HIGH BLOOD PRESSURE

ITS VARIATIONS AND CONTROL

A MANUAL FOR PRACTITIONERS

BY

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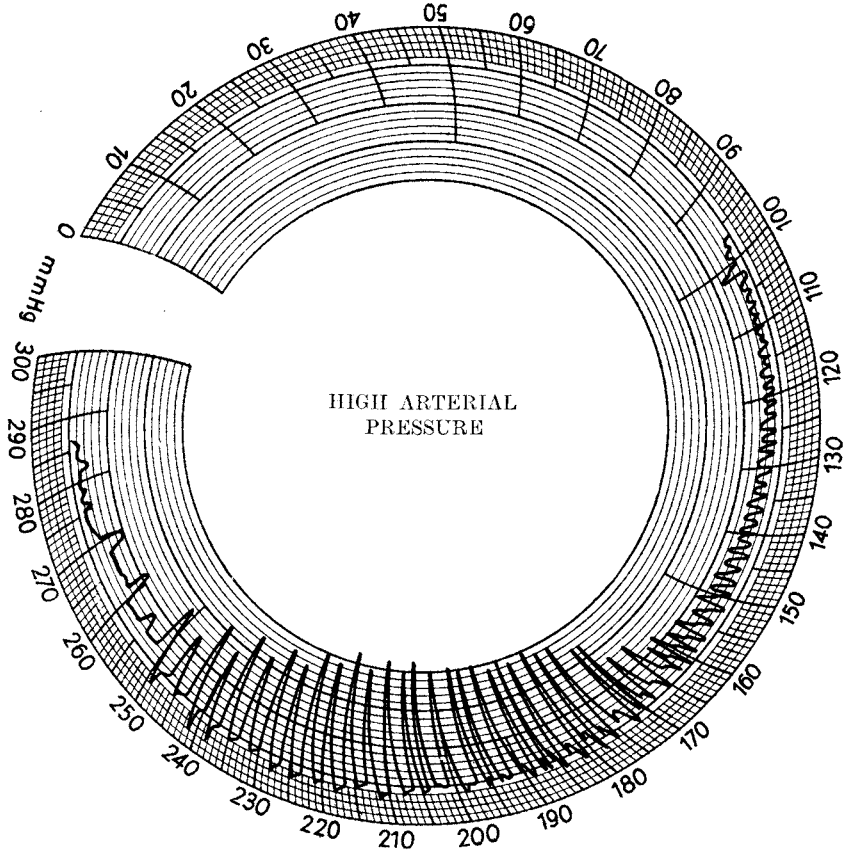

Frontispiece

GRAPHIC TRACING BY THE AUTHOR WITH THE TONOSCILLOGRAPH OF PLESCH showing high arterial pressure and *pulsus alternans* in a man aged 47. Systolic pressure is at 269 mm. Hg., diastolic at 151 mm. Hg., and mean pressure at 204 mm. Hg.

PIÆ MEMORIÆ

T. CLIFFORD ALLBUTT

HOC OPUSCULUM

J.F.H.D. —OLIM DISCIPULUS—

LIBENTER DEDICAVIT

PREFACE TO THE THIRD EDITION

It is highly gratifying to the author that the continued welcome given to this book by practitioners, teachers and students of medicine justifies the appearance of a new edition.

In the endeavour to meet the needs of those for whom the book is intended, no efforts have been spared to render it simple, concise and adequate, the chief aim throughout having been to treat " High Blood Pressure " in a practical manner.

To this end, and in order to present the subject in readily assimilable form, although the main outlines remain the same, the Third Edition has been so largely rewritten as in many respects to constitute a new book.

As the result of present-day research and enlarged personal experience, salient points have been stressed, much relevant matter has been included, and much that was becoming out of date deleted. Twenty-four fresh illustrations have been added, and seven old ones replaced.

Careful revision has been made of sphygmomanometric methods and instruments, particularly those that are objective and self-registering.

The significance of mean arterial pressure has been discussed, and standard pressures at various ages have been revised to accord with the latest observations.

The section on hyperpiesia has been considerably amplified in the light of modern work. In like manner arteriosclerosis and its relations with hyperpiesis have been dealt with.

The causation, significance, varieties, symptomatology, associations and effects of high arterial pressure states have been extensively considered, while prognosis and end-results find appropriate mention.

