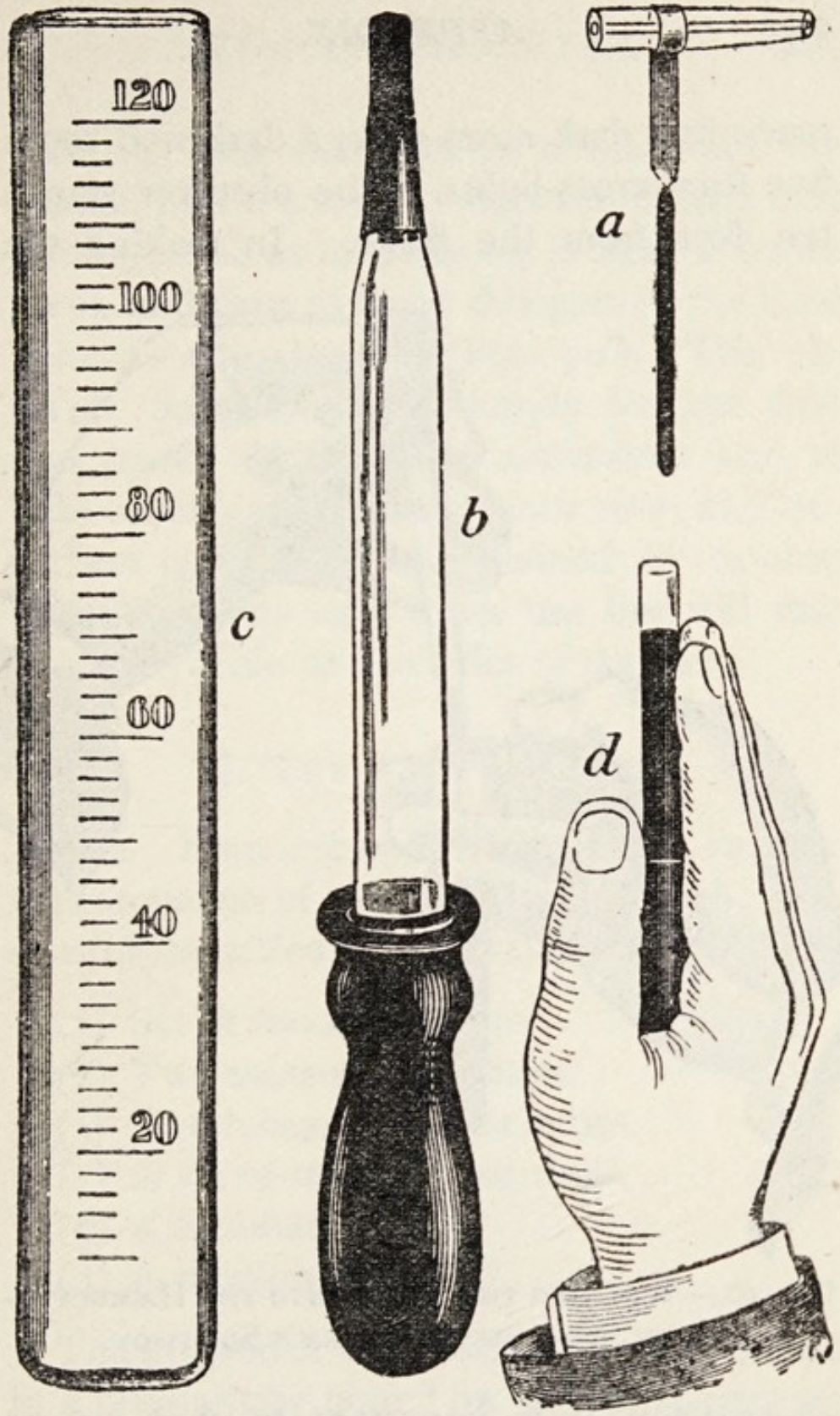

FIG. 9.—THE HÆMOCYTOMETER.

