BLOOD-PRESSURE

FROM THE CLINICAL STANDPOINT

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PREFACE

THE past few years have marked a rapid rise in the clinical value of the sphygmomanometer. This instrument is now a part of the armamentarium of almost every physician. It is opportune therefore that a book of moderate size should be produced containing in concise form, a résumé of the clinical and experimental work which has led to present popularity of the blood-pressure test.

In the following pages, the author has endeavored to present in easily accessible form, the pith of medical literature bearing on blood-pressure studies in their relation to medicine, not only in cardio-vascular and renal conditions, but also in many diseases in which clinical observation has shown the information obtained by the sphygmomanometer to be of value.

It has been thought advisable to devote a number of pages to the discussion of the circulation and its relation to the blood-pressure, together with the various methods employed in sphygmomanometry, to acquaint the practitioner with the theory of this procedure, so that deductions from his observations may be of greatest value.

The writer is indebted to a large number of authors for much of the material contained in this work, which has been obtained largely from the medical literature of the last seven or eight years. Whenever practical the full reference is given in the text, so that if desired, the facts contained in this little book may be supplemented by a study of the original.

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BLOOD-PRESSURE

INTRODUCTION

During the brief period from 1900 to 1910 our knowledge of sphygmomanometry developed from a procedure of uncertain and doubtful value, as viewed by the rank and file of the medical profession, to a method of examination equalled by but few of the many reliable methods of precision in daily use by the general practitioner.

Prior to 1900, a few pioneer physicians had developed the habit of making observations of the blood-pressure and recording their findings. These were available for their own use, and were employed to their own advantage and to the benefit of their patients. It is true also that for even a longer period the physiologist and his co-worker the pharmacologist, have been thoroughly acquainted with the value of this test, both in animal experimentation and in the sick-room.

The pioneers in the field of cardiovascular disease early appreciated the great advantage they possessed by this means of graphically interpreting arterial tension over the old method of estimating tension or blood-pressure by the finger, for it was well known and readily demonstrated that the accuracy of the tactile estimation of blood-pressure was notoriously uncertain, being dependent on several variable and uncertain factors, and the estimation so complicated that even at best an element of error amounting to from 10 to 80 mm. was frequently present.

Following the pioneer work of Janeway, Russell, Mackenzie and others, a knowledge of the value of this subject gradually spread, during which many observers, recognizing the value of this procedure, began to apply the sphygmomanometer not only to the study of cardiovascular and renal diseases, but also to a large number of disease conditions in which changes in the circulation might be expected to occur.

During the past five years, 1907 to 1912, the application of the sphygmomanometer has spread with great rapidity, and bids fair soon to become universal. Coincident with this continued activity, there has developed an immense literature bearing directly and indirectly upon changes in the circulation and in the organs relating thereto, both in physiologic and pathologic conditions. The science of medicine is so broad, its ramifications so extensive, and its literature so voluminous that few, if any, are able to accurately follow and intelligently grasp the almost daily advances in all branches of medicine. This fact furnishes the argument for the preparation of this little work, if indeed any be needed.

A careful search of modern text-books, extending to the end of 1912, fails to reveal any work dealing with this subject from the standpoint of the general practitioner, who so far has had nowhere to turn in case of emergency, or from which to glean a few important facts in his spare half hours. No effort has been made to make this volume a complete and exhaustive review of the whole study of blood-pressure with its many ramifications, but the aim is rather to provide a compendium of clinical data in such form that the material contained will be readily available for immediate use.

To properly appreciate the function of the sphygmomanometer and the changing conditions of the circulation which it reveals, it is important to have a working knowledge of the theory of hydrostatics and of the physical conditions surrounding the circulation both in health and disease, and to possess a proper appreciation of the factors controlling the cardiovascular and renal systems and the significance of the terms employed in this study. It is proper therefore to commence with a brief general discussion of the circulation, even though to some it may seem to be a needless waste of time and space.

CHAPTER I

PHYSIOLOGY

THE CIRCULATION

The maintenance of a normal circulation is essential to good health. Abnormalities in the circulation are either the result of, or result in, disease. The activity of the heart is a vital function like respiration, for if the heart should cease to beat even for a very short space of time, the circulation would fail, and the individual would die. The heart must not only act continuously, but also in an approximately normal manner in order to maintain the body in a condition of health. By failure of the circulation is meant a gradual diminution in blood-pressure, until it becomes insufficient to maintain body nutrition and offers insufficient resistance for a normal action of the heart.

Conditions affecting the action of the heart are shown by alterations in the circulation and variations in the circulation are shown by changes in the heart's action. The two conditions, heart action and a maintained circulatory equilibrium, are in every way interdependent. They can neither be separated, nor considered intelligently one apart from the other. This serves to emphasize the importance of a study of the circulation, not only in an investigation of circulatory diseases, but also in the study of diseases of the heart, particularly pathologic changes in its muscular and nervous mechanism.

We are for the most part indebted to Harvey[1] for discovering and demonstrating the true function of the heart as the main-spring of the circulation. In 1616 Wm. Harvey stated that "a perpetual movement of the blood in a circle is caused by the beat of the heart." From a perusal of the original or of the translation of Harvey's work on the circulation, it is evident that his conception was the true one, and that it forms the basis of our modern conception of circulatory physiology and pathology. It is of interest to note, however, that investigations of ancient manuscripts bearing on medicine show that some knowledge was possessed by the ancient Egyptians as shown by references to the heart, and the use of the word circulate in the Ebers papyrus. An interesting review "The Advance in Knowledge Regarding the Circulation of the Blood" has recently been published by Dr. Geo. Wm. Norris.

Between the heart and capillary system there is a large and ramifying network of blood-vessels of progressively narrowing individual caliber, but of rapidly increasing cross-sectional area, which convey the blood to every part of the body. By normal circulation we mean the normal distribution of blood to every part of the body, whereby the normal interchange of nourishment and waste is sustained in all organs and tissues. This metabolic function occurs in the capillaries, between which area and the heart the blood-vessels serve as a series of conduits. We see therefore that between the heart, as the central

[1] See Camac's "Epoch-making Contributions to Medicine and Surgery," Philadelphia, 1909.

source of supply, and the capillaries, the points of interchange, there is a wide gap. This is filled by the arteries carrying blood on its way to the terminal points of interchange, driven along by the intermittent pumping action of the heart. A rhythmically contracting heart and a volume of blood alone could not afford every part of the body its perfect supply of pabulum nor maintain an equal distribution of this fluid. We must necessarily have another mechanical factor to complete our system. This is blood-pressure. Blood-pressure is an essential factor for sustaining the circulation and maintaining heart action, and as a corollary we must recognize the arterial walls as a further factor in maintaining and regulating this circulation. This is by virtue of the elasticity and contractibility of the blood-vessel walls, whereby they may expand under an increase in pressure from within, or may contract to maintain or to elevate the pressure by a shortening of the circular muscular fibers in their walls.

A normally acting circulation is shown by a normal blood-pressure, which by virtue of being normal, shows that the heart action and the distribution of blood must be taking place in a normal manner. Therefore the study of blood-pressure becomes a most valuable and efficient guide to the state of the cardiovascular system and sphygmomanometry a most important diagnostic method. Bearing on the importance of this study A. Randle Short[1] says "It has become a truism that when feeling the pulse, it is of more importance to observe the tension, or blood-pressure, than to count the pulse rate. But only within the last few years has it been recognized how inadequately

[1] The New Physiology in Surgical and Medical Practice.

even the skilled finger can judge the blood-pressure because of the complicated factor of the variable rigidity of the vessel walls." Discussing the factor of the vessel wall, as preventing an accurate digital estimate of blood-pressure, Wm. Russell[1] makes the following significant remark: "I must, however, again add a warning note to the effect that feeling the radial is not always a reliable guide as to what the brachial pressure will read. In some cases the radial artery and its pulse would not lead one to suppose that the brachial pressure would be high. I have two such cases under observation as I write this. The radial artery being neither hard nor incompressible, and yet in both there is a steady reading from brachial of over 200 mm. Hg. On the other hand, the brachial pressure may be lower than the state of the radial suggests."

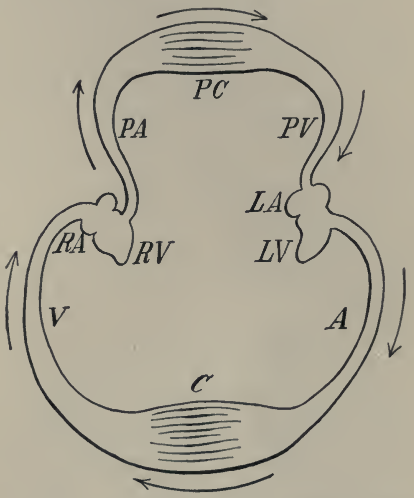

FIG. 1.—General diagram of the circulation: the arrows indicate the course of the blood: *PA*, pulmonary artery; *PC*, pulmonary capillaries; *PV*, pulmonary veins; *LA*, left auricle; *LV*, left ventricle; *A*, systemic arteries; *C*, systemic capillaries; *V*, systemic veins; *RA*, right auricle; *RV*, right ventricle. (John G. Curtis in "American Text-book of Physiology.")

In the human body we may trace the course of a given particle of blood as it leaves the right ventricle until, having traversed the entire cardiovascular system, it

[1] Arteriosclerosis, Hypertonus and Blood-pressure, page 73.

returns to the starting-point. Referring to Fig. 1, we find the course of the blood to be as follows: From the trunk of the pulmonary artery through a succession of arterial branches into the capillaries of the lungs, from there through the several branches of the pulmonary vein to the left auricle of the heart, thence through the mitral valve to the left ventricle, then by way of the aortic valve to the aorta and the general arterial tree until it finally reaches the capillaries. From the capillaries into the veins back toward the heart, through the vena cavæ and into the right auricle, through the tricuspid valve into the right ventricle, through the pulmonary valve into the pulmonary artery where the tracing of the circuit began.

In brief, the vascular system is a closed series of tubes of varying diameters, including a force pump. This tubular system is partially interrupted at two points by a series of very minute vessels, the capillaries of the lung and of the general circulation.

The condition of the arterial walls and the width of the arteries exercise considerable influence upon the flow of blood. If all the arteries of the body were fully dilated it would be absolutely impossible for the heart to maintain the circulation, because the relatively small amount of blood in the body could not begin to completely fill the vessels. The caliber of the arteries is influenced mainly by reflexes coming from various parts of the body, including the heart and the blood-vessels themselves. Stimulation of a peripheral nerve will cause normally a reflex contraction which will tend to raise blood-pressure.

The force by which the blood is driven from the right to the left side of the heart, through the capillaries which are related to the respiratory surface of the lung, is nearly all derived from the contraction of the muscular wall of the right ventricle. The force by which the blood is driven from the left side of the heart through the general circulation, including all the other capillaries in the body, is nearly all derived from the contractions of the muscular wall of the left ventricle. The contraction of the two ventricles is simultaneous. The force generated by the heart in maintaining the circulation is, to a subordinate degree, supplemented by the aspirating action of the chest wall during the respiratory act, by the pumping action of the skeletal muscles and by the elasticity or tone of the arteries themselves.

The usual systolic arterial blood-pressure, about 120 mm. Hg., is much more than is actually necessary to drive blood from the arteries into the veins. This extra pressure has a function, however, which is seen whenever the arterioles of any organ or small area relax. If the arterial pressure was barely adequate to sustain a flow, a lowered resistance in any part would seriously drain other regions. The high head of pressure, therefore, serves to keep all parts properly supplied with blood, even if an especially active part of the body is making an unusual demand.

In order to better understand the cause of maintenance of blood-pressure it is necessary first to consider the science of hydrostatics as to its effect upon the circulatory system.

Fluids are incompressible and the heart is an intermittent pump, therefore if the arteries were rigid and unyielding tubes, each increment of blood coming from the heart would be required to move all the blood in the whole arterial system, while during the heart rest, all flow would cease. This would result in the intermittent development of pressure, accompanied by periods when it must fall to zero. Such a condition would be inimical to health, as the proper nutrition and tension of the organs and tissues of the body would not be maintained. The arterial walls are, however, as already stated, not rigid but elastic and distensible, and are capable therefore of expanding under pressure to accommodate more fluid, while during diastole their elasticity and contractility tend to maintain pressure. This property gradually reduces the sharp intermittency of the flow in the arterial system, so that as we pass outward from the heart this feature becomes less marked and finally disappears before the capillaries are reached. Another factor enters here. This is the gradual tapering and extensive ramification of the arterial system. This length of vessel combined with its elasticity aids in reducing the flow to a uniform rate of flow.

A third factor is the relatively large number and minutely small diameter of the capillaries. If the vessels were short and the tubes of large diameter the alteration in flow would not occur and the blood would pass into the veins intermittently. This is shown in certain pathologic conditions where we have a capillary pulse and a transmitted venous pulse.

In considering the factors involved in maintaining a uniform flow in the capillaries, we find that they also assist in establishing and maintaining pressure, for if the heart as a pump was large enough and the arteries short enough and the outlet large enough there would be no blood-pressure. In the arterial system, we find that blood-pressure is maintained first by the pumping action of the heart which acts against the friction of the walls of the blood-vessels, second by their narrowing diameter, and third by the viscosity of the blood itself. Starting with the arterial system as a closed system of tubes, including the heart, we find that as the heart begins to beat, the blood is pumped into the arteries, and in its passage toward the capillaries it meets with resistance. This causes the pressure to rise in the arterial system, which increase in pressure brings into action the normal tone of the arterial walls. So that as the pressure rises the arteries expand to accommodate an additional amount of blood, at the same time the blood-pressure rises, this increases the pressure in the capillary system and drives more blood into the veins in a given time. Blood-pressure will reach normal and be maintained there, when as much blood passes through the capillaries during a heart cycle as enters the aorta during systole. At this time, the power of the heart is exactly balanced by the factors of volume of blood in the arterial system, its viscosity and vasomotor tone.

The term tonus or blood-pressure has been applied to indicate the amount of pressure existing within the arterial system, and this as we have seen, is dependent upon the factors just mentioned. The final and most important condition affecting blood-pressure is vasomotor tone. This is maintained through a special reflex mechanism which has for its purpose the maintenance of normal blood-pressure in spite of temporary alterations in peripheral resistance in different parts of the body. This system has the power of regulating the amount of blood reaching any part and is operated by the demand of organs and tissues for nutrition.

We have therefore at any time in any individual, five factors which go to maintain normal blood-pressure.

1. The energy of the heart.
2. Peripheral resistance.
3. Tonus.
4. Volume of blood.
5. Viscosity.

These all may and do all vary under normal conditions and in pathologic states may become greatly altered.

Not only may they vary independently of each other, but they are also so closely interrelated to the cardiomotor and vasomotor systems, that alterations in one of them may cause profound changes in another. We are as yet in possession of but incomplete evidence as to the relative value of these several factors, but enough is known to form a good working basis, which may be used to explain alterations in the circulation, both in health and disease, and which perhaps may be used to build up a rational therapy in cardiovascular and renal diseases, and other circulatory disturbances.

CHAPTER II

THE SPHYGMOMANOMETER

The use of manometers or upright tubes filled with fluid, in the study and measurement of blood-pressure in man is attributed to an English clergyman, Stephen Hales,[1] who published the results of his experiments in 1733.

The apparatus employed by Hales and his followers was naturally extremely crude and the result of their work of little practical value, and found little favor at the hands of either physicians or physiologists until about 150 years later, when the sphygmomanometer was first perfected and adapted to clinical purposes by Professor v. Basch, of Vienna, in 1876. This apparatus, as it appeared in its original form, consisted of a U-tube, one limb branched to join the tube from the pelotte, which was used to compress the artery under observation. The U-tube partly filled with mercury was provided with a scale reading in millimeters. The remainder of the tubular portion of the apparatus was filled with fluid which transmitted the pressure from the elastic membrane of the pelotte to the mercury. The original instrument of v. Basch has since undergone numerous modifications both by v. Basch himself and by others, of which the most important was the introduction of a portable metallic chamber or aneroid manometer.

In 1889 Potain replaced the water of the earlier instru-

[1] Statistical Essays, London, 1733, Vol. II.

ments with air and raised the pressure in the circuit by means of a bulb connected with the apparatus by a branch-shaped tube. During the next several years the instruments of v. Basch and Potain had considerable vogue but were not widely used because of several inherent defects in the construction of the instruments themselves.

In 1896 Riva-Rocci[1] and Hill[2] published almost simultaneously articles descriptive of new sphygmomanometers. The important feature of each of these instruments was the introduction of a rubber-bag or tube encircling the arm and inflated by a bulb or a pump. This improvement surmounted the most serious defect in the earlier instruments, which was the difficulty of accurately adapting the small round pelotte to the arm, thereby compressing the artery (the radial) directly over the bone. By the method of Riva-Rocci and Hill, the pressure is everywhere exerted at right angles to the tangent of the circumference of the arm, and the artery is therefore compressed equally from three sides against the bone.

Since this time there has been practically no change in the principles of sphygmomanometry. Improvements having been made toward perfecting the apparatus and simplifying the technic, changes having been directed chiefly toward portability, in means of circular compression, and source of pressure.

From the narrow arm-band as originally employed by Riva-Rocci (4.5 cm.—2 in.) to the extremely wide band of Von Recklinghausen, numerous investigators have determined that a cuff 11 to 13 cm. (4 1/2–5 in.) in width

[1] *Gaz. med. di Torino*, 1896, Nos. 50 and 51.
[2] *Brit. Med. Jour.*, 1897, Vol. II, p. 904.

gives the most nearly accurate readings, except perhaps in the extremely obese. A special narrow cuff may also be found advantageous for work with babies and small children.

With accurate indicators and a standard cuff the values obtained in blood-pressure studies are comparable from individual to individual and are almost entirely independent of the variations in the soft parts which overlie the vessel. (See Chapter III.)

DESCRIPTIONS OF THE MODERN INSTRUMENTS

1. **Riva-Rocci Sphygmomanometer** (Fig. 2).—The mercury manometer is of a cistern form. This reduces practically to zero the fall in the other column such as occurs

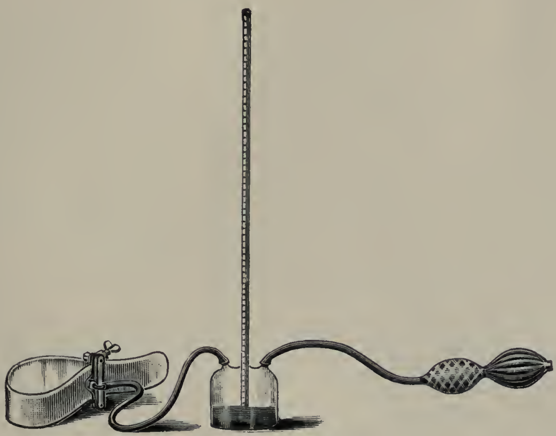

Fig. 2.—Riva-Rocci's sphygmomanometer. (Sahli and Potter.)

with the U-tube, and permits the use of a millimeter scale for measuring the height of the mercury column. The scale reads up to 260 mm. (10 in.) Hg. The cistern is of heavy glass, from which emerge two tubes, one for the inflating apparatus and the other for the attachment of the arm-band. The latter is provided with a release valve for gradually lowering the pressure in the circuit during the test.

The armlet consists of a hollow rubber tube covered with silk having a width of 4.5 cm. (2 in.), which is fastened to the arm with a special clamp. The inflating apparatus

Fig. 3.—Cook's modification of the Riva-Rocci sphygmomanometer, showing narrow arm-band in place, with cautery bulb inflator.

is a double bulb such as is employed with a thermocautery.

The apparatus stands firmly on a solid base and has a scale which is easy to read, it is easy to adjust and rapid in operation.

Disadvantages.—The size and construction of the apparatus make it not easily portable. The armlet is too narrow for accurate readings, and the elasticity of the tubular system makes diastolic readings difficult. It is not available for pressures over 260 mm. (10 in.) of mercury.

2. **Cook's Modification of the Riva-Rocci** (Fig. 3).—This is very similar to the preceding but is of lighter construction, and is provided with a jointed manometer tube

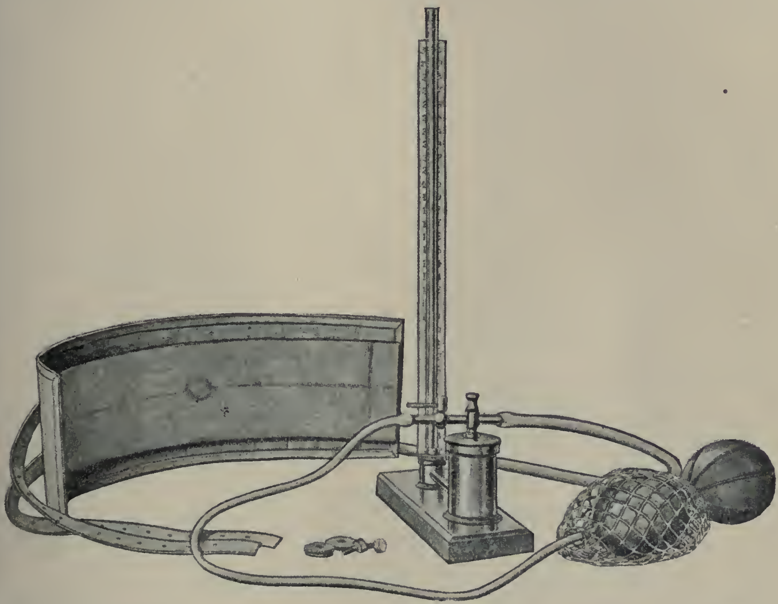

Fig. 4.—Stanton's sphygmomanometer, showing arrangement of parts, with cautery bulb inflator.

which allows the instrument to be packed in a smaller space. It employs the small armlet and the double bulb inflator.

Disadvantages.—It does not stand firmly and is easily upset and broken. Without special care in packing and transportation the mercury is often spilled. The caliber of the tube (1 mm.) is too small and the narrow cuff gives readings that are too high. The scale etched on the glass is difficult to read.

3. **Stanton's Sphygmomanometer** (Fig. 4).—This instrument was devised in an effort to increase portability, to reduce the probability of breakage and to lessen the elasticity of the tubular system, thereby improving the diastolic fluctuation of the mercury.

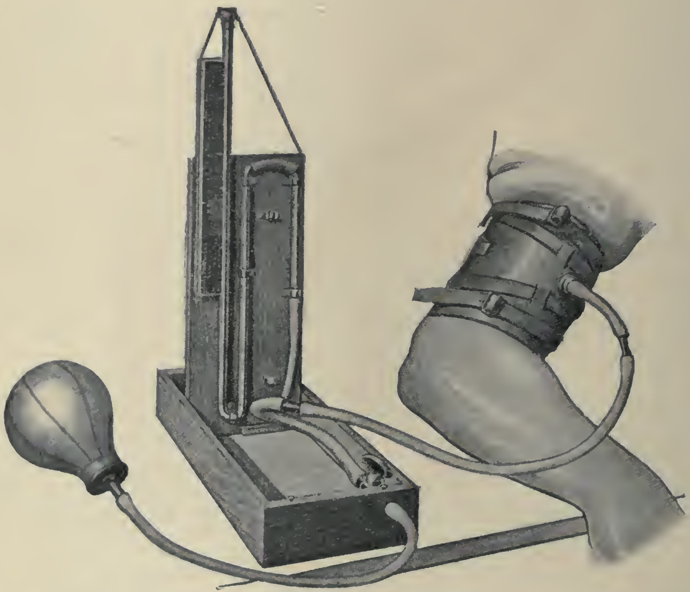

Fig. 5.—Janeway's sphygmomanometer, attached to arm, showing method of retention of cuff, arrangement of momanometer, with Politzer bag inflator.

These changes were effected by substituting a metal cistern and by arranging over this a screw joint for the attachment of the vertical glass tube; also the introduction of a stopcock in a short tube as it emerges from the cistern to serve for attachment of the inflating bulb, this eliminates the elasticity of the inflating apparatus during the diastolic reading. The instrument employs the standard 12-cm. (4 1/2 in.) cuff retained on the arm by a canvas outer cuff and buckle straps.

Disadvantages.—Chiefly the time and skill required to set up the apparatus before using, and the great difficulty in avoiding the loss of mercury during the setting-up process. Finally the cistern arrangement gives low readings in high pressures.

4. **Janeway's Sphygmomanometer** (Fig. 5).—In the construction of the Janeway apparatus we see a return to the U-tube type, first devised by v. Basch. This form appears to be a more accurate method of employing the mercury column, since in the cistern form no cognizance is taken in the change in the level of the mercury in the cistern, which must, for physical reasons, give too low readings when employed in the study of high pressures.

This instrument employs the circular compression band of standard width and a Politzer bag for inflation, thus eliminating the frequent rupture of the double cautery bulb which occurs in high pressures. Apart from this, the only original feature of this instrument is the jointed U-tube which allows the instrument, without cuff or inflating bag, to be contained in a case measuring 10 1/4×4 5/8×1 7/8 in. and weighing 2 1/2 lb.

The open end of the manometer tube is closed with a cork when not in use, and the rubber connection on the other limb leading to the attachments is compressed by closing the case, to prevent loss of mercury from the manometer. The scale is arranged to slide down into the box when not in use. The arm band is 12 cm. (4 in.) wide and is retained on the arm by an inelastic outer cuff provided with friction straps.

This apparatus is light, compact and portable and the readings are accurate.

Disadvantages.—Frail because of jointed U-tube. The mercury is easily spilled because of the loose methods employed to confine it when the instrument is not in use.

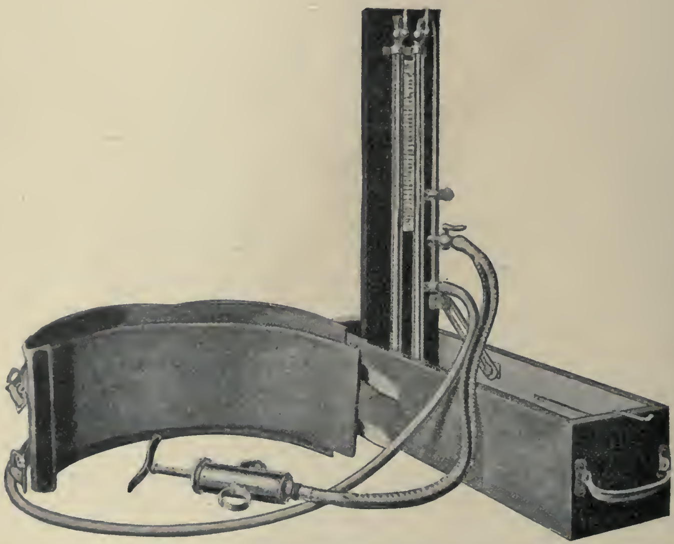

Fig. 6.—Faught's mercury sphygmomanometer, showing relation of parts, metal pump, and special expansion tubing for inflator.

The rubber connections are short-lived and must be replaced at irregular intervals.

The Faught Standard Mercury Sphygmomanometer (See Fig. 6.).—This apparatus was devised in the early part of 1909 in an endeavor to overcome if possible the shortcomings of existing instruments, the majority of which were frail, required special skill to operate, consumed too much time and were defective mechanically.

The Standard sphygmomanometer is of the U-tube type in the construction of which, all complicated parts have been either simplified or eliminated. The complete apparatus including the cuff of standard width, and the inflating pump may be enclosed in a mahogany carrying case, measuring $14 \times 4 \times 4\ 1/2$ in. The lid is hinged and locks in a vertical position to serve as the support of the manometer and the connections. Each arm of the U-tube is provided with a guard cock which remains closed, except during actual use of the instrument, thus preventing absolutely any loss of mercury, excepting where there is gross carelessness. There are no rubber connections; the upper nipple to which the pump is attached is provided with a stopcock, which must be closed during the systolic and diastolic readings. The millimeter scale, which can be adjusted to the level of the mercury is reduced one-half to compensate for the fall of the mercury in the other limb, thus the markings give the reading directly in millimeters of mercury. The pressure is obtained by the use of a metallic pump attached to the upper nipple by means of a collapsible rubber tube of special construction, which by its expansion during the operation of the pump, reduces the impact of air before it reaches the mercury in the manometer, and takes the place of the second bulb of the cautery apparatus. An escape-valve is provided for gradually lowering pressure during the test.

Disadvantages.—It being perhaps somewhat difficult for an author to see the possible defects in an apparatus of his own devising, criticisms of this instrument must therefore be left for others. It is not believed that the size of this instrument, and its weight as compared to the more recently devised pocket types of sphygmomanometer, should be considered detrimental, as many students prefer the mercury type, and use it exclusively in physiologic research.

Faught Pocket Sphygmomanometer (Fig. 7).—This instrument reverts to the type represented by the later models of v. Basch and Potain, in that four metallic diaphragms are substituted for the U-tube of the manome-

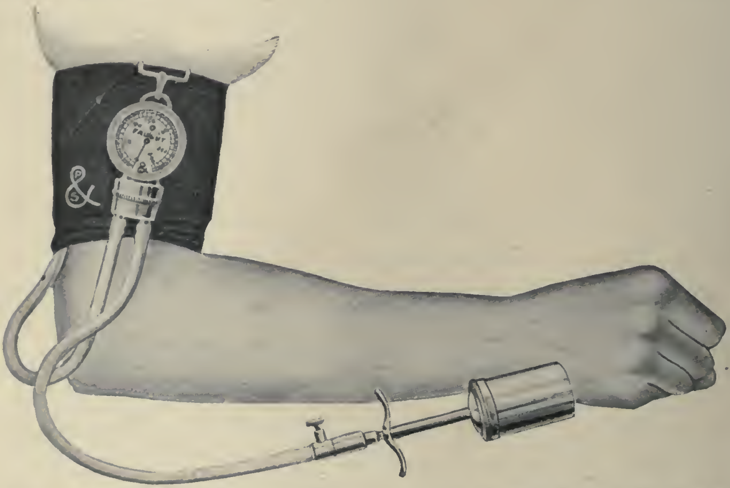

Fig. 7.—Faught pocket sphygmomanometer attached to arm showing position of arm-band, dial attached to hook and arrangement of tube connections.

ter. The result is an exceedingly compact and portable apparatus, which when folded in its case, may be carried in the pocket.

The dial is of white enamel, somewhat similar to that of a watch. The scale is graduated in millimeters of mercury, as determined by accurate callibration with a standard mercury column. The numerals are in red and black, to facilitate reading, and each individual graduation represents two millimeters, giving a working scale extending from zero to 300. No mathematical calculations are necessary to compute the pressure, which can be easily read directly from the dial.

A similar instrument devised by Dr. Rogers is graduated in centimeters only, on a scale which terminates at 260. It is therefore less convenient and less practical than the one above referred to.

The accuracy of the so-called aneroid, spring or diaphragm type of sphygmomanometer has been questioned by some, but the objections appear to be based upon a superficial knowledge of the constructional characteristics and care employed in their manufacture. Special tests made by both the author and by others have shown that the danger of these instruments suddenly becoming inaccurate is very slight, and could only result from clogging from the mechanism and would be detected instantly. The so-called "fatigue of metal" referred to by some authorities does not exist, and any error which manufacturers admit may develop in their instrument, must be due to some mechanical defect, which in the Faught Pocket Sphygmomanometer, at least, has been overcome. In order to insure accurate and unvariable readings at all points on the scale, a factor of safety of 150 mm. has been provided, *i.e.*, each instrument before leaving the factory is tested up to 150 mm. above the 300 on the scale, or to 450, after which the readings must correspond with those of a standard mercury column, and the needle after this severe test must return immediately to zero. This shows clearly that, with ordinary use it is practically impossible to distort the compression chambers of the instrument, and so render it inaccurate. It has never been found necessary to compare the Faught Pocket Sphygmomanometer with a mercury sphygmomanometer to insure its accuracy.

This instrument employs the flexible bandage cuff or arm-band, the inflatable portion of which measures 5×9 in. A small mêtal pump with exhaust valve attached is supplied and these parts in addition to the Morocco pocket case, constitute the latest and most improved type of sphygmomanometer. The needle of the indicator is extremely delicate and so sensitive that a diastolic reading may be made in any case in which a mercury manometer will accomplish it.

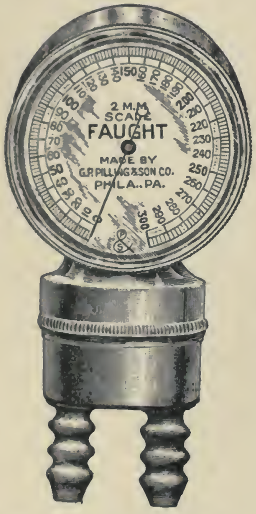

Fig. 8.—Faught pocket apparatus dial in detail.

8. **Bishop's Sphygmomanometer.**—This apparatus is very ingenious. It depends for its operation upon pressure produced by elevating a column of heavy fluid in a flexible tube, to one end of which is attached the arm-band, and to the other a small reservoir. The flexible tube is graduated in the equivalent of millimeters of mercury. The reading is obtained by attaching the cuff to the arm and then elevating vertically the tube with its reservoir until the pressure developed obliterates the pulse at the wrist. At this point the marking on the tube at the level of the arm-band gives the subject's blood-pressure in millimeters of mercury.

Disadvantages.—This apparatus while portable and compact is, on account of the length of the tube, rather awkward for one person to operate, for the same reason the reading is only approximate and is available only for systolic readings unless the auscultatory method is employed.

9. **Erlanger's Sphygmomanometer** (Fig. 9).—This instrument in its improved form is apparently the most accurate

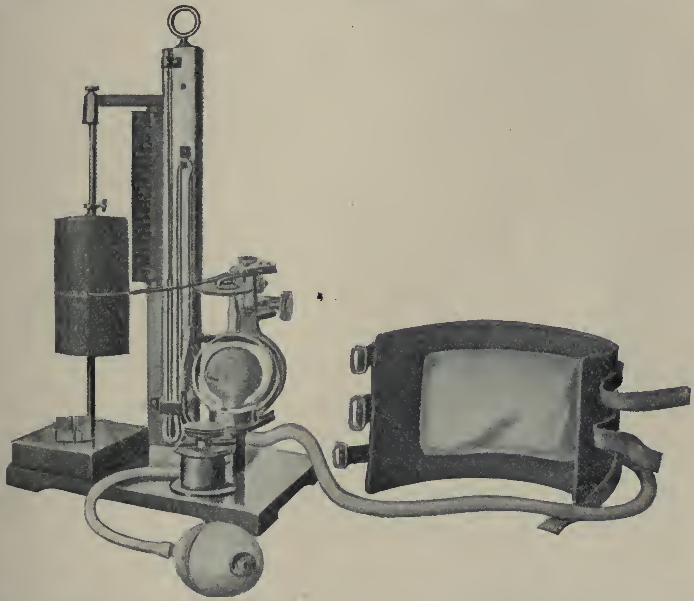

FIG. 9.—Erlanger's sphygmomanometer with kymographion in place, showing arm-band and atomizer-bulb inflator.

yet devised for determining blood-pressure, being based upon the same principle as the other instruments but both the return of the pulse and the point of maximum pulsation are made clearly visible, thus almost entirely removing subjective errors.

The construction of this instrument is more complicated than any other but the only essential difference is the addition of an original recording device. The U-tube manometer connects with a four-way tube, of which one branch leads to the armlet, and another to a special stopcock. The vertical branch communicates with the interior of a rubber bulb, enclosed within a heavy glass bulb, which in turn, under certain conditions communicates freely with the atmosphere through another tube returning to the special stopcock. The object of this glass-encased rubber bulb is to shield the delicate tambour from too sudden changes in pressure. The tambour communicates with the air in the glass bulb outside of the rubber ball, and operating an aluminum needle above the tambour, inscribes its movements on a revolving drum. This makes a tracing upon smoked paper as in the ordinary kymographion. The whole is attached to a metal base and is covered for transportation by a metal case which is somewhat larger than a microscope box and about as heavy.

The standard cuff is employed and pressure is obtained from a Politzer bag. All rubber tubing is of the high-pressure variety to afford rigidity.

The minute details of construction and the operation of the special stopcock are too extensive to include here, suffice to say that with practice in handling the instrument the readings obtained are very accurate and furnish a permanent graphic record of both systolic and diastolic pressures.

Disadvantages.—The chief fault to be found with this apparatus is from the standpoint of clinical availability. Its bulk and weight render it almost useless for clinical work except perhaps in the office or the hospital. The technic of smoking the cylinder and of making necessary adjustments consumes more time than one can generally

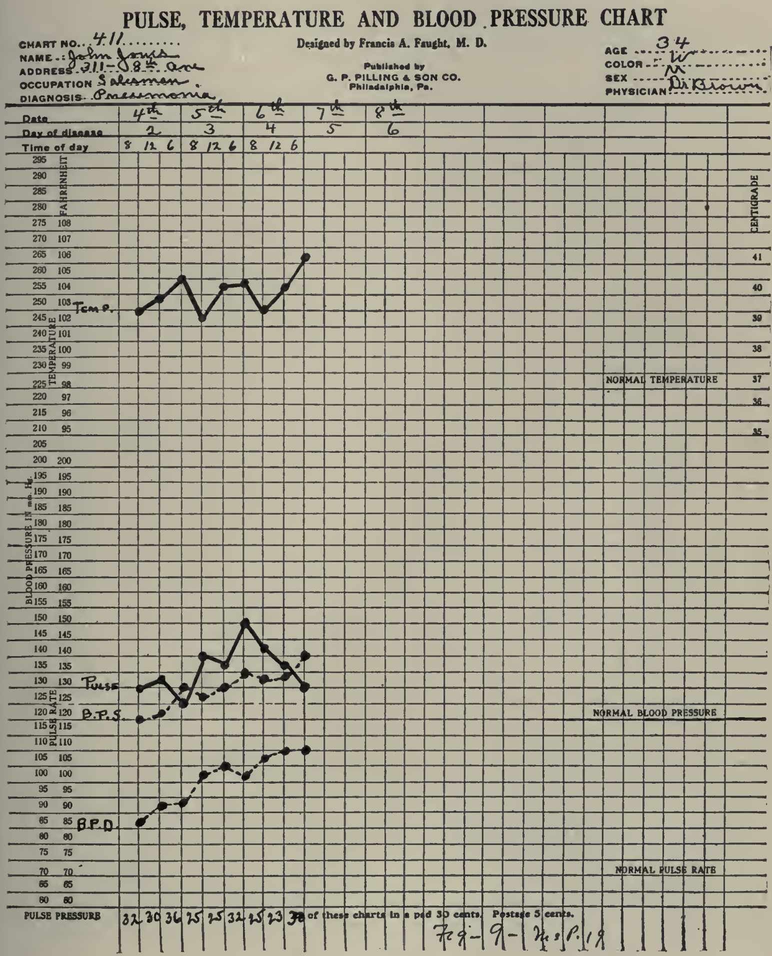

Fig. 10.—Specimen chart.

spare during the pursuit of an active practice. Also the rubber connections and the diaphragm of the tambour so often need replacing at most inconvenient times.

The value of graphite records to-day is sufficiently obvious and needs no argument. We would know little of the characteristic temperature curves of malaria or typhoid fever if we depended for our information upon a long column of figures. The course of blood-pressure is equally easy to chart and the curve thus obtained at a glance tells us much that the perusal of the usual written record would fail to convey. In both acute and chronic diseases and during operations the systolic blood-pressure and the pulse should be charted at regular intervals.

This chart is arranged in the form of a combined pulse, temperature and blood-pressure chart, the several scales being so placed that the pulse, temperature and blood-pressure curves do not become superposed. (See Fig. 10.)

The chart sheet measures 9×12 in., which is the same size as the usual hospital history sheet. The chart may be filled in, in different colors if desired to make the record more graphic, but this is not necessary to its proper keeping. These charts may be obtained in pads of twenty-five from any surgical instrument dealer at a nominal price.

CHAPTER III

THE PRINCIPLE OF THE SPHYGMOMANOMETER CIRCULAR COMPRESSION

This is the basis of modern sphygmomanometry without which the modern sphygmomanometer could not have been developed, and the immense value of this procedure lost to clinical medicine. For obviously the direct method of the physiologist is not applicable, as it requires direct connection between the vessel and the tube leading to the manometer.

It remained for Riva-Rocca and Hill, each working independently of each other, to substitute the arm-encircling cuff for the uncertain and inaccurate pelote of v. Basch.

By means of the encircling arm-band, the pressure produced within the hollow inflatable rubber portion is exerted equally from every direction against the artery. This is true whether the inflatable portion of the arm-band completely surrounds the arm or not. Physiologic experiment has shown that the tissues intervening between the surface and the artery offer a negligible amount of resistance and that observations obtained through the tissues by the modern sphygmomanometer agree very closely with those obtained by the direct method.

It has been said already that the width of the tubular cuff influences to a significant degree the reading obtained. This is easily explained by noting the change which occurs within a narrow (2 in.) and a wide (5 in.) cuff during inflation under a rigid retaining device—reference to the

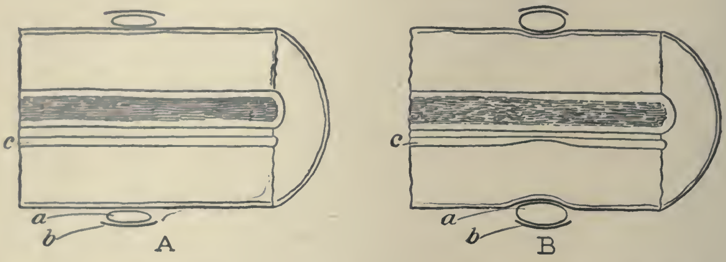

Fig. 11.—*A*. Schematic section of arm, showing narrow arm-band (*a*) with retaining device (*b*) before inflation, artery (*c*). *B*. Same showing change in form of compression band (*a*), after inflation, artery (*c*) compressed. Note great change in form and increase in circumference of compression bag. This change occurred only at the expense of a measurable amount of pressure.

accompanying illustration will aid the explanation. Fig. 11 shows a narrow armlet which allows insufficient material to indent the tissues and compress the vessel without

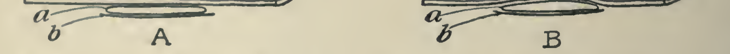

Fig. 12.—*A*. Schematic section of arm showing wide arm-band (*a*) with retaining device (*b*) and artery (*c*) before inflation. *B*. Same, showing change in form of compression bag (*a*) after inflation artery (*c*) compressed, note slight change in form of compression bag, insufficient to exert any additional force than that required to compress artery.

requiring additional pressure to expand the rubber bag, this amount being registered on the scale of the sphygmomanometer in addition to that required to compress the vessel. Chamberlain[1] has determined that this amount of error on an arm of average size is 8 mm. or more.

Fig. 12 shows wide arm-band (*A*) before compression and (*B*) after compression, where the change in form of the rubber portion is insufficient to exert pressure beside that required to indent the tissues and compress the vessel.

Influence of the Vessel Wall.—Upon this subject authorities differ. The early experiments of v. Basch[2] show that the resistance to closure of a normal radial artery scarcely amounts to 1 mm. and Janeway[3] agrees with this. On the other hand Russell[4] does not agree, but states after discussing the factors involved, that "I cannot but think that those who have thought that the vessel wall was negligible have not had the data necessary to correct opinion." The author's belief is that the vessel wall, as a factor, need not be considered from a clinical standpoint as any resistance which could be offered by a vessel even markedly sclerosed would be insignificant when compared to the alterations in pressure occurring within the vessel. I submit as proof the many high-pressure cases that are met where but little change can be demonstrated in the superficial vessels, and on the other hand, I saw a case recently whose superficial vessels (radial so far as it could be digitally traced) were absolutely rigid, so firm that one had the feeling that careless handling would cause them to break, and yet at no time did the blood-pressure register over 110 mm.