My grateful thanks are due to numerous friends for valued help ; in particular to Dr. Fortescue Fox, who finds nothing

to modify in his useful contribution on " Baths and Waters," and to Dr. Knyvett Gordon, who has kindly revised for me the section on bacteriological and histological diagnosis. I am also indebted to the Editors of the *Lancet* and the *Medical Press and Circular* for permission to utilise respective articles from my pen on " Graphic Blood Pressure Records " and on " Milestones in the History of Blood Pressure " as bases for corresponding portions of Chapters III. and XV. ; to Messrs. Hawksley & Son, Short & Mason, Ltd., the W. A. Baum Co. Inc. (New York), Boulitte (Paris), and Professor J. Plesch (Berlin) for illustrations.

Last, but by no means least, I have to thank my publishers, Messrs. Wm. Heinemann (Medical Books) Ltd., for their courteous and ever-ready help.

J. F. H. D.

LONDON, W. 1.
July, 1934.

PREFACE TO THE SECOND EDITION

THE very cordial reception accorded by the profession to the first edition of " High Blood Pressure " appears to have justified its presentation as a text-book intended primarily for the use of general practitioners.

The Second Edition includes the most recent work on high arterial pressure and the diseases of which it is a symptom. The whole book has undergone careful revision : several chapters have been amplified, whilst much that was hazarded tentatively in the previous edition is now stated definitely on the ground of further considerable experience and personal observation. Many new illustrations, charts and tables have been added.

The terms "hyperpiesis," "hyperpiesia" and "hypertonia" are still so loosely employed that in Chapter V. the author has endeavoured to clarify the relationships of these terms by a new classification based on fundamental physical laws.

In Chapter X. will be found a series of observations on simultaneous brachial pressures in pulmonary tuberculosis.

Biochemistry, which has made such important strides in elucidating many vital processes hitherto but vaguely appreciated, is rapidly adding its quota towards the better understanding of changes in arterial pressure under conditions of disturbed metabolism. This subject, as well as various modern modes of treatment, has in the new edition received due notice.

The author's grateful acknowledgments are due to many friends : in particular, to Dr. R. Fortescue Fox for kindly contributing an Appendix to Chapter IX. on " The Treatment of Arterial Pressure by Baths and Waters " ; to Dr. Henry Ellis for valuable help on the biochemical side and for useful suggestions on classification ; to Dr. Sidney Bontor

and to Dr. A. F. Bill (Davos) for renewed assistance in the arduous task of proof-revision ; to Dr. J. W. W. Adamson, Mr. H. L. Attwater and Mr. H. Robinson for various practical hints.

J. F. H. D.

LONDON, W. 1,
June, 1926

PREFACE TO THE FIRST EDITION

WITHIN recent times blood pressure has become the object of widespread and increasing attention. The reasons for this are not far to seek. On the one hand, the modern physician is studying his patients with greater care, and as an aid to accurate diagnosis and treatment makes more frequent use of the sphygmomanometer ; on the other hand, public interest has been aroused to the importance of high arterial pressure by reason of the greater prevalence of the causes that induce it. In the words of Warfield, " there can be no doubt that arterial disease in the comparatively young is more frequent than it was twenty-five years ago, and that the mortality from diseases directly dependent on arteriosclerotic changes is increasing."

The exigencies of daily professional work preclude lengthy study of the extensive, scattered, and to some extent inconclusive literature which has grown up around the subject of blood pressure, especially of late years. As is only to be expected in a matter which admits of no absolute finality by the very nature of the human elements concerned, measurements cannot be stated in precise mathematical formulæ.

The aim of this manual is to present to the general practitioner in condensed and applicable form the modern views regarding blood pressure, and thus, in some instances, to supply a lack which has been present since his student days.

The book has been written in response to numerous requests from medical men actively engaged in the practice of their profession for a concise and handy volume in which they can readily find expressed in simple terms the essentials which they require to know.

Beginning, therefore, with a consideration of basic principles and technique, I have developed my thesis so as to emphasise the importance of the true index of diastolic pressure and the necessity of recording by the most suitable

method what I have termed "the complete arterial pressure picture," thus gradually leading up to a conception of abnormal arterial pressures as symptomatic of various underlying causes, certain of which are known, whilst others are as yet imperfectly understood. In the light of these premises, I have dealt with the control of high arterial pressure, bringing it into line with the preceding contents in the endeavour to render the conclusions based on personal experience a guide and help in daily practice.