a, Blood measurer; *b*, mixing pipette; *c*, flattened graduated tube; *d*, mode of holding the tube for observation.

made in a dark room or in a darkened room free from cross-lights. The observer stands ten feet from the flame. In making the

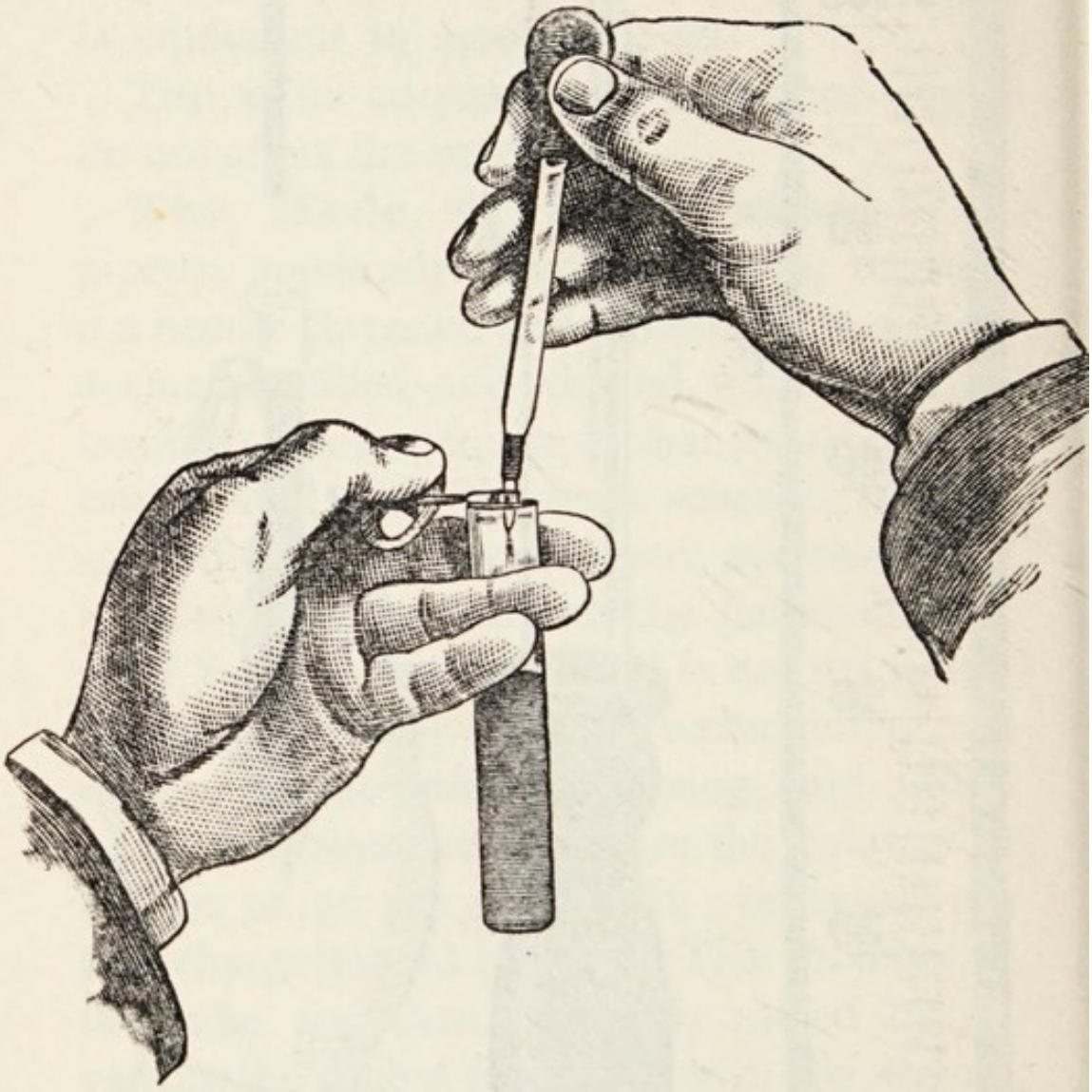

FIG. 10.—WASHING THE BLOOD INTO THE HÆMOCYTOMETER TUBE WITH HAYEM'S SOLUTION.