[1] Chamberlain, *Philippine Jour. of Sci.*, Vol. VI, No. 6, Sec. B, Dec., 1911.
[2] *Berlin klin. Wochen.*, 1887, Vol. XXIV.
[3] Janeway, Clinical Study of Blood-pressure, p. 61.
[4] Arterial Hypertension, Arteriosclerosis and Blood-pressure, J. B. Lippincott, 1908, p. 52.

It seems safe to assume that the vessel wall as a definite factor can be absolutely eliminated because all pressures are read through the vessel wall, which always being included can clinically at least be ignored.

Influence of Other Intervening Structures.—Vital tissue is perfectly elastic. Therefore any pressure applied to the surface of the body is directly transmitted to the underlying structures without loss of force.

Pressure is applied to an accessible part of the body over a large blood-vessel such as the brachial. If the amount of this pressure is sufficient to overcome the pressure of the blood within the vessel, the vessel will collapse and the pulse be prevented from passing beyond it. If the amount of the compressing force is measured and expressed in definite terms of weight (as millimeters of a column of mercury) then we can, by applying just sufficient pressure to collapse the vessel, measure the amount of force exerted by the blood in resisting this collapse.

In practice the pressure is produced by a cautery bulb or a small hand pump, and is applied to the arm by means of a hollow flat rubber bag. This is wrapped about the arm and held there by some form of inelastic cuff. Communication with a mercury manometer measures the amount of pressure applied to the vessel.

Fig. 13 *A* and *B* shows the relation of the compression bag to the artery. In Fig. *A*, the pressure within the cuff is greater than the blood-pressure within the artery, which is therefore collapsed and the pulse in the distal end of the vessel cut off. In Fig. *B* the pressure in the cuff has been reduced so that it is a fraction of a millimeter less than the systolic pressure within the vessel. Now at each systole a small amount of blood passes the constriction and will reach the distal end of the artery, where the wave can be felt by the palpating finger at the wrist.

Fig. 14 *A* and *B* represents the conditions existing between the constricting cuff and the vessel at the diastolic

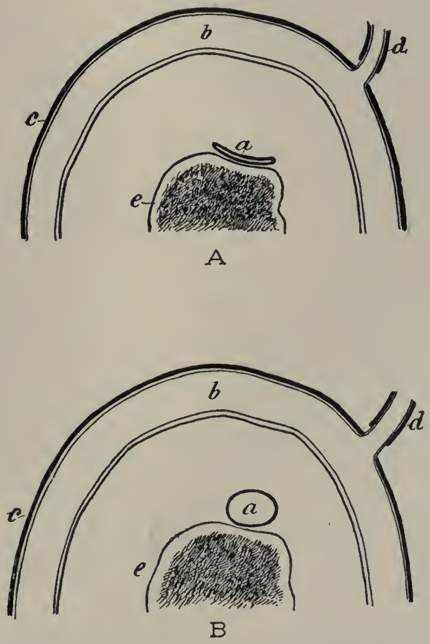

Fig. 13.—*A*. Pressure in "*b*" 135 mm. Hg., pressure in "*a*" 130 mm. Hg., B is therefore collapsed, pulse cannot pass. *B*. Pressure "*b*" 129 mm. Hg., pressure in "*a*" 130 mm. Hg., pulse passes. Diagram of relations of armlet to brachial artery. Explanation of systolic reading: a, artery; *b*, compressing armlet; *c*, retaining cuff; *d*, tube to manometer; *e*, humerus.

time of pressure. *A* represents a pressure within the cuff less than the systolic pressure in the vessel. This is insufficient to affect the vessel during the systolic period. *B* shows the artery and cuff during the diastolic period, when the pressure within the artery is at its lowest point, a fraction of a millimeter less than the pressure within the cuff. Consequently the artery is collapsed at this time. The effect of each succeeding systole is to alternate between a round and a flat vessel at the point of compression. This affects the pressure of air within the cuff

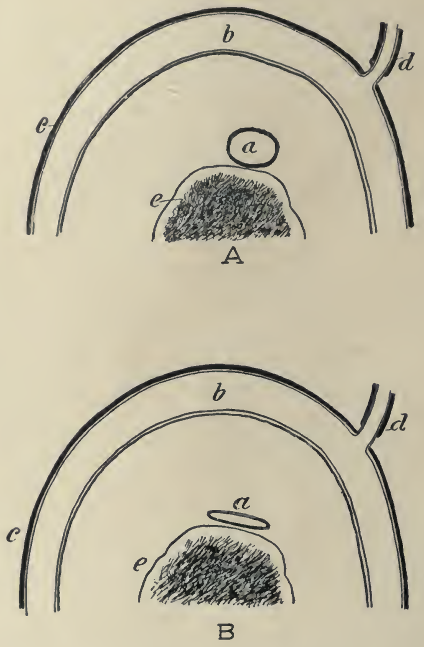

FIG. 14.—A. Systolic pressure in "*a*" 130 mm. Hg., pressure in "*b*" 101 mm. Hg., artery not compressed. B. Diastolic pressure in "*a*" 100 mm. Hg., pressure in "*b*" 101 mm. Hg., artery collapsed. Diagram of relation of armlet to brachial artery. Explanation of diastolic reading; *a*, artery; *b*, compressing armlet; *c*, retaining cuff; *d*, tube to manometer; *e*, humerus.

which is in turn transmitted to the mercury column of the manometer and becomes visible in the rhythmic fluctuation of the column of mercury which is synchronous with the pulse beat. Since the fluctuation will reach a maximum at the time when the pressure in the cuff is approximately equal to the diastolic pressure in the vessel, we are justified in considering the base of the manometer column at this time a measure of the diastolic pressure within the vessel.

Method of Application.—The practical application of the modern sphygmomanometer is a very simple procedure requiring very little experience and occupying very little time.

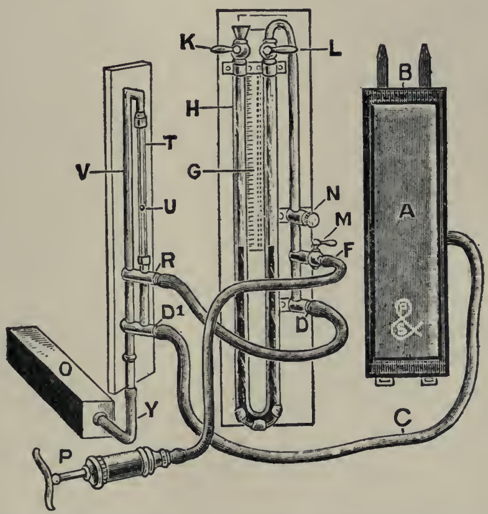

FIG. 15.—Explanatory diagram showing individual parts of apparatus: *A*, armlet—rubber; *B*, armlet—leather; *C*, armlet—connection; *D*, nipple for armlet; D^1, nipple for armlet; *F*, nipple for pressure; *G*, scale; *H*, manometer tube; *K*, guard cock; *L*, guard cock; *M*, pressure guard cock, *N*, exhaust valve; *O*, pneumatic chamber; *P*, pump; *R*, union nipple; *T*, oscillometer tube; *U*, oscillator; *V*, vertical connection; *Y*, flexible connection.

The component parts are much the same in all makes, excepting for slight differences in detail, such as the construction of the arm-band, location of attachments for tubes, etc. Therefore a detailed description of one will serve equally for all.

The accompanying illustration (Fig. 15) will serve as a key, the letters upon it being uniform throughout all the illustrations of the Faught-Pilling Instruments and including the Fedde Diastolic Indicator.

DIRECTIONS FOR OPERATING THE STANDARD SPHYGMOMANOMETER

The patient should be in a comfortable position, and either in a sitting or a reclining posture. The instrument should be upon a level surface within easy reach of the examiner.

The lid is then raised until it locks in a vertical position. If the tube from the pump is not already connected to the nipple *F* it should be firmly attached to it. The two mercury guard cocks *K* and *L* at the ends of the U-tube should be opened and the escape valve *N* tightly closed.

The hollow rubber bag of the arm-band *A* should be firmly wrapped around the bared arm of the patient and securely bound there by the leather cuff and straps *B*. The cuff should be applied snugly, but not with pressure, as it is not designed to compress the member, but only to restrain the inner rubber bag while pressure is applied to it.

The tube from the arm-band *C* is attached firmly to the nipple *D*. The cock in the nipple *F* is opened.

This arrangement forms a continuous closed pneumatic system communicating freely with the manometer tube of the instrument. Now when pressure is raised in the arm-band by the hand pump, the amount of force exerted is indicated by the rise of the right-hand column in the manometer tube H, the height of which will be indicated on the scale *G* in millimeters of mercury.

To Obtain the Systolic Reading.—With one hand find the pulse at the wrist of the arm, to which the arm-band has been applied. The fingers should be in a comfortable position and under no circumstances should be moved during the observation. Care should also be observed that the pulse is not cut off by undue pressure of the palpating fingers. The cuff should be in the same horizontal plane as the subject's heart.

Palpatory Method.—While the pulse is thus under observation, the pressure in the apparatus is raised by means of the hand bellows or pump until the pressure within the constricting band is sufficient to prevent the pulse from reaching the wrist. When this is accomplished the cock in the nipple *M.* is closed to eliminate the elastic pressure. Now by a fraction of a turn in the valve *N* the pressure in the system is slowly released. During this part of the procedure, a close watch should be kept upon the height of the mercury column and for the return of the first pulse beat at the wrist. The level of the mercury column at the instant that the pulse passes the compression band will represent the systolic pressure in the vessel under observation. It is advisable to repeat this procedure a few times to check the correctness of the finding.

Auscultatory Method. (Fig. 16.)—In 1905, Korotkow first discovered that when the bell of a stethoscope was placed over the brachial artery just below the cuff of the sphygmomanometer, a series of characteristic sounds could be heard when the pressure was gradually released. These sounds, of which three were described, were found to bear a definite relation to the character of the pulse and to the systolic and diastolic blood-pressure in the artery. When the artery is compressed, and no blood passes the cuff, no sound can be heard in the stethoscope. The first sound to appear is a clear sharp tone which corresponds to the first pulse wave to pass beyond the cuff (first phase) the third sound is dull and quite suddenly disappears (third phase). This point was believed to indicate the moment of diastolic pressure within the cuff.

Fig. 16.—Auscultatory blood-pressure test.

Subsequent observers, among them Ettinger and Goodman and Howell[1] have shown that there can usually be distinguished five phases, which are described as follows:

First phase. A sharp clear tone, which indicates the first passage of the arterial stream beyond the cuff (first phase of Korotkow).

[1] *Arch. Int. Med.*, Vol. VI, 1910.

Second phase. This same tone dulled combined with a series of faint murmurs.

Third phase. A change to another sharp clear tone, which more or less suddenly becomes dull.

Fourth phase. The moment that above-mentioned change occurs.

Fifth phase. All sound disappears (third phase of Korotkow).

Much critical work has been done to determine, if possible, the significance of these tone changes and their bearing on the circulation in health and disease. A summary of this work is as follows:

The appearance of the first sound measures accurately the systolic pressure. This point has been confirmed by records made with the Erlanger instrument.[1] The reading by this method has been found to be 10 to 15 mm. higher than the method of palpation. According to Warfield, all phases are not by any means always differentiated.

The tones are dependent upon three factors:

First, heart strength.

Second, size of artery.

Third, arterial elasticity.

The third tone normally is the loudest, and it is generally believed that a loud, long clear third phase is indicative of a strong heart, a weak third phase a weak heart, and an absent third phase a greatly weakened, dilated heart. Arteriosclerosis accentuates the third phase so that when present we may note a good third phase even with a weakened heart.

Goodman and Howell (*loc. cit.*) direct attention to the

[1] Warfield, *Interstate Med. Jour.*, Vol. XIX, No. 10, p. 860.

varying strength and quality of the sounds in cases of irregular hearts, and they believe that they were able to detect slight irregularities in force, more readily by auscult-ing the artery than by the heart itself.

Another conclusion of these investigators is that, a long drawn-out fourth phase is very significant of aortic insufficiency, in this Warfield's studies agree.

It is generally accepted that the disappearance of all sound measures the diastolic pressure. Warfield contests this and proves his contentions by studies made with the Erlanger instrument. However this may finally be settled, it would seem best to adhere to the present view, so that records made from time to time by different observers shall be comparable.

To Obtain the Diastolic Pressure.—The diastolic pressure may be obtained in several ways. The method employed will depend upon the character of the instrument used and the preference of the operator. The methods will be described in the order in which they have been devised.

1. *Visible Method.*—This depends on the to-and-fro motion imparted to the mercury in the U-tube, which occurs after the pressure has fallen below the systolic point. Having determined the systolic pressure, again raise the pressure to a few millimeters above this point and immediately close the valve M. Now allow the pressure to fall very slowly by releasing the air through the valve N.

As the mercury falls below the systolic point, there will in most cases be noted a rhythmic motion synchronous with the pulse. This gradually increases in amplitude up to a certain point, after which it decreases and finally ceases before zero pressure is reached. During this gradual fall, the base of the mercury column, when the mercury is making the greatest excursion, represents the diastolic pressure.

2. *Palpatory Method.*—Raise the pressure within the apparatus to the systolic point, then, while keeping the fingers on the pulse, allow the mercury column to fall gradually as in the first method. It will then be noted that at first the pulse is very feeble and thready in character and continues so for a time, when, as the pressure falls, it will suddenly assume the full bounding character of the pulse of aortic regurgitation. At the moment that this change occurs the height of the mercury column will represent the diastolic pressure in millimeters of mercury.

3. *Auscultatory Method.*—This is also available for the diastolic readings, and employs, as in the systolic method, a stethoscope placed over the vessels at the bend of the elbow. As the pressure is allowed to further recede within the apparatus, a series of tones may be heard (see page 47), until a point is reached when a soft blowing murmur develops, to almost immediately disappear, after which no further sound is audible. It has been found that this last sound is heard at the diastolic period, and therefore a reading of the sphygmomanometer at this moment will indicate the diastolic blood-pressure.

The auscultatory method has the advantage over all others in that it is available in every case regardless of the size and volume of the pulse, and can be applied to any make of sphygmomanometer. It should be borne in mind, however, that diastolic readings made by the auscultatory method may be from 10 to 15 mm. lower than those obtained by the visible or the palpatory methods.

A special stethoscope has recently been devised which is a great aid in performing the auscultatory method. This, as shown in the accompanying cut (Fig. 17), is a Bowles stethoscope with a button-like projection from the

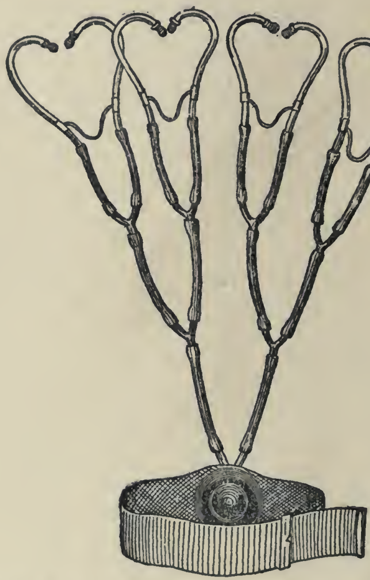

FIG. 17.—Multiple sphygmometroscope. Adaptation of multiple Bowles stethoscope for auscultatory reading of blood-pressure.

face of the diaphragm, which greatly facilitates application to the artery below the sphygmomanometer cuff. This is secured in position by a narrow cuff fastened with a friction buckle. This little apparatus is self-retaining and allows the operator the freedom of both hands with which to manage the sphygmomanometer. This is quite important as it will be found expedient to note the first disappearance of the pulse by palpation of the radial, as in the other methods, thereby preventing accidental or careless overcompression of the arm.

The accompanying illustration presents a new device to facilitate the teaching of blood-pressure readings by the auscultatory method.

The chief drawback to this method has been the seeming difficulty which the average physician has in learning to perceive and interpret the sounds heard over the artery. In thinking over this matter, the idea suggested itself that, if the sphygmometroscope was made into a multiple of say four, whereby the sounds could be heard by more than one individual at the same time, it would overcome this difficulty and make it possible for anyone familiar with the sounds heard during auscultatory blood-pressure observations to direct the attention of a small group of observers during the actual performance of the test. This has been done,[1] and the result proved highly satisfactory. No difficulty resulting from the distribution of the sound through a larger tubular system.

This device would be found valuable, particularly to the medical teacher, as it has been my experience that many students go through their clinical studies without ever actually hearing or seeing the thing demonstrated. It is applicable also in demonstrating conditions, involving marked variations in pressure, to medical societies or groups of medical men.

4. *Diastolic Indicator.*—This may be attached to any make of sphygmomanometer and is of decided advantage in determining an accurate diastolic pressure when the

[1] J. F. Prendergast, *N. Y. Med. Jour.*, vol. xcvii, No. 2, 1913.

systolic pressure is very feeble. (See Fig. 18.) Its application is very similar to Method No. 1 except that the movement of the mercury column is ignored and the movement of the pith ball in the small vertical tube relied upon to determine the diastolic pressure.

Reference to Fig. 15, will show the method of uniting the Fedde Indicator to a sphygmomanometer. It will be noted that the narrow perpendicular glass tube contains a small light ball of pith or cork which is free to move up and down within the tube.

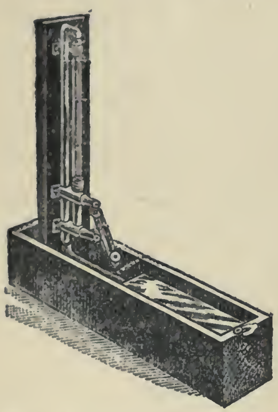

FIG. 18.—Fedde indicator as separate unit.

When determining the systolic pressure pay no attention to this indicator, as each impact of air will make the ball dance violently, but this has no bearing on the test. When the pressure has reached the systolic point close the valve *N* when the ball will begin to move slightly in rhythm with the pulse. This motion gradually increases, until it reaches a maximum as the level of the mercury column gradually falls, when quite suddenly, its motion becomes markedly less. At the moment of this reduced movement the level of the mercury will indicate the diastolic pressure.

It must be borne in mind that the indicator gives a diastolic pressure considerably lower than 1 and 2 (about 10 to 15 mm.).

Cautions.—To obtain accurate and reliable clinical data with the sphygmomanometer, it is important that some systematic technic be adhered to, and that all observations not only on the same patient, but in all cases, be made under as nearly the same conditions as possible. Attention to detail will eliminate largely the errors arising from such factors as position of the patient, presence of fatigue or mental excitement, arm used for observation, etc. It is also valuable to note the apparatus used, the width of cuff, the time of day, the pulse rate, the sex and age of the patient.

Care should also be taken to see that the observation is not too prolonged, for the interruption of the circulation in the extremity will, if continued, itself cause changes in pressure.

No single reading should be accepted when it is possible to make more than one. It is better to see a patient a number of times under varying conditions before finally deciding what his blood-pressure is.

CHAPTER IV

THE SPHYGMOMANOMETER AND METHOD OF ITS USE

FACTORS INFLUENCING BLOOD-PRESSURE

There may be still some who are loath to accept the new order of things. Old-fashioned practitioners, who either cannot or will not see anything good in the many aids to diagnosis employed by physicians. We are now in an age of development and progress, and he who does not progress, recedes, there is no middle ground. The following abstracts are particularly directed toward the skeptic, or he who has so far failed to see the value of the sphygmomanometer. Dr. Janeway asks and answers this trite question:[1] "When should the general practitioner measure the blood-pressure?" To this he replies "First, in every careful examination of the cardiovascular system. Second, in the first examination of every new patient, and the occasional examination for purposes of establishing prognosis in cases of hypertensive cardiovascular disease and in nephritis. Third, in examinations for the certification of health, such as applicants for life insurance, recruits for the army, navy, police, fire department, etc., and the examination of boys and others for competition in athletics." This summary fairly expresses the sentiment of a large number of clinicians.

[1] Theo. C. Janeway, *Albany Medical Annals*, March, 1911.

Add to this an almost unlimited field offered by many pathologic conditions in which the blood-pressure findings have been established, and the almost universal applicability of the sphygmomanometer will be readily appreciated.

In order to intelligently employ and to clinically estimate the value of blood-pressure findings in any case, we must know what constitute the normal boundaries of blood-pressure, what factors may normally influence the reading and what constitutes an abnormal or pathologic blood-pressure.

The Normal Blood-pressure.—Mechanical difference in instruments apart from accidental error, due to defective manometers (which is now rare) must be considered, especially when comparison is made between figures, obtained some years ago and now. In the early days of sphygmomanometry, the width of the arm-band, and the method of application of pressure was not critically considered, so that, except when indicated, it cannot be determined whether figures refer to pressure tests made with a 4-, 8-, 12- or 16-cm. cuff, or whether any cuff at all was used, as with the early instruments of v. Basch and Potain.

All instruments employing the mercury scale or its equivalent, will under the ordinary conditions give similar readings. The chief cause for difference is in the use of cuffs of varying width. The standard cuff as now accepted by most authorities is one having a width of compression surface of 4 1/2 to 5 in. (11 to 13 cm.). This, in all but the most obese, will give uniform pressure readings, which by actual experiment have been found to correspond closely to the figures obtained by the direct introduction into a vessel of a canula communicating with a mercury manometer.

The cuff of Riva-Rocci, and as employed by Cook, in his simplification of the Riva-Rocci apparatus, measures 8 cm. in width. This has been found to interpose some resistance of its own, due to stretching of the rubber of the cuff, so that readings obtained by it are from 6 to 10 mm. higher (depending on the circumference of the arm) than those obtained by the standard cuff. Therefore all figures obtained by the narrow cuff must be corrected, by the subtraction of 6, 8 or 10 mm. before they can be compared to the standard reading.

This difference has been carefully figured out by Chamberlain[1] and others.

The sphygmomanometer of Potain is not graduated in mm. of Hg. at all, and therefore cannot be directly compared to the figures obtained by other instruments. Potain in his work on blood-pressure gives the normal with his instrument as 150 to 190 for men and 140 to 180 for women. The readings with the Gaertner tonometer range from 10 to 20 mm. below the standard.

If we accept the 12-cm. cuff as standard and employ it for a basis of comparison, we find that a number of so-called physiological, or normal factors influence blood-pressure readings, and that in any study of blood-pressure, these must be considered.

It has been established that the blood-pressure is influenced normally by:

Age.

Sex.

[1] *Philippine Jour. of Sci.*, December, 1911.

The time of day.
Size and temperament.
Digestion.
Muscular development.
Muscular exertion.
Mental worry or fatigue.

These several factors will be considered in order, and an effort made to outline their influence.

In connection with this subject, it seems necessary to include in this group a consideration of tobacco and alcohol indulgence, because of their general employment by men at least. They should always be noted in an estimation of blood-pressure.

Age and Sex.—Janeway in more than 2,000 blood-pressure determinations has found the high normal limit of systolic pressure, with very few exceptions, to be 145 mm.; his figures for women are 10 mm. less.

The Diastolic Pressure.—There are few reports to be found in literature bearing upon this. Janeway on the basis of several hundred readings of about 200 cases believes the diastolic pressure to be from 25 to 40 mm. below the systolic pressure in a normal individual. This holds good only during repose, for posture, exertion, etc., affect the two pressures unequally.

H. P. Woley[1] reports his examinations of 100 healthy subjects between the ages of fifteen and sixty years. The results are shown in the accompanying chart (Fig. 19). Except for slight variations the figures obtained are in close accord with the results of other observers. Lauder

[1] *Jour. A. M. A.*, Vol. LV, No. 2, p. 121.

Brunton states[1] that the normal pressure in children between eight and fourteen years is 90 mm., in youth from fifteen to twenty-one, 100 to 120.

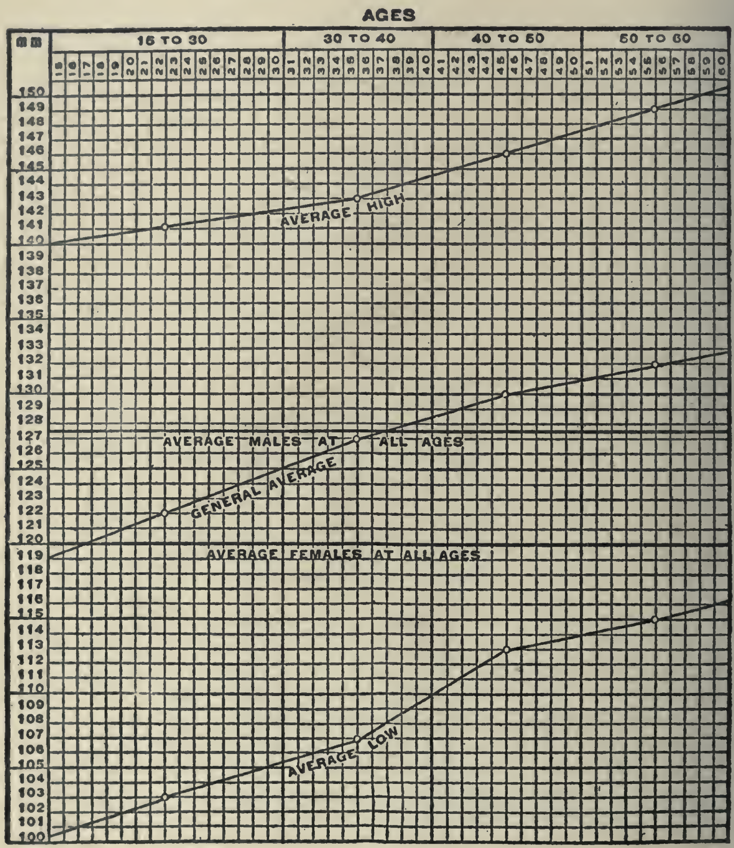

Fig. 19.—Woley's chart showing effect of age on blood-pressure, giving mean, high and low average.

Krehl[2] and Cook give from 75 to 90 mm. as the normal systolic pressure during the first years of life.

[1] *Lancet*, Oct. 17, 1898.
[2] Clinical Pathology.

Periodic Variations.—The respiratory and Traube-Hering and the other less rhythmical but apparently spontaneous fluctuations in the normal blood-pressure must be borne in mind in all clinical experiments. The respiratory waves are usually very slight during quiet breathing and need not be considered.

Exact figures are wanting. According to Janeway[1] this variation amounts to as much as 30 mm. Ordinarily 5 to 10 would be a liberal estimate.

Daily Variations.—The record of blood-pressure taken at frequent intervals throughout twenty-four hours shows variations from the average level so striking and of such great extent and long duration that they cannot be easily explained. These are shown in the accompanying figure (Fig. 20).

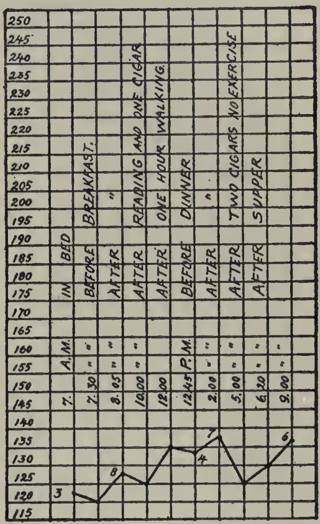

Fig. 20.—Record of systolic pressure variations occurring during the working hours of a young healthy man.

The record shows the effect of physical and mental strain on blood-pressure, these varying in different individuals and in the same individual from day to day. Janeway believes that this variation may reach 60 mm. of Hg., although I have never seen such great fluctuation.

Old Age.—As early adult life is passed we have to deal with those progressive changes in the cardiovascular system which are the inevitable result of the wear and tear of every-

[1] Clinical Study of Blood-pressure.

day life, and which show themselves in a gradually progressive reduction in arterial tonicity, a lessened functional activity of the eliminative organs, particularly the kidneys, and degenerative changes in the myocardium. The inevitable result of these is a gradual elevation in the systolic blood-pressure. We now have to establish new normals by which we may determine the pathological. For this purpose the author published in 1909 the following rule,[1] which will serve as a practical guide: "Consider the normal average systolic pressure at age twenty to be 120 mm. Hg., then for each year of life above this add 1/2 mm. to 120." Thus at age sixty the average normal systolic pressure would be 140 mm. Hg., and this estimate adheres closely to figures given by Woley, Janeway and others (see page 59). Of course figures obtained by this, or any other arbitrary formula, are possessed of great elasticity and may be so modified by other so-called physiologic factors as to lose their identity entirely.

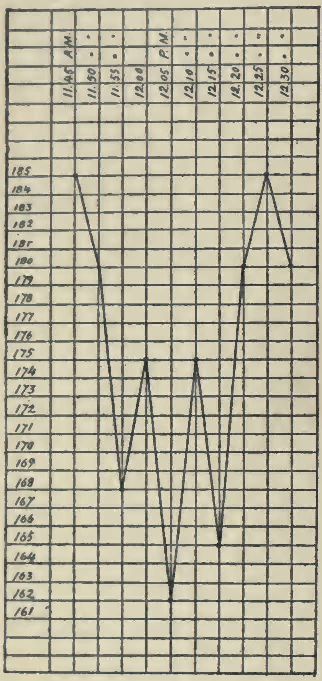

FIG. 21.—Rapid variation in blood-pressure, occurring in forty-five minutes, patient sitting quietly in office.

Size and Temperament.—With the standard armlet the factor of size of the individual (adult of course) does not

[1] The Sphygmomanometer and its Practical Application.

enter. Temperment on the other hand does undoubtedly affect the reading, because in nervous persons it is often impossible entirely to remove the effect of psychic influence; allowance must therefore be made for an abnormally high reading in such persons, particularly when the reading fluctuates to a surprising degree in a limited period of time (Fig. 21).

Sleep.—Authorities agree that the lowest blood-pressure during the twenty-four hours occurs in the first hours of sleep and that a gradual rise occurs toward morning.[1] During the day there is a physiologic rise which reaches its maximum in the evening. (See author's chart.)

Posture.—This should not be confounded with the influence of gravity which may be eliminated by making all observations with the cuff on the level of the heart. Authorities differ, perhaps because of the many possibilities of error arising from accompanying physical and mental effort. The weight of opinion, however, seems to show that pressure tends to rise as the individual passes from the standing to the head-down posture.[2]

Factors Influencing Blood-pressure.—POSTURE: The following series of observation were made upon twenty-two healthy medical students.[3]

[1] Brush and Fairweather, *A. M. Jour. Physical,* Vol. V, p. 99.

[2] O. Z. Stephens, *Jour. A. M. A.,* Oct. 1, 1904; A. M. Sanford, *Jour. A. M. A.,* Feb. 15, 1908.

[3] O. Z. Stephens, *Jour. A. M. A.,* Oct. 1, 1904.

Ten Centimeter Cuff. Pressure in Millimeters of Mercury

Systolic Pressure and Pulse Rate[1]

Systolic pressure	Standing	Sitting	Supine	Head down	Right lateral	Left lateral
Right arm	132.6	133.3	152.5	166.2	155.0	110.0
Average	130.8	131.7	150.4	165.6	143.5	133.0
Left arm	130.0	130.0	148.3	165.0	114.0	156.0
Pulse rate	86.0	82.0	68.7	65.8	68.1	69.1

Systolic and Diastolic Pressures[2]

	Standing	Sitting	Supine	Head down
1—Arm Systolic	84	90	94	100
Diastolic	70	70	76	80
2—Arm Systolic	126	124	132	134
Diastolic	110	110	112	115

From these observations the following conclusions can be drawn as to the effect of posture upon blood-pressure, pulse pressure, and pulse rate.

These observations show that there is little change in pressure between the standing and the sitting posture; occasionally there is a rise of a few millimeters, possibly due to an increase in the intra-abdominal pressure. Between the standing and recumbent the rise may be as much as 20 mm. Between the standing and the head-down (Trendelenburg) the rise may reach 35 mm. Hg. Most observers note a compensatory lowering of pulse rate, and it is upon these two factors (change in pressure and change in pulse

[1] O. Z. Stephens, *Jour. A. M. A.*, Oct. 1, 1904.

[2] Sanford, *Jour. A. M. A.*, Feb. 15, 1908.

rate) that the so-called functional tests have their basis (see page 164).

Prolonged rest in bed, in one accustomed to be up and about (unaccustomed rest of Gumprecht) especially if there be a tendency to high pressure, causes a rapid and marked fall, with the establishment of a new systolic level.

Emotion and Excitement (Vasomotor).—In determining psychic influences in blood-pressure, temperament plays an important part. The temporary pressure-raising effect of fright, fear, apprehension or other form of mental influence, must always be recognized. Vasomotor changes from the application of heat and cold, and those occurring in the arm from prolonged pressure of the arm-band, may amount to 5 or 10 mm. and must not be ignored. Every effort should be made before and during the test to eliminate these several disturbing factors. This may be done by establishing a proper understanding between the patient and physician, by repeating the test at another sitting if necessary, and by performing the test with as little delay as possible.

Muscular Development and Exercise.—It is believed that in the muscularly well developed, the normal systolic pressure may be from 5 to 15 mm. above that of a physically weak individual.

It has been long known that muscular work usually increases systolic blood-pressure. This is sudden and sharp in the healthy and may cause an elevation of from 5 to 15 mm. This rise becomes less marked as subjects become accustomed to performing the act or acts and this reduction in the excitability of the cardiovascular system is one of the beneficial effects of training. When the effort is moderate and prolonged (as in walking) the systolic pressure may rise from 5 to 10 mm., but soon becomes adjusted to a new level upon which additional exertion has little, if any effect, until a condition of fatigue is reached. Fatigue after prolonged exertion results in a fall in pressure which progresses until a dangerous fall in pressure may occur.[1] During moderate exercise in a normal person, the systolic and diastolic pressures tend to become more widely separated, *i.e.*, the pulse pressure becomes greater;[2] upon this physiologic fact is based the work test of Graupner[3] (see Chapter XII).

Passive movements and massage produce no appreciable effect on blood-pressure (Eichberg) (see page 246).

Altitude.—(See Chapter VI.)

Diet and Digestion.—The difficulty of arriving at definite conclusions concerning the effect of this factor is great. Some authorities report a fall, others a rise after eating. No intelligent conclusion can therefore be arrived at for the present.

The ingestion of large amounts of fluid, particularly beer, usually will cause a temporary rise of from 10 to 20 mm.

The Influence of Temperature and Baths.—From a practical standpoint the influence of the external temperature is insignificant and may be ignored. The effect of baths will be fully treated in Chapter XIX.

Atmospheric Pressure.—(See Altitude.) Pomeroy[4] states that the result of nearly all experimental data show that the effect of diminished barometric pressure upon the

[1] Kavenstein, *Zeitschr. f. klin. Med.*, 1903, Vol. L, p. 322.
[2] Krehl, "Clin. Path.," 1905, 3rd Edition.
[3] Die Messung der Herzkraft, 1905.
[4] *Interstate Med. Journal*, 1911.

human organism is to lower the blood-pressure and that the result is not transient but is permanent during the continuance of the low barometer.

Alcohol and Tobacco. *Alcohol.*—Clinical evidence so far shows that a moderate daily use of alcoholic drink does not materially influence blood-pressure. Large amounts of beer, owing to the bulk of fluid causes a temporary rise of from 5 to 15 mm. (See Arteriosclerosis, page 118.)

Physiologically, alcohol is not a stimulant, and direct injection into a vein does not cause a rise in pressure; on the contrary large doses cause a diminution in blood-pressure from vasodilatation.[1]

Tobacco.—The alkaloid of tobacco, nicotin, is, next to adrenalin, the most powerful vaso-constrictor known (see page 270). Cook and Briggs[2] have shown a temporary rise in blood-pressure following smoking, and yet we have the apparent paradox that those who indulge in excessive smoking have a subnormal blood-pressure. The moderate use of cigars has been found by many, including the author (Fig. 22), to cause reduction in pressure, while continuous smoking resulted in a rise from 5 to 15 mm.

Conclusions.—This collection of facts relating to the many transitory factors influencing the normal blood-pressure level would, on first thought, lead the reader to the conclusion that after all little can be learned from the clinical study of blood-pressure, because of the apparent difficulty of separating the real from the false variations. This is not true. We must be in possession of a working knowledge of the physiological and environmental con-

[1] Cushny, Pharmacology and Therapeutics, Phila., 1903.
[2] *Johns Hopkins Hospital Reports*, 1903, V. XI.

ditions affecting our observations that we may, by properly gauging and excluding them, arrive at a clearer conception of the condition of the cardiovascular renal system, and follow more intelligently the effect of therapeutic measures.

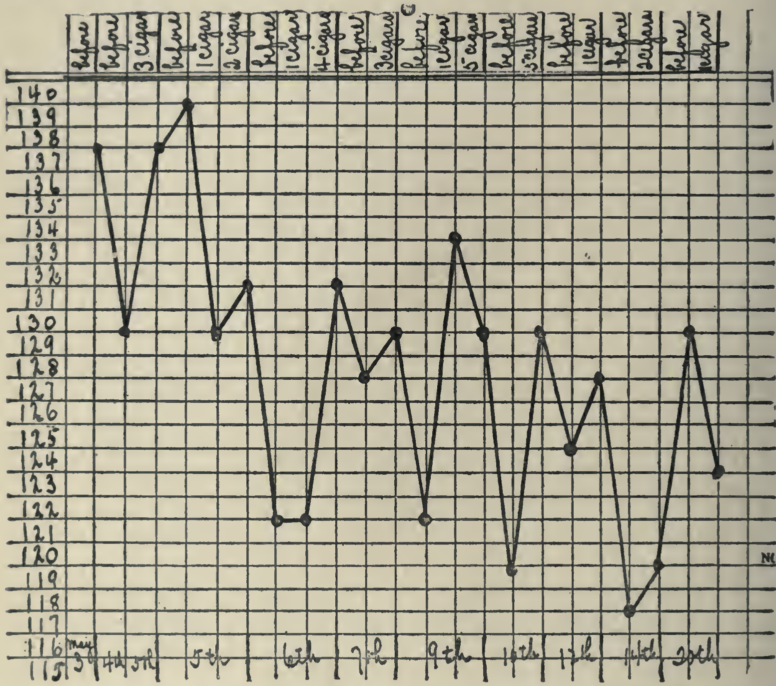

FIG. 22.—Showing the effect of moderate smoking on one accustomed to the use of tobacco, after a brief period of abstinence. Tracing shows very well the sedative effect of a moderate amount of tobacco, and the pressure-raising influence of several cigars smoked in rapid succession. Also the general downward tendency of the curve would suggest that the individual became gradually reaccustomed to the use of the drug.

Furthermore, it will be noted that these obscuring factors are all confined to a comparatively short division of the scale of the sphygmomanometer, while the truly significant variations are often measured by alterations of from fifty to a hundred or more millimeters of mercury.

CHAPTER V

TERMS, DEFINITIONS, ETC.

Before proceeding to a discussion on the subject of blood-pressure in its relation to the human organism in health and disease, it is necessary and indeed essential that the several terms employed should be defined, and that their relation to the different events in the cardiac cycle should be definitely understood. These will here be taken up and discussed serratim.

Capillary Blood-pressure.—The pressure of the blood in the capillaries is low because of the resistance offered to the progress of the blood by the fine bore of the vessels, and because of the relatively large cross-sectional area of all the capillaries compared to that of the aorta and great vessels.

If one press with a blunt object upon the skin just below the matrix of the finger-nail, the ruddy surface becomes pale, because the capillaries are flattened by the force applied and the blood driven out of them. If delicate weights or a spring be used to apply the pressure, then the force which is just sufficient to whiten the tissues can be measured, and the amount of pressure which approximately counter-balances the pressure within the capillaries can be definitely determined.

The capillary pressure, measured by this means, has been found to be much lower than the pressure in the arteries, and considerably higher than that in the great veins. This capillary pressure has been found to equal that required to sustain a column of from 24 to 54 mm. of Hg.[1]

From our knowledge of the physiology of the circulation, we know that the flow of blood through the capillaries is one of the several factors controlling blood-pressure, for it is probably largely through the power of the capillary vasomotors that alteration of the flow of blood through important organs and in different parts is either maintained or compensated for.

Further we believe that the splanchnic area with its vast cross-sectional area, is the most important capillary region. The condition of the capillaries (the volume and rate of blood flow through them) is a part of the factor of peripheral resistance, and has been considered under that head (see page 20).

From a practical standpoint, we as yet have no means of studying the condition of, or the changes in, capillary pressure.

VENOUS PRESSURE

Venous pressure is taken to be that pressure existing in the great veins that are in close relation to the heart and which in a large measure determines the amount of blood entering the right auricle. Of late, venous pressure has been attracting more and more attention, due to advances in our knowledge of the mechanism of the heart and relation of the pressure within the great veins to the volume output of the heart.

[1] Am. Text-book of Physiol., p. 377.

Normal venous pressure is lower than the pressure either in the arteries or capillaries.

Measurement of Venous Blood-pressure.—Howell[1] describes a practical method of venous pressure estimation which, however, is only applicable to the superficial vessels of the extremities. The apparatus consists of a light rubber bag connected with a water manometer, which is placed around the upper arm and held there by a few turns of bandage. Another cuff, made of rubber dam, likewise connected with a water manometer, surrounds the forearm. Air is pumped into the first bag until the veins of the upper arm being obliterated, venous congestion in the forearm is produced, a rise in the water column in the second manometer resulting. When this occurs the pressure in the first manometer, representing the venous blood-pressure, is read off. In making an observation it is necessary that the arm be maintained at the level of the heart. The readings are influenced also by the temperature of the air, the thickness of the skin and the prominence of the superficial veins. These modifying influences are to be allowed for in considering the result, but as the usefulness of venous pressure estimations centers especially in the following of individual cases (as the patients improve or grow worse) the sources of error mentioned are not of great importance. The average normal venous pressure by this method proved to be 7.6 cm. of water. In a series of cardiovascular cases the pressures ranged from 7 to 25 cm., the average being 13.9.

Lauder Brunton[2] suggests a simple way of roughly esti-

[1] *Arch. Int. Med.*, Feb., 1912.
[2] Therapeutics of the Circulation, P. Blakiston's Son & Co., 1908, p. 84.

mating venous pressure. This is done by noting the height above the level of the heart at which the veins of the hand become empty. Normally they should do so at about the level of the third rib, or above. The greater the venous pressure the higher must the hand be raised before the veins will empty. Regarding the effect of alteration in venous pressure upon systolic blood-pressure, Janeway[1] quotes A. Quirin, whose experiments in changes in intraabdominal pressure (forcing blood to right heart) showed that, up to a certain point, increasing abdominal pressure raised arterial pressure. But beyond a certain point, a fall in arterial pressure occurred, finally leading to death if compression of the abdomen was continued. It may safely be accepted that we are dealing with an abnormally high venous pressure, when there are evidences of general right-sided venous engorgement—superficially engorged veins, large liver, cyanosis, etc. In this connection Bishop points out that in determining the degree of arterial hypotension, the chief factor is the approximation of venous and arterial pressures, and that a low pressure need not be regarded as pathologic, unless the venous pressure is abnormally high, and that it is this alteration between the relation of arterial and venous pressures that determines the degree and seriousness of general venous congestion.