By reason of their importance, arterial pressures in pulmonary tuberculosis and in relation to life assurance have received special notice in separate chapters. A review of the historical aspects of blood pressure estimation has been taken out of chronological order and relegated to the end of the book, so as not to break the sequence of more practical issues, but at the same time to render it available to those interested in the development of modern methods and technique.

In a volume of this size it has been thought well to avoid loading the text with notes of the mass of literature consulted. Should the reader desire further information touching various matters upon which considerations of space have forbidden me deeply to dwell, a list of the chief references is appended. In medicine there is no "always" and no "never." Hence I crave indulgence if the reasons for the faith that is in me are not, on every occasion, set forth in detail, or if, within the compass of these pages, individual mention of other writers to whom I am also indebted has not always been possible.

To colleagues and friends who have helped me with practical suggestions I tender my grateful thanks, in particular to Dr. Sidney Bontor for much sound advice and valuable criticism, and to Dr. Peter Miles for kindly reading through the proof-sheets.

J. F. HALLS DALLY.

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LIST OF ILLUSTRATIONS

HIGH BLOOD PRESSURE
ITS VARIATIONS AND CONTROL

CHAPTER I

General Considerations

"Life is short; art is long; experience is fallacious, and judgment difficult."
HIPPOCRATES: *Aphorism I.*

"Every man's own reason is his best Œdipus, and will upon a reasonable truce find a way to loose those bonds wherewith the subtleties of error have enchained our more flexible and tender judgment."
SIR THOMAS BROWNE: *Religio Medici.*

MEDICINE is an art, and, since the human factor precludes reduction of clinical data to exact mathematical formulæ, the use of a single method offers no royal road to success.

The fundamental characters of the circulation are still acknowledged to be in the main identical with those portrayed by the immortal Harvey,[1] to whom, incidentally, the capillary circulation was unknown, yet, particularly during the past half-century, detailed investigation, aided by the employment of instrumental methods, has had the effect of modifying many of our previous concepts. Among the appliances which aim at a higher degree of precision than can be attained by the unaided senses, however specialised, of the physician, the sphygmomanometer has met with a large and increasing measure of recognition.

The following pages constitute an attempt to indicate the position of sphygmomanometry in clinical medicine, and at the same time to assess both its advantages and its limitations. A good servant but a bad master, its findings must ever be viewed in their due perspective. To disdain, however, to profit by its aid, no longer accords with the prestige of a self-respecting practitioner of the healing art.

Present-day clinical estimations of arterial pressure in man

are made by applying to some superficial artery an external pressure sufficient either to arrest the flow of blood entirely or to permit it during a part only of the cardiac cycle. The method ordinarily adopted is to inflate with air a rubber bag fastened around a limb over the chosen artery, usually the brachial above the elbow. The bag is connected with some form of pressure indicator, either mercurial or aneroid, whose scale is commonly graduated in millimetres of mercury.

In everyday clinical investigations the pressures at two phases only of the cardiac cycle are measured—the diastolic (minimal) and the systolic (maximal) pressures.

The diastolic pressure is the lowest pressure that occurs in any cardiac cycle. It may be taken as a measure of the peripheral tissue resistance and of the factors due to elastic contraction of the arterial walls.

The systolic pressure is the highest pressure that occurs in an artery during cardiac systole. It indicates the force of ventricular contraction, and varies widely in response to the numerous factors which influence blood pressure in general.

MacWilliam and Melvin [2] find that impressions derived from digital examination of the pulse as to (*a*) the pressure in the vessel between the beats (diastolic pressure) and (*b*) the extent of the change at systole (differential pressure) are in some cases specially fallacious and more deceptive than impressions as to compressibility of the pulse (systolic pressure). "An enormous pulse pressure (for example, 120 mm. in a case of aortic regurgitation) may be present when the finger fails to give any adequate conception of such a pressure wave along the artery, and the measured diastolic pressure may be very high when the impression given by digital examination is that it is rather low."

Whilst I am of opinion that *skilled* palpation of the superficial arteries is helpful where no instrument is available, and that the trained finger can gauge extremes with some approach to precision, nevertheless intervening pressures present many pitfalls, for, after all, it is only by instrumental methods that blood pressures can accurately be measured and, what is of even greater importance, recorded.