observation it is important to shut out as much as possible the diffused light of the candle. The most satisfactory way to do this is to place the lower end of the tube,

with its long diameter in a line with the candle, in the concavity between thumb and forefinger, so as to make as it were a frame for it, and then to bring this part of the tube *quite close* to the eye (Fig. 9 *d*). The observer must look out sharply for the first appearance of the faint transverse line of light which dilution brings into view, and the earliest indications are obtained by turning the tube on its axis, when the line will first become visible at the sides of the tube.

II. The Lymphometer.

The hæmocytometer, adapted to the measurement of systemic tissue-lymph, consists of the following apparatus :

(1) Set of lancet prickers.
(2) Two measuring pipettes.
(3) Two hæmocytometer tubes.
(4) A set of stout rubber rings.
(5) A fimbriated tube.
(6) A nipple pipette.

The method of observation [1] was described in a preliminary note "on the measurement

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[1] See the Oliver-Sharpey Lectures, *The Lancet*, vol. i. 1904.

of tissue fluid" read before the Royal Society, June 11, 1903. It is based on an examination of two samples of blood; one before, and the other after compression of the tissues of the finger by stout rubber rings. The first sample is believed to contain a certain admixture of tissue fluid; for the needle, in puncturing the capillaries, must penetrate the areolar tissue spaces which surround them, and perhaps also the lymphatic capillaries of origin under the epithelium,[1] and so must liberate lymph, which mingles with the blood. On the other hand, it has been concluded that the second sample is freed from that admixture, as, after removing the rings which squeeze the lymph away from the capillaries, the blood instantly returns to the vessels, whereas an appreciable interval elapses before fresh lymph is formed.

A puncture is made on the dorsum of the finger near the root of the nail—this part being much more amenable to efficient compression than the pad of the finger—and the blood is measured off by one of the automatic pipettes; after which the three rings are

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[1] "The Lymphatics," by G. Delamere, P. Poirier, and B. Ciméo; translated by Cecil H. Leaf, M.A., M.B., F.R.C.S. Eng.), 1903, p. 3.

slowly rolled with pressure over the finger and are lodged beyond the phalangeal joint. The metal tube is then passed over the finger up to the joint, and the rings are rolled on to it, and are thus removed without compressing the distal parts of the finger. Now the blanched finger suddenly reddens, when (the tissue fluid having been squeezed away by the rings) the blood may be extruded by pressure through the original puncture without fear of disturbing its quality. It is then measured off by the other pipette, and each sample is washed into its respective tube, and the percentages of corpuscles are read in the manner previously described (*see* p. 244). This method of enumeration enables the finest differences between the two samples to be discriminated. When tissue-lymph is present the percentage quantity is indicated by the higher reading of the tube containing the sample after compression; but when it is not present the tubes read alike. A nominal difference of two points may be regarded as practically indicating a minimum quantity of tissue fluid.

Besides affording a measure of the tissue-fluid in the finger, I have found this method useful in giving a ready all-round estimate

of the condition of the blood, by taking the mean of the two observations, *e.g.*—

92 after compression,
80 before „ = 86.

These observations occupy but a few minutes; and the practical advantages derived from them are considerable in at once settling the question of anæmia (in its broad sense), spanæmia and chlorosis; and in detecting polycythæmic or apoplasmic anæmia in which the blood is concentrated and therefore reduced in volume (*see* "Blood and Blood-pressure," 1901, pp. 98–102).[1]

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[1] The Hæmocytometer and Lymphometer are made by Mr. Lovibond, the Colour Laboratories, Salisbury.

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