Pulmonary Venous Pressure.—We have no method of precision by which the degree of pulmonary venous pressure may be determined. Clinical phenomena alone will indicate this.

[1] Clinical Study of Blood-pressure.

ARTERIAL PRESSURE

By arterial pressure is meant the degree of pressure exerted by the blood flowing within the arterial system. In any individual, arterial blood-pressure at any instant depends upon five separate factors. (See Chapter I, page 22.)

1. The energy or pumping power of the heart.
2. The peripheral resistance.
3. The elasticity of the arterial walls.
4. The volume of the circulating blood.
5. The viscosity of the blood.

All of these vary under normal conditions and in pathological states the changes may become very great. Not only may they vary independently of each other, but they are capable of such complicated interaction through the vasomotor and cardiomotor systems, by which one directly influences the other, that there is still much conflicting testimony. We cannot yet reduce the study of blood-pressure to a definite basis; we can, however, correlate that which is definitely known, and deduce from this a fairly satisfactory working hypothesis.

1. **The Heart Energy.**—The heart is a force pump of intermittent action. The left ventricle during systole forces a volume of blood into the arterial system, during diastole the blood is distributed through the arterial tree into and through the capillary system. Any increase in the rapidity of discharge from the heart in the volume output (ounces per minute) will, all other factors remaining constant, result in an increase in blood-pressure. Conversely, any diminution in the rate or volume output will cause a reduction in blood-pressure. On the other hand,

a compensatory relation between the rate and volume output may permit either to be altered without any appreciable alteration in blood-pressure.

2. **Peripheral Resistance.**—Peripheral resistance is that force present in the arterial system, which has a tendency to retard or prevent the forward movement of the circulating blood. This in the living body is composed of the combined factors of diameter of the conducting tubes, surface friction, distance from the heart and branching of the conducting tubes. It is obvious that any obstruction at the outlet of a distensible tube will increase the pressure of the fluid flowing in that tube. A familiar example of this is the common garden-hose fitted with an adjustable nozzle. The same physical law holds good for the arterial system. Increased peripheral resistance means higher pressure, diminished resistance lower pressure, this is invariable unless some compensating change occurs in the force of the heart.

The other factors, length of conducting tube and friction are so insignificant that they may be left out of all clinical considerations, without introducing an appreciable amount of error.

The Arterial System.—The arterial system is composed of a series of vital tubes, which branch and rebranch continually from the heart to the capillaries. The arterial wall, due to its circular muscular coat, is not only a simple elastic tube but being vital has the power of contractility. This contractility is one of the essential characteristics of arteries and it is due to this function that we have a condition called arterial tone or tonus.

Tonus is a condition of the arterial wall caused by the inherent tension of the muscular fibers. This muscular coat is found throughout the arterial system down to the smallest arterioles, and is under the control of the vasomotor system. Through the mechanism of the vasomotor system the vessel walls are capable of altering their diameter in response to proper stimuli. This power to change diameter is the essential mechanism which controls peripheral resistance.

The degree of tonus is also affected by the composition of the circulating blood. (See Chapter I.) Under normal condition the tonus (peripheral resistance) is controlled by the balance between two opposing sets of nerve fibers—the vasoconstrictors and the vasodilators. The constrictor fibers are the more important, since they are always active; their activity is chiefly concerned in maintaining the normal degree of tonus or vasoconstriction. This tonus is absolutely essential to the maintenance of the circulation, vasomotor (constrictor) paralysis result in such wide-spread dilatation of the arteries that the heart fails because pressure is lowered to such an extent that it fails to pass the capillaries and the veins fail to deliver sufficient blood to the heart to stimulate contraction. Variations in vasomotor tone are constantly occurring in different parts of the body in response to local demands for such a change. This is a physiologic necessity, since functional activity of any part (as muscular exercise) must always be accompanied by increased blood supply.

The varying relation between dilatation and constriction determines the amount and extent of alteration in blood-pressure. The first effect of vasodilatation or vasoconstriction in a small area is either a reduction or an elevation in pressure only in the vessel supplying that part. When this vasomotor change involves a large area, then the alteration in pressure may reach the aorta. Ordinarily, a compensatory change in other areas (notably the splanchnic) occurs which counter-balances the change in the affected area and so sustains normal blood-pressure.

Vasomotor tone, both local and general is under the control of a reflex system, which is markedly affected by many reflexes of remote origin. These may produce either a rise or fall in blood-pressure. These changes may follow stimulation of either sensory or motor nerves. Even the sensation of pain may cause marked temporary alteration in pressure.

One of the most important clinical facts to be remembered when studying blood-pressure is that of all the parts of the vascular system, the abdominal vessels, controlled by the splanchnic nerves, have the greatest effect on blood-pressure. This is due to several reasons, first the great size of this vascular area. It is believed that the abdominal vessels, when dilated, are sufficient to contain almost all the blood in the body. A second reason is that of all the vasomotor areas the splanchnic vasomotors are most easily affected by reflexes from any sensory nerve.[1]

3. **Elasticity of the Vessel Wall.**—The elasticity of the vessel wall is due to the elastic fibers contained in the adventitia, and to the elastic lamina found in the larger vessels which limit the intima, both externally and internally.[2] Were it not for this elastic quality of the arteries, the heart would be called upon to do a great deal of unnec-

[1] Janeway, p. 22.
[2] Russell, p. 2.

essary work, which would absorb a vast amount of valuable energy, and the flow of blood throughout the arterial system would be intermittent, as the heart at each beat would be required to drive the whole volume of blood forward and through the capillaries. A condition obviously incompatible with normal physiology in the body.

Let us now consider how this elasticity affects the circulation and blood-pressure.

Considering a partially filled arterial system let us observe what occurs when a volume of blood is projected into this system by the ventricular systoles. In the beginning the elastic walls of the vessels make room for this change by expanding, while some accommodation is also obtained by the onward passage of blood toward the capillaries. Since it is easier for the arteries to expand than for the whole mass of blood to pass on through the capillaries, the increments of blood are largely stored in the arterial system, thereby tending, by the increasing tension of the arterial walls, to increase blood-pressure. Up to a certain point it is easier for the accommodation to occur by further expansion. When the capacity of the arteries to expand under pressure is approached the stretched muscular coat will become tense and stiff. Now at this point each systole will drive a larger portion of the blood forward through the capillaries, and an increasingly smaller amount will be stored in the vessels by a further yielding of the wall. Normal conditions of pressure will be reached and maintained when the blood accommodated at each systole by arterial expansion exactly equals the amount of blood passing through the capillaries during the cardiac cycle. When this balance of force occurs the blood-pressure will rise no further.

Anything altering this relation, either by increasing the output of the ventricle or by obstructing the flow through the capillaries, or *vice versa*, will cause the blood-pressure to change. The same is true of alterations in the normal elasticity of the arterial system.

Thus during each cardiac cycle, the heart muscle does work in maintaining the capillary flow against capillary resistance, and in causing expansion of the arterial wall. A part of the manifest energy of the heart thus becomes for a time potential in the stretched fibers of the arterial wall. The moment that a systole is at an end, the stretched elastic fibers recoil and continue the work of the heart in maintaining the arterial flow against capillary resistance.

As this potential energy becomes expanded the pressure gradually falls and it would eventually reach zero were it not for the rhythmically recurring cardiac systole which causes the pressure to again rise.

The elasticity of the vessels is very perfect and is capable of standing a pressure greater than by any chance could possibly be developed during life. According to Janeway[1] quoting Grehant and Quinguard, the carotid artery of a dog is capable of withstanding a pressure twenty times greater than the normal pressure, without tearing. For the human carotid the lowest pressure at which rupture occurs is 1.29 meters of mercury, at least eight times the ordinary carotid pressure of the normal circulating blood.

4. Volume.—Compared with the full cubic volume contents of the arteries, capillaries and veins combined, the volume of blood is surprisingly small. In the normal

[1] *Loc. cit.*, p. 24.

individual the volume capacity of the vascular system is so reduced, that the blood is maintained at all times under a considerable pressure. This is due to the continuous contraction of the walls of the blood-vessels which has been considered (page 21) under the head of vasomotor tone or tonus.

While a certain amount of blood, probably about three-fourths that of the total volume of blood, is necessary to support the circulation, still it has been found that a large amount of blood can be withdrawn (see Venesection, page 263) and that the pressure rapidly returns to a point at or near normal. On the other hand Worm Müller[1] has shown that an amount of fluid greater than the total blood volume of the body can be transferred into the vessels, without increasing the blood-pressure above a point frequently reached under normal conditions. Therefore it would seem that except for great changes, the volume of the circulating blood has only a slight and temporary influence on normal blood-pressure.

5. **The Viscosity of the Blood.**—The viscosity of the blood is a factor that up to this time has been omitted almost entirely in considering the variations, normal and pathological in blood-pressure. For purely physicial reasons the factor of viscosity is of upmost importance, since variations even when slight must affect enormously the resistance offered to the passage of blood through the arterial system, and therefore must profoundly affect blood-pressure. It will probably be found as experimentation is carried further that the viscosity of the blood is an important factor affecting blood-pressure, and that the development of methods for

[1] Quoted by Janeway, p. 26.

its modification or control will mark an epoch in the study and treatment of diseases involving blood-pressure changes.

THE BLOOD-PRESSURE WITHIN DIFFERENT ARTERIES

As the arteries rapidly diminish in size from the aorta to the periphery it would naturally be supposed that the arterial pressure would rapidly undergo a similar reduction. Experiment has shown this not to be the case, on the contrary, we know that the blood-pressure within the arteries except the very smallest, diminishes very slowly as the distance from the heart increases, and therefore the blood-pressure is obtained by the modern sphygmomanometer from the brachial or the femoral, approximates very closely that existing within the aorta, near the heart.

Thus when we say blood-pressure (arterial) we mean the pressure found in one of the larger superficial vessels and we have found that this represents very closely the aortic pressure.

Clinical terms employed in blood-pressure studies:

The pulse.
Systolic pressure.
Diastolic pressure.
Mean pressure.
Pulse pressure.

The Pulse.—From our knowledge of the action of the heart, we know that blood is forced into the aorta at regular intervals, and that each change of blood entering the aorta is felt throughout the arterial system in the form of a wave which is styled the pulse and which may be felt as a rhythmically recurring impulse (due to transitory increase in size of the vessel) in all palpable arteries.

The propagation of this wave throughout the arterial system implies a change in diameter of the vessel with a resulting stretching of the vessel wall (see Elasticity, page 18) caused by the increased increment of blood entering it. This further stretching of an already stretched vessel wall can only occur through an increase in pressure within the vessel sufficient to cause the stretching which is left under the finger. It is a self-evident fact, then that there occurs alternately, in regular rhythmic cycle, a rise and fall in blood-pressure throughout the arterial system. Corresponding to the ventricular systole and diastole, the highest and lowest points of this change in pressure are termed respectively, systolic blood-pressure and diastolic blood-pressure.

Systolic Blood-pressure.—This term is applied to the blood-pressure within a given vessel, when the maximum force is exerted within it during ventricular systole. This is the pressure meant when "blood-pressure" is referred to. It is capable of considerable variation, through reflex and other causes, without passing the boundaries considered as normal in clinical medicine. It may also vary widely either above or below the normal limits, under the influence of many pathologic conditions. (See Chapter IV.)

Diastolic Blood-pressure is the degree of pressure exerted within a vessel under observation, during cardiac diastole (immediately preceding systole) and represents the lowest pressure occurring in the vessel during the cardiac cycle. Like the systolic pressure, the diastolic pressure varies within certain limits in health, and widely in pathologic states.

Mean Blood-pressure.—Clinical observation has established the fact that the mean blood-pressure, as determined by the sphygmomanometer, corresponds closely with the arithmetical mean of the systolic and diastolic pressure, *i.e.*, the sum of systolic and diastolic pressure divided by two. (See Fig. 23.)

Pulse Pressure, Pulse Range or Amplitude.—These synonymous terms are employed to designate the total variation in pressure occurring in a given vessel during a cardiac cycle. This variation may be determined then

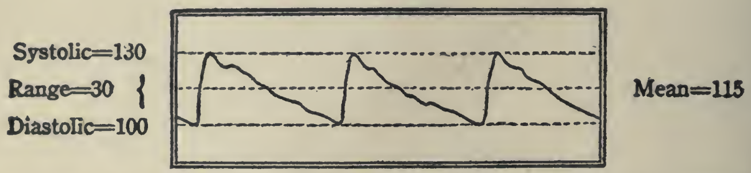

Fig. 23.—Normal pulse tracing: showing relation of systolic, diastolic. pulse pressure and mean. Pulse pressure equals 30.

by subtracting the estimated diastolic pressure from the estimated systolic pressure, and varies in health between 25 and 40 mm. Hg.

The determination of pulse pressure is of greatest importance in the study of diseased conditions, particularly in the estimate of cardiac muscular efficiency and in determining the prognosis of certain valvular and blood-vessel diseases and toxemic states.

CHAPTER VI

CLIMATOLOGIC AND RACIAL INFLUENCE

Altitude.—In approaching the subject of the effect of altitude on blood-pressure and pulse rate, a sharp line must be drawn between the influence of changes in altitude (atmospheric pressure) upon normals and other individuals, particularly the tuberculous, otherwise confusion will surely follow, because the great bulk of clinical data demonstrates that altitude affects normal and pathologic individuals differently.

Healthy Individuals.—Gardner and Hoagland[1] at an altitude of 6,000 ft., measured large numbers of normals who had lived in Colorado for more than a year, and concluded that the average blood-pressure was slightly lower than at the sea level.

They also show that prolonged residence at that altitude does not materially affect blood-pressure.

Experiments showed that an ascent from 6,000 to 14,000 lowered pressure and increased pulse rate. The fall was apparently a permanent one.

Smith at Ft. Stanton[2] (6,200 ft.) states that "It has been scientifically established that blood-pressure is lowered with increased altitude."

Pomeroy in the *Interstate Medical Journal*, in order to determine the degree of variation in the systolic and dias-

[1] *Trans. Am. Climatological Assn.*, 1905.
[2] Reprint No. 51, Public Health Rep., P. H. and M. H. Service.

tolic blood-pressures, caused by altitude, average the studies of eighteen observers, dating from 1878 up to the present time and found that the fall of systolic blood-pressure ranged between 1 and 22 mm., and the diastolic fall between 1 and 11 mm.

Schneider and Hedblom[1] present a very concise and accurate summary of present knowledge bearing on this point.

1. A considerable elevation in altitude tends to lower systolic and diastolic blood-pressure and to increase the heart rate.
2. The fall of systolic pressure is slightly greater and more certain to occur than the fall of diastolic pressure.
3. A rise in diastolic pressure occurs in some individuals.
4. The influence of such factors as psychic states, eating and exercise may obscure the findings.
5. The fall in blood-pressure and increase in heart rate are more marked in the early part of stay in higher altitudes.
6. With prolonged stay in higher altitudes the heart rate probably returns more nearly to normal than the blood-pressure of all individuals.
7. High altitudes do not affect in the same degree all individuals.
8. Small elevations in altitude do not materially influence blood-pressure.
9. Those individuals most affected by high altitude seem to sustain the greater fall in systolic blood-pressure and the greater acceleration in heart rate.
10. The heat of the summer season probably accelerates the pulse rate.

[1] *Am. Jour. Physiol.*, Vol. XXIII, No. 2.

Tuberculosis.—LeRoy S. Peters, pointed out in 1908[1] that altitude usually caused a rise in blood-pressure in the tuberculous. He made his observations at an altitude of 6,000 ft. (For effect of tuberculosis on blood-pressure see Chapter XIV.)

Bullock[2] confirms the observations of Peters. The blood-pressure raising effect of altitude on persons suffering from pulmonary tuberculosis appears to be of distinct advantage to the patient, as it directly combats the blood-pressure reducing acting of tuberculo-toxins by altering metabolism, modifying and stimulating tissue change, and aiding elimination.

B. R. Hooker[3] shows in his reports of respiratory cases that placing patients in the open air increased the blood-pressure from 5 to 10 mm.

Influence of Climate.—Weston P. Chamberlain has recently reported in the *Philippine Journal of Science*[4] an exhaustive study of the effect of climate and race upon the normal average blood-pressure readings. The study is based upon 6,128 blood-pressure observations on 1,042 white men and 552 Filipinos all in good health and ranging in age from twenty to forty years. The average systolic pressure of 5,368 readings on 992 persons, was 115.6 mm. and the pulse rate taken simultaneously averaged eighty-one beats per minute. The average age was 26.6 years. Comparing this average with that of Woley (see page 60) it is found to be 7 mm. lower and compared to Bachman[5]

[1] *Arch. Int. Med.*, Aug., 1908.
[2] *Jour. A. M. A.*, June 19, 1909.
[3] *Med. Rec.*, Jan. 28, 1911.
[4] Dec. 1911, Vol. VI, No. 6, Sec. B.
[5] *New York Med. Jour.*, 1911.

3 mm. lower. While the pulse rate in Chamberlain's series was nine beats per minute above the average accepted as normal in temperate climates for all ages. He found that the blood-pressure has a tendency to be lower than the averages given above, during the first three months stay in tropical climates.

Chamberlain's Table.—Average systolic blood-pressures and pulse rates, based on 5,368 observations of each which were made on 992 American soldiers serving in the Philippines; arranged according to age. (12.5 cm. armlet.)

Age period, years	Average age, years	Number of men showing pressures from— 91 to 100 mm.	101 to 110 mm.	111 to 120 mm.	121 to 130 mm.	131 to 140 mm.	141 to 150 mm.	151 to 160 mm.	Total number of men	Average pressure	Average pulse rate
										mm.	
18 to 20......	19.4	1	12	13	8	1	1	.....	36	115.0	78
20 to 25......	22.8	32	156	165	87	22	5	2	469	114.3	82
25 to 30......	27.2	16	73	108	70	13	3	3	286	115.9	81
30 to 35......	32.6	2	34	42	23	7	1	.....	109	116.7	80
35 to 40......	37.5	.....	9	24	14	8	3	.....	58	120.5	81
Over 40......	43.1	2	3	17	7	2	2	1	34	119.6	79
Totals or averages	26.6	53	287	369	209	53	15	6	992	115.6	81

Racial Influence on Blood-pressure.—Chamberlain also reported[1] a series of observations conducted to determine the effect of race upon average systolic blood-pressure and obtained the following result:

Average blood-pressure of 100 Filipino scouts, 115.0
Average blood-pressure of 100 Philippine soldiers, 115.9

and states that "we may, therefore, conclude that the mean

[1] *Loc. cit.*

blood-pressure for Filipinos during the period of fifteen to forty years (average twenty-five years) is 115 or 116 mm. and that it does not differ from the pressures at the same age for Americans residing in the Philippines."

CHAPTER VII

THE RELATION OF BLOOD-PRESSURE TO ATHLETIC LIFE AND EXERCISE

This is the age of athletics—never has there been such a wide-spread devotion to outdoor sports and athletic contests since the days of ancient Greece. Old and young alike are awakening to the value of recreation and exercise, and have turned to track and field sports, sometime without pausing to consider the advisability of such exertion, or without ascertaining their fitness to participate in the more strenuous forms of exercise. Undoubtedly to so participate might lead into grave danger. Each should learn and know his limitation and be governed thereby.

Parents of growing children are beginning to appreciate the value of a more definite knowledge of their children's physical fitness, both as a guide to avoidance of future physical defects and weaknesses and as an index of the character and amount of exertion that can be safely indulged in.

This problem is frequently brought to the physician for solution, by the following question: "Doctor, my boys are going to boarding school this fall, and I am anxious to know whether their physical condition is such that they may indulge in track work, football, basket-ball, etc."

The answer is difficult, as to make a definite reply is to shoulder a great responsibility. The age, muscular development and general build, heredity, past history and idiosyncrasy of the applicant will of course enter into the decision, but the chief factor is the condition of the cardiovascular system and of the kidneys. This same question is put in a modified form by persons of all ages relative to the danger of bicycling, golf, cricket, swimming, etc., and is even more difficult to answer. At this point I can do no better than quote extensively from Robert E. Coughlin.[1]

The four ages to consider will be (1) early life, including infancy, boyhood, youth and adolescence up to the twenty-first year; (2) manhood, from the twenty-first year up to the fortieth year, (3) middle age from the fortieth to the fifty-fifth year; (4) beyond middle life up to old age.

In a discussion regarding athletics in boys' schools a middle ground seems to be occupied by the Medical Officers-of-Schools Association said *The Hospital* (April 3, 1909) in an editorial, "These medical officers adopt the extreme position of neither side; that is, they advocate neither grandmother coddling nor the Spartan survival-of-the-fittest attitude of many athletic persons. They point out that neither age nor distance is in any way an exact criterion of the strain inflicted on any given boy by any given race. The quarter mile is a far more exhausting race for most boys than are the long-distance races, and to this we would add the half mile, in which school boys have done at various times very notable performances, but, now and then with considerable detriment to themselves.

"The Association's recommendation that the plan of running all the boys, old and young, over the same course or distance is not to be recommended, but we are not sure that in this particular we quite agree. For if a separation is made, it must be on some rough line, such as age, and that

[1] *Medical Record*, April 2, 1910.

means that a compact, well-developed youth capable of any exertion may be sent into a junior division to set the small boys a hot pace over a short run, while an overgrown and much less precocious boy a month older may be put to compete with the most athletic of his fellows over a long distance. When all the boys run together, the best runner may finish a five-mile cross-country run half an hour before the worst, but at the same time this gives those who, by reason of youth or retarded development, cannot excel at this exercise, a chance to complete their run and benefit by it without undue strain. Provided there is a thorough medical examination of every boy on entering competitive sports, and that the effects of various games on the younger boys are carefully supervised, that certain common sense rules, which boys themselves do not appreciate are enforced upon them, then the risk of ordinary school exercise, including cross-country runs and flat races not exceeding one mile, is so reduced as to be quite beneficial."

Dr. Tyrrell Brooks of Oxford says it was his experience that the most vigorous undergraduates came from schools whose athletics were of the most strenuous type. Of the organs likely to be endangered from excess in athletics, the heart is the chief. Valvular damage, due to over-exertion is very rare, but it is to be remembered that slight dilatation of the heart is difficult to estimate. It is almost certain that the natural resilience of the heart is so great in boyhood that mere muscular exercise can hardly seriously damage the heart. Special care in permitting active exercise after convalescence from acute illness is a very important precaution.

In summing up the work of Dr. Benedict and Dr.

Carpenter, done in Wesleyan University, a medical editor has the following to say: "The human body is a machine of such a degree of efficiency that one-fifth of the energy expended by it can be utilized as work, and that this efficiency is constant in men of all types. The strongest and the most thorough is able to do more work than the novice, this is not because his muscles are of such a quality that he can get more work out of them from the same amount of energy, but because he is able to put more energy in the shape of tissue changed into the action. It would seem then that training, besides preparing the heart to stand greater strain, acts to increase the subject's power of using up his tissues and by giving him more muscular tissues to use rather than by teaching him to conserve his energies. To adopt a metaphor from the mechanical world, the professional has a more powerful engine because he is able to use more fuel, not because he wastes less steam.

From the twenty-first year to the fortieth, little may be said of the evil effects of athletics as most young men have by this time passed out of athletic life and gone into their chosen vocations. The eagerness to make a living and the desire to succeed in life compels them to be up and doing. As a rule degenerative changes are not at this time so apparent, and though the man may be working under high pressure, no notice is taken of such changes until middle life is reached. This is the time, however when the habits of life are formed.

HOW TO DETERMINE PHYSICAL FITNESS

The most practical means which we have at our disposal with which to corroborate and qualify the results of a careful physical examination, are by urinalysis and by a study of blood-pressure—including also the diastolic, the mean, and the pulse pressures. Applying also, except in adolescents and youths, the so-called functional tests. A pathologic urinary finding will of course be given its proper value, and will be correlated to the other evidence. Studies of the blood-pressure, its normal behavior under strain, its changes in pathologic heart valve and heart muscle conditions, have been carried out by many investigators, usually in connection with the physical departments related to colleges and other institutions. These have developed some very interesting and valuable data, which may now be employed in examinations for physical fitness.

Apart from the discovery of valvular defects and functional murmurs, there is little that can be deduced from the usual blood-pressure test in early life, up to about the time the boy or girl enters college, from about the age of sixteen years, on to middle or early old age. The ability of an individual to withstand strain, without danger, depends largely upon the integrity of the cardiovascular system, so that special examinations are usually directed toward this system.

The discovery of a pathologically high blood-pressure (see Chapter IV for this determination) would move the individual's age limit forward, so that if when he is forty there is a marked degree of hypertension, he would have to go into the fifty- or sixty-year class. The discovery of arteriosclerosis even in the absence of a hypertension should suggest the same caution. We know from experience and have had confirmed by a systematic investigation the effect of various forms of exercise on the systolic, diastolic, pulse pressure, and on the pulse rate. As demonstrated by O. S. Lowsley[1] who found that in healthy young athletes the blood-pressure and the pulse pressure are greatly increased during exercise and remain above normal even at the conclusion of very exhaustive work. After exhausting exercise there is a period of subnormal blood-pressure and the more exhausting the exercise, the more marked and prolonged this phase will be. (See Tuberculosis, Chapter XIV, Page 182.) The rapidity with which the pulse rate drops to normal is also determined by the degree of exhaustion. There is often observed, however, a secondary rise in rate during the period of subnormal pressure. Very violent exercise of even a few seconds, as in the running of a hundred-yard dash causes a much more pronounced and lasting negative phase than does more prolonged but moderate work.

We have seen that in the work test, as suggested by Graupner, this fact is used as a basis of the experiment, and that this has been corroborated since by others—including Boardman Reed[2]—that the condition just described is only possible in a heart which has not had its normal reserve power either involved or destroyed through chronic arterial or chronic myocardial change. While in the weak heart, whatever the cause, the rise in pressure does not always occur, and even if a rise is noted, the blood-pressure falls before the pulse rate—and remains down. This test of Graupner's can therefore be applied to those who are about to enter strenuous athletic contests, where, if the

[1] *Am. Jour. Physiol.*, March 1, 1911.
[2] *South. Calif. Pract.*, August, 1910.

normal relation of pressure and pulse exists, the individual may safely be allowed to participate.

Lowsley suggests also that the duration of the negative phase is a fair index of the strain on the circulatory system, and that the test might be used in determining the fitness of any individual for the performance of certain forms of exercise. He believes that if the negative phase passes within an hour, that the individual is well within the "hygienic limit" but that if it lasts more than two hours, it is a sign that the margin of safety has been exceeded—such measures as these should be of considerable help to physical directors in deciding the qualifications even of those who are apparently sound.

Another method, suggested by Masing,[1] is to note the relative effect of exercise on the systolic and diastolic pressures; a normal circulatory apparatus will yield a systolic pressure greater in proportion than the diastolic, in other words, the pulse pressure or amplitude will be increased, while in a defective cardiovascular system the systolic and diastolic pressures, even when raised, will tend to approximate. Janeway cites a case where the pressure in a healthy man, age twenty-six (a) at rest was 135 systolic, diastolic 100, pulse pressure 35; (b) after running up three flights of stairs, systolic 175, diastolic 120, pulse pressure 55, indicating a normal cardiac strength, capable of maintaining its tone under strain. On the other hand a man apparently in good health gave a systolic (c) at rest of 140, diastolic 100, pulse pressure 40. After two minutes exercise, (d) systolic 155, diastolic 125,

[1] E. Masing, *Deut. Arch. f. klin. Med.*, 1902, Vol. LXXIV, and later commented upon, Janeway, p. 122.

pulse pressure 30, showing a defective musculature and imperfect heart tone; in this case violent exercise would probably give rise to permanent dilatation.

According to Gibson this may be expressed graphically as follows:

(a) $\frac{PP}{SP} - \frac{35}{135} - 1/4$

(b) $\frac{PP}{SP} - \frac{55}{175} - 1/3$

Fraction larger after exercise—good heart.

(c) $\frac{PP}{SP} - \frac{40}{140} - 1/3$

(d) $\frac{PP}{SP} - \frac{30}{155} - 1/5$

Fraction smaller after exercise—poor heart.

Summary.—After prolonged and severe exertion, the blood-pressure falls to a point below the normal as determined before the exertion.

After a brief period of mild or moderate exercise, the blood-pressure immediately afterward shows, in the majority, a rise followed quickly by a fall near the normal—perhaps a little above or a little below.

The fall denotes fatigue, the failure to rise probably indicates muscular heart weakness or, at least, temporary dilatation, as is shown to be the case in marathon runners and foot-ball players.

F. Gelsbock believes that low pressure immediately after mild exercise is due to muscular heart weakness, he also believes that arteriosclerosis may result from long-continued high blood-pressure occurring in athletes.

Danger to athletic individuals in real life is to the vascular system, this is because such individuals do not continue their systematic exercise, so that metabolism and elimination become defective (Gelsbock).

CHAPTER VIII

HYPOTENSION

Definition.—This term is employed to designate alterations in arterial blood-pressure in which the pressure curve remains below the established normal minimum. The actual level of this pressure will be affected to a degree by the age and other physiologic factors, which control the normal level of pressure. (See page 59.)

We must also admit the possibility of a relative hypotension, in which the curve of pressure, while being above the established normal, is yet so far below a previous long-continued high pressure, as to prevent the physical phenomena of a pathologic low pressure. This point is discussed more fully below.

In order to fully comprehend the discussion which follows, some form of clinical classification of low blood-pressure must be formulated. The following seem to be the accepted subdivisions of this class of conditions.

These several forms of hypotension are terminal hypotension, essential hypotension, primary or true hypotension and relative hypotension.

Terminal Hypotension.—The term is used to indicate that abnormal lowering of tension in the circulation which indicates the approaching end of one life.

With the approach of death from any pathologic condition, the blood-pressure tends more or less rapidly toward zero. The rate of which this arrives and its relation to the actual cause of death, is determined by so many factors about which almost nothing is known, that but little really definite can be yet determined upon. According to Janeway, pressure as low as 60 mm. (5 cm. cuff) may persist, in protracted illness for several days before death. In such a case the hypotension may be of some value as a sign of impending dissolution, but as a rule the terminal fall in pressure is usually a matter of hours or minutes.

Essential Hypotension.—Occasionally there are cases which seem to have a constitutionally low blood-pressure. These cases show no definite signs of disease, and no discoverable cause can be assigned for the condition. Though sometimes it may develop later that this state was in reality an early sign of tuberculous infection. This point will be developed later. Such individuals are frequently unequal to any particular effort either mental or physical.

Primary or True Hypotension.—This is closely allied to the preceding, but is distinguished from it by the appearing of some assignable cause—other than the pre-existence of some condition causing hypertension. Bishop defines primary or true hypotension as occuring in those cases whose pressure-reducing mechanism has failed, when there has been no previous overdemand for pressure.

Relative Hypotension.—This term would seem to be a necessary one and should be applied to those cases whose actual pressure, while still above the estimated normal, has fallen from a former pathologic high level to such a degree that symptoms due to the fall have developed. A fairly common example of this is the frequent occurrence of edema or other signs of circulatory failure following injudicious attempts to reduce a high pressure.

The same condition obtains in a failing cardiovascular system, when the pressure has been for a long time high. See chart, Fig. 24. Here also we may have most serious and distressing symptoms, pointing to circulatory failure, and yet the pressure may be found still above the estimated normal level.

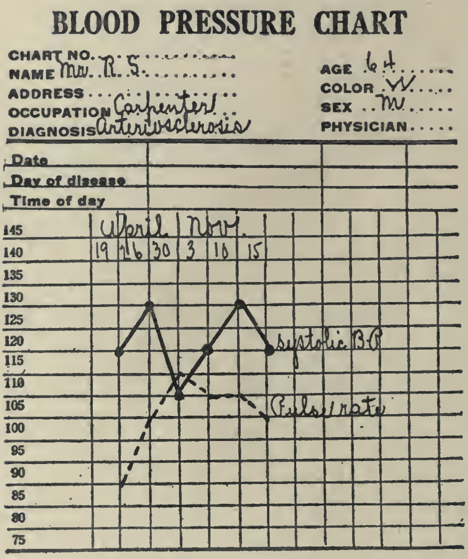

Fig. 24.—Hypotension of lost compensation (relative hypotension). Arteries markedly sclerosed, heart showed myocardial degeneration, pulse always rapid. Cerebral symptoms marked, treatment had very little effect.

Causes of Hypotension.—In considering the etiology of the condition of hypotension, we find that it occurs in many diseases and conditions, as for example in wasting diseases, in toxemias, acute and chronic infections, in certain conditions of the heart, and in circulatory depression from any cause as in shock, in collapse, in cardiac asthma, during and after hemorrhage and in a number of metabolic diseases of which diabetes is an example.

The Lower Normal Limits.—The limits are, of course, largely arbitrary, depending as they do upon so many variable and varying factors. To maintain their full value, they must be modified to conform to our knowledge of the many so-called physiologic factors active in each individual case. (See Chapter IV.)

Experience teaches that 105 mm. may be taken as the low limit of normal blood-pressure in young men, and 95 mm. as the normal low limit in young women. This will of necessity be modified slightly by the age, occupation and muscular development of each individual. The only way to estimate the degree of abnormality in the blood-pressure is to apply the knowledge obtained from experience in examining a large number of cases. Therefore it is usually advisable to employ the blood-pressure test as a routine in all cases, in order to develop one's ability to interpret the significance in each individual case.

Extreme Low Pressure.—The lowest blood-pressure in an adult, compatible with life, has been reported by Neu to be from 40 to 45 mm., and this only occurred with subnormal temperature accompanied by unconsciousness. He has observed and recorded recovery after a temporary fall in pressure as low as 50 mm.

Conditions Accompanied by Hypotension.—In the present state of our knowledge of this subject, it is impossible to lay down arbitrary laws or to make a positive statement regarding the absolute level in persons, occurring under the different conditions, which are dependent upon so many varying factors. In the statements which follow it should be remembered that the figures apply only to the majority of cases of the types discussed.

Diseases of the Heart.—In the majority of valvular lesions of the heart where compensation is good, the effect on blood-pressure is very slight, so that this test is here chiefly of prognostic value. The two exceptions to this are aortic regurgitation and mitral stenosis.

In *aortic regurgitation* we find a persistent and uniform high systolic pressure combined with an exceptionally low diastolic reading, which results in a characteristic pulse pressure, and upon this alone diagnosis may be made. This large pulse pressure found in aortic disease is further argumented when accompanied by arterial sclerosis, chronic myocarditis, or chronic kidney diseases (see Fig. 25).

Mitral Stenosis.—Extreme narrowing of the valve orifice occasionally may so reduce the volume of blood passing through the heart that the blood-pressure is lowered simply because the heart is able to pump only a fraction of its normal amount.

In other organic conditions of the heart, and in the last stages of valvular cases the tendency of the pressure is downward, when it is due to failing circulation and venous stasis. From a clinical standpoint, Bishop[1] makes an important point when he states that failure of the circulation in heart disease does not become a matter of anxiety during acute attacks of valvulitis, during which time the patient is at rest, but that it becomes more serious when the patient resumes his occupation, and that even then the low arterial tension should not be regarded as pathologic except when it is but little above venous pressure, as shown by venous

[1] Heart Disease and Blood-pressure, 1907.

congestion and enlarged liver, etc. This emphasizes the point made that the actual pressure level found does not always measure the degree of pathogenicity of the case. Each case has its own particular law and must be studied and treated according to the conditions present.

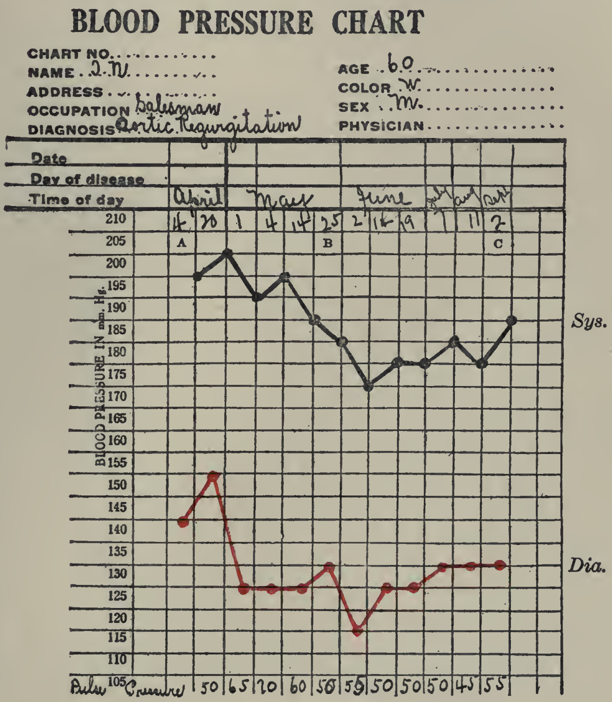

FIG. 25.—Aortic regurgitation with moderate arteriosclerosis, symptoms the result of high pressure. A to B show effect of treatment. B, all drug treatment stopped. B to C, patient resumed occupation, general measures continued. Note large pulse pressure characteristic of this disease.

Alterations in Heart Rate.—These do not, as a rule, influence blood-pressure. We believe that blood-pressure has a marked influence on pulse rate. The action of the heart and pressure being in the relation of a force pump to a water supply; as the demand for increased pressure or sustained pressure arises, the pump responds with greater force and energy, for this mechanism is so delicately balanced, that under ordinary conditions, very little fluctuation in pressure occurs.

Two conditions however have been found to have an influence on blood-pressure, the cause and significance of which are as yet obscure.

1. **Paroxysmal Tachycardia.**—The pulse rate may be from 150 to 300, the heart sounds good and the pulse small, sometimes the pulse rate cannot be counted, the blood-pressure is usually found to be low, probably because the shortness of diastole does not allow the proper filling of the ventricles; the venus pressure is high. In the intervals the circulation is apparently normal (Krehl).

2. **Bradycardia.**—The effect on blood-pressure is variable, depending on the cause and on other conditions if present. When extreme, blood-pressure is always lowered; patients with dyspnea cannot exert themselves, and even change in posture may precipitate attacks of syncope (Krehl).

Finally, in close relation to the circulation in diseases of the heart, as has been found by Krehl and others that in the last stages of arteriosclerosis, wide-spread dilatation of the splanchnic area together with failure of the heart to respond to the demands made upon it, there results a gradually falling blood-pressure, when therapeutic measures have little or no effect. (See Terminal Hypertension.)

Shock and Collapse.—Closely allied to this condition, at least from a mechanical and physiologic standpoint, is shock and collapse.

In both these conditions we find a sudden and dangerous decrease in blood-pressure. This is due to one or two conditions. First the overwhelming of the vasomotor system, by circulating toxins, which cause vasomotor paralysis. This is collapse. On the other hand, shock would appear to result from a failure of vasomotor tone, the result of reflex stimulation through the sympathetic system. Experimental evidence shows that the circulatory disturbance occurring at the height of infections, depends absolutely upon paralysis of the vessels, and not upon any damage to the cardiac mechanism (Crile).

Cardiac Asthma.—This term implies a severe attack of dyspnea occurring in an individual having heart disease. During the attack the pulse is rapid, soft and irregular in force and rhythm. The blood-pressure is usually below normal during the height of the attack, speedily regaining its former level as the attack subsides.

Hemorrhage.—The degree of low pressure following hemorrhage usually bears a direct relation to the amount of loss and rapidity with which the bleeding occurs. The one exception to this is in cerebral hemorrhage when the blood-pressure usually reaches very high levels. Cases have been reported in which the pressure has reached 400 mm. In the cases of acute hemorrhage, such as occurs from wounds, during typhoid fever, in tuberculosis and epistaxis, the amount of lowering may be so great as to endanger life. If the loss supervenes upon an already weakened state or during collapse when the vasomotor system is crippled or paralyzed, the hypotension may be the direct cause of death. It is noteworthy that this fall, even when great, is usually transient, and the value of this sign therefore decreases in proportion, as the time between the hemorrhage and the observation is prolonged.

Altitude.—The disagreement existing between reports of the effect of altitude on blood-pressure is probably due to the fact that some observers have reported observations on healthy individuals while others observed only pathologic conditions. (See Chapter XIV, Tuberculosis, page 83.) In a healthy individual nearly all competent observers agree that high elevations cause a moderate reduction in blood-pressure. It is pertinent to quote here part of the summary of the very able article of Schneider and Hedblom.[1] Among their conclusions the following bear on blood-pressure:

1. Considerable elevation in altitude tends to lower blood-pressure and to increase the pulse rate.

2. The fall is greater during the early periods of residence in high altitudes. The fall in high altitudes is between 1 and 22 mm.

3. Change in altitude does not affect each individual to the same degree, a slight elevation does not affect blood-pressure and psychic influences may modify the reading.

The danger of high altitudes to those having a low pressure, especially if accompanied by a weakened physical condition, is probably due to a further reduction of an already existing hypotension.

Paresis.—Hypotension is the rule, unless kidney complications exist.

Infections.—*Tuberculosis.*—In uncomplicated pulmonary tuberculosis the systolic pressure tends to fall, and the diastolic pressure to remain stationary or to rise. Tesser

[1] *Am. Jour. Physiol.*, Vol. XXIII, No. 3.

and others report the occurrence of hypotension in uninvolved members of families with tuberculosis taint.

From a diagnostic standpoint, the symptoms of hypotension when otherwise unexplained should suggest a careful examination for tuberculosis, particularly in the lungs. In an established case, the chief value of this test is in prognosis, where the data compiled by Haven Emerson may quite safely be relied upon.

Haven Emerson[1] stated that hypotension in tuberculosis is marked and constant in advanced cases, almost always present in the moderately advanced cases, and frequently enough found in the very early or doubtful cases to warrant its use as a valuable differential sign, and further, hypotension is progressive as the process advances and rises with progress toward recovery, the pressure returning to normal in cases that are cured. Continued hypotension never persists in the presence of evident improvement in the tubercular process.

Reitter has suggested that the occurrence of hypotension associated with evidence of nephritis is suggestive of renal tuberculosis. On the other hand, Sezary[2] does not find any relation between the condition of the suprarenal glands and the low blood-pressure found in tuberculosis. He believes that low tension is at first due to the direct effect of the action of the soluble toxins of the tubercle bacillus, and that not until later do the suprarenals become involved. He cites cases examined at autopsies which showed almost complete destruction of the suprarenals, and yet during life showed a relatively high blood-pressure.

[1] *Arch. Int. Med.*, 1910.
[2] *Abstract, Jour. A. M. A.*, Vol. LIV, No. 15.

Typhoid Fever.—The symptoms of hypotension probably more frequently accompany the average case of typhoid fever than any other acute infection. The systolic pressure is usually 100 or less, decreasing as the disease progresses and toxic phenomena occur. The diastolic pressure tends to remain at the original level or to rise slightly; this causes a reduction in pulse pressure, which is usually significant evidence of a weakened heart muscle, calling attention to the need for complete rest and more active stimulation. The effect of the common complication of typhoid fever, namely hemorrhage, is to produce a further rapid fall in pressure, the amount of this indicates roughly the extent of the hemorrhage. The development of peritonitis after perforation forces the pressure up to or above the original normal level. In this disease particularly, it is important to keep blood-pressure records. Systematic observations will be of great value in differentiating hemorrhages from perforation, and will serve as a guide to the general management of any case, pressure will rise and will remain high in the presence of nephritis but will fall again when peritonitis follows perforation.

In the study of a large series of cases, Joseph H. Barach[1] mentions among others the following important factors which briefly summarizes our knowledge of blood-pressure during typhoid fever.

1. The blood-pressure falls below the normal after the patient has taken to bed and stays down until convalescence is established, when it returns toward normal.

2. Typhoid fever is a disease with a blood-pressure below 100.

[1] *Penna. Med. Jour.*, July, 1907.

3. The blood-pressure is governed by factors of its own and bears no constant relation to pulse rate or temperature.

4. In diagnosis the blood-pressure may be of value in differentiating this disease from others, after we know the behavior of other diseases in this respect. In the diagnosis of the complications it has a value.

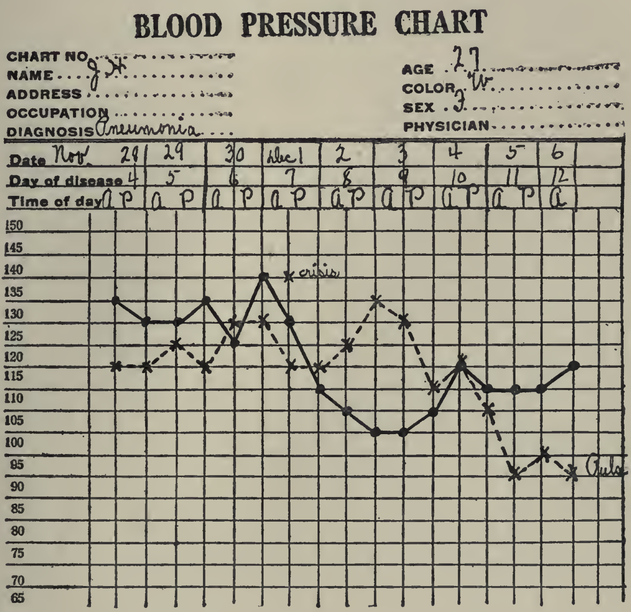

FIG. 26.—Showing close relation between pulse rate and blood-pressure and application of Gibson's rule. Symptoms of collapse developed after crisis and continued until normal relation was reestablished during convalesence.

5. In prognosis the blood-pressure chart is of value. A steadily falling pressure means great danger. As long as the blood-pressure keeps up to a reasonable level, we feel that there is reserve power to work with.

Pneumonia.—The pressure in pneumonia depends on the severity of the case and the degree of toxemia, and also on the various modifying influences which may affect the case. It may be stated as a general rule that during the first day or two of the disease, the blood-pressure is slightly above normal, following which it falls more or less rapidly to a condition of hypotension of about 100 or 90 mm. (See Fig. 26.)

The blood-pressure test seems to be of very significant value when employed in conjunction with the pulse rate.

As expressed by Gibson[1] it offers a valuable aid in prognosis and a reliable guide to treatment. He says "When arterial pressure expressed in millimeters of mercury does not fall below the pulse rate expressed in beats per minute, the fact may be taken as of excellent augury, while the converse is equally true." These observations have been confirmed by G. A. Gordon[2] and H. A. Hare[3]. No case of pneumonia should be treated without the blood-pressure test being regularly employed. Just as observations of the pulse or the temperature are regularly taken.

Cholera.—Hypotension is the rule. Low blood-pressure during the stage of collapse is a very valuable guide to the necessity of transfusion. The blood-pressure is always below 100. The most satisfactory treatment, or the one most likely to combat complications, such as uremia, in administering the intravenous solution of adrenalin. By this means in one epidemic the death rate was reduced almost one-half.[4]

[1] *Edinburgh Med. Jour.*, Jan., 1908.
[2] *Edinburgh Med. Jour.*, 1910.
[3] *Therapeutic Gazette*, June, 1910.
[4] Leonard Rogers, *Therapeutic Gazette*, Nov. 15, 1909.

Cerebrospinal Meningitis.—Robinson[1] noted that blood-pressure was unusually high during the acute state and in those showing severe symptoms, and was low in mild cases and in convalescence.

Abram Sophian[2] depends absolutely on the sphygmomanometer as a guide to the value and safety of lumbar puncture and serum injections.

Other infections in which the pressure is usually low are diphtheria, scarlet fever, measles and acute rheumatism. Here the sphygmomanometer may be of value in conjunction with other symptoms in diagnosis, prognosis and treatment.

Miscellaneous Conditions.—A condition of hypotension usually exists in all *wasting diseases* and *cachectic* states and commonly is seen in carcinoma and general paresis. In these conditions it is a natural result of a general toxemia and the gradual failure of function in the entire body, which includes a weakening muscular system, a gradually lowering vasomotor tone and a diminution in the quantity and quality of the blood.

In *diabetes* the pressure is usually subnormal, unless complicated by nephritis and arteriosclerosis.

In *Addison's disease* the blood-pressure is extraordinarily low because of the destruction of the suprarenal glands.

In *epileptic coma* the blood-pressure is always low; here it constitutes a valuable differential sign between this condition and uremia (Edgecombe).

Edgecombe[3] reports his studies of a number of miscel-

[1] *Arch. of Internal Med.*, May 5, 1910.
[2] *Jour. A. M. A.*, March 23, 1910.
[3] W. Edgecombe, *Edit. Medical Record*, April 29, 1911.

laneous conditions in which the blood-pressure may be of value.

(1) Subjects with poor circulation, with cold hands and feet and liable to chilblains. These may gain a temporary rise by means of baths, massage and exercise, while at the same time the circulation improves, but it is difficult to effect any enduring rise in the general level of the blood-pressure for they are prone on cessation of treatment to relapse to their former state.

(2) Cases of pure neurasthenia, having as their prominent characteristic, profound fatigue, either somatic or psychic or both. It is not easy to say whether the low blood-pressure in such cases of neurasthenia is the cause or effect of the extreme fatigue. A rise in blood-pressure is an almost invariable accompaniment of improvement.

(3) Tobacco poisoning. Tobacco usually has the effect of raising the blood-pressure with this apparent anomaly, that heavy smokers frequently have subnormal pressure.

(4) In cases of dilated heart, with or without valvular disease, the pressure will sometimes be found low, and a rise in pressure is one of the indications of the progress of the case toward recovery.

(5) There are many examples met with of the so-called gouty or rheumatic manifestations of lumbago, sciatica, or neuritis which show a blood-pressure somewhat below normal. Many of these cases have a subnormal acidity of the urine, and are liable almost constantly to a copious deposit of phosphates which leads to, or is accompanied by, a state of nervous depression.

(6) The clinical symptom phosphaturia, in whatever

condition it may occur, is generally accompanied by a low blood-pressure.

(7) Young subjects with "rheumatoid arthritis" frequently have a blood-pressure below normal, which rises as the condition improves.

The chief practical value of the sphygmomanometer in the light of our knowledge of hypotension is the valuable aid derived, both for differential diagnosis, prognosis, and as a guide for treatment. It alone can give timely warning of the onset of the hypotension accompanying vasomotor paralysis from shock or any other cause. In acute diseases the blood-pressure test should be taken daily.

Effects and Danger of Hypotension.—The direct effect of a falling blood-pressure is the accumulation of an abnormal amount of blood in the veins, and a slowing of the current in the arteries. This will affect the capillary circulation and interfere with the nutritive and secretory processes which depend upon it. The most serious effect is on the heart, as it has been shown that complete loss of vasomotor tone soon leads to death, because of the gradual accumulation of nearly all the blood in the body on the venous side, so that the heart has no blood upon which to act.

"Low blood-pressure due to general prostration is not to be regarded as a disorder of the circulation, except insofar as the circulation fails to respond to the demand made upon it. Thus in shock it is the nervous system that is at fault, not the circulatory apparatus" (Bishop).

CHAPTER IX

HYPERTENSION, PRESCLEROSIS OR ESSENTIAL ARTERIAL HYPERTENSION

The term hypertension, in its general acceptance, has come to mean any condition in which the blood-pressure is maintained at a level above normal. It would seem best, at least for clinical purposes to limit the term hypertension to that condition first described by Huchard and termed by him presclerosis, and to designate all other high pressures, which are either dependent upon, or accompanied by distinct and easily recognized arterial kidney or heart changes, as true high blood-pressure. Thus we limit the term hypertension to a condition of blood-pressure dependent largely if not solely, upon a muscular change in the arterial walls and capillaries, whereby they are temporarily narrowed and constricted, as contrasted to true high pressure, when there is a permanent pathologic change either in some part or all of the cardiovascular renal system, and which can never be entirely overcome by treatment. (See Fig. 27.)

By adhering to this distinction we are immediately enabled to appreciate the etiology of each of these conditions, and also to explain the difference in the results obtained by methods directed toward their relief, or the relief of the symptoms caused by them.

In the class designated as high blood-pressure, the diagnosis is made for us and our chief concern is to determine the cause, to arrest the progress of the disease and to relieve symptoms.

In hypertension, the diagnosis is often difficult, but when once made we are usually in a position to relieve the condition more or less completely and to cure the disease causing it during the time that the patient adheres to a restraining hygiene.

BLOOD PRESSURE CHART

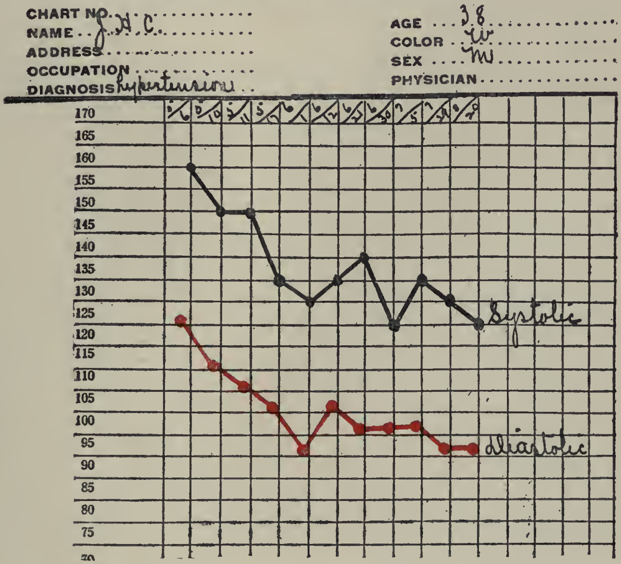

FIG. 27.—Illustrates pure hypertension and the effect that may be expected to follow measures directed toward relief of underlying toxemia.

It would seem reasonable to consider the condition of hypertension or presclerosis, as a distinct disease entity, not forgetting its varying etiology. While on the other hand, we should never lose sight of the fact that true high pressure is a symptom only, merely a small part of the whole pathologic picture, and that it should never possess

8

the entire field of our vision. True high blood-pressure may be a salutory and beneficent condition not to be interfered with lightly or illadvisedly. Hypertension is always harmful and every effort should be made to control and reduce it.

Hypertension is a subtil condition often lurking where least expected. There may be no change in the palpable arteries except a barely distinguishable narrowing and stiffening when rolled under the finger, a hardly noticeable change in the aortic second sound, possibly a faint systolic whiff at the aortic cartilage, while the urine remains practically normal. The blood-pressure will be found to be from 140 to 180 mm. depending upon the degree of arterial contraction.

By many, a progressive change in the arteries and a gradually rising blood-pressure is looked upon as a normal condition in those past middle life or in early old age.

Hypertension is, I believe, always a sign of the beginning of a pathologic change which according to Huchard, Russell and others is the danger signal, a warning that some alteration must occur in the daily life of the individual presenting the condition, or else the at first curable condition will progress and eventually merge into and become a case of cardiovascular renal disease.

A permanent increase in blood-pressure in a young adult, or in one in early middle life in the absence of discoverable organic change, in the heart, blood-vessels or kidneys, is always a sign of a chronic toxemia; a poisoning arising from some error in metabolism or deficiency of elimination, either intestinal or urinary or both. Such a rise in blood-pressure is rarely discovered, except by the routine estimation of blood-pressure of all patients coming under the physician's observation, or perhaps through examinations for life insurance. In the early stages of this condition, symptoms referable to the condition are rare, or if mentioned are attributed to overwork, or mental worry, neurasthenia, etc., on the other hand, one may by careful questioning elicit suggestive symptoms, such as fleeting dizziness, tinnitus aurium, disturbed sleep, cold hands and cold feet, gastric distress and flatulency, constipation, lack of interest and of power of concentration, diminished desire to be up and doing, distaste for physical exertion and weakened tolerance for substances which affect the brain, such as alcohol and tobacco.

The blood-pressure does not need to be greatly increased, in order to injure the heart, and to cause permanent change in the blood-vessels and in the kidneys. The amount of work required of the heart to overcome the resistance of a few mm. Hg. mounts up surprisingly. (See page 21.)

Thus increased work even in the comparatively young, may result in degeneration. Cases have been reported in which arteriosclerosis has been found at autopsy in persons under thirty years of age.

Treatment.—In bad cases, rest in bed with massage. In mild cases this preliminary is not required. The important rules for diet are:

1. To reduce the total amount of food. Many cases are in men and women who are overfed and underexercised; here properly supervised exercise is most valuable.
2. To reduce the amount of protein in the dietary.
3. To limit the amount of fluid taken, and to employ

measures that will promote elimination from the skin, as sweating by means of hot baths, hot packs, Russian baths, vapor-cabinet baths, and electric light baths. Nauheim baths may be given carefully, if there is little arteriosclerosis and no signs of nephritis. Autocondensation may reduce hypertension and should be tried. Calomel and saline purges, not too frequently repeated, are beneficial and may safely be prescribed every seven to ten days. The effect is to prevent and remove intestinal putrefaction and to lower blood-pressure by taking fluid from the body —washing the blood.

Regulation of habits, including relief from business worries and excessive anxiety, attending constantly to work beyond one's capacity—this is particularly the case with physicians who form a large part of this class. Increasing the period of recreation and hours of sleep, in other words, demanding a life of moderation in all things.

The condition of hypertension persists in most cases after the development of the true high pressure which accompanies arteriosclerosis, and it is in all probability this factor which in a large measure determines the extent of reduction, accomplished in the treatment of arteriosclerosis. This has been ably demonstrated by Russell[1] and others. Measures which accomplish a reduction in pressure amounting to 10 to 40 mm. in high-pressure cases, is usually followed by relief from subjective symptoms and are beneficial, reductions in pressure which result in edema or other untoward symptoms are probably due to a toxic effect, and are evidence of depression or myocardial weakening and are dangerous.

[1] Loc. cit.

Syphilis as a cause of hypertension is well recognized; it acts in the same manner as other circulating toxins and therefore may be looked for as a factor in the production of hypertension. (See also Chapter XIV.)

CHAPTER X

ARTERIOSCLEROSIS

A careful review of literature upon arteriosclerosis, its causes and treatment is most discouraging, as no two authorities appear to agree even upon the most fundamental and important points. This is quite remarkable when we consider the relatively frequent occurrence of this disease, and the almost unlimited opportunity offered for its study. Even theories as to the main causative factor differ. We are unable therefore to treat the subject as scientifically as might be desired; however, much general information is available which will serve as a guide to the study and treatment of this disease a knowledge of which may possibly be a stimulus to others to carry the work further.

Causes.—The broad underlying cause of arteriosclerosis is some irritant poison or a toxemia which may vary both as to origin and nature, depending largely upon the surroundings and personal habits of the individual that forms the basis of the study. The more common causes of general and prolonged toxemia are chronic infections (chiefly syphilitic); the introduction of toxic agents, as alcohol, tobacco, coffee, lead; the development of autotoxemias from disturbance in metabolism either through improper or excessive dietary or overmental strain (emotional and nervous), insufficient physical exertion, resulting in maldigestion, gout and that large group of ill-defined metabolic disturbances termed autointoxications.

Excessive physical exertion, while recognized as a cause of generalized arteriosclerosis does not always lead to this condition. Usually additional factors must be considered, such as the added strain caused by simultaneous over-indulgence in food, alcohol, tobacco, late hours, etc.

According to the observations of Coughlin[1] the effect of competition in active athletics does little or no harm to the cardiovascular system when indulged in by those in training, and who are under competent observation. From his study he concludes "that there is a close relationship between the athletic life and degenerative changes in the vascular system, especially in heart and arteries, when the voluntary and involuntary muscles are not kept in tone by regular systematic exercise, particularly in middle life and beyond. (See also Chapter VII.)

Occurrence.—Arteriosclerosis is usually encountered in the second half of life although not infrequently well-marked cases are met with before the age of thirty and an occasional apoplexy occurs in the twenties. The tendency to arterial change seems to be on the increase, as both the age of incidence (development of symptoms) and the percentage of cases encountered are advancing.

Incidence of apoplexy, organic heart disease and chronic nephritis from the statistics of the Penn Mutual Life Insurance Company in 1908. These three diseases comprise 25.1 per cent. of the total mortality of the Company.

	1900	1908
Apoplexy	7 per cent.	9.5 per cent.
Heart disease	8.6 per cent.	9.3 per cent.

This seems largely the result of the high tension and

[1] R. E. Coughlin, *N. Y. Med. Rec.*, April 2, 1910.

artificial life led by the average business and professional man of to-day. For the same reason men are more susceptible than women. All statistics bear out the fact. Huchard[1] investigated the cause of arteriosclerosis in 2,680 cases out of 15,000 patients under his personal observation. From this study he finds the causes according to their relative frequency as follows: gout, uricemia, character of food, syphilis, tobacco poisoning, worry, mental overexertion and alcohol. He dwells particularly upon the fact that the abuse of meat in diet is a powerful and frequent cause of arteriosclerosis, as it easily produces within the body poisons which have a selective action for the tissues composing the arterial wall. Herz[2] finds that almost invariably his cases of arteriosclerosis are in those who take life too seriously and either from ambition or necessitylive an especially strenuous life. Herz sent out a series of questions to a large number of physicians in Austria in an effort to determine the leading factors productive of arterial change[3] and from 822 replies he deduced the following statistics.

Number of cases resulting from:

Emotional and nervous	150
Physical exertion	146
Age	138
Alcohol	133
Tobacco	88
Syphilis	77
Heredity	72
Metabolic disturbances	19
Coffee and tea	13
Infections, etc.	7

[1] *Medizin klin. Berlin*, August 29, V., No. 35.
[2] *Medizin klin. Berlin*, January 16, VI, No. 3.
[3] *Wien. klin. Wochen.*, Vol. II., XXIV, No. 44.

From a general survey of the etiology of arteriosclerosis, we fail to find *the* cause, but it will be seen that we may, however, divide the causes of arteriosclerosis into three more or less correlated groups—toxic, physical and infectious. The latter two often involving some phase of the former, so that it may be said, broadly speaking, that arteriosclerosis is usually the result of some form of toxemia.

Pathology.—The term arteriosclerosis is too loosely employed by the average physician. This has led to great confusion in the reporting of cases and to the compilation of statistics. It is often impossible to learn precisely what condition an author is discussing, so that the benefit of careful research are often lost to the reader. The two conditions usually confused are, atheroma and diffuse generalized arteriosclerosis, and less often the condition of pure hypertension, as found before any permanent change has occurred in the vessel wall. (See Page 112.)

The pathologist has more than once pointed out clearly these different conditions and has correlated them with the physical signs. Among them Russell has made most careful studies of the condition of the vessels, and their relation to chronic disease of the heart, kidneys, cerebral system, to blood-pressure. According to Russell,[1] atheroma is a local or patchy affection of the arteries characterized by a local thickening and degeneration of the intima. This soon undergoes a form of fatty degeneration which is termed, atheroma. Later these patches become the seat of a calcareous deposit and in the larger arteries atheromatous cysts and ulcers may be formed with local

[1] Wm. Russell, Arterial Hypertension, Sclerosis and Blood-pressure, J. B. Lippincott Co., 1910.

sacculations. These changes may be so extensive, especially in the aortic arch that a local bulging occurs to which the name aneurysmal bulging has been applied.

Atheromatous changes are quite common in the cerebral and coronary arteries but comparatively rare in the radials. When present in the radials, they give rise to local thickenings, which give an irregular nodular feel to the vessel. They are never symmetrical. Russell believes that the character of these changes is very suggestive of a low-grade infection, and assigns a primary microorganismal implantation as their origin.

Arteriosclerosis, on the other hand, may be roughly defined as a thickening of the arterial wall with a diminution in the size of its lumen. The changes which have led to this when examined in detail are seen to consist of (1) a marked thickening of the intima, due to hypertrophy of the muscle fibers; (2) a thickening of the intima without atheromatous degeneration; (3) and in some cases a fibrous thickening of the adventitia. The muscular coat may show some degeneration but the prevailing notion that in such thickened vessels the muscle coat is replaced by fibrous tissue (fibrous degeneration) is erroneous (Russell).

These changes are not confined to limited areas of the vessel wall as in atheroma, but affect uniformly a large portion of the vascular system and are usually distributed throughout the body for instance in the coronary and renal arteries.

Cases are encountered where both processes are met in combination. These usually occur late in life, the atheromatous changes generally being confined to the large vessels and aorta.

The clinical study of blood-pressure and its relation to visceral involvement would seem to bear witness to the accuracy of Russell's deductions and conclusions, for it will be recognized that were this change one of pure fibrous degeneration with destruction of the muscular tissue in the vessel walls, then measures directed toward relieving hypertension (contraction of the muscular wall) would be useless. As proof of this and of the value of such measures, we have only to review the evidence found in every-day practice, where such measures affect reduction in a larger majority of cases.

The experiments of Pearce[1] in the artificial production of arteriosclerosis in rabbits resulted in the production of merely an atheromatous change in the aorta and the formation of necrotic areas in the elastica and media, which subsequently became impregnated with lime salts (typical atheroma). They showed no evidence of arteriosclerosis.

Adler and Hansel[2] endeavored to produce arteriosclerosis by the injection of massive doses of nicotin. These efforts resulted in the destruction of small areas of intima and muscularis without the production of a diffuse contraction or thickening of the vessel.

These facts further emphasize the present belief that arteriosclerosis is a generalized alteration in the blood-vessels, resulting from a prolonged but mild irritation by a circulating poison and not from a local injury or low-grade infection of isolated areas in the larger vessels.

Clinical Manifestations.—There exists great confusion among pathologists as to the exact nature and process of

[1] *Jour. Exp. Med.*, Vol. VIII, p. 74, 1906.
[2] *Assn. Am. Physiol.*, May, 1906.

arteriosclerosis, so that it is not possible, at this time, to give an exact or rational definition of the disorder from the pathologic standpoint. Clinically the elevation of pressure in arteriosclerosis affords a method of distinguishing between this disease and atheroma with which it is so often confused. Atheroma is really a senile affection coming on in persons between sixty and eighty years and involves the blood-vessels only. Arteriosclerosis on the other hand may attack persons between thirty and sixty years of age and is largely a visceral complaint involving as it progresses, the heart, kidneys and nervous system. Although Oppenheim[1] has reported two cases of undoubted arteriosclerosis in boys of nine and ten years of age. The first died of spontaneous rupture of the aorta probably of the syphilitic origin while the second case was undoubtedly due to autotoxemia.

With the study of atheroma we are but little concerned as this condition must be looked upon as a more or less natural process due to the changes caused by advancing years, and not particularly related to those factors which are recognized as producing arteriosclerosis.

Gull and Sutton's original conception of this disease as an "Arteriocapillary Fibrous" is incomplete. From the viewpoint of the pathologist, the clinician and the therapeutist, we must recognize the multiplicity of the lesions involved in arteriosclerosis and admit the condition as a joint involvement of the heart the blood-vessels and the kidneys, in what may be termed cardiovascular renal disease. Jump has recorded a study of a large number of autopsy records in which arteriosclerosis was present in 69 per cent. and a chronic kidney lesion in 71 per cent. We can

[1] *Virch. Arch.*, Vol. CLVIII, No. 2.

usually by appropriate study of each case, determine the predominating type.

Clinically three stages may be identified (1) the presclerosis of Huchard (Hypertension of Brunton) in which the nervous mechanism of the vascular system is affected by circulating toxins, causing an arterial spasm, and an elevation of pressure (see page 112); (2) the cardioarterial type in which permanent changes have occurred in the blood-vessels, including the coronary cerebral and renal; and (3) the final stage including heart and kidney failure. Careful distinction must also be made between the pathologic lesion and the clinical picture. Simple atheroma may remain for a long time as a symptomless anatomic change. In cardiovascular disease on the other hand, the subjective symptoms predominate and the physical signs may often be obscure. Renal insufficiency is an early and almost constant accompaniment of the latter disease and is present even in the absence of demonstrable albuminuria. This fact accentuates the importance of eliminative treatment during the whole course of the disease.

Clinically, arteriosclerosis may begin in the kidneys, in the heart or in other organs, but there cannot be arteriosclerosis (general) without both cardiac and renal involvement.

Of greatest importance in the diagnosis of arteriosclerosis, is the recognition of the condition termed by Huchard, presclerosis, because of the brilliant results obtained from treatment in these cases. (See Chapter IX.)

Symptomatology.—In the cases with pipe stem or tortuous arteries and ringing aortic second sound, seen in elderly individuals with interstitial nephritis, the diagnosis is made for us, and the treatment is of little avail. It is in the unsuspected subtil ones showing none of these characteristics, with little or no palpable change in the peripheral arteries, doubtful change in the valve sound, perhaps a little roughening in the second sound, with a normal or practically normal urine, that early diagnosis gives brilliant results. These cases may and usually do show gastrointestinal symptoms of a chronic nature, which may be found by diligent search, but which often fail to obtain proper consideration at the hands of the physician. These cases when the sphygmomanometer is properly used show a blood-pressure with a systolic range of from 160 to 250 mm. Hg. (See Fig. 28.)

In persons, entering upon, or in early middle life, this increase in blood-pressure is, in the absence of demonstrable nephritis, usually the only sign of a chronic poisoning arising from some deficiency of elimination, either intestinal or urinary, or both. With regard to an exact symptomatology of early generalized arteriosclerosis, the clinical signs and subjective symptoms may simulate almost any known disease and cause nearly anything from a fleeting dizziness to gangrene of the extremities. Some of the symptoms are not infrequently attributed to neurasthenia. These are vague, unpleasant feelings or fullness in the head, slight momentary dizziness, cold hands and cold feet, sleep unrefreshing and disturbed by dreams, gastric distress and flatulence coming on in one or two hours after meals, constipation and loss of power of concentration and interest in business affairs. The general vitality and power of resistance of the body fails, and tolerance for substances which affect the brain, as alcohol and tobacco is diminished. The patients tire easily. The gastric symptoms increase in severity and any exertion after meals bring on attacks of gastric and heart pain,

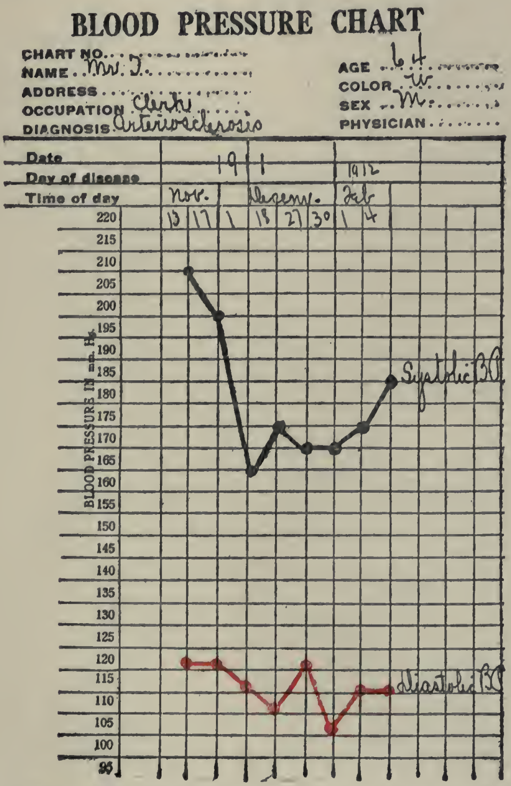

FIG. 28.—Case sought advice because of the recurrence of an old sciatica. Treatment was largely directed toward a chronic intestinal toxemia. The marked fall in pressure resulting from a relaxation of hypertonus, while the further failure to bring the pressure below 165 indicates the failure of such measures to affect a permanent change in the vessel walls.

which are only relieved by resting. The cases gradually become incapacitated for work. They are nervous, lose weight and move slowly, the evidence of involvement of all the organs in the arteriosclerotic process, notably in the brain, heart, eyes and kidneys. Extreme cases give all the classical symptoms and signs which go to make up the syndrome of cardiovascular renal disease. By this time the diagnosis is as easy as the treatment is difficult.

Klemperer a few years ago reported that in a short space of time, he had seen fifty-one doctors between the ages of twenty-eight and forty who gave signs of arteriosclerosis. These cases presented symptoms usually classed as neurasthenic. They were irritable and could not accomplish their work without some form of stimulation, they had been living a high-tentioned life, without sufficient rest and recreation, living two years in one and carrying a blood-pressure of 160 to 180. (Chart, Fig. 29.) For such to continue under the same strain would soon result in marked and incurable cases of cardiovascular disease, terminating in death, long before their alloted time.

Cases of arteriosclerosis which have sustained a high pressure over considerable time show periods of great depression with severe headaches, nausea and sudden vertigo. This is due to the irritation and diminished nutrition of the cerebral centers from the high pressure and the narrowed arteries. Eventually these symptoms become more or less constant, memory fails and insomnia ensues, while life becomes a burden.

Often it is not until these cases suffer a cerebral hemorrhage or show signs of cardiac weakness, that they are even suspected of having anything more than a nervous condition. Routine observations of blood-pressure and a practical knowledge of the early signs of this disease would have made a diagnosis in time to obtain benefit from preventive measures. The routine estimation of blood-pressure and the preparation of a daily chart is of great value in the study of suspicious cases such as simple chronic

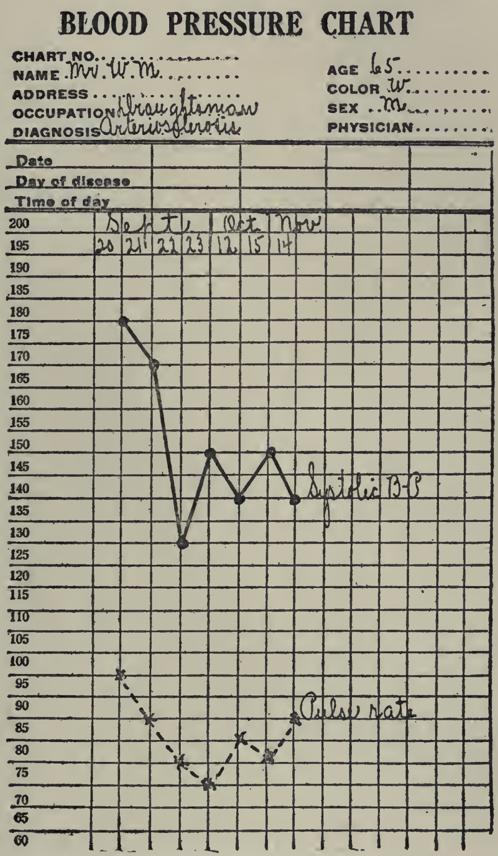

FIG. 29.—Symptoms chiefly cerebral, September. 20, patient slightly delirious, some muscular weakness on left side of body. Chart shows effect of active treatment continued until September 23. Remainder of chart shows lowered pressure maintained by physical measures after patient resumed his activity.

bronchitis with emphysema which are often explained and their etiologic factors supplied, by demonstrating the presence of continued high pressure. Indeed the blood-pressure need not be very much elevated to injure the heart and other organs, for an increase of a few millimeters if long continued entails an enormous increase in the daily work of the heart.

In considering the subject of arteriosclerosis, Daland[1] points out that we must differentiate clinically between (1) the normal thickening of the arterial wall, which occurs after the age of forty, and which progressively increases with advancing years, (2) the atheroma which is a characteristic of the aged and (3) arterial spasm from any cause which when long continued results in a permanent thickening of the vessel walls and a reduction in their lumen. This change being greater than that expected for the given age of the individual.

Pathologically we have to consider the exciting agent or agents which alter the condition in order to separate that of syphilitic origin from the autotoxic or alimentary and renal. Very often clinically in the absence of confirming history, clinical signs and characteristic symptoms, this differentiation is impossible.

Diagnosis.—For diagnostic purposes we may assume that the arterial wall may usually be demonstrated by palpation to be thickened after the age of forty years (Daland). This seems very practical from a diagnostic standpoint when we consider that a demonstration of the state of the vessels is purely a relative comparison and that to ignore this premise would lead into error resulting in a diagnosis of arteriosclerosis in patients having vessels with no more than a normal degree of thickening.

[1] *Monthly Cycl. Pract. Med.*, Vol. X, p. 145, 1907.

Observation is, therefore, first directed toward a study of all accessible vessels by means of inspection and palpation, not forgetting those of the retina by means of the ophthalmoscope. The study of the radial arteries gives most valuable information, but it must not be forgotten that the fibrotic process may be inconspicuous in the peripheral arteries while well advanced in the internal arteries, more especially the splanchnic and cerebrals; and occasionally fibrosis may be advanced in the peripheral vessels with but little or no change in the important internal ones. On account of variations in size and situation of the radial arteries both should be examined. It must also be remembered that the excessive deposition of adipose, or the presence of edema, may prevent successful examination of the radial arteries. It seldom happens, however, that radial sclerosis is diagnosed when absent, the error is usually on the other side.

It is important to separate true sclerosis from pure hypertension as the impression under the finger in these two conditions is quite similar. Arterial spasm usually occurs in the young and palpation of the vessel wall reveals a vessel which feels thicker and smaller than normal, while the lumen appears to be diminished. The common causes of this condition are (1) acute uremia, occurring in the course of acute parenchymatous nephritis as in scarlet fever and similar infectious process; (2) in certain cases of severe acute intestinal toxemia; (3) in certain cases of irritating chemical poisoning.

Apart from the result obtained from palpation, the cardiac and renal signs of arterial spasm may exactly simulate arteriosclerosis and we are therefore compelled to rely upon the knowledge of the cause and duration of the condition to determine the degree of arterial change.

Reliance should not be placed upon the radial arteries alone, but for diagnostic purposes we should use the temporal, the carotid, the brachial, the abdominal aorta, the femoral and the dorsalis pedis.

J. N. Jackson[1] rightly calls attention to the routine measurement of blood-pressure and its importance in every-day practice. He cites as examples, cases in which although the patient did not feel particularly ill, yet the presence of a high blood-pressure demanded a grave prognosis, the correctness of which was later established. It has been stated by Robertson and others that a case with a blood-pressure of over 200 mm. which is constantly maintained, when accompanied by kidney involvement will be very unlikely to live beyond the two-year limit, however there are no definite statistics covering this point.

Elevation of Blood-Pressure.—Having determined the condition of the blood-vessels, the blood-pressure tests may then be applied. In the presence of arteriosclerosis the systolic pressure will be found above that determined as normal for the age of the individual. This elevation need not be great. A continued hypertension of 20 or 30 mm. unless explained upon other ground should be considered pathologic and calls for explanation. It should, however, be remembered that cases will be met having very hard and firm peripheral vessels showing a normal or subnormal systolic blood-pressure.

I distinctly remember one case of over fifty years of age

[1] *Boston Med. and Sur. Jour.*, Nov. 2, 1911.

having the most rigid and pipe-stem radials that I have ever palpated, yet at no time was the systolic pressure found to be over 100 mm. Hg. Again the hypertensive effect of arteriosclerosis may be counteracted by the hypotensive effect of an associated thyroid disease or a deranged adrenal system, as in Addison's disease. (See page 109.)

A comparison of the systolic and diastolic pressures and an estimation of the pulse-pressure is of distinct value in the study of all cases. The physical changes produced in the dynamics of the circulation by the less elastic vessel walls, will in the presence of a normal heart show an increased pressure often as high as 60 and occasionally 100 or more, as the advanced and extreme cases are met.

Examination of the heart in pure early arteriosclerosis (before the kidney has become much damaged) will reveal only some slight accentuation of the second aortic sound. The studies of Romberg and Hasenfeld[1] found hypertrophy of the left ventricle in only a small proportion of cases of arteriosclerosis before an associated nephritis had developed.

The temperature, as is the case in most chronic diseases, will usually be found subnormal, although Stengel[2] called attention to the occurrence of continued fever in certain cases and he holds that when there is no other assignable cause for the fever, it is probably due to the arteriosclerotic process.

Examination of the digestive tract will often reveal slight departures from normal probably dating back for many years, and the results of test-meal examinations will

[1] *Deut. Arch. f. klin. Med.*, Vol. LIX, 1897, p. 193.
[2] *Medicine*, Detroit, June, 1906.

show reduced gastric secretory activity with abdominal distention and often most obstinate constipation.

A consideration of the patient's history and recent general condition may reveal periods of mental lassitude and irritability with headaches coming on after mental or physical excitement or at a certain time each day. There are often momentary attacks of dizziness often accompanied by nausea and followed by profuse perspiration and a period of weakness. Insomnia, loss of memory, melancholia and other nervous symptoms as a gradual loss of mental vigor and bodily tone unite to form a well-known picture of the average cause of established arteriosclerosis.

Treatment of Arteriosclerosis.—Certain causes of arteriosclerosis demand besides the general therapy as applied to the condition of sclerosis (chiefly toxemia) a therapy devoted to the particular causal agent, when this is ascertainable. There are cases of arteriosclerosis, dependent upon gout, diabetes mellitis, alcoholism, nicotinism and syphilis, and more rarely upon lead and other poisons. Without going into detail, it is sufficient to point out that when the relation of the arteriosclerotic process to any of the above diseases has been established, it is imperative to treat not only the condition itself but also, to direct treatment toward the relief or removal of these exciting causes.

All authorities agree that the general treatment should combine a reduction of diet and stimulation of elimination, through the skin, bowels and kidneys together with the removal of all undue physical and mental strain.

From another standpoint the treatment can be divided into (a) that directed toward the prevention of the development of the disease in those predisposed by heredity or environment; (b) that directed toward arresting the production and to the relief of the conditions as found.

When the disease is recognized in the early stage before the heart is seriously involved and before the kidneys show positive evidence of an interstitial nephritis, much can be gained by careful regulation of hygiene, by careful living and by modification of diet. Work, both physical and mental, should be reduced and the patient taught to live on a lower plane.

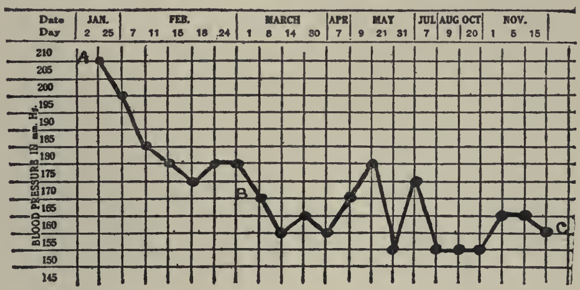

FIG. 30.—This blood-pressure chart, taken from a case of arteriosclerosis shows effect (1) from *A* to *B*, combined drug and rest treatment; (2) *B* to *C*, effect of dietetic hygiene combined with systematic daily walking, no drugs, patient attending to business.

Dietetic regulation with measures directed toward maintaining renal efficiency should always be the basis of sound treatment (Fig. 30).

An excessive milk diet is often beneficial in effecting marked reduction in a dangerously high pressure. So also a meat diet, but the ultimate effect of the meat diet may cause an aggravation of the disease, as shown by the nervous and other serious symptoms.[1]

By emphasizing strongly the dangers of worry and of

[1] J. M. King, *So. Calif. Med. Jour.*, Aug., 1910.

undue strenuousness and by urging patients to take greater advantage of the sunny side of life and to refrain from a too serious view of themselves and their responsibilities, many cases of arteriosclerosis can be avoided.[1]

According to the observations of Huchard,[2] the ideal diet directed toward the reduction of hypertension, and the prevention of arteriosclerosis is one composed chiefly of vegetables and milk and one from which sodium chlorid is largely eliminated.

General Dietetic Directions.—A good general rule of diet is that while meat is not to be prohibited a diet composed chiefly of milk, vegetables and fruit is indicated; absolute milk diet is not good when prolonged but may be used for the relief of certain symptoms. It is important that the daily supply of food should be taken in small quantities and at frequent intervals. Alcohol, tea, coffee and tobacco need not usually be prohibited entirely, although with heart pain and in angina tobacco should never be allowed. The reduction in diet should not be carried to a point of causing a feeling of subjective weakness, and should not rapidly reduce body weight, except in the obese.

Limiting the amount of water taken undoubtedly spares the heart and vessels, but the amount should not fall below 1,500 c.c. per diem and even when there is edema not below this figure for more than three consecutive days, otherwise kidney elimination will be reduced and the case suffer accordingly.[3]

[1] M. Herg, *Medizin Klin., Berlin*, Jan. 16, VI, No. 3.
[2] *Bul. de l'Acad. de Med.*, Jan. 21. 1907.
[3] A. Strasser, *Wien klin. Wochen*, April 8,1909.

Special Diet.—The fact that too abundant diet may be a cause of arteriosclerosis should be kept in mind. Moreover the food should contain as small amounts of toxic substances as possible, as these upon entering the circulation cause a narrowing of the vessels (hypertonus, and thereby cause an increase in pressure.

Sausages, spiced and pickled meats, rich foods, strong broths, caviare, ham, and foods containing much nuclein as roe and sweet breads, strong cheese, liquors and strongly alcoholic drinks should be absolutely forbidden.

Baths, Climate, Etc.—(See also Chapter XIX.) Very few patients with arteriosclerosis do well in an altitude of 3,000 ft. and over. One important fact must always be remembered—the danger in arteriosclerosis of any sudden alteration of blood-pressure, particularly any sudden increase, hot or very cold baths are therefore contraindicated, on the contrary baths of moderate temperature in cases of arteriosclerosis, with a very good or compensated heart are very beneficial. These baths may be taken, at any of the resorts, where such treatment is given, or at home, by means of a simple warm bath accompanied by general friction of the body, or by a warm pack which increases cutaneous dilatation and increases elimination through the increased production of perspiration.

Medication.—(See also Chapter XIX.) The condition of the intestinal tract is of utmost importance; daily evacuation must be had, if necessary by means of laxatives, or cholagogues, supplemented by salines or saline waters (Hunyadi or Pluto) as the study of the blood-pressure curve indicates.

Drug Treatment.—(See also Chapter XIX.) Directions toward the relief of high pressure and modification of the condition of the pathologic vessels, have been most disappointing, and the reports of observers who have tabulated their findings, are so at variance that little dependable knowledge can be obtained from them. This is in part due to the fact that many have endeavored to accomplish the impossible (the removal of sclerotic tissue) and partly to the great variety of causes underlying the development of the condition.

It is far from the author's intention to discourage drug therapy, therefore it seems advisable to give a brief résumé of the opinions of recognized authorities and thus allow the reader to draw his own conclusions from them.

Huchard[1] places his dependence in the treatment of this condition in the following order.

Diet, muscular exercise, massage, particularly abdominal and precordial massage (massage does not produce elevation of blood-pressure, see page 246), baths and high-frequency currents. Drugs, the nitrites with theobromin to assist elimination through the kidneys. He believes heart tonics unnecessary until the condition is far advanced, but when indicated the periodic administration of digitalis is advised.

Edgecombe[2] says that the effect of thyroid extract is powerful and rapid.

On the other hand, Huchard[3] relies almost wholly upon dietetic and hygienic measures, and says that the abuse of drugs, especially of the iodids and of digitalis is especially

[1] *Bul. de l'Acad. de Med.*, Jan. 21, 1907.
[2] *N. Y. Med. Rec.*, July 16, 1910.
[3] *Jour. A. M. A.*, Vol. LII, No. 14.

to be avoided and also the abuse of the so-called "anti-sclerotic serums," high frequency currents and climatic and some mineral water "cures."

Beverly Robinson agrees with Huchard when he states that "the larger my experience and the more I watch cases of pronounced arteriosclerosis especially in men and women past middle life, the less frequently I prescribe either digitalis or the iodids. If a cardiac tonic or stimulant is required, strophanthus, caffein and nux vomica are preferable by far, and are not likely, in small or moderate doses, to do positive injury. To lessen hypertension, where it is clearly indicated, by reason of headache, fainting attacks, pallor and general nervous irritability, sweet spirits of niter, in small or moderate doses, added to water is the least injurious and most useful drug I have known, not excepting nitroglycerin and the nitrites."

Henry Jackson[1] in moderate uncomplicated arteriosclerosis, depends chiefly on dietetic measures and the reduction of mental and physical exertion. One drug he considered of value as a preventive measure and in some cases as curative, potassium iodid, to be taken 1/2 gr. three times a day for three out of four weeks in a month, or by substituting strontium iodid in the same dose when potassium upsets the stomach. He employs saline cathartics and sees benefit in high frequency currents. (See Chapter XIX.)

[1] *Boston M. and S. Jour.*, Aug. 11, 1910.

CHAPTER XI

DISEASE OF THE KIDNEYS

It is beyond the scope of this work or the author's ability to analyze and classify the complicated relations which the light of recent knowledge has shown may exist between chronic diseases involving the heart, the blood-vessels and the kidneys. Most careful study during life often fails to fully illuminate all cases, in some of which only at post-mortem is the true condition demonstrated, when it is far from rare to have our clinical diagnosis absolutely reversed by the pathologic findings.

In a large per cent. of cases met and studied clinically we find coincident involvement of the kidneys, the heart and the blood-vessels, so that our ability is often taxed to the utmost in an effort to give each factor its true value, and to assign each to its proper place in therapeutic management.

The views here set down have been reached by a careful review of recent literature, bearing upon the relation of these several organs to the clinical picture, particularly chronic nephritis, as viewed by the author, in the light of his experience with this class of cases.

Etiology.—According to Stengel,[1] Bright's disease may be one of the complications of the general disease, arterio-sclerosis, when it will usually be found as a well-developed

[1] *Medicine*, Detroit, Mich., June, 1906.

case, contributing largely toward the terminal stage, which comprises circulatory and organic failures and terminal infections. Stengel in discussing the association of albuminuria and slight renal change with general arteriosclerosis concludes that, at least from the clinician's standpoint for prognostic purposes, comparatively little significance attaches to the occasional presence of slight traces of albumin in arteriosclerosis when phenomiæ, such as high blood-pressure and polyuria are wanting.

Clinical Classification.—Allibert in the *British Medical Journal* (April 15, 1911) classifies clinically the kidney relation to generalized arteriosclerosis as follows:

1. Associated with chronic nephritis. (a) Chronic generalized arteriosclerosis not due to high blood-pressure, constantly found associated with contracted kidney and arteriosclerosis of varying origin in which the kidney condition is not constant. (b) Secondary generalized arteriosclerosis due to high pressure. (c) A few mixed cases which reveal kidney disease, supervening on a general arteriosclerosis, but not originally due to Bright's disease.

2. Not associated with Bright's disease. (a) Arteriosclerotic kidney, in which the kidney changes are secondary to general arteriosclerosis. High pressure may be considered as (1) obligate with contracted kidney, (2) irregularly associated with the other kinds of nephritis and (3) occasionally as independent of chronic nephritis.

This classification while amply covering all conditions will be in most cases very difficult to apply. Usually we will have to be content with a broader generalization, and to give precedence to the prominent symptoms which demand therapeutic management, in order to relieve the patient and possibly arrest the progress of the disease.

Pathology.—Marcuse[1] asserts that renal inflammation causes an increased resistance to the blood current in both kidneys, in consequence of which a compensatory hyperemia of the suprarenal capsules is produced through the inferior suprarenal artery, which is a branch of the renal artery. We know that hyperemia of an organ can bring about a hypertrophy of that organ and can assume naturally that a hyperemia or hypertrophy of the suprarenal capsules increases their functional activity and results in an increased amount of adrenalin in the blood. In this way the increased functional activity of the suprarenal capsules brings into causal connection general increase of blood-pressure and hypertrophy of the left side of the heart.

The belief of Hiatt[2] is that the rise in blood-pressure in cases of autointoxication is caused primarily by the action of these poisons on the smaller ganglionic endings of the splanchnic nerves in the vessel walls, and these poisons have a selective action for the vasoconstrictor nerves and that the first effect of this action is a rise in the blood-pressure of the portal vessels. The connections of the nerves supplying these vessels is such that there is a general reaction. An efferent impulse is sent out to the entire vascular system from the reflex centers located in the spinal cord. This results in acceleration of the heart beat, both through the sympathetic ganglia and the accelerator nerves of the heart. Thus we have a faster beat and an increased peripheral resistance, these two factors will raise the pressure to an

[1] *Berlin.klin. Wochen.*, July 19, 1909.
[2] H. B. Hiatt, *Archives of Diagnosis*, N. Y., April, 1911.

extent that requires a more forceful beat to overcome it. The more forceful beat, in the presence of the conditions mentioned, causes a rise in pressure and thus a vicious circle which involves the kidneys is established.

Richard C. Cabot[1] in the fullness of his experience, presents a very useful and practical classification of the several degrees of disturbed renal function as follows:

1. Renal irritation, presence of albumin and casts.

2. Renal insufficiency, which may exist with or without cause, and shows itself chiefly in the physical characteristics of the urine and the condition of the rest of the body (oliguria, dropsy, and uremia).

3. Nephritis, which shows itself in the postmortem condition of the kidney. These three sets of changes are frequently associated, but the association is by no means invariable.

Renal Irritation.—Following violent exercise, often causes albumin and casts to appear in the urine. The microscopic findings often being as various as those of an acute nephritis. These cases subside entirely, have no great elevation in blood-pressure and postmortems have shown that such a urine is consistent with normal kidneys.

Renal Insufficiency.—The kidney cannot perform its normal function, either of elimination or of excretion or both. The condition is characterized by diminution in twenty-four hours urine and by the appearance of dropsy. High blood-pressure and cardiac hypertrophy are natural sequential accompaniments. Often the larger portion of urine is secreted at night, and is of lower specific gravity. Albumin and casts may or may not be found.

[1] *N. Y. Med. Jour.*, May 12, 1906.

Nephritis.—Acute and chronic interstitial nephritis are hard to determine without careful and painstaking study. Postmortems show wide discrepancy between the clinical and pathologic findings.

Pathologic reports bearing on the relation of elevated blood-pressure to postmortem findings in the heart, blood-vessels and kidneys, seem to demonstrate conclusively that the blood-pressure may be accepted as a safe guide and that the statement of Emerson,[1] "that persistently and constantly high blood-pressure is evidence of either acute or chronic nephritis," is probably in a large majority of cases correct.

Roger L. Lee[2] reports both clinical and pathologic (autopsy) findings in fifty-three cases seen in the wards of Massachusetts General Hospital, all of whom showed systolic blood-pressure of over 160. He found high pressure associated with kidney lesions in thirty-eight cases or 71 per cent. Seven who showed kidney lesions also had a systolic blood-pressure, ranging from 165 to 240 mm. Their ages were between twenty and forty-nine years.

High blood-pressure existed with arteriosclerosis in thirty-seven cases or 69 per cent. General arteriosclerosis was associated with lesions of the kidneys in twenty-eight cases or 52 per cent. There was only one case of high pressure with arteriosclerosis without kidney, cerebral, or cardiac lesion; this showed only hypertrophy and dilatation. The case was sixty years old and the blood-pressure was 210.

Of cases with high blood-pressure without kidney lesion, these were 15 or 28 per cent. Among these seven showed

[1] *Jour. A. M. A.*, June 6, 1909.
[2] *Jour. A. M. A.*, Vol. LVII, No. 15, p. 1179.

cerebral lesions, four had cerebral hemorrhage and seven showed cardiac lesions. The blood-pressure varied from 175 to 260.

H. D. Jump in International Clinics (Series 21, Vol. I) reports the pathologic findings of a series of high-pressure cases 71 per cent. of which showed some permanent kidney change, while arteriosclerosis was present in 69 per cent. of cases. All cases which showed a repeated and constant pressure over 200 showed some form of nephritis. Jump believes that a systolic blood-pressure above 160 is always to be viewed with suspicion.

Signs and Symptoms.—Chronic interstitial nephritis in its well-developed form is usually the result of a gradually progressive process, leading up to a clinical picture which is too well known and too easily recognized to require more than passing comment. It is particularly the early states of this disease, with which we are concerned. Our chief effort and desire is to reach an early provisional diagnosis, so that preventive or prophylactic treatment may be instituted, at a time when proper management may be reasonably expected to arrest the progress of the degenerative process in the kidneys, and so to indefinitely prolong the individual's period of usefulness and life.

Blood-pressure.—A permanent elevation of both systolic and diastolic blood-pressure is the most prominent and characteristic sign of well-developed chronic nephritis. Sawada[1] states that he has never seen a case of hypertension of more than 170 mm. in simple arteriosclerosis. Romberg[2] considers that persistent high blood-pressure in a sus-

[1] *Deutsch med. Wochen.*, 1904, No. 30.
[2] *Kong. f. Int. Med.*, 1904, No 60, p. 17.

pected case establishes a diagnosis of chronic interstitial nephritis. From the author's experience, in the light of postmortem findings, it seems very doubtful whether a high arterial pressure, from arteriosclerosis or any other cause, can persist over a long period of time, without giving rise to the chronic congestion and permanent degenerative changes in the kidneys; with a clinical picture known as chronic Bright's disease. The pressure is higher than that seen usually in any other chronic disease. Sphygmomanometric observations daily confirm this. A systolic blood-pressure of more than 200 mm. (standard cuff) is not uncommon, and I have seen several cases with a reading of over 300 mm. (Fig. 31). Two of which have been under observation for more than a year, and will be referred to later. A second salient feature of this disease, is that the diastolic blood-pressure does not show a proportionate elevation, but is usually from 60 to 90 mm. lower, thus making an increased pulse pressure (evidence of unnecessary overwork of the heart). Factors such as marked general arteriosclerosis or aortic regurgitation will further accentuate this sign. While myocardial weakness, or a failing heart will be shown by a fall from the marked elevation and a gradually narrowing pulse pressure.

This fact emphasizes the importance of considering the blood-pressure and its factors only as signs or indicators, and of the necessity of always viewing the case as a clinical whole. Otherwise if too much importance be placed on the sphygmomanometer findings, we may fail to recognize signs of a failing circulation which may mark the beginning of the end. A moderately high blood-pressure and a moderately increased range; coupled with stationary physical signs and symptoms, usually indicate a well-sustained circulation and a fairly adequate renal function. If a lowering of systolic pressure and an approximation of the

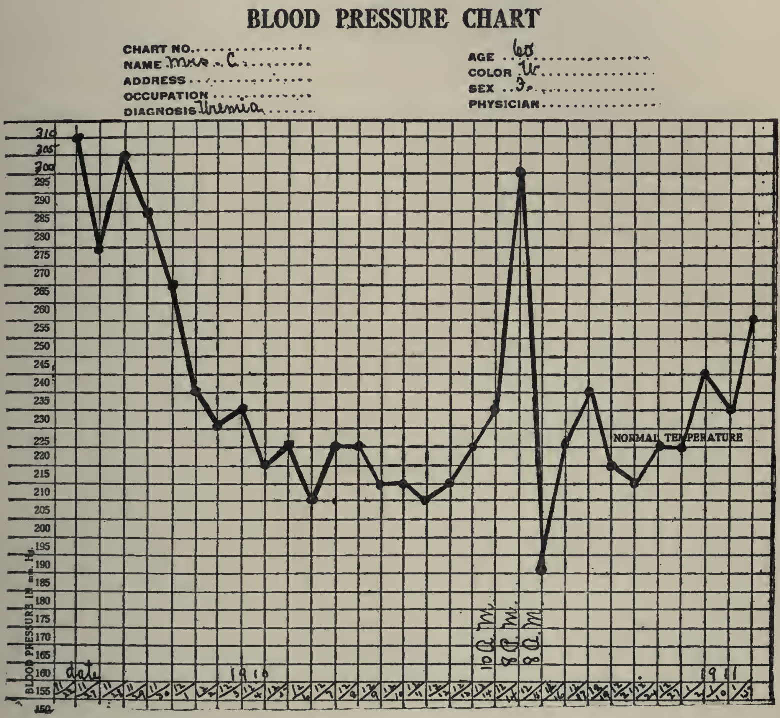

FIG. 31.—Uremic paralysis, resulting from chronic interstitial nephritis. Treatment consisted of hot wet packs, nitroglycerine, 1 per cent. solution, one minim every hour, and magnesium sulphate. Treatment stopped on December 1, and sodium nitrite substituted, December 7, nitrite stopped, hot packs continued. Following sudden rise on December 14, thirty minute pack, two minims nitroglycerine, repeated every two hours, resulted in profound fall as shown, with no bad effect. Subsequent treatment consisted of thirty-minute vapor baths and weekly purge in spite of which there occurred a gradually rising pressure.

diastolic pressure should follow, this should give warning of a failing circulation, progressive renal inadequacy and all that these changes imply. The high pressure of chronic nephritis from a clinical standpoint at least would seem to be a wise provision in many cases, one which is necessary in order to maintain a fairly adequate renal secretion. The system seems after a time to become readjusted to the heightened pressure, so that sudden changes, through accident or misguided treatment, result in disaster. My memory of two cases emphasizing this point is quite vivid. The first always began to suffer from edema and effusion when the pressure was forced down to any extent, and the second complained of a return of cerebral symptoms, when he had, contrary to orders, markedly reduced his pressure, by too long-continued use of sodium iodid. This chart (Chart No. 36, page 238) demonstrates the change in blood-pressure which was noted.

Until quite recently it has been the custom to consider albumin and casts of certain varieties in the urine as evidence of chronic nephritis. A condition of serious import and one that endangered life. In the light of recent pathologic findings, we can now classify these cases and properly value them to the urinary findings in at least a majority of cases, and this, especially before the usual symptoms have developed, is accomplished through the agency of the sphygmomanometer.

That there is still much to learn concerning the borderline cases is amply shown by pathologic reports (see page 114) and since we have a means of precision, which is simple in operation, and which may be called to our aid, it would seem almost unnecessary to urge the importance of sphygmomanometry, as an aid to this study. Careful study of pathologic urinary findings is essential because of the great frequency of the association of chronic renal change with high blood-pressure. As almost every case of blood-pressure above 200 mm. will at some time show albumin and casts (one or both) it would be advisable to differentiate between an actually chronically contracted kidney, and the arteriosclerotic (secondary) kidney, which is well recognized, both from a prognostic and a therapeutic standpoint, to be of less serious import.

A. E. Elliott[1] reports a series of sixty cases of chronic nephritis with an average systolic pressure of 190. The highest was 285 mm.

Elliott concludes that renal permeability to albumin seems to be overcome when the blood-pressure reaches or exceeds 200 mm. Hg., so that in cases of very high pressure, whether there is nephritis or not, either of primary or secondary nature, we usually find albumin in the urine.

Prognosis.—To arrive at a satisfactory prognosis, one must be in possession of all the facts, not only the important and self-evident signs, but the minor details of the life and habits of the individual. With a full knowledge of the case, prognosis became merely a process of weighing and deciding, pro and con. For example, as Cook has emphasized the outlook in case of a robust looking man of fifty-five with a blood-pressure of 200 mm. even with no appreciable arterial degeneration, is not so good as in the case of a man of sixty-five with rigid arteries and a lower pressure.

Treatment.—The treatment of chronic disease of the kidneys cannot be outlined, much less reduced to a definite routine. Each case is a law unto itself, requiring special

[1] *Jour. Am. Med. Assn.*, April 1, 1907.

study, often some experiment and calling for particular measures and peculiar management. This outline will serve merely to recall to the physician's mind some measures which are of value in the average case, and from which may be selected a combination of measures suitable to each particular case.

Diet and hygiene are of foremost importance, while drug methods are often secondary, and may in many cases be entirely dispensed with. Blood-pressure reduction by drugs should be attempted only with great caution, except when necessary to prevent or relieve a uremic attack.

Symptoms of overpressure are usually relieved by a reduction of 20 or 30 mm. and do not reappear, as long as this reduction is maintained. Englebach[1] emphatically warns us of the dangers of nitrites both in edema and in cases of advanced Bright's disease.

Diuretics are of value in so far as they increase elimination, and may be of aid in relieving the heart from unnecessary additional strain. Heart tonics are usually unnecessary before the nephritic condition is well advanced, but when indicated, the intermittent administration of digitalis will often prove of value.

Among other remedial measures of value in combating certain conditions arising in chronic nephritis may be mentioned calomel and saline cathartics, alkaline mineral waters, enteroclysis, hot baths of various kinds, and venesection followed by intravenous saline infusion.

Iodids in small doses are of value when a syphilitic history is obtained or suspected.

[1] *Interstate Med. Jour.*, June, 1911.

In uremia, venesection, enteroclysis; or hypodermoclysis, and chloral gr. x to xxx by rectum; morphin 1/4 to 1/2 gr. hypodermatically.

Huchard[1] regards renal insufficiency as a very important symptom of early arteriosclerosis and one that should command our best efforts for its relief, consequently we should reduce to a minimum the alimentary toxins which are the chief cause of this difficulty. Here diet regulation plays an important part, as it also does in lessening the tendency to excessive arterial tension. The ideal diet is one composed chiefly of vegetables and milk and one from which sodium chloride has, as far as possible, been eliminated.

A fair amount of active physical exercise is desirable, but when impracticable massage may be substituted.

Overexertion, overstrain and undue exposure are to be avoided.

[1] *Bul. de l'Acad. de Med.*, January 21, 1907.

CHAPTER XII

MYOCARDIAL DEGENERATION

Definition.—From a practical standpoint it would seem advisable to employ the general term myocardial degeneration, to the exclusion of all others, when discussing from a clinical standpoint, the pathologic changes which may occur in the heart muscle; for, while we recognize pathologically a sharp line of demarcation between acute and chronic inflammation, and between fatty degeneration, fibroid degeneration, or fibrosis, weak heart, senile heart and chronic cardiac insufficiency, in the majority of cases there is no way by which these various conditions can be distinguished from each other clinically. Any attempt to separate the various forms of myocardial change, by a clinical study of the case, is merely an expression of ignorance, for the symptoms supposed to indicate different forms of myocardial disease, may be caused by the same pathologic conditions. Also various pathologic changes may give rise to an identical train of symptoms so that all efforts to clinically classify must necessarily fail. On this account it appears best to consider them all together, not only from the standpoint of symptomatology, but also for purposes of prognosis and treatment, which with few exceptions are essentially the same for all. These exceptions whenever well marked will be indicated.

Occurrence.—Of the various forms of myocardial change the pathologic entity known as fibroid myocarditis is probably the commonest disease of the heart muscle. It is usually responsible for permanent dilatation. It results from repeated strain, as in chronic nephritis, arteriosclerosis, from gourmandizing and from manual labor. Such changes have been produced in the heart by experimental adrenalin myocarditis (Pearce, Fleischer and Loeb). This affection is more common in men than in women, occurs most frequently in those past their prime, but is commonly observed five to ten years earlier than fatty degeneration.[1] These two conditions are usually classified, clinically as chronic myocarditis, yet while they may occur as acute conditions, following certain acute infections, they are usually chronic in character, the symptoms may, however, occur suddenly and be very acute.

In cases of great emaciation, the heart may be weakened by actual atrophy of the organ. Such hearts are small, have a muddy brown color, do not maintain their shape, and are functionally very weak.

Etiology.—Chronic myocarditis is a condition of the heart muscle resulting usually from some alteration in the circulatory system. Leslie Thorne Thorne[2] says that the two most common forms of tissue degeneration resulting from hypertension are those of atheroma and fatty degeneration.

The modus operandi of degeneration is probably that of a disturbed blood supply to the heart itself, due to a narrowing of the coronary arteries. It is therefore essentially a chronic progressive process and from the very nature of the change, when once the process has become fairly

[1] E. Fletcher Ingals, *Boston Med. and Sur. Jour.*, Vol. CXLV, No. 18.
[2] *Lancet*, June 4, 1910.

well started is but slightly amenable to treatment. The whole subject of chronic myocarditis is one of muscular integrity. The diseased heart dilates from slight overstrain, one which under ordinary circumstances would be harmless, so that a short run for a car, running upstairs, lifting weights, etc., may result in a more or less permanent dilatation.

Bruce[1] dwells upon the frequency of cardiac degeneration, associated with glycosuria, and also the frequent relation of gout to chronic myocarditis.

A most important factor, never to be forgotten, especially in this strenuous age, is the affect of the constant strain of responsibility borne by business men, legislators, professional men, etc. In these the development of high pressure is particularly common. This results in cardiac enlargement with more or less insufficiency, all of which marks the beginning of the end, unless the stress of life is reduced.

In the same class of cases, on account of indiscretions in diet, and sedentary habits with insufficient exercise, the intraabdominal vessels are subjected to abnormal and prolonged strain, this leads in time to sclerosis of their coats, increased blood-pressure, cardiac overwork and eventually to degeneration of the myocardium.

Fatty degeneration of the heart is due in most cases to the same conditions which cause atheroma of the aorta, and disturbance in the coronary circulation. This is one of the natural results of advancing age, where it is generally dependent upon the long-continued action, of such irritations as chronic autointoxications, habitual use of alcohol, toxic effects of tobacco, coffee, etc.

[1] *Lancet*, July 15, 1911.

Chronic diseases of the kidneys by increasing the resistance in the arterioles, raises blood-pressure; this produces general arteriosclerosis from which the coronary arteries are not exempt, and leads inevitably to myocardial degeneration. Less commonly we find fatty degeneration following protracted wasting diseases, exhausting discharges or anemia from repeated losses of blood. Acute fatty degeneration usually results from the toxins of diphtheria and other acute infectious processes, and occasionally from phosphorous or mercurial poisoning.

In the study of all diseases of the heart, we are chiefly concerned with the function of the heart muscle, and this function is intimately concerned with the several factors of irritability, rhythmicity, conductivity, contractility and tonicity (Lauder Brunton).

Unfortunately of that about which we desire to know most, we actually know the least. Thus far no accurate means of measuring these several factors especially the ability of the heart to carry on its circulation, against the odds of valvular disease, arterial disease and disease of the heart itself, has yet been found. The many methods which have been suggested from time to time, all fall short of any great degree of accuracy.

In this discussion we are concerned chiefly with the function of contractility and tonicity or tonus. By tonus is meant the power of the heart muscle to resist overdilatation, during the diastolic period. The normal heart begins to dilate under exercise, but if the exercise is moderate this is transient, and is quickly overcome by the normal tonus. In the athlete as a result of training, any exercise to which the individual is accustomed actually causes the heart to become smaller, this is due to an increase in the reserve power. The heart of the athlete is often hypertrophied and such hypertrophy in the light of recent experimental evidence is a true hypertrophy (Kuelbs).

While a strong heart tends to decrease in size during exercise, the weakened heart tends to increase, or in other words to dilate. This is due to a deficiency in tonus; consequently a heart in which the muscle is diseased will dilate upon comparatively slight exertion. In fatty degeneration there is always diminished tonicity, therefore, diminished tonus is an important factor in the production of permanent dilatation.

In this connection the venous pressure plays an important part, as venous pressure is increased by exercise, and is particularly high during straining, heavy lifting, etc., this is frequently a factor in the production of overstrain, because a high venous pressure keeps the right heart dilated, and if the tonicity is low, the heart muscle will remain dilated. According to the researches of Louis M. Warfield[1] the most important factor in the production of chronic dilatation is constant repetition of the strain. Even a mildly diseased heart may recover from considerable strain, provided the strain ceases at once, or that time is allowed for the heart to return to normal size before the second strain occurs. On the other hand, if repeated strain occurs to a heart already dilated, having low tonicity, then permanent damage results. The border line between true heart failure and complete recovery depends to a large degree upon the period of rest after strain.

[1] *Interstate Med. Jour.*, p. 994, 1911.

This brings us back to the first proposition, which is, that the normal function of the heart muscle depends primarily upon the integrity of the muscle itself, and that if this is strong, it would be able to stand an immense amount of strain without becoming permanently damaged. We see, therefore, that the essential factor in the production of a chronically dilated and weak heart is a reduced tonicity the result of alterations in the condition of the heart muscle itself. That these changes are usually either of fibrous or fatty degeneration, which eventuate in loss of heart strength and permanent dilatation.

Pathology.—*Fatty degeneration* of the heart muscle includes two conditions, one in which the action of the organ as a whole is impaired by a superabundance of fat, which does not necessarily replace directly the muscular fibers, this fatty deposit penetrates between the muscular fibers, impedes their action mechanically and may, through pressure impair their nutrition; resulting ultimately in the destruction of the fibers themselves by true fatty degeneration. This condition generally occurs in obesity and is termed fat heart. A heart in this condition is always weak and although it may do its work fairly well from time to time, will eventually give way under some strain. In the other condition owing to some obstruction or inflammation of the coronary arteries, nutrition is interfered with and the muscle fibers become more or less replaced by fat. This constitutes true fatty degeneration.

Parenchymatous degeneration is a cloudy swelling, essentially acute in nature, usually occurs in infections among which rheumatism and diphtheria are well known examples. It usually does not result in permanent impairment of the heart muscle.

In *fibroid degeneration*, the muscular fibers are more or less completely replaced by connective tissue. This is not a general change in the heart muscle, but usually occurs in limited areas. These are found most frequently near the apex of the left ventricle in the interventricular septum or in the papillary muscles.

Not infrequently the heart muscle becomes hypertrophied in the early stages of any of these conditions in an attempt to overcome the functional deficiency, but later, disease of the coronary arteries, reducing the normal blood supply of the heart muscle, causes the fibers to degenerate, the heart wall becomes weakened and dilated, and incompetence follows.

After death, the dilated heart will show under the microscope either fatty or fibroid degeneration, or both, involving the muscle fibers themselves, together with varying amounts of intermuscular fat.

Valvular Disease Accompanying Chronic Myocarditis.— Hearts, the subject of valvular lesions are not necessarily weak hearts, though lesions of the different valves, naturally produce different effects upon the functional power of the heart. Valvular lesions in general affect the heart only in so far as they force the heart continually to do more work to maintain the circulation than the normal heart should be called upon to do. The normal heart has a large reserve power and auxiliary force for use in emergency. The addition of a valvular lesion constantly encroaches upon this reserve power. Thus in aortic disease, we may actually have a more powerful heart than normal, while its reserve power is very much less or may be entirely exhausted.

A very important point to bear in mind is that it is not the valve lesion which determines the failure, but the condition of the heart muscle. One must not be misled into believing that hearts with a defective valve are as strong as normal hearts, this is not so, as they often break down under strains which would have no effect upon normal hearts. The very fact that there is a valvular lesion means that eventually the myocardium will become diseased, and that this will determine the functional power of the heart. This again brings us around to the point already made, that the pathology of heart disease is the pathology of the myocardium. It is well known that many persons with valvular disease are able to be about and to attend to their daily tasks without inconvenience, and we are frequently so interested in the valvular lesion itself that we are apt to neglect to study the condition of the heart muscle, which is the true indication of the physical condition of the case. Thus, we often fail to notice warning signs, until some accident develops the symptoms of heart failure. This emphasizes the necessity of careful examinations, made at regular intervals when possible, during which particular attention should be paid to an estimation of the functional capacity of the heart muscle. By this means, we may be able to put off for a long time the symptoms of heart failure.

Heart Failure.—Broken compensation does not occur in a normal heart, however severe the exercise may be. A normal heart muscle is, however, capable of becoming acutely dilated. In this condition the pulse may be so rapid that it cannot be counted, and nausea and vomiting may occur. The patient may faint from the exertion, but so far as we know, such hearts return after a time to normal size and by virtue of the normal tonicity of the heart muscle no permanent damage results. Even hearts with evident valvular lesions do not break down, except temporarily, unless the ventricle is diseased. According to Warfield "One may therefore lay down the axiom that broken compensation is dependent upon an excessive strain, placed upon a heart whose muscle is the seat of pathologic changes, which have weakened the muscle." Symptoms indicative of cardiac degeneration usually occur several years before the fatal termination. In the presence of a blood-pressure above 200 these symptoms rarely extend over a period of more than two years. The usual symptoms of heart failure occur often without warning, when the patient is seized with sharp precordial pain accompanied by faintness and dizziness, following which he may sink back in his chair or fall to the floor and expire before any assistance can be rendered.

Symptoms.—An early and apparent sign of early myocardial change is the development of peculiarities in rhythm. Careful investigation will often show that the function of rhythmicity has been interfered with, causing intermittence, irregularity and extra contraction, or extra-systoles, this means damage to the heart muscle.

F. R. Nuller[1] is of the opinion that since we so frequently find arteriosclerosis in heart failure in later years, in those who earlier showed extra-systoles, we should therefore pay

[1] Harvey Lecture, 1906–07.

more attention to such a symptom, as it is probably a stage in the evolution of these diseases.

In many cases beginning myocardial change may be perceived by studying the two aortic tones, the first sound in the aortic area, may be unchanged, weakened or accompanied by a murmur, while the second sound may be either intensified or diminished. A systolic aortic murmur occasionally may be due to a blood state, but in a person of middle age, it is strong evidence in favor of alterations in the aortic wall, and in the myocardium. Should these findings be associated with thickened peripheral arteries and an elevated blood-pressure, slight but persistent, the conclusion is warranted, that myocarditis is present, especially when in addition, there have been subjective signs of muscular weakness. Where the transverse diameter of the heart can be shown to be increased the diagnosis of chronic myocarditis is insured.

Two most important signs which are frequently overlooked are feebleness of the muscle sounds and diminution of the force of the impulse against the chest wall.

Not infrequently the degenerative process progresses without symptoms and it is not discovered until an attack of dyspnea or fainting occurs or a paroxysm of angina pectoris proves immediately fatal. Fortunately these prominent and serious warnings usually precede the fatal termination by several months, yet many or nearly all of them may be absent.

As a result of feeble circulation or of venous congestion, or the development of emboli, many other symptoms and signs may appear in individual cases, which if the physician is on his guard, may readily be traced to their true source.

Ingall calls attention to a significant symptom which sometimes occurs in myocarditis, namely, pseudoapoplexy. In this the patient becomes suddenly unconscious and falls, following which there develops paralysis, but the symptoms usually pass off in a few hours or at most in a few days. The well-known Cheyne-Stokes respiration is an occasional early symptom; other symptoms, to be borne in mind are, a comparatively rapid loss of weight in a person who has been inclined to stoutness, pallor, a swollen and congested appearance of the ears and lips, pain in the head, unexplained disturbances of the stomach and bowels. Pain over the aorta following exertion and relieved by rest, dyspnea with muscular weakness, accompanied by changes in frequency and heart rhythm. The significance of these symptoms is increased, especially if the history points to such remote causes of myocarditis as typhoid fever, chronic malaria, scarlet fever, diphtheria, syphilis, gout and alcoholism.

Diagnosis.—We have as yet no positive means of diagnosing the condition of the heart muscle, which often after the most careful examination fails to show signs of disease, will, when it comes to postmortem, show myocardial changes where the heart apparently carried on the circulation completely. The explanation of this is, that the heart was never called upon to perform any work beyond its capacity. Many of these individuals have high blood-pressure and this sign antidates any other positive physical finding. Routine observation of the blood-pressure in mature individuals will furnish valuable aid in recognizing these cases early in their development, since every case of persistently high blood-pressure is potentially, if not actually, a case of myocardial disease. Again there are mild grades of breathlessness or slight oppression in the percordial region, following exertion, which indicates that the heart cannot carry even a small extra load without suffering dilatation. The history and examination of these cases is of great importance, especially in regard to the past accidents as acute infection, including syphilis. The etiologic factors of alcohol, overindulgence at the table, excesses in tobacco, profound mental strain, worry, lack of out-door exercise and chronic intestinal toxemia, must all be considered. The development of cardiac symptoms in such persons in the absence of definite lesions should be looked upon as presumptive evidence of some degree of chronic myocarditis.

Severer grades show dyspnea, cough, pain over the liver, swelling of the feet, scanty urination and fluid in the serous cavities. Examination shows that the heart is dilated, the muscle sounds feeble, the rhythm, gallop or embryocardial in type and the rate intermittent, irregular or both.

The symptoms of myocardial degeneration are frequently mistaken for functional affections, especially when they occur in those who have not yet reached middle life. Symptoms of heart weakness developing in young persons are likely to be functional, but in a person past fifty years of age they are usually the result of some organic change.

The most important differential feature is often brought out by exercise. Exercise has little immediate effect on functional disturbances, but when it increases the pain, dyspnea and cardiac disturbance the myocardium is degenerated. In the study of these cases, one must not neglect to consider symptoms that are referable to other organs, such as the brain, the kidney and the liver. Among the recent developments in the study of organic disease of the heart muscle, several so-called functional tests have been devised, and have been found of great value by many observers.

Graupner's Test.—This is based upon the physiologic fact that a given amount of exercise, such as ten bending movements, or running up a flight of stairs, causes both an acceleration in the pulse rate and a rise in blood-pressure, but the latter does not occur coincidently with the former; or if, as in some cases the pressure does rise first, it fails to rise again after the pulse has returned to normal. It is this secondary rise which indicates a good heart muscle. A not too seriously affected heart may show a rise in blood-pressure immediately after the exertion, but with the slowing of the pulse the pressure will be found to have fallen to a level lower than before the experiment. The sphygmomanometer is required for an accurate demonstration of these changes in pressure, which may be recorded in definite units of measure for future reference and comparison.

Shapiro's Test.—This is based upon the alteration in pulse rate occurring in normal individuals by change of posture from the standing to the recumbent. Normally, the number of pulse beats per minute is from seven to ten less in the recumbent position, but when chronic myocarditis develops this difference tends to disappear, so that in seriously weakened hearts the pulse may be as rapid in the recumbent as in the standing posture.

Cautions.—It is not advisable to apply Graupner's test to patients with excessively high blood-pressure, in those of apoplectic tendency or in those with high-grade arteriosclerosis. The test is unsafe in those with a systolic pressure of 200 mm. or over. In such cases there is danger of ocular or cerebral hemorrhage or acute dilatation of heart.

The test will be difficult if not impossible of application in women unless all tight clothing is removed.

Valvular disease is not necessarily a contra-indication in this test, as the condition of the myocardium seems to be the only important factor, except in aortic regurgitation with high pressure, so that the presence of valvular lesions need not detract from the value of the information obtained by this test.

Treatment.—(For details of measures affecting blood-pressure, see Chapter XIX.) Patients should be cautioned to do nothing that increases dyspnea and to rest immediately whenever shortness of breath occurs. In severe cases the patient should be kept in bed, until the heart has had time to rest and regain some of its lost tone. Excessive effort of all kinds both mental and physical must cease. If the affection follows some acute infectious disease, as pneumonia, or from a chronic infection, or from syphilis, treatment should be first directed to these conditions. If it has resulted from excesses of any kind, these should be at once abandoned. Alcohol in all forms, tobacco and coffee, should for a time at least be prohibited in practically all cases; elimination should be encouraged by the proper use of saline laxatives and diuretics, the skin should be kept clean by warm baths, gentle massage and, when indicated, diaphoretics. The digestive functions should be carefully guarded and a light but nutritious diet outlined. A temporary change in climate is sometimes of great value.

The question of the usefulness or harmfulness of drugs will have to be determined in each individual case. The value of digitalis, depends upon whether its action on the tonus of the heart muscle outweighs its constricting effect on the blood-vessels. Hirschfelder, believes that this advantage outweighs the disadvantage, and conforms to the view of Cloetta.[1]

Probably the safest drug to use in all cases is strychnine or nux vomica and this in combination with digitalis will often be all the direct medication required. At first they can be given in moderate doses but the quantity may be steadily increased, while the effects are carefully watched, until the desired result is obtained, or until the beginning of toxic symptoms develop. Other drugs commonly recommended for strengthening the heart muscles are usually disappointing, although occasionally they may give surprising results and should therefore be tried when other measures fail. Caffeine citrate has somewhat the same action as digitails, but is less reliable. Theobromin may be tried, particularly when caffeine causes insomnia. Spartine sulphate in doses of from 1/2 to 2 gr. three times a day may be found valuable for its effect on heart rhythm and on urinary excretion. According to Ingalls chloroform may be inhaled with perfect safety and great relief, but should be used with great caution, the same applies to morphin. Other heart remedies such as strophanthus, adonis vernalis, and convallaria majalis have proven unreliable and should not be used.

The nitrites are valuable in overcoming the dangers resulting from high pressure, but before medication of this

[1] *Arch. f. Exper. Path. u. Pharma.*, No. 209, 1908.

sort is begun, careful studies of the blood-pressure should be made and only if blood-pressure is found to be high should the vasodilators be tried. When the blood-pressure is high, relaxation of the peripheral circulation may throw the balance in favor of the heart, after which, with rest, elimination and judicious tonic treatment the case may recover. Anginoid pains, cardiac asthma, acute dyspnea, palpitation and arrhythmias sometimes yield more readily to these drugs than to any other emergency medication.

After the subsidence of the acute symptoms, and after the danger period has passed, exercise should be begun. The exercise treatment depends upon the assumption that a properly estimated amount of mild exercise will stimulate the heart, increase its tonus and its output, lower the pulse rate thereby increasing its period of rest. The border-line between improvement and harm from exercise is easily crossed, for a strain which is slight may stimulate the heart to stronger contractions and improve its tonicity, while a strain which is too great even if only in a slight degree will weaken and dilate it. This fact renders exercise treatment and bath treatment of heart disease very dangerous weapons in inexperienced hands. The myocardium may be strengthened first by massage, next by appropriate resistance movements, followed, if improvement warrants it, by moderate walking on the level, and by light gymnastics. The effects of these measures upon the physical signs, particularly the pulse and blood-pressure and upon the subdictive signs will show the rapidity with which increasing exercise is indicated. The CO_2 baths give almost the same effect of strain upon the heart as is produced by exercise, they increase the systolic output and the blood-pressure may or may not be affected. The effect of the Nauheim bath is fatiguing and should be used with great discretion.

CHAPTER XIII

ACUTE INFECTIONS

In the study of infectious diseases, the routine use of the blood-pressure test offers an almost unlimited field of usefulness, which in the light of present knowledge, no physician can afford to neglect. Naturally this test offers little in the way of diagnosis, but for prognosis and as a guide to treatment there is so much positive evidence that it furnishes constant and a most reliable aid.

In acute infections the basis for application of the test is the experimental evidence of the vasomotor relation of collapse. Sajous[1] has brought forward a theory of the relation of the adrenal gland to the dangerously low blood-pressure found in the terminal stages of acute infections, especially in pneumonia and typhoid fever. Sajous quotes Goldzicher who reaches the conclusion that in septicemia the appearance of low blood-pressure is to be ascribed to insufficiency of the adrenals. This relation if found to be the true explanation when generally recognized may yield a rich harvest of recoveries.

In the study of infectious diseases, single observations are valueless because of the lack of normal figures for comparison. Careful daily observations should be made and if the pressure tends toward a dangerous hypotension, the periods of observation should be shortened to meet the requirement. These should be carefully recorded on a

[1] *Monthly Cyclo. Pract. Med.*, Dec., 1911.

suitable chart (see page 37) as are the pulse and temperature. The combined chart referred to will be found practical for this purpose.

Gibson predicts that the use of the blood-pressure test will be the guide for treatment in all infectious diseases.

Pneumonia.—There is no uniformity in the blood-pressure findings in pneumonia, some observers finding hypotension and others hypertension. The truth of the matter is probably that the pressure varies with the degree of the toxemia and with the gravity of the case. The following statement of Gibson[1] is significant; it offers a very valuable method in prognosis and shows the way for an improvement in the therapeutics of this disease. He says "Where arterial pressure expressed in millimeters of mercury does not fall below the pulse rate expressed in beats per minute, the fact may be taken as of excellent augury, while the converse is equally true." This observation has been confirmed by G. A. Gordon[2] who states that in no case out of his series was there a fatal result when the blood-pressure kept above pulse rate. In fifteen cases only one recovery occurred when the pulse fell below.

Hare[3] also corroborates this assertion.

This lowering of the pressure is probably due to a toxic vasomotor paralysis of the splanchnics. Forchheimer[4] says that with a healthy heart vasomotor paralysis is the most common cause of death in pneumonia, and that this mode of death may occur irrespective of health and disease of the heart. The first manifestation of this con-

[1] *Edinburgh Med. Jour.*, January, 1908

[2] *Edinburgh Med. Jour.*, January, 1910.

[3] *Therapeutic Gazette*, June, 1910.

[4] *Jour. Amer. Med. Assoc.*, October 30, 1909.

dition is a lowering of the blood-pressure with an increased rapidity of the heart, and under such conditions it becomes necessary for measures to be taken to increase pressure. (See Chapter XVIII.)

Typhoid Fever.—Daily estimations of blood-pressure are an absolute necessity to the proper and intelligent conduct of a case of typhoid fever and a chart should be carefully prepared and followed. In the absence of pre-existing cardiovascular or renal complications, typhoid fever if uncomplicated, is always accompanied by low pressure, due to the effect of the bacterial toxins causing vasomotor paresis of the splanchnics. This hypotension is slowly and regularly progressive with the development of the toxemia and gives us an exact indication for the use of stimulants. From the end of the first week of the disease, the pressure commences a gradual fall which continues usually until the establishment of convalescence, unless complications should intervene (see below). The pressure is usually below 100 and may often fall to 90 or 85; at the same time the diastolic will be lower, but usually not in proportion to the systolic depression; the pulse pressure is diminished.

Perforation.—Crile[1] and Cook and Briggs[2] note that in typhoid fever with perforation and peritonitis, there is an early and decided rise, which is followed by a fall as toxemia increases. This was found to be the invariable rule by Crile in twenty surgical patients.

Hemorrhage.—There is a rapid fall in blood-pressure without the initial rise, by which fact it may be separated

[1] *Jour. A. M. A.*, May 9, 1905.
[2] *Johns Hopkins Hos. Rep.*, Vol. XI, 1903.

from the preceding. The degree and rapidity of the fall in some measure indicates the extent of the hemorrhage. The pressure tends, upon the arrest of hemorrhage, to return rapidly to almost the level noted before the hemorrhage occurred.

Pneumonia is another complication in which there has occasionally been noted a rise in pressure.

Joseph H. Barach,[1] in discussing the significance and value of the blood-pressure test in typhoid fever, arrives at the following conclusions.

1. It has shown us that the blood-pressure falls below the normal after the patient has taken to bed and stays down until convalescence is established, and then returns toward normal.

2. That typhoid fever is a disease with a blood-pressure below 100.

3. That the blood-pressure is governed by factors of its own and bears no constant relation to pulse rate or temperature.

4. That in prognosis the blood-pressure chart is of value. A steadily falling pressure means great danger; as long as the blood-pressure keeps up to a reasonable level, we may feel that there is reserve pressure to work with.

Diphtheria.—The effect of the diphtheria toxin upon muscular tissue throughout the body, and upon the heart muscle in particular, has long been a grave concern of the practicing physician, heart death after diphtheria being an all too frequent sequelæ. The routine estimation of blood-pressure therefore becomes an important prognostic measure, particularly in this disease.

[1] *Penna. Med. Jour.*, July, 1907.

As in other infections, the blood-pressure tends toward subnormal during invasion, with a gradual return toward normal during convalescence.

From a clinical study of 179 cases of diphtheria Rolleston[1] found a subnormal pressure in sixty-three cases or 35 per cent., the extent and duration bearing a direct relation to the severity of the faucial attack. The highest readings were found during the first and the lowest during the second week. The normal tension was usually reestablished by the seventh week. Evidence of dyspnea (partial asphyxia) in laryngeal cases caused an elevation in pressure. Tracheotomy in these cases was followed by an immediate fall of 20 to 40 mm. The effect of serum administration was a rise in pressure in 40 per cent. of cases. Albuminuria did not cause a rise in pressure, except in one case with uremia.

In studying the relation of blood-pressure in diphtheria to myocardial alterations Bruchner[2] examined critically 200 cases of this disease. He found that mild cardiac involvement did not affect the normal blood-pressure curve, that cases with irregular blood-pressure showed various clinical pictures. Every case of marked fall in pressure was associated with definite signs of myocarditis. Falls as much as 50 mm. (Gärtner's Tonometer) appeared only with severe myocarditis. This was the greatest drop in which recovery occurred. A steady progressive fall in pressure was present in the fatal cases. In every case, with one exception the marked falls in pressure were accompanied simultaneously by signs of cardiac involve-

[1] J. D. Rolleston, *Brit. Jour. of Children's Diseases*, October, 1911.
[2] *Deutsche med. Wochen.*, Oct. 28, 1909.

ment; in one case only did the fall precede the clinical signs.

Scarlet Fever.—The blood-pressure shows a moderate rise at the onset of the disease and thereafter closely follows the pulse and temperature curve. After the seventh or eighth day the pressure may be below normal. Complications have a marked effect upon blood-pressure. Cases showing albuminuria generally show hypertension. This rise in pressure is accompanied by slowing of the heart's action. With the subsidence of the kidney irritation the pulse rate increases and the blood-pressure returns to normal.[1]

In acute nephritis secondary to scarlet fever there is practically always a marked rise in arterial pressure. Butterman[2] has observed a rise of more than 50 mm. within twenty-four hours after the development of an acute nephritis.

Other Acute Infections.—In the other acute infectious diseases there is little to state that is of practical importance regarding the blood-pressure, because many of them are so mild as to have no appreciable effect upon arterial tension, and also because observations as far as they have been made, shed very little light. In general it may be stated that the development of toxemia from any cause, results in depression of the normal pressure curve which tends to return to normal with relief from the toxemia.

Therapeutics.—Apart from the usual treatment employed in the conduct of a case of any particular acute in-

[1] J. Davidson, *Lancet*, Oct. 19, 1907.
[2] *Arch. f. klin. Med.*, Vol. LXXIV, p. 11.

fection, certain measures have been found of value in directly combating a dangerously falling blood-pressure.

In typhoid fever it will be noted that the bath treatment or one of its several modifications has a marked and beneficial effect on falling blood-pressure and when it acts favorably should be employed for this purpose, apart from the necessity occasioned by the height of the temperature.

At the present writing, students of clinical medicine do not place much dependence in such measures as injection of strychnin, digitalis, or alcohol, for critical studies seem to show these to be without effect. (See Chapter XVIII.)

Adrenalin or epinephrin and pituitary extract now possess the field and are strongly advocated by many observers, among whom may be mentioned Sajous, Goldzicher, Gibson[1] and Brown.[2]

Sajous[3] states that the adrenals show a special susceptibility to certain infections and that the treatment of low tension resulting from this condition (hypoadrenia) gives some surprising results.

The dosage and methods of administration as laid down by different observers are quite elastic. On an examining of literature we find the following are suggested.

Gibson advocates hypodermoclysis or intravenous transfusion of large amounts of normal saline (large amounts are not more efficient than small,—author) a pint of normal saline to which 1 c.c. of 1–1000 adrenalin solution has been added, to be repeated as required. Others recommend the hypodermatic administration of 10 to 24 minims adrenalin

[1] *Loc. cit.*
[2] *Am. Med.*, Vol. VI, No. 50, p. 563, *et seq.*
[3] *Monthly Cyclo.*, Dec., 1911.

in emergency and small repeated doses, as a routine. In average cases the glandular suprarenalis sicca of the U. S. P. is advised.

Brown[1] advises the intramuscular injection of epinephrin as a means of sustaining the peripheral circulation in adynamia with hypotension occurring during the course of infections. Brown warns against administration by the mouth as uncertain, and shows by experiment upon a series of patients of between twenty and fifty years whose arteries still had the power of contraction and dilatation. All these patients were given 15 minims every twenty minutes for four hours, the blood-pressure being taken every hour thereafter until it returned to the low point. He found that the pressure was maintained above the point recorded before injection for about four hours, and that the rise after injection (intramuscular) was noticeable in fifteen minutes after the injection—and the maximum rise was recorded in from one and three-fourths to two hours from the time of the first injection. A second series of doses given to some of these patients resulted in a second rise, sustained for about the same length of time, thus demonstrating that the blood-pressure could be maintained for a considerable time if it should become necessary.

The bulk of evidence sustains the value of this drug especially any method which allows the drug to be administered gradually enough to have a sustained action. Thus very gradual continuous administration, as by the addition of 1 or 2 c.c. in the Murphy method, or the intramuscular methods of administration.

[1] *Loc. cit.*

L. Rinon and De Sille[1] advocate the administration of 1/5 gr. pituitary extract, as required, as extremely efficient in counteracting depressed arterial tension, producing diuresis and greatly improving the general condition.

Cholera is a disease of subnormal blood-pressure probably giving the lowest pressure readings found in any of the infectious diseases. Leonard Rogers[2] looks upon a pressure below 70 mm. systolic, in man and a little lower in women, as an indication for measures to combat the dangerous fall of blood-pressure. He believes the blood-pressure test a valuable guide in the treatment of the stages of collapse and in combating post-choleraic uremia—as before the use of this test the mortality from this cause was 13.2 per cent. whereas afterward it was reduced to 6.9 per cent.

Cerebrospinal Meningitis.—Abram Sophian,[3] in a study of an epidemic in Dallas, Texas, in the winter of 1911–12, first employed the blood-pressure test as a routine in this disease. This idea developed from the fact that a previous study of the cerebrospinal pressure made at the time of lumbar puncture failed to give the required information, which would increase the safety of fluid removal, and be a guide to the injection of antimeningococcic serum. In a study of 200 cases with nearly 700 blood-pressure observations, he found almost constant results in blood-pressure change on injecting serum, and recommends the employment of this knowledge as a guide to the amount of fluid to be withdrawn and also to the quantity of serum that can safely be given.

[1] Quoted by Sajous. *Loc. cit.*
[2] *Therapeutic Gazette*, Nov. 15, 1909.
[3] *Jour. A. M. A.*, Vol. VIII, No. 12, p. 843.

The withdrawal of fluid does not have a uniform effect on blood-pressure in meningitis, usually there is a fall in pressure (about 10 mm. in adults, 5 mm. in children occasionally it is greater) depending to some extent upon the quantity of fluid withdrawn.

The suddenness of the fall is also an indication of the amount of fluid to be removed, and the speed with which this can safely be accomplished. In cases where there is no change in blood-pressure, as much fluid as possible may be allowed to escape, until the normal cerebrospinal pressure is reached (roughly estimated, one drop escaping from the needle in every three to five seconds).

As a rule as soon as the injection of fluid is begun, the blood-pressure begins to fall and falls steadily (a rise in pressure is rare). After the pressure has dropped considerably, say 20 to 30 mm., the fall in pressure becomes relatively faster if more fluid is injected. This is a danger signal. Sophian finds that a total drop of 20 mm. in an adult with an average blood-pressure of 110 to 120 mm. is a safe indication to stop injection.

He also found that the previous use of adrenalin tends to prevent the fall in pressure, but prefers not to use this drug except in cases with an initial low pressure.

G. Canby Robinson[1] finds that when the symptoms are severe the blood-pressure tends to be high. During convalescence and when the symptoms are mild the blood-pressure is low. The blood-pressure seems to bear some relationship to the severity of the disease and should be a valuable guide in prognosis as well as in treatment.

[1] *Arch. Int. Med.*, May 6, 1910.

CHAPTER XIV

CHRONIC INFECTIONS

Tuberculosis.—The value of the blood-pressure test as an aid to the early diagnosis of pulmonary tuberculosis will be more apparent as the application of the sphygmomanometer becomes more universal.

The blood-pressure is uniformly subnormal in pulmonary tuberculosis and often also in tubercular infections of other regions.

Lauder Brunton has noted the constant relation of low blood-pressure to pulmonary tuberculosis, and believes that hypotension may be a guiding sign before any physical sign is present in the lungs.

Cook in this connection makes the following significant statement: "When low blood-pressure is persistently found in individuals or in families, it should put us on our guard for tuberculosis." Many cases of so-called idiopathic low blood-pressure have later developed the importance of these observations, by showing after a longer or shorter time signs of pulmonary involvement.

A complete study was made by Haven Emerson[1] on the status of the blood-pressure test in tuberculosis. He says that hypotension is universally found in advanced cases of pulmonary tuberculosis. Hypotension is found

[1] *Arch. Int. Med.*, April, 1911.

in almost all cases of moderately advanced pulmonary tuberculosis, or in early cases in which toxemia is marked, except when arteriosclerosis, the so-called arthritic or gouty diathesis, chronic nephritis or diabetes (doubtful if uncomplicated, author) complicate the tuberculosis and bring about a normal pressure or a hypertension. Occasionally a short period of hypertension may precede or accompany hemoptysis in a patient ordinarily showing hypotension.

Emerson emphasizes that hypotension should be sought for in subjects just as carefully as it is the custom to search for pulmonary signs. Hypotension when it is present in tuberculosis increases with extension of the process. Recovery from hypotension accompanies arrest or improvement. Return to normal pressure is commonly found in those who are cured; continuation of hypotension seems never to accompany improvement.

Prognosis.—Emerson believes prognosis can be as safely based on alterations in blood-pressure as on changes in the pulse or temperature.

My own observations as far as they have gone, show that a study of diastolic blood-pressure in tuberculosis or in tuberculous suspects is equally as important as the systolic pressure reading, for we find that, provided other reasons for the change can be eliminated, a slightly lowered blood-pressure which is persistent, combined with a reduced pulse pressure is very suggestive of the effect of the toxemia from tuberculous infection. Also the relation of the pulse pressure to rest or to exercise in cases of pulmonary tuberculosis, is a most valuable guide to the amount of exertion that may be safely permitted.

A valuable and most complete study of the effect of exercise upon tuberculous patients has been made by L. S. Peters and E. S. Bullock.[1] A definite plan was outlined and careful study and accurate records made. Six men were used. Three were excellent cases both pulmonarily and physically, two fairly arrested far advanced cases; and one a new recruit, with normal temperature, but poor physical condition. The points in this report are so well taken and the table shows so graphically the results obtained, that they are copied here in full.

"All were started with a fifteen minute's walk the first day. The pressures on starting of the three able-bodied men were 138, 132 and 148 respectively. On their return the pressures were 138, 144 and 153. After an hour's rest the readings were 138, 142 and 158, showing that apparently the exercise was not harmful. The two fairly well arrested, far advanced cases, started out with pressures of 164 and 124, returning with 146 and 130, and after resting 164 and 118. The first man was not used to any exercise in any form, as is well shown in a drop of 18 mm. Hg., with a return to the original after an hour's rest. The overexertion in the second man is evident, for we find after resting that there is a drop of 6 mm. from the original reading recorded after the return from exercise."

"The new recruit started with 146, returned with 138 and after rest his reading was 127. The overexertion in this instance is well illustrated from the pressure findings and was further substantiated by the marked fatigue, breathlessness, and rapid heart action of the individual himself. This experiment was carried on for a period of six days,

[1] *Med. Rec.*, Sept. 14, 1912, p. 463.

each day's exercise being graded by the previous day's results in blood-pressure. The table of these findings, which we here append, will show at a glance that we are able to control the readings by an increase, a decrease, or a repetition of the exercise. Whenever a man showed a drop of 6 or more mm. Hg. after rest or a marked drop on returning, even though this disappeared after resting, we decreased the exercise. If there was a slight drop after returning we repeated the same exercise the following day or until we maintained an even standard, when the walk was increased. It is interesting to note that in one of the three excellent cases the pressure remained practically the same even up to walks of one and a half hours, and later this same man took walks of two hours in the morning and two in the afternoon with no change in pressure and no evil results. The other two after a few repetitions were able to do the same."

Improvement in subjective symptoms follows the effect of blood-pressure elevation, and persists if the pressure can be maintained at a higher level than that existing before such treatment.

Case No.	Blood-pressure			Pulse			Remarks
	Before	After	After rest	Before	After	After rest	
1	138	138	138	....	......	......	1st day. 15 minute walk.
	138	141	150	92	98	80	2nd day. increased to 30 minutes.
	140	139	150	98	92	76	3rd day. Increased to 45 minutes.
	142	140	140	96	106	80	4th day. Increased to 1 hour.
	140	142	138	88	88	76	5th day. Increased to 1 hour, 15 minutes.
	142	138	140	88	88	76	6th day. Increased to 1 hour, 30 minutes.

Case No.	Blood-pressure			Pulse			Remarks
	Before	After	After rest	Before	After	After rest	
2	132	144	142	......	......	......	1st day. 15 minute walk.
	126	122	127	74	80	......	2nd day. Increased to 30 minutes.
	126	135	126	66	76	68	3rd day. Increased to 45 minutes.
	132	134	116	78	80	84	4th day. Increased to 1 hour.
	126	130	126	70	80	72	5th day. Cut to 45 minutes.
	126	124	136	73	76	75	6th day. Repeated 1 hour walk.
3	146	138	127	......	......	......	1st day. 15 minute walk.
	142	124	132	120	120	100	2nd day. Repeated 15 minute walk at slower pace.
	140	145	130	120	118	96	3rd day. Cut to 10 minutes at slow pace.
	132	145	142	120	120	112	4th day. Repeated 10 minute walk.
	142	150	140	116	120	108	5th day. Repeated 10 minute walk.
	138	146	142	118	120	104	6th day. Increased to 15 minutes.
4	148	152	158	......	......	......	1st day. 15 minute walk.
	146	146	135	88	84	72	2nd day. Increased to 30 minutes.
	145	142	146	80	80	72	3rd day. Repeated 30 minute walk.
	150	140	142	80	100	72	4th day. Increased to 45 minutes.
	145	140	158	84	80	76	5th day. Increased to 1 hour.
	140	142	138	76	80	80	6th day. Increased to 1 hour, 30 minutes.
							This man unused to exercise.
5	164	146	164	......	......	......	1st day. 15 minute walk.
	146	134	144	118	120	106	2nd day. Cut to 10 minutes.
	140	150	138	100	104	100	3rd day. 10 minute walk.
	146	146	138	90	98	110	4th day. Increased to 15 minutes.
	144	144	152	110	100	100	5th day. Repeated 15 minute walk.
	138	144	146	104	104	92	6th day. Increased to 25 minutes.
6	124	130	118	......	......	......	1st day. 15 minute walk.
	115	114	110	100	120	108	2nd day. Repeated 15 minute walk.
	122	117	118	100	100	84	3rd day. Increased to 20 minutes.
	115	110	115	100	110	100	4th day. Increased to 30 minutes.
	122	110	112	88	110	96	5th day. Increased to 45 minutes.
	115	114	114	96	96	88	6th day. Decreased to 30 minutes.

Syphilis.—As a primary cause of arteriosclerosis, syphilis is too well known to demand more than passing consideration here. In any history this disease should never be overlooked, but should be given due consideration as a predisposing cause in the production of those lesions of the cardiovascular and renal systems that are associated with hypertension. It is believed by many that a moderately

BLOOD PRESSURE CHART

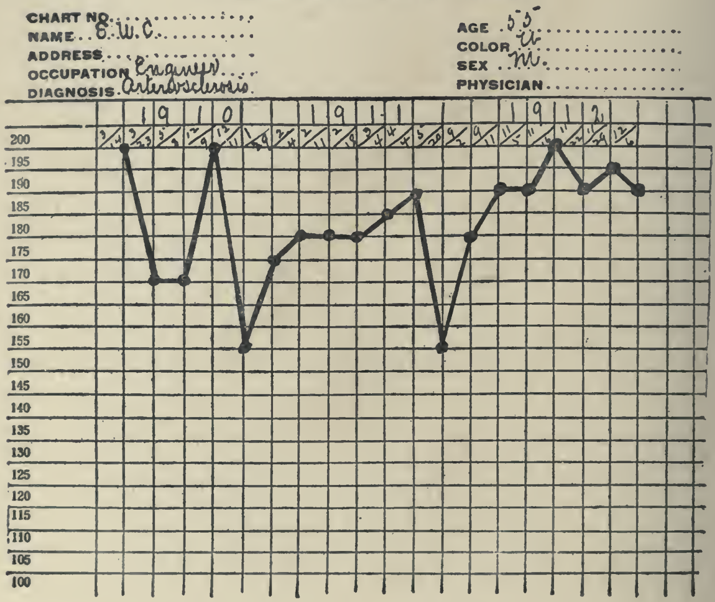

FIG. 32.—Arteriosclerosis, probably of syphilitic origin. Patient very stout. Symptoms: complains of dizziness, dyspnea, physical weakness; urine shows trace of albumin, low specific gravity, few granular cases. Potassium iodid in small dose effected first reduction to 170 with a rise following cessation of treatment. Second fall followed use of potassium iodid, purging and baths. Baths and weekly purge continued till April 4, 1911. Third fall followed administration of mercury and iodid, plus baths. Subsequent treatment is having less effect on pressure level, but symptomatic result continues good.

high blood-pressure which can be traced to a previous syphilitic infection is more amenable to treatment and gives more satisfactory results than continued high pressure from other causes. This, however, remains to be proven. In the meantime, antisyphilitic treatment, particularly the use of the iodids, should be vigorously carried out.

In this connection it may be of interest to note that in a small number of cases examined before and after the administration of Salvarsan ("606") by the author, very little if any effect from the injection of the drug was noted.

During the early acute stages syphilis acts like any other general infection, in that it is usually accompanied by a moderate reduction in blood-pressure with some narrowing of the limits of pulse pressure. These changes are, as a general rule, so slight that they need not be considered in the care of the case; therefore, from a practical standpoint, the blood-pressure in acute syphilitic infection is of little value. (See Fig. 32.)

CHAPTER XV

RELATION OF BLOOD-PRESSURE TO METABOLIC AND MISCELLANEOUS DISEASES

The diseases found in this group have been so placed because, while the blood-pressure findings are of no special value in diagnosis, except as they relate to complications of the heart, arteries and kidneys, they are nevertheless of some assistance in arriving at the proper prognosis, and in guiding treatment. The test should therefore be employed in them.

Addison's Disease.—Several years ago, Janeway reported two cases of unquestioned Addison's disease, in which the systolic pressure tended downward. Recently Gibson[1] reports very low pressures in his series of cases. Two cases seen by me showed a marked degree of hypertension. A. Randal Short ("New Physiology") discusses the subject from the physiologic standpoint, and shows that if the suprarenal veins are clamped for a few hours, thereby preventing the entrance of adrenalin secretion into the circulation, the blood-pressure rapidly falls. As the pathology of Addison's disease involves a degenerative process of the adrenal glands, we have the probable explanation of the low blood-pressures found in this disease. Improvement in subjective symptoms follow the effect of blood-pressure elevation, and persist if the pressure can be maintained at a higher level than that existing before such treatment.

[1] *British Med. Jour.*, Dec. 10, 1910.

Aviation Sickness.—In the *Medical Press and Circular* for August, 1911, reference is made to a communication by Crouchet and Moulinier to the French Academy of Sciences, in which they report their observations upon a number of aviators. They note two varieties of trouble resulting from flights in aeroplanes. The first is due to the altitude attained, and depends on differences in atmospheric pressure, in temperature, and changes in the chemical composition of the air found at high altitudes.

The second factor, which need not be discussed here, is the actual physical effort put forth. They consider the rapidity of ascent and of descent as most important, and recommend a reduction in the speed at which these changes should take place. The effect of ascent begins to be shown when a height of 1,500 (4,500 ft.) meters is reached, which causes quick, short respiration and tachycardia. There is usually a sensation of headache and moderate deafness. During descent there is a sensation of discomfort like that which accompanies a sudden descent in an elevator, violent palpitation and great noise in the ears. On landing the aviator is not free from the above sensations for a considerable time. Respiration quickly returns to normal but arterial hypotension which they found in most cases to be quite marked, persists for a long time after the flight is finished.

Auricular Fibrillation.[1]—The readings taken by Silberberg from eight patients, all of whom were typical examples of cardiac irregularity due to auricular fibrillation, show the wide range of blood-pressure which the individual case of auricular fibrillation may possess. The blood-pressure

[1] M. D. Silberberg, *Bos. Med. and Surg. Jour.*, April 6, 1912.

reading of smallest beats varied from 80 to 160; maximum blood-pressure ran from 100 to 210. This variation Silberberg says is of importance, because in the cases in which it occurs, single observations of blood-pressure taken in the ordinary manner, possess only a restricted value as an index, and the error introduced is continued if comparative observations are confined to a reading of the most forcible beats only.

Cardiac Asthma and Pulmonary Edema.—The frequently occurring attacks of dyspnea found in heart and kidney cases are usually accompanied by hypertension. In their most severe form, true edema of the lungs develops. In this connection Amblard[1] states that recent experimental research is amply confirmed by clinical findings, and that we may accept it as a fact that in high pressure cases, further elevation in blood-pressure, due to insufficiency of the left ventricle usually precedes attacks of acute pulmonary edema, therefore careful attention to the blood-pressure, both systolic and diastolic, as the means of determining an increase in pressure, or a functional failure of the heart, would direct attention to the need of immediate reduction in the maximal arterial pressure.

Cerebral Hemorrhage.—Large hemorrhages into the brain case when accompanied by symptoms of general compression, slow pulse, coma and altered breathing are accompanied by hypertension, which bears a direct relation to the amount of increased intracranial tension. It is of great importance when examining an unconscious patient to know the origin of the coma and the gravity of the case. No single piece of evidence is as clear or as re-

[1] *Presse Médicale,* August 12, Vol. XIX, No. 64.

liable as the systolic pressure. If this is extreme (between 200 or 300 mm.) the brain is undergoing dangerous compression and danger to life is imminent. In cases where progressive increments of hemorrhage are suspected, nothing can be more valuable than frequently repeated blood-pressure tests, which, by demonstrating a progressively rising pressure, would indicate a rapidly extending area of cerebral compression. So employed the blood-pressure test is a safe guide to the need and urgency of decompression, whereas on the other hand, a stationary or falling pressure without increase of symptoms, such an operation need not be considered. Reports seem to show that hemorrhage in the anterior fossa of the cranium have least effect on blood-pressure, and those into the posterior the most.

In cerebral hemorrhage, Norris has reported, a systolic pressure as high as 400 mm.

Differential Diagnosis.—Many authors, among them Jump,[1] point out the fact that in both cerebral hemorrhage and in apoplectic coma, the marked hypertension occurring in these two conditions, would serve to distinguish them from embolism, in which the blood-pressure is low, and further that a gradual increase in intracranial tension, such as would be caused by a slow-growing brain tumor, has little or no effect on general blood-pressure.

Cheyne-Stokes Respiration.—Pollock[2] reports a series of blood-pressure estimations in fifteen cases of Cheyne-Stokes respiration arising from various causes, which confirm the earlier observations of Cushing that in Cheyne-Stokes respiration with increased endocranial tension, the

[1] *International Clinics*, Vol. I, p. 49, 21 series.
[2] *Archives of Internal Medicine*, Vol. IX, No. 4, 1912.

blood-pressure is low during the period of apnea and high during that of hyperpnea, as well as the demonstration by Eysner that this fact is of clinical value in the differentiation of Cheyne-Stokes respiration with increased endocranial tension from other types. In the cases with increased tension, the blood-pressure began to rise slightly before respiration commenced and began to fall after the summit of respiratory activity was reached, whereas in the other cases, the pressure began to fall after the beginning of respiration and rose as respiration diminished.

General Paresis.—Communications on the subject of blood-pressure in this disease are few, and insufficient in number to produce reliable statistics, although with few exceptions, they point to a moderate hypotension in this disease. The best clinical report that I have been able to find is by A. Schmigergeld from studies made at Ward's Island, N. Y.,[1] who arrives at the following conclusions:

1. The blood-pressure in general paresis is very variable.
2. In the majority of cases it seems lower than normal.
3. There exists no relation between the mood of the paretic tone and the state of the tension.

Lead Poisoning.—The effect of chronic lead intoxication frequently results in permanent changes in the arteries and kidneys, resulting in a secondary hypertension. There is, however, a form of hypertension occurring in lead poisoning, as evidenced by the typical colic, which is always accompanied by a moderate elevation in blood-pressure, which may remain elevated for several days, succeeding the attack. (A primary hypertension.)

[1] *New York Medical Journal*, August 28, 1909.

With the knowledge of exposure to lead, followed by an attack of pain with high blood-pressure, we may be aided in difficult cases by the blood-pressure test to separate lead colic from renal and hepatic colic, in which the blood-pressure is low.

Momburg Constriction.—Dr. Fred L. Adair[1] has studied twenty-three cases in an effort to determine the effect of abdominal constriction by the Momburg tube on blood-pressure, pulse, etc. Cases showing abnormalities of the heart, blood-vessels or kidneys were excluded and all observations were made in the supine posture without anesthesia. While of necessity the duration of application was short, the femoral pulse was always obliterated. The detail findings of this series are shown in the table on page 194, and correspond in general with the results of earlier observers, notably Wolff. The most dangerous period appears to be when the tube is removed, and this is most dangerous in those presenting arterial change, cardiac disease, anemia and vasomotor instability.

Neurasthenia.—(See Hypotension, Chap. VIII.) Neurasthenia or the fatigue neurosis resulting from lack of nervous energy and instability of the sympathetic nervous system is naturally, when uncomplicated, accompanied by hypotension. We may include under this head the psychic instability of blood-pressure, so beautifully discussed by Schrump[2] where he shows that before we may arrive at a decision that a low blood-pressure is pathologic, we must make sure that it is not psychogenic. He also makes the interesting statement, that a rise in pressure of psychogenic

[1] *Surg., Gyn. and Obst.*, 1912, p. 112.
[2] *Deutsch. med. Wochen.*, Dec. 22, 1910.

origin affects chiefly the systolic pressure; as the mind does not seem to have an influence upon the diastolic pressure, which is unaltered. Psychic instability is almost constantly present, in all individuals to some degree, but is much more marked in the neuropath. It is sometimes difficult to determine by one examination a normal from a pathologic alteration in blood-pressure, and it may become necessary to divert the patient's attention and to repeat the test at a subsequent time. Furthermore, it must not be overlooked that the period of absolute rest which usually begins the treatment of grave neurasthenia, is itself a cause for a lower blood-pressure. The degree to which the pressure falls in this condition depends somewhat upon the gravity of the disease and the temperament of the patient but is usually moderate.

I have been unable to find any reference to a hypotension lower than 80 mm. systolic in neurasthenia.

The treatment of this disease when successful may be indicated by a gradual return of the pressure to normal. It must be borne in mind that complicating nephritis may so affect the blood-pressure, as to render the findings of no value.

Prolonged Epistaxis Associated with Increased Vascular Tension.—Harold Hays[1] notes the frequent association of prolonged and profuse epistaxis to high blood-pressure and has found this condition usually associated with two classes of circulatory disease.

1. Arteriosclerosis involving the arterial system and the myocardium.
2. Valvular disease, or congenital deformity of the heart.

[1] *N. Y. Med. Jour.*, March 4, 1911.

In this first group, the epistaxis seems to be the direct result of the high arterial tension, and is both a warning sign of impending apoplexy, and a beneficial act on the part of nature to relieve a dangerously high blood-pressure. This fact should lead to inquiry into the state of the circulation, particularly in all persons of advancing years, who show a tendency to epistaxis, especially if uncontrollable by the usual means. Relief from both the loss of blood and the danger attending a markedly elevated pressure may best be accomplished by measures directed toward controlling the hypertension. This in Hays experience is best accomplished in emergency by large doses of morphia.

Renal and Biliary Colic.—Abdominal pain accompanying these two conditions has no effect upon blood-pressure, unless obscured by a complicating nephritis. This fact should help to differentiate them from tabes and from lead colic, both of which give a marked hypertension.

Shock.—(See Surgery, Chapter XVI.)

Tabes Dorsalis.—Lewellys F. Barker recently reported some cases of this disease in which the blood-pressure varied between 190 and 215 mm. Hg. Other authors have had similar experience, noting the rise usually during the paroxysm of abdominal pain. Jump[1] calls attention to this important differential point, that while with abdominal pain in gastric crises of tabes the blood-pressure is nearly always markedly elevated, it is usually low or normal in renal or biliary colic.

[1] *International Clinics.* Vol. I, Series 21, p. 49.

Adair's Table of Pulse and Blood-pressure (Momburg Constriction)

Case	Pulse					Blood-pressure				
	Before	During		After		Before	During		After	
		Maximum	Minimum	Maximum	Minimum		Maximum	Minimum	Maximum	Minimum
1	95	97	97	88	88	116–118	132	122	118	112
2	104	103	103	106	106	144	148	90	144	124
3	86	96	96	94	94	114–116	114	114	116	116
4	84	120	110	72	72	122	126	98	128	118
5	86	104	98	92	84	146	154	138	146	130
6	86	78	72	92	74	128	166	138	128	110
7	88–102	122	116	98	98	144	148	111	140	140
8	98–120	136	120	96	88	124	110	100	124	110
9	96	104	80	120	80	120	140	120	120	96
10	88	92	88	72	72	126	144	126	122	120
11	84	120	92	88	84	124	136	130	126	92
12	86–104	160	116	135	90	125	175	154	128	100
13	66	120	72	112	56	106	136	128	106	70
14	64	84	58	96	60	110	128	122	124	90
15	70	116	64	104	56	116–118	184	158	122	110
16	84–88	120	100	128	80	118	156	102	136	114
17	72	132	110	88	64	120	166	160	150	120
18	64	112	96	88	64	110	120	114	124	116
19	80	96	68	?	?	110	116	108	?	?
20	76	104	76	100	64	107	115	104	?	?
21	98	132	114	88	72	136	182	157	128	116
22	70	92	84	84	60	110–112	126	115	114	110
23	82	92	84	84	72	138–140	166	156	132	124

Thoracic Aneurysm.—In thoracic aneurysm the *pulsus differens* may be definitely determined by the blood-pressure test, taken upon both arms. When taken by the finger one may be greatly mislead by the apparent findings. As an example of this, in one case of undoubted aneurysm of the last third of the arch the left radial seemed distinctly smaller than the right, and the signs and radiograph showed an aneurysm located apparently so as to interfere with the flow of blood through the left subclavian, but the sphygmomanometer showed an average of 5 mm. higher on the left side and an autopsy showed the sac just below the subclavian.

In the differential diagnosis between thoracic aneurysm and dilatation of the arch of the aorta, O. K. Williamson[1] says the latter shows a greater increase in blood-pressure than the former, and if the difference in pressure in two arms is 30 mm. or more, it speaks strongly for aneurysm. Between aneurysm and mediastinal tumor a difference between the two sides of 20 mm. or more indicates aneurysm. While these reports as far as I know, have not been confirmed, and as I have had no experience in the matter, they must be taken with some question, but may prove of value in aiding the elucidation of difficult cases.

[1] *Lancet*, Nov. 30, 1907.

CHAPTER XVI

BLOOD-PRESSURE IN SURGERY

I can introduce this subject in no better manner than by presenting the following extract from a recent article by Joseph C. Bloodgood[1] of Baltimore, whose powers of observation and accuracy of deduction have made him an authority on surgical pathology.

"In view of the fact that at the present time our scientific methods of accurately estimating the vital resistance of the patient and the factors of safety are to a certain extent so unreliable and the factors themselves so numerous and the problems themselves so complicated, it is my opinion that every patient should be given the benefit of the doubt and prepared for the operation with the greatest care, that the operation be performed under the least dangerous anesthetic, that the manipulations of the operation be made with the least degree of trauma and loss of blood and that the operative treatment be planned to reduce as far as possible any depressant factors and to give the patient the benefit of any improvement in treatment. In general it is my experience that as a rule, certain things are neglected in the majority of cases. These are the more careful investigations of the general condition of the patient—the estimation of the kidney function and the blood-pressure record. The time is fast coming when the individual will expect and demand these more

[1] *Penna. Med. Jour.*, January, 1912, p. 256.

modern, more exact methods of diagnosis. During the last year, I have paid considerable attention to routine blood-pressure records and at the present time, I am getting the impression that the blood-pressure will warn the surgeon of the danger line before the pulse or the respiration. My respect for the blood-pressure record is increasing daily and I would urge all surgeons to use it in extraordinary operations and handicapped patients. But to learn to interpret blood-pressure records one must employ them at all operations as a routine.

When the blood-pressure falls to 100 or lower, it is time to stop the operation and give the saline immediately. One point I wish to make clear which many surgeons do not seem to be familiar with, the patient seems in fair condition at the end of the operation, but no blood-pressure record is taken. He is lifted to the stretcher, carried to his room and when put to bed he is found to be in collapse requiring hurried treatment. This can be avoided in most cases, if after the operation is finished and the bandage adjusted, a blood-pressure record is taken. If this record is much lower than that taken at the beginning of the operation, it is an indication that the patient should not be transported, but kept quiet on the table and given the salt solution by all three methods. It is important therefore to investigate the patient before he is lifted from the table to be transported, and to begin the saline treatment then, if indicated. I am confident that this would prevent many of the cases of collapse or sudden vasomotor shocks which are observed after the patient reaches his bed. The surgeon must be familiar with the manipulations which produce shock. Nothing helps him more to estimate this than the blood-pressure. It is to be remembered that anything that either diminishes or increases the blood-pressure is a stimulation which sooner or later will lead to exhaustion and a fall of the blood-pressure. It is the uniform rate of pulse and respiration and the uniform blood-pressure that indicates an operation with the least degree of shock. During the entire resection of the colon with anastomosis, if done without tension on the vessels and nerves, one will observe very little change in the pulse, respiration or blood-pressure, but the moment one pushes the intestines away to suture the rent in the posterior peritoneum caused by the removal of the colon, the quiet patient moves, the pulse and respiration are more rapid, the blood-pressure rises at once, and if the patient's factors of safety are small, the blood-pressure quickly falls and the patient is in shock."

The danger of any anesthetic depends chiefly upon its effect on the circulation, and examinations with the sphygmomanometer of patients under anesthesia show that the ability to withstand its prolonged administration depends upon the power of the vasomotor and cardiomotor systems to maintain the blood-pressure at or near the normal level. In other words, as long as a fair blood-pressure is maintained during anesthesia, its administration can be safely continued. This, of course, may be modified by the pathologic condition leading up to the operation, and the patient's general physical condition at the time that the anesthetic is administered. The employment of the sphygmomanometer has placed the administration of anesthetics upon a firmer foundation, and has in every way borne out the result of clinical experience, as to the relative safety of the anesthetics commonly employed. To-day the administration of anesthesia, except possibly of the shortest duration, without routine blood-pressure studies lays the surgeon open to censure, if untoward effects follow. Conversely the surgeon who employs the sphygmomanometer protects himself in the event of deaths under anesthesia.

The importance of this study was originally developed and demonstrated by Crile in 1903[1] but only during the past year or two has it become generally accepted. It is safe to say that in the near future, the sphygmomanometer will have almost as wide application by surgeons as it now has by medical men. Blood-pressure observations can usually be made without difficulty by the anesthetist, although the undivided attention of another assistant, as a student or nurse, who can easily be trained to make these observations, should be used when possible. Observations made during surgical operations should occur from two- to five-minute intervals and when necessary an expert assistant can give blood-pressure reports once every minute.

In grave cases the value of the test becomes greater as the interval of observation is shortened for it is possible for serious changes in the circulation to occur in a very short space of time. The observation to be of greatest service should be charted and kept in view of the surgeon. The value of these observations lies not only in the facility with which dangerous alterations in blood-pressure may be detected, but also in the fact that the effect of respiratory and stimulating measures may be noted, so that efficient dosage may be employed. These observations will be further increased in value if the pulse rate is taken at

[1] G. W. Crile, Blood-pressure in Surgery, Philadelphia, 1903.

regular intervals and reported on the same chart with the blood-pressure, for it is known that a falling pressure with a rising pulse rate is an indication for immediate action. In the study of blood-pressure under anesthetics it is necessary to obtain the patient's normal systolic pressure before the anesthesia is begun, and this should if possible be obtained the day before, or at least previous to the patient's final preparation and appearance in the operating-room. Observations made immediately before the anesthesia will frequently show an abnormally high pressure and an accelerated pulse rate. This may be accounted for by the stimulating effects of excitement and fright on the cardiomotor and vasomotor centers.

It must also be borne in mind that the blood-pressure level will be affected by rest in bed, and by restricted diet which usually precedes surgical operations.

From a surgical standpoint, the study of the systolic blood-pressure alone is necessary, because the object of the observation is to follow changes in the vascular tension.

In order to intelligently employ the sphygmomanometer during surgical operations, the surgeon must appreciate the influence of the ordinary steps of surgical procedure, as compared with the extraordinary and dangerous manifestations. As far as reliable information is available in literature the following may be stated: Pain practically always causes a temporary rise in blood-pressure. In abdominal pain in which the splanchnic nerves are involved, the pressure increases greatly on account of constriction of the splanchnic vessels. H. Curschmann[1] believes that by this method we may be able to differentiate between the

[1] *Münch. Med. Woch.*, October 15, 1907.

causes of abdominal pain. He draws the conclusion from certain observations which he made that pain from gastric and intestinal crises in tabes and in lead colic caused the pressure to run up to 170 to 200 mm., to drop again to normal as soon as the pain ceased. In pain from gastric ulcer, gall-stones and appendicitis there was only a very moderate increase. He further made this very interesting observation that pressure rose 8–15 mm. from electric stimulation of the thigh in normal individuals, but if the part stimulated were analgesic, from hysteria or spinal-cord disease, there was no rise. He thinks this indicated the reality of the sensory disturbance in hysteria. The rise from stimulation would serve to distinguish between feigned and pathological conditions.

Influence of Operative Procedures. *Skin Incision.*—All cutting of the skin involves the irritation of peripheral nerves, which according to Janeway reflexly stimulates vasoconstriction, which shows itself by a slight rise in the pressure curve. On the contrary, Lull and Turner,[1] working in the Jefferson Clinic at Philadelphia, found that the skin incision resulted in a fall in blood-pressure and that this was more marked when the patient was but slightly anesthetized. They offer no explanation, but it might easily be explained on the ground that the effect depends upon the character of nerve cut, as it is known that pressor fibers occur in mixed nerves. There is room for more extended observation on this point. Whatever the effect may be, it seldom amounts to more than 10 mm. and therefore does not demand great consideration. Crile from his experience states that a fall occurs when the nerve trunk is

[1] G. F. Lull and C. H. Turner, *Therapeutic Gazette*, 1911, p. 94.

irritated and that serious depression of blood-pressure follows stretching of the sciatic. This he believes is due to reflex dilatation of the splanchnic area. The same investigator has shown that manipulation of serous cavities usually cause a sharp fall which may at times be dangerous. Lull and Turner demonstrate that incision of the peritoneum causes a transitory fall in blood-pressure. In this connection they make a very important suggestion, which if true will considerably modify present surgical custom and teaching. They contend that during operation, involving severence of nerve trunks or their branches, the dangerous fall may be modified and the procedure rendered more safe by withdrawal of the anesthetic at the moment the incision is made. Janeway maintains that incision in the peritoneum usually causes a sharper rise than skin incision, and that subsequently the curve is downward, depending on the extent and duration of the operation and the amount of manipulation and exposure of the viscera.

As might be expected, simple *paracentesis abdomini* causes a fall in pressure, due largely to a release of intra-abdominal pressure, which allows the splanchnic area to become overfilled.

Capps and Lewis[1] noted that almost invariably aspiration of a pleural effusion caused a marked fall in blood-pressure, sometimes to an alarming degree. They concluded that such a marked change is not a simple result of altered intrathoracic pressure, but is caused by two separate reflexes, one a cardio-inhibitory and the other a vasodilator.

Gynecologic Operations.—Reliable observations, as far as

[1] J. A. Capps and D. Lewis, *Am. Jour. Med. Sci.*, Dec., 1908.

I am able to learn agree with the original studies of Crile, which showed that manipulations of the pelvic organs caused a rise in pressure and that this rise was proportionate to the severity of the traumatism. The reports of observers employing chloroform as an anesthetic are unreliable because of the uniform depressing effect of chloroform itself.

Cord and Brain Operations.—Crile states that dural incisions have little or no effect upon blood-pressure curve, but that irritation such as sponging of the spinal or cerebral dura mater causes a sharp fall. Operations for decompression usually cause some reduction in pressure. The amount depending somewhat upon the extent and nature of the operation.

Hemorrhage.—Carl J. Wiggins[1] has found a frequent determination of the pulse pressure, in cases of suspected internal hemorrhage of great value in differentiating this complication from others accompanied by a falling blood-pressure. This author finds that almost uniformly a progressive decrease in pulse pressure and a rising pulse rate after surgical procedures are indicative of continued bleeding, and that the converse if persistent (after several observations) indicates a cessation of hemorrhage.

In all operations control of hemorrhage is an important factor in maintaining blood-pressure. When hemorrhage is slight and well controlled the effect on pressure is usually unimportant and does not call for special treatment. On the other hand, operations accompanied by considerable bleeding may result in severe and dangerous hypotension. The tendency to shock is greatly increased by hypotension

[1] *Arch. Int. Med.*, Sept., 1910.

from any cause during anesthesia, but if shock is successfully combated, pressure soon returns to a safe level.

Influence of Anesthetics on Blood-pressure.—Discussing in the abstract the action of anesthetics, Guy, Goodall and Reid remark that blood-pressure may be lowered by (1) depression of the heart (a) by vagus inhibition, either by direct stimulation of center by the drug, or by reflex stimulation through the nervous system. (b) By weakening of the heart muscle. 2. Dilatation of the vessel wall or paralysis of vasomotor tone. Blood-pressure may be elevated by (1) stimulation of the heart (a) by excitement (b) by stimulation of the heart by the drug (2) stimulation of the vasomotor centers (a) by the action of the drug (b) by asphyxia.

Experiment and clinical study show that the different anesthetics in general use affect the circulation and blood-pressure in different ways, and that the extent of the depressing effect of the anesthetic on blood-pressure determines in a great measure the relative danger of the anesthetic.

In the following paragraphs an effort has been made to indicate the action of different drugs used in the production of anesthesia and to show what blood-pressure changes may be expected to occur under them.

Ether.—The opinion of all observers that ether even in large amounts seldom produces a significant fall in blood-pressure, has recently been confirmed by the careful observations of Guy, Goodall and Reid.[1] Experimental study upon animals shows that very large amounts of ether may be given before any serious effect is produced on the cardio-

[1] Wm. Guy, Alex. Goodall and H. S. Reid, *Edinburgh Med. Jour.*, August, 1911.

motor or vasomotor systems. The earliest and most efficient indicator of approaching danger is a marked fall in blood-pressure. During the administration of ether before the full anesthetic effect is obtained there is a moderate rise due to mental excitement and muscular activity. When the state of full anesthesia is reached the pulse and blood-pressure return to normal level. As the patient comes out of the anesthetic a moderate rise is often observed. The administration of oxygen to hasten the return to consciousness always causes a sharp rise in pressure.

Chloroform.—Almost without exception chloroform causes a reduction in blood-pressure, which may occur suddenly and be dangerous even after small amounts. Chloroform if given in too concentrated form may cause a sudden and severe fall in blood-pressure, from fatal inhibition of the heart by direct stimulation of its inhibitory center (Guy, Goodall and Reid).

Chloroform is dangerous in all stages of its administration, the greatest danger is at the beginning of the administration. Struggling by the patient seems to increase the bad effect.

Nitrous Oxid.—Nitrous oxid, when given alone, usually causes an elevation in blood-pressure, due to the partial asphyxia induced. This rise is not so marked when rebreathing is allowed (Guy, Goodall and Reid) and is almost entirely eliminated when a gallon of oxygen is inhaled first. This is a point of value in cases of essential hypertension. Although the employment of oxygen in this way curtails by a few seconds the available period of anesthesia.

Nitrous Oxid-ether Sequence.—This condition causes a gradual elevation of pressure, until the stage of complete anesthesia is reached, when it has the same effect as outlined under ether anesthesia.

Nitrous Oxid Combined with Oxygen employed for continuous anesthesia as recently advocated and successfully

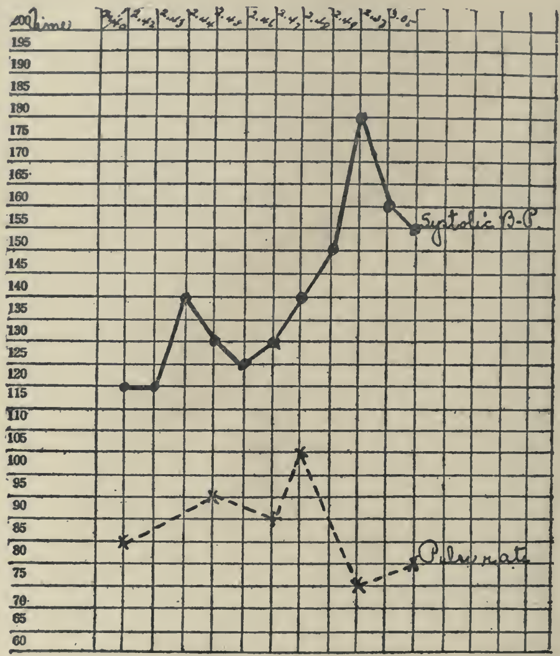

FIG. 33.—Anesthesia chart. Nitrous oxid and oxygen, patient young, adult male, white. Duration of administration, ten minutes. 2.40, Patient in chair; 2.42, anesthesia begun; 2.44, analgesia established; 2.46, complete anesthesia begun; 2.48, root extracted; 2.49 nitrous oxid stopped 50 per cent. oxygen administered; 2.50 patient conscious; 2.05 patient left chair. Attention is directed to primary effect of N_2O, to gradual fall during analgesia and the marked rise immediately following the administration of 50 per cent. oxygen.

practised by Teter and others, produces a primary rise in blood-pressure, which immediately falls to normal, as the state of analgesia is reached. The proper control of the effect by the oxygen allows the pressure to be maintained at normal indefinitely. Any increase in the amount of oxygen or the withdrawal of the nitrous oxid usually causes a sudden and marked elevation in blood-pressure, which persists for from five to fifteen minutes after the return to consciousness (Fig. 33).

Ethyl Chlorid.—The administration of even 3 or 4 c.c. of this anesthetic has been followed by serious consequence, 5 c.c. has been known to produce death and any amount over this is considered dangerous. Its effect is that of a powerful inhibitor of both heart and blood-vessel tone causing constant fall in blood-pressure. The pulse is not usually much affected but a dangerous hypotension is usually accompanied by a rapid and small pulse. The association of oxygen with ethyl chlorid seems to prevent the hypotension thereby rendering the effect of this anesthetic less dangerous.

Cocain.—Crile reports very little change in the circulation from cocain injections employed in the usual manner in safe dosage. Fear and fright may cause the curve to become irregular and show a slight rise. Cushing states[1] that cocain injections into the spinal cord generally induce dangerous hypotension.

In conclusion it may be stated that the effect of any anesthetic upon the circulation is of little importance unless blood-pressure is materially affected, and that any disturbance in blood-pressure resulting from the anesthetic is a symptom of great importance. An anesthetic which affects the blood-pressure but slightly and only when pushed to saturation is certainly to be preferred.

[1] Harvey Cushing, *Annals of Surgery*, 1902.

OPHTHALMOLOGY

The blood-pressure test has lately found great favor among the ophthalmologists, particularly those devoting their time to operative work.

Among the earlier careful studies into the value of this test, from an ophthalmologic viewpoint, in prognosis, diagnosis and treatment was one made by Fox and Batroff[1] and their findings have since been fully corroborated by many careful observers, among them L. C. Peter,[2] in 1911. The same author[3] directed attention to the close relation of high blood-pressure, chronic interstitial nephritis and albuminuric retinitis, showing that in some degree at least, there was a direct relation between the amount of increased tension and the severity of the symptoms produced.

The studies of Fox and Batroff were directed largely toward demonstrating the relation between retinal hemorrhages and high arterial pressure. From a study of 100 cases, they concluded that "the true or exciting cause of these hemorrhages in a very large proportion of the cases is a sudden transcient or a persistent abnormal elevation of the arterial pressure." And further that "the blood-pressure should be carefully and frequently studied in this class of ophthalmic cases; first with a view to determining the presence of one of the most frequent causal conditions, secondly to permit us to intelligently direct the treatment. The oculist, therefore, often being the first physician to be consulted, should study these patients with the internist, in order that the most comprehensive knowledge possible should be available for the sufferer."

[1] Colorado Medicine, May, 1909.
[2] L. C. Peter, *Penna. Med. Jour.*, March, 1911.
[3] *N. Y. Med. Jour.*, Aug. 20, 1910.

The summary of the findings of Fox and Batroff's series of 100 cases of hemorrhage is as follows:

Eighty per cent. occurred coincidently with other diseased conditions in which hypertension is the rule. The majority of retinal hemorrhages were found in persons suffering from chronic interstitial nephritis 40 per cent., the next most common relation was arteriosclerosis 27 per cent. and as is well known that these two pathologic conditions are rarely met independently of each other we may say that 67 per cent. of cases of retinal hemorrhage occurred in cases of cardiovascular-renal disease.

These authors are confident that high arterial tension is an important factor in the production of acute glaucoma, and cite a case with pressure of 265 mm. They strongly advocate the reduction of pressure by bleeding in all high-pressure cases before attempting operative procedures and cite a case in proof of this argument. Peter's later article reiterates this statement.

Jackson[1] and John Dunn[2] are also emphatic in stating that no case of essential glaucoma, either acute or chronic, should be considered fully examined until the blood-pressure has been carefully studied by a sphygmomanometer. In this belief Peter heartily concurs.

Dunn also discusses certain other cases of ocular disease, in which the use of the sphygmomanometer should never be neglected, as its revelations will not only be helpful in the proper understanding or existing ocular conditions and suggestive in prognosis and treatment, but will prevent blunders which, without this restraining influence, would

[1] *Am. Jour. of Ophth.*, Dec., 1909.
[2] *Arch. of Ophth.*

be committed. As a rule the higher the arterial pressure, the less favorable is the eye for a surgical procedure.

In corneal ulcers the blood-pressure test may give us information as to why the treatment does not succeed, often being explained by the presence of chronic kidney disease. A high blood-pressure will tell when not to operate in senile cataracts, or at least when the danger of hemorrhage may be reduced by preliminary blood-pressure reducing measures. On the other hand, with very high blood-pressure in persons past middle life, look out for retinal hemorrhages.

Peter says "that occasionally one will find early retinal disturbances and only a moderate increase in blood-pressure 150 to 170 mm. in young adults without other symptoms." He reports two cases of this type, but believes that these cases are so few that they really tend to confirm the now accepted view that increased blood-pressure is one of the earliest premonitory signs of arterio-sclerosis, and chronic diseases, and that this combination of conditions is the cause of early retinal and arterial changes as well as of the later phenomena.

Peter again calls attention to another group of cases in which hypertension plays an important rôle, namely, spasm or ataxia of the retinal artery or branches, which was first brought out by Zentmayer in 1906.

The value of the sphygmomanometer in the hands of the ophthalmic surgeon is now firmly established and he would be distinctly negligent, who would continue his professional career without the aid and guidance of the information derived from the blood-pressure test.

CHAPTER XVII

BLOOD-PRESSURE IN OBSTETRIC PRACTICE

The Value of Sphygmomanometry.—The obstetrician of the present day must have constant recourse to the blood-pressure test if he would maintain the lead in his profession. The sphygmomanometer now ranks with urinalysis in the examination of pregnant women. In the blood-pressure test we have a most valuable means of detecting early toxemias, which often lead to the eclamptic state. The blood-pressure test is capable of early furnishing very definite indications of departures from normal metabolism in the pregnant women. This is usually evident before the development of any physical signs, or of any noticeable change in the urine. From a pathological standpoint, it is evident that the close relation between the kidney and blood-pressure should be a valuable guide in this condition, since alterations in metabolism and the overproduction of waste products and the development of special toxins in the blood will show themselves in a gradually rising blood-pressure.

Many obstetricians (Hirst, Baily) are now most emphatic in insisting that reading blood-pressure observations should be made a part of the periodical examination of pregnant women and that with the development of suspicious signs and advances toward the end of the gestation, the intervals between the tests should be shortened, and that the test should not be omitted during puerperium, as in this state women may develop serious toxemia and eclamptic attacks.

Patients should be required to submit to the blood-pressure test at least as often as the urine is examined. Indeed it would be well to apply the sphygmomanometer at every convenient occasion. Employed in this way, with the records properly charted, the blood-pressure tests will furnish a far more adequate guide to the seriousness of a pregnancy nephritis and the urgency of inducing labor, than the usual urinalysis (see Fig. 34).

Blood-pressure during Pregnancy.—A series of examinations, made by John C. Hirst[1] showed that the average systolic pressure at rest, in non-pregnant women showing no signs of heart or kidney lesions, was 112 mm. Hg. In another series of 100 pregnant women, who had no evidence of kidney disturbance or any other sign of toxemia, gave an average pressure of 118. This average remains practically unaltered up to seven and one-half months after which a slight gradual rise occurs so that by the middle of the last month of pregnancy the average normal pressure is 124 mm. Hg. Usually with subsidence of the uterus, the pressure shows a slight fall. These findings coincide with the observations of H. C. Baily[2] who made 1,135 systolic readings on 145 normally pregnant women. Rather strangely his average systolic pressure in the early months of normal pregnancy was also 118 mm. Hg. Naturally the individual readings vary greatly within certain limits, which Baily believes to be insignificant unless it exceeds 30 mm. above the average, or reaches

[1] *N. Y. Med. Jour.*, June 11, 1910.
[2] *Sur., Gyn. and Obst.*, Vol. XIII., No. 5, p. 485.

above 148. Arthur J. Benedict[1] believes that a pressure of over 125 mm. Hg. in pregnancy is not normal, but indicates toxemia. I have been unable to find any other observer drawing this narrow margin, and therefore feel that this is an unnecessary narrow limit for normal variation in pregnancy. Baily in studying cases after the onset of labor noted that the pressure usually rose during the first and second stages remaining at 140 to 150 mm. Hg. between pains.

Hirst had noted that a fall of pressure coincides with rupture of the membranes, sometimes amounting to 50 or more millimeters, usually accompanied by marked relief from headache and epigastric symptoms. This is only temporary, as the pressure gradually rises as labor continues. There is a second fall of 60 to 90 mm. immediately after the child is born, which is also temporary, the pressure returning to almost the level attained before birth. Profuse hemorrhage or the supervention of exhaustion will interfere with this rise, the degree of reduction in pressure indicating the seriousness of these complications. Obstetric operations, according to Cook and Briggs,[2] which involve the introduction of the hand into the vagina or uterus, and instrumental deliveries, cause a sharp reflex rise which has been known to result in rupture of a cerebral vessel.

John Cooke Hirst[3] states that the earliest and most constant sign of toxemia in the latter half of pregnancy is a high and constantly rising blood-pressure (Fig. 34), and

[1] *Brit. Med. Jour.*, Dec. 3, 1910.
[2] *Johns Hopkins Hospital Reports.*, 1903, Vol. XI., 451.
[3] *New York Med. Jour.*, June 11, 1910.

this symptom precedes albuminuria and all the constitutional signs of an impending eclamptic attack.

According to the observations of Baily, blood-pressure in early toxemia may be low; here apparently toxic substances are circulating in the blood which have a marked influence on the vomiting center, but little effect on the vasomotor apparatus.

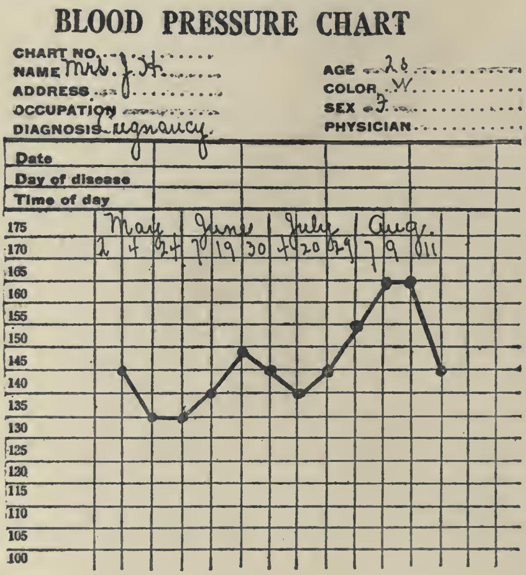

FIG. 34.—May 2, case showed albumin, scanty urine, headaches and dizziness, symptoms relieved by hot pack and purgation at irregularly repeated intervals. Premature induction of labor advised ,but declined. Normal delivery on August 9. Treatment controlled subjective symptoms, but did not much effect the tendency to a rising blood-pressure.

In the development of toxemia in the latter months, there is usually present a blood-pressure rising principal or a harmone action, or else blood-pressure is raised to increase the natural resistance of the body. He also noted that in the fulminant type of fatal toxemia, in the latter months the blood-pressure may be very low. In exceptional cases Baily has shown that convulsions may occur and yet the blood-pressure be no higher than 155, and that eclamptic toxemia may be even more severe when the pressure is very low. This elevation should never exceed 150 mm., and the pressure should fall after labor is finished. If the pressure exceeds this, it is a warning of the pre-eclamptie condition. If abnormally high pressure persists in the third stage, or there is little or none of the normal decline, measures for relief must be instituted almost as urgently as if the seizures were present.

T. M. Green[1] conveniently divides toxemia of pregnancy in three divisions:

First, moderate increase in blood-pressure.

Second, marked increase in blood-pressure.

Third, extreme increase of blood-pressure.

To these may be added the fourth, which is suggested by the studies of Hirst and of Baily, namely: extreme eclamptic condition in which the blood-pressure may be low.

In the first two, symptoms disappear and blood-pressure falls after delivery. In the third and fourth, blood-pressure continues abnormal, and the disease usually progresses to a rapidly fatal termination.

The blood-pressure seems to bear definite relation to the type of case, and its frequent observation should be of great value both in prognosis and in treatment.

According to Hirst, the highest pressure noted by him in a toxemic case without eclampsia was 192 mm. The highest in eclampsia was 320 mm. How high he was un-

[1] *Boston M. and S. Jour.*, April 28, 1910.

able to determine because the mercury ran out of the top of the tube before the pulse was shut off.

To summarize our present knowledge of the relation of blood-pressure findings, I can do no better than quote in full Hirst's summary, which is as follows:

First, the normal blood-pressure in normal healthy non-pregnant women will not vary much from 112 mm.

Second, the normal blood-pressure in healthy pregnant women will average close to 118 mm. A slight increase over these figures is to be expected in the last month of pregnancy.

Third, blood-pressure in toxemia in the first half of pregnancy associated with pernicious vomiting is usually low.

Fourth, blood-pressure in the latter half of pregnancy, associated with albuminuria and eclampsia, is invariably high.

Fifth, a high and rising blood-pressure is an invariable and very often the earliest sign of toxemia in the latter half of pregnancy.

Sixth, upon the rupture of the membranes, there is an immediate fall of pressure of from 60 to 90 mm. This fall is temporary only, but is attended with marked relief in the headache and epigastric pain these patients so frequently complain of. The relief from these symptoms lasts, however, for some hours after the pressure returns to near its original height, which is shortly after the first fall. A similar fall, by much slighter, is noticed after a sweat bath.

Seventh, there is a second fall of from 60 to 90 mm. after the child is born. This again is only temporary, and in from fifteen to thirty minutes, if a patient has not bled profusely, the pressure returns to about its level before the birth.

Eighth, usually in eclampsia, the pressure remains high for forty-eight hours after the birth then begins to subside and reaches the normal of from 118 to 124 mm. in from seven to ten days after delivery.

Ninth, as far as it is possible to lay down any rules in these cases we may say that a blood-pressure of below 125 mm. could be disregarded, a pressure of from 125 to 150

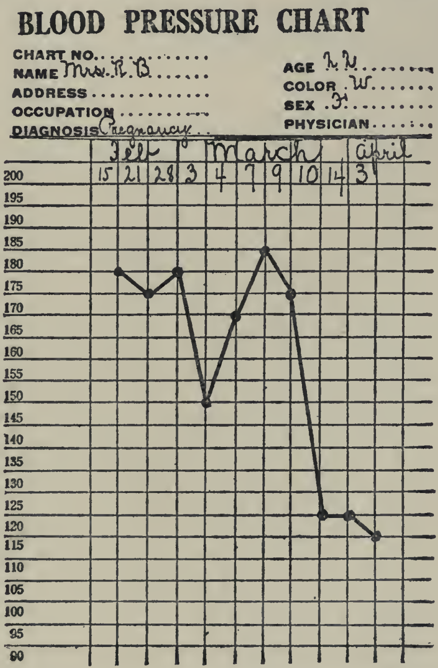

FIG. 35.—Term calculated to March 30, labor induced March 9. February 5, ankles edematous, marked gastric irritation, large amount of albumin in urine. This condition not relieved by treatment. March 7, urine boiled nearly solid. March 9, labor induced; March 10, delivered; April 3, albumin absent, patient normal.

mm. needs careful watching and moderate eliminative treatment, and that a pressure of over 150 mm. needs usually active eliminative treatment, and will in all probability, especially if it shows a tendency to climb higher, require the induction of premature labor. (Fig. 35.)

CHAPTER XVIII

BLOOD-PRESSURE IN LIFE INSURANCE

Since the publication of the author's review of the blood-pressure situation among life insurance companies in 1909[1] the value of this test as an aid in determining the acceptability of life insurance risk has rapidly increased, so that at the present time very few, if any, large insurance companies fail to appreciate the value of this procedure in life insurance examinations.

By this test we may very early detect signs of beginning pathologic change in the cardiovascular system and in the kidneys often before there is any demonstrable evidence of departure from normal either in the physical signs, personal history or urine. This is chiefly because the apparent character of the pulse, and the examination of the superficial vessels, does not always portray the actual condition of the general arterial tree or the degree of arterial tension. We fail to learn that the true condition of the arteries may not have been apparent, that the heart has begun to hypertrophy, and that chronic nephritis or cerebral arterial rupture may develop at any time. Clinicians have agreed that the estimation of arterial tension or blood-pressure by the usual means is most unsatisfactory, and in any case unreliable and often misleading. Even the most experienced have been unconsciously led into grave error by depending

[1] The Status of the Blood-pressure Observations in Life Insurance Examinations, *New York Med. Jour.*, July 23, 1910.

upon tactile sensations when the sphygmomanometer should have been employed.

To quote from Wm. Russell[1] we find the following very significant statement: "I must, however, again add a warning note to the effect that feeling the radial pulse is not always a reliable guide as to what the blood-pressure will read. I have two such cases under observation, the radial being neither hard nor incompressible, and yet in both there is a steady reading of over 200 mm. Hg."

Many times we may feel a soft and compressible radial where there exists marked sclerosis of the aorta and of the splanchnic area. Here only the blood-pressure test reveals the true situation. In other instances the reading of the sphygmomanometer may explain the significance of an apparently simple headache, a mild attack of indigestion, or transitory attacks of vertigo in an apparently healthy individual, by demonstrating that these cases have suffered from a long-continued toxemia, which has resulted in an unsuspected pathologic change in the cerebral or general vessels.

From the subjective standpoint, it is now well recognize that such pathologic changes may be present in the cardiovascular and renal systems, long before any suggestive symptoms are complained of by the individual, or if any complaint is made, the symptoms are usually attributed to some trivial cause.

Normal or Ordinary Variations.—It is necessary to recognize in this connection the activity of such usual but unimportant factors as alimentary hypertension, so well described by Russell, occurring in normal vessels, and due

[1] Arteriosclerosis, Hypertonus and Blood-pressure, 1908.

to errors in diet of either quantitative or qualitative origin. These respond immediately to the correction of such errors together with stimulation of the eliminative functions. Of further interest, particularly to the life insurance examiner, are the so-called physiologic variations depending on age, sex, mental and physical excitement, fatigue, etc.

These must all be taken into consideration in estimating the character and class of risk. (See Chapter IV.)

Such variations need not confuse the examiner, as they all occur within a range sufficiently restricted to prevent them from obscuring the issue. The only one which needs special consideration is the age factor. To determine this, many tables have been suggested and devised in an effort to indicate the normal average systolic pressure for any given age. While these are correct and can be applied, they are difficult to employ and hence are unsatisfactory, as their use entails reference to a table or the carrying of many figures constantly in mind.

Formula to Estimate Normal Pressure.—To simplify this, the author suggested a formula, based upon a large number of observations of his own and of others, which can be universally applied. The average obtained by the formula agrees closely with the experience of most observers, and since its first publication in 1910[1] it has been extensively quoted and is now employed by at least one insurance company. (The Provident Life and Trust Company, Philadelphia.) As originally suggested, it was as follows: "Consider the average normal systolic blood-pressure in the male at age twenty to be 120 mm. of Hg.; for each year

[1] The Sphygmomanometer and its Practical Application, Pilling Co., Philadelphia, 1910.

of life thereafter 1/2 mm. to 120." Later it seemed advisable to eliminate the fraction, and this was done by changing the phraseology to read as follows: "Consider the normal average systolic blood-pressure of a male, age twenty to be 120 mm., then add 1 mm. to every additional two years of life." In both the formulas the result is the same, thus at the age thirty the normal average systolic blood-pressure would be 125, sixty, 140 mm., etc. It is sufficiently established to pass without question that the normal average blood-pressure for females at the same ages is approximately 10 mm. less than that for the male.

Permissible Variations.—It is not sufficient to establish a normal average with which to rate the risk but it is necessary also to determine what variations above and below this shall be permitted to pass as normal. Unfortunately with the evidence at hand, this question cannot be definitely answered, for existing statistics do not agree. As far as can be gathered from many published reports of blood-pressure tests, a variation of 36 mm. in normal individuals is deemed not to exceed normal. If we accept this, then a variation of 17 mm. above or below the normal average may be allowed. Thus at age twenty any reading of over 137 or below 103 would call for explanation, while at age thirty the permissible variation lies between 157 mm. and 123 mm. In all determinations of blood-pressure, the factor of the diameter of the cuff employed and the type of instrument used in making the test must be considered, assuming, of course, that the accuracy of the instrument itself is beyond dispute.

At the present time the accepted standard for the width of cuff is between 4 1/4 and 5 in. (11 cm. to 13 cm.). A cuff of narrower width gives higher readings in proportion to the narrowness of the cuff.

Applications.—As a routine measure, the left arm should be employed and be bared to permit application of the cuff. Both patient and operator should be in comfortable positions, preferably the sitting posture. Nervous individuals should be assured of the harmlessness of the test, and have their attention diverted from the proceeding. Time also should be allowed to permit the circulation to become quieted, as after rapid walking, stair climbing, etc.

In the presence of a developing arteriosclerosis, the blood-pressure need not be greatly increased. An elevation of 30 to 40 mm. above that estimated as normal for the individual is significant and demands explanation. On the other hand a rise of even this amount should never be hastily assigned to arteriosclerosis, or the risk rejected without further study. When there is any doubt as to the accuracy of his finding, the operator should apply the test to the patient upon a subsequent occasion, before making his report.

Nephritis.—Bearing in mind the difficulty of early diagnosis in cases of chronic nephritis by a single urinalysis, particularly in individuals apparently in normal health, the importance of a blood-pressure test will be apparent, because it is recognized that we cannot have permanent kidney change without a constant elevation in blood-pressure, and even in the presence of albumin or casts, we may question their true significance. Here a persistently high blood-pressure, say 150 mm. or over, in an individual below middle age will settle the question at least in regard to the risk. The presence alone of scanty albumin and casts in the urine is not conclusive evidence of a diseased kidney, as these elements may come from any number of transitory and comparatively unimportant complications. The blood-pressure test will serve as a check, so that the applicant with a normal blood-pressure whose urine has occasionally shown albumin and casts will not immediately be rejected, and such individuals will be given the benefit of the doubt and the company thereby relieved from committing grave injustice.

Besides the physiologic variations already mentioned, the examiner employing the blood-pressure test must endeavor to control as much as possible the conditions surrounding the observation, otherwise the data as forwarded to the home office may be misleading. Every effort should be made to find what is the actual blood-pressure of the individual. More than one observation should be made when necessary in order to avoid reporting an abnormally high pressure, influenced temporarily by emotion, violent exercise, digestion posture or alcoholic stimulation.

Overweights.—The overweights demand careful consideration by the insurance examiner. This is a group which shows an unfavorable mortality in life insurance statistics, particularly in the higher ages. It should be remembered that the amount of adipose tissue covering the vessels does not materially affect the reading, as cases of very large arms present readings of normal or even below, so that findings of high pressure should be attributed to some other cause. In a person of modern overweight in whom nothing in the physical examination or history indicates rejection, the final decision is often made upon the relation of the blood-pressure test. Accepting this when the pressure is found normal, and declining when the pressure reduces or passes high normal limit.

Chronic Myocarditis.—This is probably the most difficult condition to diagnose which is met in the course of insurance work. Its possible presence must always be borne in mind and every effort made to eliminate it in the examination, particularly in those past middle life, and in those presenting past history of hard physical labor, excessive brain work, alcoholism and syphilis. This will of course not be difficult to recognize, when the disease has progressed sufficiently to affect the general health of the individual. It is in the early stages, where the usual method examination fails to reveal it, that the sphygmomanometer is of greatest value. In the early cases the systolic pressure need not be materially affected, so that recourse must be had to the functional tests of Graupner and Shapiro, and to a study of the diastolic and pulse pressures, by which changes in normal reserve of the heart, and the strength and volume of its output can be estimated. (See page 164.) Regarding the question of diastolic and pulse pressures, there is but little definitely known, although several conditions are now recognized as affecting these readings, which can be applied in health examinations and used to advantage in the work of the insurance examiner. Thus arteriosclerosis, on account of diminished elasticity of the blood-vessels, will show an increased pulse pressure (over 40 mm.) and the more extensive this change in the vessels, the greater will the pulse pressure be. This condition may be demonstrated in a suspect even before the systolic pressure has permanently passed the normal high limit of health.

Incipient Tuberculosis.—The presence of a slightly lowered blood-pressure accompanied by a slight elevation in pulse rate, with or without fever, combined with a history of slight loss of weight, is very suggestive evidence of an existing pulmonary lesion. In tuberculosis the blood-pressure is usually low and the pulse pressure diminished.

In this connection Haven Emerson[1] states that hypotension is found in almost all cases of moderately advanced tuberculosis and that it has been found by many observers in early doubtful or suspected cases with or without physical signs of the disease of the lungs, and that it is considered by competent clinicians as a most useful sign. Cook also states that low blood-pressure, if persistently found in individuals or in families should put us on our guard for tuberculosis. In applicants of light weight and a blood-pressure of 100 or under and of poor family history, the risk is bad. (See also page 179.)

Blood-pressure in Relation to Mortality.—Dr. J. W. Fisher of the Northwestern Mutal Life Insurance Company, has produced some very valuable work[2] by drawing conclusions from a study and analysis of the mortality statistics of that Company beginning 1907 and continuing until the middle of 1911. The report in full, more than confirms present opinions regarding the value of the blood-pressure test in the study of the cardiovascular and renal systems.

From a study of 2,668 insured taken from the actuary's tables giving blood-pressure readings between 140 and 149 mm. Hg., had 81.85 expected deaths, 31 actual deaths, a

[1] *Arch. Int. Med.*, 1910.

[2] *Medical Record*, October 21, 1911.

percentage of 37.87 which was slightly below the normal death rate of the company on exposure of two years. He shows another table of mortality records of 527 insured persons with a blood-pressure reading of 150 mm. Hg. and over with 22.19 expected deaths and actual deaths 12, which is about 35 per cent. in excess of the general average mortality of the company covering the same period and 10 per cent. higher than the general average mortality during the first five years of exposure covering the twenty years period 1885 to 1905.

He further shows a mortality record of 782 persons, declined for insurance, in whom the blood-pressure averaged 171.03 mm. Hg., 21.61 expected deaths with 32 actual deaths, a percentage of 155.27 or almost four times greater than the general average of the company. In another table are shown 366 cases rejected in which there were reported no other impairments than high blood-pressure at the time the application was received at the home office. The expected deaths were 10.14, the actual deaths 14, or 138.17 per cent. of the table. Efforts made to follow carefully these 366 cases in order to secure data as to the subsequent physical condition of these applicants, more than justified the opinion that the sphygmomanometer was one of the earliest, if not the very earliest, means of detecting departures from normal in this group of cases, as many impairments were later discovered or developed in a large number of cases rejected for high pressure only.

CHAPTER XIX

METHODS OF CONTROLLING BLOOD-PRESSURE

Causes of Failure.—A large number of the unsuccessful results in the treatment of cardiovascular renal diseases can be traced to one or more of the following causes:

1. The diagnosis is not made sufficiently early.
2. The case may have been poorly or incompletely studied.
3. The predisposing causes have not been found.
4. As a result, the condition is but imperfectly understood.
5. The therapy is irrational because it is based upon an incomplete knowledge of the case in question, plus a deficient knowledge of therapeutic methods by drugs or other measures.
6. Too great dependence has been placed upon drugs alone, especially the vasodilators, to the neglect of the newer so-called physiologic methods.

It may be said in general that while drugs are at times invaluable in the treatment of pathologic circulatory condition, especially in emergency, their value is usually much overestimated. The secret of successful treatment usually lies in a careful study, an early and complete diagnosis, rigid supervision and regulation of the individuals habits, rather than attempts to lower blood-pressure and relieve symptoms by the employment of drugs. A properly conducted study will sometimes yield gratifying results even in advanced cases, and at times in those cases commonly regarded as hopeless.

The most satisfactory results naturally follow complete examination immediately following the appearance of the first suggestive sign or symptom of impairment of the circulatory apparatus. This should be followed by a careful estimate of the functional power left in the impaired organs and the immediate adoption of a life and habits suited to the limitations determined. Thus we attempt to produce an adjustment of the individual's life which is an equivalent to relative good health. By correct diagnosis the full meaning of this phrase is meant and not the mere statement that the patient has "cardiovascular renal disease."

To arrive at a correct diagnosis, one must take a full history including a complete analysis of social history and personal habits, carefully considering both business and social activities, making a complete physical examination, including blood and urine examinations, and the blood-pressure, not omitting the functional tests. In fact the success of treatment depends chiefly upon the completeness in which the problem of each case is studied. Next upon the intelligence with which the remedies are employed and only secondarily to the particular remedial measures applied.

The material presented in this chapter has been carefully complied from literature which appeared during the past three years, and represents broadly the various measures recognized to be of value in combating the dangers of this condition. The reader must, however, not lose sight of the fact that hypertension or elevation of blood-pressure is very rarely a disease by itself, which is to be combated purely for the effect which the measures employed may have upon it. On the contrary, hypertension is as a rule merely a symptom, occurring in the course of certain pathologic conditions developing within the human economy, and bearing close and often important relation to disease in certain systems or organs. Too much stress has been laid upon this one symptom, and the tendency of late has been to speak of hypertension as though it were the whole disease, and the main object of therapeutic attack. Such a condition is unfortunate and greatly to be deplored, as such an attitude obscures the vision of the investigator often leading him into serious error. Only occasionally is hypertension the most important symptom calling for relief. On the other hand, it may be the only cheering symptom in an otherwise unpromising anamnesis, where it is often a wise provision on the part of nature to augment or maintain the activity of certain organs notably the kidneys and to preserve their function which would otherwise suffer from an insufficient circulation.

Direct therapeutic measures aimed at distinct pathologic conditions, will not be considered as they are beyond the object and scope of this book. This chapter will consist more of a resumé of existing literature and will be more in the nature of a reference chapter to be consulted when knowledge of the relative value of certain measures is desired, and when the effect of any particular drug is in doubt.

The classification of drugs and other therapeutic measures which follows is somewhat arbitrary, and is more a matter of convenience than of science.

Measures Employed to Reduce Blood-pressure.—Under this heading will be discussed first those drugs directly influencing blood-pressure through their specific action on the arterial wall, or on the vasomotors—the vasodilators; second, a miscellaneous group of drugs which are valuable chiefly for their secondary effect on reducing blood-pressure; and third, a group of physiologic or drugless measures which have recently been employed with success in combating hypertension and the symptoms resulting therefrom.

The Vasodilators.—This group of drugs belongs to that large and indefinite class known as depressomotor. It has a distinctly sedative action upon the spinal cord and other centers, and acts chiefly by reducing nervous irritability.

The several drugs belonging to this group, while having much in common, vary in their selective activity, thus while they all have a tendency to reduce arterial pressure, this effect in many instances occurs only after the administration of a toxic dose.

The most important vasodilators are:

Amyl nitrite	Mannitol hexanitrate
Nitroglycerin	Vasotonin
Potassium nitrite	Diuretin
Sodium nitrite	Agurin
Erythrol tetranitrate	

They act chiefly by causing dilatation of the arterioles and capillaries with consequent reduction in arterial blood-pressure. Besides varying in the amount of the drug required to obtain a physiologic action, these drugs differ greatly, also in their rapidity of action, the amount of reduction and the duration of effect obtained. It is important, therefore, to consider individually the more commonly employed members of this group.

Amyl Nitrite as a representative member of this group will be discussed critically. On account of its volatility, this drug is usually dispensed in glass pearls. These are to be crushed and the fumes immediately inhaled. The first effect of inhalation is hurried and panting breathing, followed by progressive muscular weakness and cutaneous flushing. Toxic doses gradually reduce reflex activity until death occurs from respiratory failure.[1]

Effect on Circulation.—The pulse is increased in frequency and the arterial blood-pressure is rapidly diminished. This action is due to a dilatation of the small vessels from the direct action of the drug circulating in the blood upon the walls of the arterioles and capillaries (Experiment of Brunton). At the same time the drug has a minor influence on the vasomotor centers.

Administration.—This is usually by inhalation, but it may be by the mouth or hypodermatically. Dose by inhalation 1/2 mm.; by the mouth two to three drops on a lump of sugar to be taken instantly; hypodermatically 1 to 3 mm. The drug is comparatively free from danger; as much as two drams given within two hours have been without serious effect (Wood).

All the members of the vasodilator group have essentially the same action on the circulation, varying slightly because of particular minor characteristics of the individual drugs. Space will not allow a more extended discussion here.

The following table has been constructed from the most recent literature covering clinical investigations upon

[1] H. C. Wood, "Therapeutics," J. B. Lippincott Co., Philadelphia.

the effect of these drugs. A study of the table will indicate clearly the relative value of the several drugs included in this group. The selection of the particular drug to be employed will depend upon the character of the case, the urgency of immediate action, and the effect desired. For a more complete consideration of these drugs in the treatment of disease with high arterial pressures, the reader is referred to other chapters in this work. This table has been constructed from the clinical statistical reports of Wallace and Ringer,[1] Matthiew,[2] J. L. Miller,[3] and Lauder Brunton.[4]

Drug	Effectual dose	Begin effect	Max. effect in	Mm. reduct.	Duration	Dose, interval
Amyl nitrite	1–3 mm... inhalation.	1 min.	2 min.	20–40	7 min......	P. R. N.
Nitroglycerin	1–2 mm...	2 min.	2 min.	20–40	30–40 min...	1–2 hrs.
Sodium and potassium nitrate	1/2 gr....	10 min.	6 min.	5–30	1-1/2 hrs...	T. i. d.
Erythrol tetranitrate	0.5–1.5...	15 min.	4 min.	15–50	4–6 hrs.....	4–6 hrs.
Mannitol hexanitrate	1 gr......	....	....	......	6 hrs.......	4–6 hrs.
Vasotonin	....	....	....	20–40	4–6 hrs.....	....
Diuretin	5 gr......	....	....	10–20	4–6 hrs.....	4–6 hrs.

Before employing any drug in this group, it should be carefully ascertained that the drug, particularly sodium nitrite, is strictly fresh, as failure to obtain the desired effect may be entirely due to the use of an inactive preparation. Tablet preparations are known to vary greatly in strength and should be of standard make. This defect can, according to some observers, be avoided by the employ-

[1] *Jour. A. M. A.*, No. 20, p. 1629.
[2] *Quart. Jour. Med.*, No. 2, p. 261.
[3] *Jour. A. M. A.*, May 21, 1910.
[4] *Loc. cit.*

ment of fresh chocolate tablet preparations. Sodium nitrite in solution rapidly loses its activity and should not be kept for more than one week. All these drugs may be employed hypodermatically when desired, but for continued use should, if possible, be given by the mouth.

According to Wallace and Ringer, it may be stated that, as a general rule, the higher the original pressure, the greater is the fall, and that an increase of the dose within safe limits seems to increase the fall. They were able in their experiments to obtain a reduction in pressure in every case, and the effect of an equal dose upon the pressure in arteriosclerosis was the same as the effect of an equal dose upon a normal individual. My own experience does not substantiate this.

Daniel Hoyt[1] arrives at the same conclusion, but advocates the use of larger doses than those generally employed, attributing failure to obtain satisfactory results to insufficient dosage or the employment of inactive preparations. This difficulty is largely removed when the clinician employs the sphygmomanometer to check his results.

Rudolph[2] notes that the effect of the vasodilators may vary from day to day, and in this connection Miller[3] brought out a very interesting as well as a most important point in the clinical action of these drugs, namely, that wide variation in their effect may occur not only from day to day, but that different drugs of the same group may affect the same individual differently. He reports the following specific instances:

[1] *International Clinics*, Vol. I, 1912.
[2] *Brit. Med. Jour.*
[3] *Loc. cit.*

Case 1.—Sodium nitrite had no effect whatever, nitroglycerin caused a reduction of 50 mm., erythrol tetranitrate resulted in a rapid fall of 110 mm., the patient going into collapse.

Case 2.—Nitroglycerin and erythrol tetranitrate had very little effect upon the pressure while a reduction of 65 mm. followed the usual dose of sodium nitrite.

Case 3.—Nitroglycerin caused a fall of 30 mm., sodium nitrite a fall of 20 mm., and erythrol tetranitrate a fall of 15 mm.

C. H. Lawrence[1] in one case saw a rise of pressure after the employment of mannitol hexanitrate which precipitated an attack of angina.

Vasotonin.—Muller and Fellner[2] report both animal experiments and clinical observations concerning the effect of vasotonin upon blood-pressure. Vasotonin is a combination of yohimbin and urethene. On animals it lowered the blood-pressure by dilating the peripheral vessels. There was no depression on the heart muscle, of the vasomotor center or upon the respiration. Fellner reports action on thirty cases of increased arterial tension.

They gave vasotonin subcutaneously in doses of 1 c.c. in some cases daily, in others every other day. The course of treatment comprised from twenty to thirty injections. They found that the remedy consistently produced a fall of blood-pressure with a marked improvement in the subjective symptoms. Thus, for instance, there was immediate relief in milder cases of angina pectoris and in cardiac and bronchial asthma. The bad cases of angina pectoris

[1] *Boston Med. and Sur. Jour.*, November 2, 1911.
[2] *Therap. Monatschrift*, 1910, XXIV, 285.

required longer treatment, but all improved and no unpleasant symptoms occurred. The use of this preparation has been confined chiefly to Germany and so far American observers have failed to obtain the uniformly favorable results reported abroad. If we are to believe foreign reports of the effect of this drug on man and the lower animals, we would expect to find a fall of from 20 to 40 mm. lasting from four to six hours, and that three or four injections given upon successive days will maintain the blood-pressure at a lower level than the original for six or seven days.

H. D. Arnold[1] reports the study of a small series of cases in which the effects were exactly the opposite. The injection of the drug was always followed by a rise in pressure and was occasionally accompanied by more or less serious disturbances. In one case it brought on an attack of angina pectoris. The duration of this rise averaged four to six hours. In the light of this dissenting evidence, small as it is, the drug cannot be recommended and if used at all should be followed with great care.

Diuretin.—W. H. Hamberger[2] following the lead of Romberg, Buch and others, strongly advocates the use of theobromin preparations, particularly theobromin sodium salicylate or diuretin. He finds this drug particularly valuable in hypertension resulting from arteriosclerosis of the abdominal vessels and reports his find in a series of experiments upon animals. This table so clearly shows the action of this drug, that it is given herewith:

[1] *Boston Med. and Sur. Jour.*, Vol. LXV, No. 18.
[2] *Interstate Med. Jour.*, Vol. XVIII, June, 1911.

Drams sodium theobromin salicylate per kw. of animal	Effect on blood-pressure expressed in mm. Hg.	
	Rise	Fall
0.0066	10 mm.	0
0.0125	0	0
0.022	0	22 mm.
0.040	0	30 mm.
0.062	0	36 mm.
0.066	0	42 mm.
0.125	0	65 mm.

The effect on the heart was not constant; as small amounts usually caused a moderate slowing of the rate, while large amounts accelerated the pulse rate and caused a marked depression in blood-pressure. Hamberger does not consider his results conclusive, but as the drug is apparently free from harm, it should be tried, particularly in the case of so-called splanchnic-sclerosis. Dosage 20 to 40 gr. a day.

Agurin.—Another theobromin preparation may be employed in the same conditions in which diuretin is indicated. Dosage 20 to 40 gr. a day.

Miscellaneous Drugs. *Veratrum Viride.*—This drug is classified with the heart depressants. Its chief physiologic action is upon the circulation, and in practice it is used chiefly to decrease the force of the heart. It is "a prompt, thoroughly efficient, and at the same time very safe remedy" (Wood).

In chronic cardiac diseases it is indicated in precisely those cases in which digitalis is contraindicated. The contraindications to the use of this drug are cardiac weakness and general adynamia. When used in excess it may cause alarming symptoms which simulate shock, but even in very large doses it is seldom fatal (Wood). In this respect it is far less dangerous than aconite. Its physiologic effect is shown in a slow pulse rate, a diminished force of the heart's action, and vasodilatation.

Administration.—Fluidextract, one to three drops, tincture three to six drops. It should be given at intervals of two or three hours, when continued effect is desired, and its activity may be hastened by gradually increasing the dose until the physiologic limit is reached. In some cases annoying vomiting may occur.

Aconite.—The action of aconite on the circulation is very decided. It is not a vasodilator but accomplishes a fall in blood-pressure through its action on vagus, causing at the same time a slowing of the pulse, which after full doses becomes small and rapid.

The drug may be safely used in cases of high pressure with an hypertrophied heart where the valves are in good condition and in eclampsia. When however there is dilatation, or myocardial degeneration, it becomes an extremely dangerous remedy, and should perhaps never be used unless with great caution and only after a careful study of the condition of the heart. Aconite is a much more dangerous drug when employed in circulatory conditions than is veratrum viride.

Administration.—Tincture, five to ten drops every three hours, fluidextract two to four drops every three hours.

Iodin.—Iodin and the iodids are supposed to beneficially influence degenerative changes in the vessel walls and have long been advocated for the treatment of high blood-pressure, apart from those cases resulting from syphilitic infection, where of course it is indicated. The profession is however by no means united, as to the efficiency of these preparations which at present do not find general favor in the treatment of arterial tension. Many believe

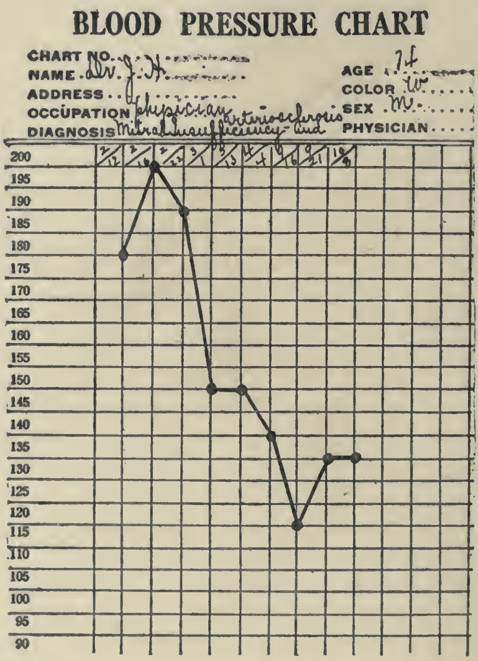

FIG. 36.—Chart shows dangerous effect of continued overuse of sodium iodid. Iodid was begun in doses of 5 gr. three times a day, and was continued until April 1. Pulse became irregular and patient was very dizzy. Strychnin, 1/30 gr., was begun on April 14, patient then left city for summer. Did not return until September 16, during which time contrary to orders, he persistently took between 25 and 30 gr. sodium iodid, and returned in very bad condition. The rising pressure of September 21 is from the combined use of strychnin and digitalis.

that any effect following the employment of this drug is due to the employment of other measures, such as improved hygiene, the elimination, rest, etc. One drawback to the continued use of this drug and its salts, is the irritation which its use causes in the digestive tract.

Iodin is usually administered in the form of potassium or sodium iodid, and as there is no difference in their effect upon the circulation and, as a rule, sodium iodid is better tolerated, the sodium preparation should be employed. No advantage has been found in the use of larger doses than 2 to 5 gr. daily, given in milk or diluted with water. Some observers, however, recommend the use of an ascending dosage beginning at 6 gr. and gradually increasing to 21 gr. a day. When used in this way an intermission of one week should occur in every four weeks of its administration. Excellent results have been reported in some cases of hypertension, but there was no proof that they were not of syphilitic origin. The accompanying chart shows a remarkable and at the same time dangerous effect from the overuse of iodid. (Fig. 36.)

The disagreeable effect of iodid can often be reduced by the addition of 5 gr. of sodium bicarbonate to each dose.

Arsenic.—This drug in doses of 1/5 gr. arsenic trioxid, has been reported favorably by some observers, among them Balfour. To obtain an effect the drug should be administered over a long period of time.

Trunecek's Serum.—Trunecek devised a serum to be used subcutaneously in arteriosclerosis. Its composition is said to be as follows:

Sodium chloride	10 grm.
Sodium sulphate	1 grm.
Calcium phosphate	0.75 grm.
Magnesium phosphate	0.75 grm.
Sodium carbonate	0.40 grm.
Sodium phosphate	0.30 grm.

One gram of this is dissolved in 15 c.c. of sterile distilled water. Treatment is begun by hypodermic injections in the region of the buttocks of 2 c.c. of the solution every other day, being increased in amount by 1 c.c. each injection until the dose of 8 c.c. is reached. The mixture has also been given per rectum and by the mouth.[1] The originator recommends this for use only in arteriosclerosis, but other authorities have used it in hypertension resulting from causes other than arteriosclerosis. Potter has made extensive studies with this substance without results, its trial can do no harm. Maximum daily dose, 10 c.c.

Thyroid Extract.—As pointed out repeatedly for many years by O. T. Osborne of Yale, a deficiency in thyroid excretion causes a rise in blood-pressure, and any increase in adrenal secretion has the same effect, hence the deduction that thyroid is of value in reducing blood-pressure. The thyroid gland seems to be a part of the mechanism of internal secretion which regulates blood-pressure, and probably is concerned in maintaining the normal low level. Thus small doses of the dried gland, 1 to 3 gr. of the official preparation a day, tends not only to lower blood-pressure, but in some cases by stimulating the action of the individual thyroid gland, serves to maintain for a time a better secretion. Possibly when benefit results from the use of iodin or the iodids, this is because of their stimulating effect on thyroid secretion. In cases of hypertonus and those showing a moderate degree of arteriosclerosis, with little or no cardiac or renal involvement, this drug may prove of great service in reducing and maintaining a more normal level of blood-pressure. It has also

[1] *American Practitioner*, April, 1912.

been used with benefit in high pressure in eclampsia. It should never be used in large doses or over a long period of time, and then not unless the patient is under close observation with frequent blood-pressure tests.

Salicylates.—All salicylates in large doses reduce blood-pressure, but are rarely employed for this effect. According to Hamberger[1] experiments with intravenous and hypodermatic injections of sodium salicylate in dogs it materially lowered blood-pressure and he suggests its use in this manner for this purpose in man.

Calomel.—Lauder Brunton[2] advises the employment of calomel in half-grain doses three or four times a day to relieve hypertension.

Rumpf recommends the restriction of the calcium content in diet, as a means of controlling high blood-pressure, but so far as I know his work lacks corroboration.

Anesthetics.—(See Chap. XVI, page 204.) Ethyl chlorid may bring about a dangerous fall in blood-pressure when used for anesthesia, even when used in small amounts, its employment is contraindicated in myocardial degeneration, and had even better not be used when there is a possibility of this condition being present, and with great caution in all cases showing hypotension. Under no circumstances should ethyl chlorid be used for its effect on blood-pressure.

Chloroform.—This drug always produces a fall in blood-pressure, which progressively increases with the duration of its administration, when used in concentrated form, as little as 3 c.c. has been known to cause a dangerous fall. Its employment is dangerous in all degenerative conditions

[1] *Interstate Med. Jour.*, p. 667, 1911.
[2] *Lancet*, Oct., 17, 1908.

and hypotonus, and while advocated by some clinicians as an emergency remedy to reduce high pressure, it had better be let alone, especially if anything else is at hand which will accomplish the same result.

Chloral may greatly relieve the symptoms of high blood-pressure, even without materially altering the level. Indeed it does not as a rule have much effect on high blood-pressure.

Hypophysis Extracts.—According to Lewis, Miller and Matthiew[1] the intravenous injections of the pars intermedia cause a decided rise in blood-pressure, injections of the pars nervosa cause a slight primary rise followed by a marked fall, accompanied by marked slowing of the pulse. Extracts of the anterior lobe give a primary fall followed in most cases by a secondary rise in pressure to a point above the original level, while the use of several parts of this gland give promise of being of value in the treatment of both high and low blood-pressure. There is as yet insufficient evidence either of an experimental or clinical nature to warrant its recommendation for general adoption.

Morphin in doses of 1/8 to 1/4 gr. hypodermatically may be relied on to lower blood-pressure and is a most valuable remedy in emergency, but not for continued use. Employed judiciously it may prolong life.

Potassium Bicarbonate, 10 gr. in a glass of water every morning, is recommended by Lauder Brunton[2] to keep blood-pressure down; he also suggests "10 gr. KNO_2, 10 gr. Na_2CO_3, and 1/2 to 2 gr. $NaNO_2$ in a powder dissolved

[1] *Arch. of Inter. Med.*, June, 1911.
[2] *Lancet*, Oct. 17, 1908.

in some hot aperient water, as tending not only to be laxative, but to keep the blood-pressure down, and this may be continued daily for years."

Physical Measures.—Under the head of physical measures valuable in controlling and in reducing high pressure, we find:

Rest.
Exercise.
Massage.
Diet.
Hydrotherapy.
Electrotherapy.
Venesection.

Rest and Posture.—Pre-eminently rest is the first essential in the treatment of all cardiovascular and renal conditions. It is always safe and generally beneficial to begin every course of treatment by rest. The term rest as here used may be purely relative or may mean absolute recumbency. The degree of rest enforced will depend entirely on the physician's judgment as based upon experience and the extent of his knowledge of the case and its requirements; no set rule can be adhered to blindly.

In the cases suddenly developing signs of incompetency, with dyspnea, a large heart, venous congestion, etc., the decision is obvious; it demands absolute rest and mental relaxation—nothing else will do. First and foremost, all unnecessary strain must be removed from the overburdened and dilated heart. This alone may suffice to break the vicious circle, allow the heart muscle to regain its lost tone and so pave the way for a period of at least relative health.

Rest in bed alone will often be sufficient to reduce a dangerously high blood-pressure. I have repeatedly seen a pressure of over 200 mm. fall to and maintain a new level of from 15 to 25 mm. lower. Occasionally even a greater reduction than this will be effected by this measure.

Effects of Sleep and Rest on Blood-pressure.—Brooks and Carroll[1] studied this question in sixty-eight patients showing average systolic pressure, in thirty with low pressures and in twenty-nine with abnormally high pressures. The results are, in a general way, illustrated in the cases with average pressure, in which readings taken between one and two hours after the beginning of sleep showed an average drop of 24 mm. Hg. Three hours after the awakening in the morning there was still an average depression of 12 mm. and from this time the pressure gradually rose during the day until usual highest level was reached in the afternoon. The greatest nocturnal fall in pressure took place in those individuals having the highest initial systolic reading. Disturbance of patients during the first sleep was found to delay, but not necessarily prevent the maximal fall in pressure; frequent interruption did, however, prevent it. Special tests were made to determine whether the sleep drop could be artificially increased in order to secure a lower general pressure curve in cases of hypertension; potassium bromide in doses as high as 120 gr., and chloral hydrate, up to 50 gr. each night, did not, however, increase the degree or persistence of the fall. Physical rest in general did not appear to alter materially either supernormal or normal blood-pressure, but the authors were led to believe

[1] *Archives of Int. Med.*, Aug., 1912.

that in mental or psychic rest profound changes in pressure occur, and that this factor largely determines the undoubted benefit derived from rest in cases of high pressure.

Exercise.—In certain cases, particularly that of the active business and professional men, it is not more rest, but more exercise that is needed. These are the cases in which, if seen sufficiently early, much may be accomplished toward permanently arresting the trouble, provided of course, that the patient is ready and willing to continue a new rule of life. These cases probably belong to those classed as true hypertonus, with tonic contraction of the circular fibers of the arteries (see page 21), with but little or no permanent pathologic change and where the kidneys show only signs of irritation. Here complete relief often follows a carefully regulated diet, combined with an increased amount of daily exercise. This should not be begun suddenly, nor be too strenuous. Walking first, to be followed later by light gymnastics or golf. Such measures should always be carefully followed by the sphygmomanometer.

In institutions and hospitals devoted to the treatment of chronic cardiovascular and renal diseases, the exercise methods of Schott and Ortel are carried out under competent supervision, and, under proper guidance, accomplish much good in educating the heart muscle to withstand more strain and to improve cardiomuscular tonus. It is not advised that the individual physician seeing at best but few cases, should attempt these special exercise treatments. A great deal can be accomplished by systematized walking as shown in the chart of a case appended herewith. (See Fig. 30.)

Massage.—General massage is usually well borne and is valuable in the treatment of cases showing failing compensation or defective heart tone. This treatment acts by emptying the venous side of the circulation and so relieves the left side of the heart, it also dilates the superficial capillaries, thereby further aiding in the distribution of the blood. Massage of the chest may influence favorably the tone of the heart itself, but deep pressure upon the abdomen should be avoided in order to escape a rise in blood-pressure and all movements should be graduated to the strength of the individual.

Both Eichberg[1] and A. Strausser[2] advocate the employment of massage in the treatment of cardiovascular diseases, and Eichberg has shown that massage movements even if prolonged do not effect a rise in blood-pressure.

Dietetics.—Much has been said and many dietetic outlines have been advocated in the treatment of circulatory disturbances. Their chief object is to diminish nitrogenous intake, to reduce putrefactive changes in the intestines which produce auto-intoxication; and, secondarily, to relieve the strain on a dilated and defective heart muscle, by reducing dangerously high pressure through limiting the fluid intake which eventually modifies the total amount of fluid in the body.

Foods.—A safe general rule to follow is, that while nitrogenous food is not to be prohibited, the amount should be greatly reduced, and a vegetable, farinaceous and milk diet substituted.

An absolute milk diet cannot be continued over a long

[1] *Jour. A. M. A.*, Sept. 19, 1908.
[2] *Wien. med. Wochen.*, April 8–15, 1909.

period because it is impossible to give sufficient nourishment without overstepping seriously a safe maximum of fluid ingestion. Excessive fluid sometimes being a factor in the production of the high pressure.

A short period of absolute milk diet (2 quarts) is useful for the relief of certain symptoms, and may guardedly be employed with benefit. When employed it should be given at two- or three-hour periods and never in large quantities at one time. The addition of some flavoring or the preparation of junket will render the employment of milk less irksome to the patient.

In the treatment of cardiovascular cases, the best results generally follow a number of small meals taken at frequent intervals (three to three and one-half hours). This prevents possible harm of throwing a heavy strain on the heart and blood-vessels through the digestive apparatus, which might easily disturb a poorly balanced circulatory equilibrium.

Alcohol, tea and coffee are usually prohibited entirely, at least for a time. An exception to this may be a heavy drinker, who cannot get along at all if his habitual potations are suddenly and entirely interdicted. As a substitute for coffee, postum may be employed; and recently a patented process has been used in Germany by which the coffee bean is freed of 90 per cent. of its caffeine. In this the taste of the coffee is not materially changed, but the effect upon the heart and blood-vessels is decidedly lessened. Elsner[1] and others report the use of this preparation during a period of several years with a great deal of satisfaction.

Tobacco.—Tobacco in the form of pipe, cigarettes and

[1] *Boston Med. and Surgical Jour.*, No. 7, 1910.

cigars has the power of raising blood-pressure with the apparent paradox that the habitual smoker has usually a low pressure. Arterial disease tends to augment the effect of smoking on arterial pressure. It is often a point of delicate decision to determine the amount of harm resulting from the use of tobacco, and the proper amount of restriction in the use of the drug necessary in each particular case. When in doubt the best rule to follow is to carefully restrict and control the patient's habits in this regard. In cases with a history of anginoid attacks tobacco in all forms should be prohibited entirely.[1]

The habit of chewing tobacco is much more harmful than smoking because of the greater amount of the active principle, nicotin, which enters the system. Its use should, therefore, not be tolerated.

In restricting diet, no definite rule can be laid down which can be followed safely in every case. Each case has its own peculiarities and the physician should endeavor to determine intelligently the restrictions to be made and what things may be allowed with safety in a given case. One should be careful in any dietetic scheme to avoid a caloric reduction below the needs of the individual, otherwise much harm may be done, for it is impossible to build up a strong heart upon insufficient nourishment.

L. F. Bishop[2] makes the following suggestions which may serve as a valuable guide in the preparation of a dietetic list in hypertension and chronic heart disease.

First, he suggests that every student of the subject should address a letter to the Superintendent of Docu-

[1] A. Strausser, *Wien. klin. Wochen.*, April 15, 1909.
[2] *N. Y. Med. Jour.*, March 4, 1911.

ments, Government Printing Office, Washington, D. C., enclosing ten cents and asking for *Bulletin* No. 28 on the "Chemical Composition of American Food Materials."

Secondly, the principal to be remembered is that an adult requires from 14 to 20 calories per pound, body weight, according to the amount of work he does. The weight is to be estimated by the normal weight for the height of the individual. For example, a person 5 ft. 7 in. tall ought to weigh 150 lb.; at light work he will require an average number of heat units per pound 17, $150 \times 17 = 2{,}550$ calories. If a healthy man has more than this, he will accumulate fat; if he has less he will become run down, and a weak heart cannot be built up on insufficient nutrition.

Bishop submits the following dietary covering a period of five days, which allows a fair caloric intake:

DIET

	Calories	Protein
Luncheon:		
1 cup of bouillon	40	10
2 slices of mushroom on toast	50	2
1 tablespoon of potatoes	100	2
1 plate of endive and lettuce salad	125 (oil)	
1 saucer of rhubarb.		
1 piece of gingerbread	230	4
Dinner:		
1 plate of vegetable soup	50	3
3 tablespoons of stewed tomatoes.		
1 large tablespoon of potatoes	110	2
2 large tablespoons of beans	60	4
2 tablespoons of Indian pudding	175	8
Lactose with each meal	300	
Average breakfast	315	8
Total for day	1,565	43

JANUARY 21.

Breakfast:			
1 orange	40		
1 small bowl of wheat berries	160		4
2 slices of toast	115		4
1 cup of weak coffee.			
Luncheon:			
4 large fried scallops	60		8
2 tablespoons of creamed potatoes	220		4
1 plate of cabbage and lettuce salad	125	(oil)	
2 tablespoons of preserved peaches	40		
1 cup of weak tea.			
Dinner:			
1 plate of vegetable soup	50		3
3 small slices of bread	230		8
2 tablespoons of potatoes	220		4
2 tablespoons of spinach.			
1 plate of scallop and lettuce salad	140		4
1 small piece of pumpkin pie	250		4
1 small piece of cheese	120		8
Lactose with each meal	300		
Total for day	2,070		51

JANUARY 22.

Breakfast, practically as before	315		8
Dinner:			
1 plate of vermicelli soup	120		4
4 small potatoes	200		4
2 tablespoons of gravy.			
3 stewed onions	100		4
Ice cream	320		8
Cake	230		4
3 small slices of bread	230		8
Tea:			
1 cup of bouillon	40		10
1 tablespoon of fried potatoes	110		2
3 slices of bread	238		8
1 plate of lettuce and celery salad	125	(oil)	
1 cup of weak tea.			
Ice cream	320		8
Sponge cake	230		4
Lactose with each meal	300		
Total for day	2,870		72

January 23.

Breakfast, as before	315		8
Luncheon:			
3 tablespoons of macaroni	100		3
2 tablespoons of spinach.			
3 small slices of bread	230		8
1 plate of lettuce and endive salad	125	(oil)	
1 piece of pumpkin pie	250		4
2 pieces of cheese	120		8
1 cup of weak tea.			
Dinner:			
Large plate of farina soup	50		2
3 tablespoons of macaroni	100		3
2 tablespoons of potatoes	220		4
3 pieces of preserved peaches	40		
3 slices of bread	230		8
Lactose with each meal	300		
Total for day	2,080		48

January 24.

Breakfast, as before	315		8
Luncheon:			
1 plate of lettuce and endive salad	125	(oil)	
2 tablespoons of potatoes	220		4
2 tablespoons of fried hominy	120		4
3 pieces of preserved peaches	40		
1 cup of weak tea.			
Dinner:			
Large plate of vegetable soup	50		3
2 tablespoons of boiled potatoes	180		4
2 tablespoons of stewed peas	100		7
2 tablespoons of rice pudding	175		4
Lactose with each meal	300		
Total for day	1,625		34

N. B.—One glass Sauterne with each luncheon.

Average for five days: Protein, 49; calories, 2.040.

Roughly speaking the average helping of meat contains 25 grm. of proteid, an egg contains 8 grm., as does also a glass of milk. An ordinary helping of rice, potatoes, bread or hominy contains about 4 grm., thick cream, butter or oil contain practically no proteid, but are very rich in heat units. Green vegetables do not count one way or the other. In cardiovascular disease, milk sugar is a valuable addition to diet, for many reasons. A sufficiency of calories can be roughly judged by watching the weight of the individual; if the weight is maintained the caloric supply is certainly sufficient.

Water.—Water properly employed may be of great value in the treatment of cardiovascular and renal diseases, but like any other good thing, it can be overworked. Cases are on record where apparently the only causative factor in the production of chronic interstitial nephritis was continued excessive water drinking. Usually it is advisable to limit the amount of water, especially in very high-tension cases or where there is a tendency to edema. This will spare both the heart and blood-vessels, but the amount should not be reduced below 1,500 c.c. per day, and even when there is edema this should not be continued for more than three consecutive days below this figure (A. Strausser).

Balfour[1] sums up his experience in the dietetic treatment of chronic myocarditis as follows:

"There should never be less than a five-hour interval between meals.

"No solid food should be taken between meals.

"The principal meal should be taken in the middle of the day.

"All food should be taken as dry as possible."

In the matter of the interval between meals, however, authorities differ; an equally competent observer advised the employment of five small meals a day (see above).

[1] The Senile Heart.

Hydrotherapy.—L. T. Thorne[1] proves by the citation of many cases that the majority of painful and dangerous symptoms which are usually attributed to hypertension are in reality the result of cardiac insufficiency and dilatation, dependent upon pathologic conditions of which hypertension is one valuable sign. He rarely employs drugs, but values most such physical measures as will reduce arterial tension, and at the same time improve the tonicity and contractility of the heart. For this he depends chiefly upon a course of natural baths or their artificial substitutes so prepared as to resemble in chemical composition the natural baths of Nauheim.

Hydrotherapy in the treatment of cases of high blood-pressure, particularly those accompanied by arteriosclerosis, accomplishes its result chiefly through regulation of the circulation. Properly used, such methods may, under certain conditions check the progress of disease by breaking the vicious circle in which the patient is involved. The primary effect of plain water, either hot or cold applied to the surface of the body, has been found by most observers to cause an initial rise in blood-pressure. This elevation usually amounts only to a few millimeters, and is followed speedily by a reaction, accompanied by lower pressure, from a relaxation of hypertonus and diminished peripheral resistance, caused by an increased flow of blood through the capillaries.

Cold Baths.—The careless application of cold to high-pressure cases may be dangerous. Its effect should first be ascertained by rubbing cold water over portions of the body. Cold applications can only be used with safety in cases of

[1] *Practitioner*, July, 1911.

early arteriosclerosis and cold douches should be used with extreme caution, as they do not as a rule give as good results as rubbing or ordinary bathing. In this connection cold sea-water baths should not be indulged in by arteriosclerotics nor by those having myocardial degeneration.

The Scottish douche (alternate application of hot and cold water) frequently gives good results in hypertension, if the contrast between the temperatures employed is properly graduated.

Hot Baths.—The temperature of hot baths in cases of arteriosclerosis should not exceed 37 or 38° C. Extreme changes in temperature of baths is also contraindicated in arteriosclerosis, because of the danger in any sudden change in pressure, particularly any sudden increase arising from capillary contraction which causes increased peripheral resistance.

Hot-air baths and electric-light baths are probably as good as the direct application of heat, and should be employed whenever practical.

In the hypertension accompanying acute nephritis, with the usual subjective symptoms, I have seen great benefit follow a properly given electric-light bath, the temperature being allowed to rise in the cabinet to 125° F. to be maintained for from fifteen to twenty minutes. Under these circumstances an immediate fall in pressure occurs often amounting to from 15 to 30 mm., occasionally more, and this fall is usually lasting in character, often persisting for twenty-four hours. The effect upon the patient is always most satisfactory, the subjective signs immediately disappearing. Elimination is increased while the pathologic elements in the urine are diminished.

The proper administration of an electric-light bath depends upon the intelligent use of the sphygmomanometer. By this instrument, and by its aid alone can the immediate effects of the bath be measured, so that its duration and the period of its administration may be definitely calculated. Hydrotherapeutic measures sometimes accomplish good results when drug medication absolutely fails. This was well shown in one case, where nitroglycerin was given to the point of intolerance, without effect upon blood-pressure, while the electric-light bath speedily reduced the pressure and easily maintained a reduction of 45 mm.

Miller recently has reported a practical series of clinical studies on the effect of the sweating process in high-pressure cases. In his series all patients reported, sweated profusely for at least thirty minutes. The method of producing the sweat varied. The blood-pressure was taken just before the sweat discontinued. Three out of five cases showed a reduction in pressure ranging from 13 to 20 mm. In one case it did not return to previous level until a lapse of four hours. Patients always felt better after the sweating. Dyspnea (uremic) is generally relieved even when the pressure is not reduced.

A number of patients were given one or more daily sweats for two or three weeks, the pressure recorded daily; results varied, in some there was no change, some showed a gradual fall. One case which had been over 210 for several years, came down to 180 (Fig. 31).

In chronic cases the sweating process is not lasting in its effect, as the pressure soon returns to original level when sweats are discontinued. Poststernal oppression is relieved more often by sweats more than by other measures.

Table 5.—From Miller. Effect of Sweating on Blood-pressure

Case	Blood-pressure before sweating	Blood-pressure after sweating	
1	160	140	Four hours before it reached previous level.
2	190	190	
3	170	170	
4	190	175	Two hours before it reached previous level.
5	185	172	

The sudden application of cold or chilling after a sweat is dangerous. In one case Miller has reported a rise of 60 mm., followed by transitory numbness. Overreduction of pressure may be followed by untoward results, although this does not always follow, as shown in the chart No. 31, page 147.

The Nauheim Treatment.—The basis of the Nauheim treatment in circulatory disorders is rest, hydrotherapeutic measures and exercise. Its chief value in the treatment of circulatory disorders comes from its effect on the heart muscle. Acting upon the heart, it increases tonus and reduces dilatation. Acting upon the circulation, it dilates the arterioles and capillaries thereby relieving a high peripheral resistance and obtaining a more uniform distribution of blood. These baths do not always produce a reduction in blood-pressure, and they may be followed by disastrous results. In this connection it is important to sound a warning note. Neither the oxygen nor the CO_2 bath should be used without a working knowledge both of what is desired and what such treatment may be expected to accomplish.

Application.—The chief hydrotherapeutic method employed at Nauheim is the complete immersion of the patient in a bath of natural brine, which is charged with free CO_2 gas. The most important constituents of this bath are sodium chlorid and calcium chlorid. The temperature of the bath is varied according to experience. The patient remains immersed for a period of from four to fifteen minutes, is then carefully dried, without chilling, and required to rest in bed for an hour. The baths are given on alternate days; the course usually occupies six weeks. Baths of similar character are given under medical supervision at Glen Springs, N. Y., where the methods are much the same as those at Nauheim and the benefits derived probably as good.

According to Dr. John M. Swan, formerly of Glen Springs, the effects to be expected from the proper use of carbonated-brine baths are as follows:

1. Diminution of the size of the heart.
2. Slowing of the pulse.
3. Reddening of the skin.
4. Slowing of the respiration.
5. Reduction in the size of the liver, if that organ has been the seat of passive congestion.
6. Improvement in the muscular quality of the heart sounds.
7. The disappearance of hemic murmurs, or those due to dilatation of an orifice.
8. Increase in the intensity of those murmurs which are dependent on valvular defect or deformity.

The chief indication for the use of the carbonated-brine bath in the treatment of chronic heart disease is in cases of myocardial weakness, with low pressure. In such cases we expect to get a retarding of the pulse, an improved heart-muscle sound and a rise in blood-pressure.

In cases of senile heart, with high blood-pressure and evidence of general arteriosclerosis, carbonated-brine baths, if given at all, should be stopped at once upon the development of an increase in blood-pressure, whether this is shown by subjective symptoms, or by the sphygmomanometer. In cases where the beneficial effect of the bath is in doubt, danger may be prevented if the temperature of the bath is kept above 98° F.; otherwise the strong brine should be omitted or diluted and the CO_2 gas left out. According to Swan, CO_2 baths are contraindicated in cases of advanced arteriosclerosis, chronic nephritis, aneurism of the large arterial trunks, and in the terminal stages of broken compensation with edema.

After the diseased myocardium has had an opportunity to recuperate, and to regain some of its lost tone by rest and the bath treatment, it is often advisable to provide exercise under proper supervision in order to help the heart perform its normal functions in as nearly a normal fashion as possible. This is in the nature of a special training of the muscles to be developed. Two systems have been devised which apply graduated work to the heart: first, Schott method or resistance movement; and, second, Ortel method of graduated hill climbing. These consist of a number of exercises of increasing severity, arranged so that the increased work imposed on the heart is very slight, but is increased in proportion as the heart muscle learns to bear the strain. For more complete descriptions of these methods and their applications, the reader is referred to works devoted to hydrotherapy and the treatment of heart diseases.

Oxygen Bath.—A mode of treatment that has recently been advocated and favorably reported upon by a number of observers is the oxygen bath. According to reports the effect of the oxygen bath is very different from that of the CO_2. In the CO_2 bath the skin becomes reddened from dilatation of the superficial vessels, while in the oxygen bath the cutaneous vessels are constricted and the skin becomes pale. The oxygen bath at 95° F. reduces both pulse rate and blood-pressure, and the effect of the CO_2 bath upon blood-pressure is variable. In arteriosclerosis these baths are said to have beneficial effect, among which is a moderate reduction of blood-pressure. According to the conclusions of A. Wolfe[1] the respective effects of the oxygen in the CO_2 bath upon the human body are as follows:

1. The temperature of the water in both instances has a material bearing upon its influence on blood-pressure.

2. At 93 or 94° F. neither bath has much influence on blood-pressure if this be not pathologically changed. The CO_2 bath at 94° tends primarily to increase a pathologic blood-pressure, whether this was at first a hypo- or a hypertension.

3. The normal pulse is but little altered by either bath, while the CO_2 reduces it more often in less degree than the oxygen bath, when the pulse is originally abnormal.

In employing the oxygen bath, the patient should not enter it immediately after active exercise or mental excitement, and unnecessary movement should be avoided while

[1] *Zeit. f. Physiol. u. Viet. Therap.*, Vol. XIV, 1910.

in the bath. He should be carefully dried and then should lie down immediately for an hour. The duration of the bath, depending upon the effect desired, should be from ten to twenty-five minutes, and should be given on alternate days. The bath is contraindicated in low blood-pressure accompanying the last stages of arteriosclerosis. Also for those with mitral defects or marked anemia.

The ingredients for the oxygen bath (sodium per borate and magnesium borate) can be obtained in the open market under the name of "perogen" bath.

Electrotherapy.—Much has been said, and, if possible, more has been written, upon the subject of electrical treatment for the reduction of arterial hypertension. A careful review of literature up to the time of writing shows that there is considerable divergence of opinion upon the value of such measures. First, in any case, we must determine the cause of high pressure and the desirability of reducing it.

Here, as in the study of other remedial agents, a systemic employment of the blood-pressure test is essential to the proper interpretation of the results, as it is only by this means that the psychic element can be eliminated, which some authorities aver is the only benefit derived from the use of electrical currents in the treatment of hypertension.

William Benham Snow[1] is conservative in his statements regarding the value of such measures, and largely confines himself to the consideration of the control of early cases of hypertension by autocondensation and other electrical measures.

He divides all cases presenting the symptoms of hypertension into the following seven clinical groups:

[1] *Jour. Adv. Therap.*, June, 1909.

1. The aged and feeble, partly compensated arteriosclerotics with low-pressure readings. (These are not benefited by electrical treatment—author.)

2. General arteriosclerosis, so wide spread that autocondensation fails to affect the reading, sequlæ cannot be avoided and electrical treatment is useless.

3. Arteriosclerosis of advanced age, fifty to sixty years, pressure above 200 mm.; autocondensation and hygienic measures cause a reduction to 165 or 160 mm., when it may be maintained by diet and occasional electrical treatment. There is a corresponding improvement in general health. Electrical treatment is valuable in this class if it can be continued indefinitely from time to time in order to maintain the reduction.

4. Arteriosclerosis in adults of thirty-five to fifty-five, pressure 150 to 170 mm., with or without beginning chronic nephritis. Here fifteen minutes treatment, 400 milliamperes by autocondensation, produces marked fall; with frequent treatments and correction in diet the tension often returns to normal, the physical condition appears normal and urine clears up. (These cases are probably those of true hypertension of Brunton, those which do not have permanent arterial change or chronic intestinal nephritis—author.)

5. Same as class four, except an earlier stage of hypertension—(author).

6. Young adults, chiefly athletes, who have developed a work hypertrophy and consequent moderate degree of hypertension (Snow fails to state effect of treatment—author.)

7. Compensatory hypertension occurring in parenchymatous nephritis, cirrhosis of liver, fever, after excessive

exercise, etc. (Condition about the same as 4, no uniformity in results of treatment—author.)

Snow states that D'Arsenval high-frequency and static-wave currents act locally upon the neuromuscular mechanism. The methods of D'Arsenval may be either autocondensation or autoconduction, by both of which methods the patient is placed in a field of hypotensive stresses where the high frequency to a greater or lesser extent surges through the tissues of the body, and are remarkably active in lowering arterial tension. "This effect is probably induced by a complex action of the current." Acting conjointly:

1. Upon metabolism, promoting tissue combustion and elimination, as demonstrated by an increase in solids in the urine, and

2. Upon the vasodilator centers which control peripheral resistance by which hypertension is relaxed, as demonstrated by the sphygmomanometer.

A twelve-minute administration of 400 milliamperes is, as a rule, followed by a reduction of from 10 to 15 mm.; occasionally a fall amounting to fifty occurs.

"Autocondensation is indicated in all cases in which hypertension is not compensatory and is contraindicated in all compensatory cases" (Snow).

Dosage 300 to 400 milliamperes from twelve to fifteen minutes duration repeated daily or on alternate days.

Van Allen[1] claims that high-frequency currents reduce the blood-pressure by removing the exciting causes, that is, by preventing autointoxication.

[1] *Albany Med. Annals.*, June, 1911.

It must be remembered that all efforts at reduction of high blood-pressure should be based upon a carefully made diagnosis, and that the indications for interfering with the circulation must be clear, otherwise one must expect to have failures. In some cases even disaster will follow ill-advised efforts to modify blood-pressure. A safe rule to follow is to watch the patient, study the effect of pressure changes upon him and cease all measures that fail to produce benefit, both in the evident physical condition of the patient and in his own subjective signs.

Venesection.—Miller,[1] after carefully studying the effect of venesection on both normal and pathologic cases, arrived at a conclusion similar to that stated by Mackenzie some years before. Miller found the rapid withdrawal of 300 c.c. or more from a normal individual is followed by a transitory fall in blood-pressure, but all persons do not react in the same way. The effect depends partly on the rapidity with which the blood is withdrawn—500 c.c. withdrawn slowly may have no effect on blood-pressure.

Butterman[2] bled ten students, withdrawing from 200 to 480 c.c., and nine showed reductions varying from 5 to 30 mm. Patients with hypertension do not necessarily all react in the same way.

The accompanying table taken from Miller's article above shows what may be expected in efforts to reduce hypertension by this means.

[1] *Jour. A. M. A.*, Vol. LIV, No. 21.
[2] *Arch. für klin. Med.*, 1902, LXXIV, No. 1.

Effect of Bleeding on Blood-pressure (Miller)

Case	Blood-pressure before bleeding	Amount of blood withdrawn c.cm.	Blood-pressure after bleeding	
1	200	500	200	
2	190	500	185	
3	160	600	150	Two hours later 160
4	185	500	170	Two hours later 180
5	220	450	210	

CHAPTER XX

BLOOD-PRESSURE ELEVATORS

Hypotension is often an important complication in acute infections, especially in pneumonia and typhoid fever. So also in shock, after hemorrhage, during anesthesia and under surgical operations a dangerously low pressure may develop and demand the employment of measures capable of controling it. (See Chapter VIII on Hypotension.)

A knowledge of the usual therapeutic measures employed in such conditions, and the effect which may be expected from them, should form an important part of the readily available knowledge of both surgeon and physician.

The routine employment of the blood-pressure test has thrown much light upon the action of blood-pressure among drugs, and has resulted in the elimination of many which have long been empirically employed. At the same time new and valuable remedies have been added to the list of those available for combating dangerous hypotension.

The varying origin and character of the drugs employed make the scientific division of this group impossible, so that the arrangement herein found is largely based upon the activity and reliability of the several drugs, as demonstrated both experimentally and clinically by the sphygmomanometer.

Adrenalin.—While reports bearing on the efficiency of adrenalin as a supporter of failing blood-pressure are conflicting, a critical study shows that this drug is probably our chief support in emergency, and that it may in many cases be relied upon, when properly employed, to support a failing circulation for a sufficiently long time to tide the case over a crisis.

Adrenalin may be administered by the mouth in doses of from fifteen to fifty minims, by hypodermic in doses of three to ten minims, and by hypodermoclysis and intravenous injections in varying dosage, depending upon the rate of flow through the needle and the extent of effect desired. The action of adrenalin when given by mouth is extremely unreliable and it is doubtful whether absorption from the stomach takes place with sufficient rapidity to allow much of the drug to be absorbed before its activity is reduced or destroyed by the fluids in the digestive tract.

MacKenzie recommends the hypodermic method for emergency use, but he believes frequent repetition is necessary if any sustained action is desired as the action is largely local, as the product is rapidly destroyed after entering the blood stream. The researches of W. Straub[1] confirm the assumption that adrenalin has no cumulative action, and says that it is probable that this substance is destroyed with great rapidity, as it vanishes from the blood completely, just as rapidly as its action subsides. Its action is further exclusively local, that is, it acts on the vessels only by direct contact. This we think proves that the continuous infusion of a weak solution of adrenalin is the only rational method of employing the drug, when continued effect is desired. Straub found it possible to

[1] *Münch. med. Wochen.*, Vol. LVII, No. 26.

send the solution continuously into a vein and thus keep blood-pressure up permanently, as long as it was continued, the effect being dependent on the concentration of the solution, and not on the absolute amount of adrenalin infused.

In the low blood-pressure of shock, Pearce and Eisenberg[1] recommend the slow intravenous administration of adrenalin salt solution (1–40,000) combined with a pure cardiac stimulant such as digitoxin. They obtained relatively rapid and permanent improvement. In this same connection A. Randal Short[2] found that the addition of adrenalin to normal salt solution in strength up to 1–20,000 would restrain the caliber of the vessels even when the vasomotor center was powerless and that apparently hopeless cases recovered under this treatment.

In contrast to this testimony Brooks and Kaplan[3] have reported two cases where adrenalin was used as the therapeutic agent for a prolonged time. They found that during continued administration adrenalin gradually lost its power, and they therefore do not accept the common belief that adrenalin will, over a prolonged period, maintain a constant elevation of pressure.

Pituitary Extract.—A. Randle Short believes that pituitary extract is of more value than adrenalin. When given hypodermatically in doses of 1/5 gr. t.i.d. it is extremely efficient in counteracting at once depressed arterial tension, it appears also to promote diuresis.

J. Campbell McClure[4] finds the effect of the drug much more prolonged than that of adrenalin. It can therefore

[1] *Arch. Int. Med.*, Aug., 1910.
[2] *Loc. cit.*
[3] *Arch. Int. Med.*, Oct. 15, 1909.
[4] *Practitioner*, Dec., 1911, p. 829.

be employed hypodermically and can be employed advantageously over a long period of time.

Digitalis.—This is another drug still under dispute. On account of local irritation, it is usually employed by the mouth in doses of from five to ten minims of tincture. The usual preparations of this drug are extremely variable and are not to be depended upon unless coming from a reliable source. I have seen less effect follow the administration of twenty-minim doses of a poor preparation than was obtained from five minims of a good active one.

Digitalis is slow and cumulative in action. According to Boos and Lawrence[1] its full action on blood-pressure cannot be expected in less than twenty-four to thirty-six hours. It cannot therefore be considered as valuable in emergency, when employed for its effect on blood-pressure. It is also well known that many cardiovascular cases stand digitalis poorly, particularly those having marked myocardial weakness. Caution is therefore always necessary during its administration. Fatal syncope has followed the overuse of digitalis (Brunton). Brunton also warns against its use in advanced Bright's disease, and in threatened apoplexy. He believes that the danger may be reduced by proper combination with vasodilators. The same author[2] tabulates the physiologic and toxic effects of digitalis on the circulation as follows:

A. Physiologic:

1. Increase in heart power.
2. Nervous irritability.

B. Toxic:

[1] *Interstate Med. Jour.*, Vol. XVII, No. 6.

[2] Therapeutics of the Circulation, 1908.

1. Heart muscle fails.
2. Vessel musculature fails, causing,
3. Increased blood-pressure, pulse slowed.
4. Blood-pressure stays up, pulse irregular and rapid.
5. Heart feeble, beat more regular.
6. Vessels dilate, blood-pressure falls.

J. M. Mackenzie,[1] on the contrary, found that only in exceptional cases does digitalis raise blood-pressure, even when carried to the physiologic limit. The only cases in which he found an increase in blood-pressure were cases of extreme dilatation of the heart with edema. Here a slight rise in pressure accompanied the improvement.

Caffein.—The immediate effect of caffein on the circulation is to elevate blood-pressure and to increase the heart rate. These effects were demonstrated by J. D. Prichard, in a series of pharmacologic experiments.[2] Large doses decrease cardiac tone and lower blood-pressure; while toxic doses may cause death by acute cardiac dilatation. Caffein must therefore be employed clinically in moderate doses only, when it has a more prompt, but less lasting action than digitalis. Coffee by the mouth or rectum has the same action as caffein, because of the presence of this drug in it. Tea also has the same action but to a less extent for the same reason.

Theobromin according to Mackenzie has an action similar to caffein, in that it raises blood-pressure and at the same time accelerates pulse rate.

Strychnin, in the light of recent pharmacologic studies and clinical investigations with the sphygmomanometer,

[1] Dis. of Heart, 1910.

[2] *Cleveland Med. Jour.*, Jan., 1912.

has no appreciable effect on blood-pressure, as it has very little effect on the tone of the vessel, but acts chiefly on the heart (Mackenzie).

Oxygen by inhalation is of particular value in the fall in pressure, occurring under prolonged anesthesia. Its administration is most valuable in emergency; where it has been shown to rise pressure from a dangerous 75 mm. to 150 mm. after ten minutes inhalation. In the nitrous oxid-oxygen anesthesia, the cessation of N_2O and the giving of 50 per cent. is followed by an immediate rise in pressure and a return to consciousness.

Nicotin.—While this drug is not employed clinically for its effect on a falling blood-pressure, nevertheless experiment has shown that next to adrenalin this is the most powerful blood-pressure elevator known (Mackenzie). In animals the effect of nicotin is shown by a slowing of the heart and a profound elevation of blood-pressure. The blood-pressure raising effect can be seen in the unaccustomed smoker. (See Page 68.) The relation of nicotin to the production of arteriosclerosis has been demonstrated by Careman, Aub and Briger[1] who have shown that nicotin in small doses (0.0035 gm. to 0.0075 gm. in cats) caused an increase in adrenalin secretion and a rise in blood-pressure.

[1] *Jour. of Pharm. and Exp. Therap.*, March, 1912.

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