Thus digital investigation is inadequate. Further, it may even be most misleading in cases where the pulse is "small," where hypertonia is present, or where the limb is adipose. In short, for a large proportion of cases palpation of the pulse will afford no more exact information than that yielded, in the absence of a clinical thermometer, by a conjecture as to the height of a patient's temperature by the feel of the skin. To measure arterial pressure takes less time and trouble than to test the urine, and often proves of equal or greater value.

It is now generally realised that departures from physiological blood pressure standards are symptomatic of some underlying condition.

As regards transitory variations, abundant evidence is forthcoming that these constitute on the part of the organism an expression of reaction capable of modification within a wide normal range to meet the varying physiological needs of daily life.

Apart from such simple and well-recognised causes, we have to consider variations in blood pressure, whether temporary or permanent, which arise from pathological conditions the primary nature of which cannot always be fully determined. In like manner, these deviations are symptomatic of the underlying cause or causes upon due appreciation of which successful management must depend.

The pressor effect of supernormal arterial pressure would appear to be exerted primarily upon the central nervous system rather than upon the peripheral circulation. Impaired blood supply to the vasomotor centre in the medulla is admitted as a cause, but in many cases cannot be ascribed to arteriosclerosis of the vessels supplying the centre. The pathological cause, as suggested by Cushing, is of pituitary origin, and the primary causes inducing these effects, at any rate as regards hyperpiesia, are probably psychical or toxæmic.

Hence we are forced to abandon the older view that abnormal blood pressure of itself is a state which necessarily calls for drastic treatment by drugs in order either to raise the pressure when unduly low or to lower it when unduly

high, for if the nature of the cause be not understood, efforts on these lines will not only produce no improvement in the patient's health, but, on the contrary, are apt to induce subjective discomfort, if not indeed actual harm, even to the extent of culmination in a fatal issue.

In control of abnormal pressure states, the best prospect of relief to the patient is derived from close co-operation between the medical attendant and the consultant. In many cases improvement is necessarily gradual, so that careful assessment and revision of the effects of therapy at definite intervals are advisable.

Are the Results of Clinical Blood Pressure Estimation accurate?

Clinical estimations of blood pressure are approximate because they are indirect, and should not be regarded as exact in the strict mathematical sense. Nevertheless, thanks to the increasing perfection of present-day technique, it is now possible to determine both diastolic and systolic pressures with equal facility and accuracy, so that the results are almost as exact as those obtained in laboratory experiments by direct measurement, and for clinical purposes may be regarded as perfectly reliable.[3, 4, 5, 6, 7]

Carefully recorded blood pressure readings furnish us with information which is often of the greatest practical help, and, as personal experience grows, their value and importance become increasingly evident. Not only is this true for circulatory disturbances, but for many general diseases in the elucidation of which the sphygmomanometer is often of the greatest assistance and will save the practitioner from making many mistakes.

Being satisfied that the present-day methods of estimating arterial pressure are reliable, in the next place it is necessary to consider the value and significance of the results obtained. Properly to assess these we must know :—

1. The best practical method of taking arterial pressures (Chapter II.).

2. The fundamental factors in their causation and maintenance (Chapter IV.).

3. The physiological limits within which arterial pressures may vary (Chapter V.).

4. The causes and significations of departures from the physiological standards for age and weight (Chapters VIII., IX. and X.).

5. The nature of the steps to be taken when necessary to control such departures (Chapters XI. and XII.).

CHAPTER II

The Clinical Estimation of Blood Pressure

"I regard the measurement of blood pressure as the most important of all the resources that have been added to our armamentarium in the last fifteen years." Cabot, 1913.

When should Blood Pressure be taken ?

1. At the first examination as a guide to diagnosis, prognosis and treatment.

2. Subsequently at intervals to register progress and to ascertain the value of certain lines of management, particularly in (*a*) cardiovascular disturbances; (*b*) renal disease; (*c*) hyperthyroidism; (*d*) pulmonary affections, especially tuberculosis; (*e*) psychical states; (*f*) cerebral and abdominal injuries and diseases; (*g*) obstetrics, including eclampsia, and gynæcology; (*h*) anæsthesia and surgery, before, during and after operation; (*i*) intravenous injections for syphilis.

3. At any examination involving physical or mental fitness.

4. All subjects above middle age should be encouraged to present themselves for sphygmomanometric investigation at least once yearly, since one is thus enabled to discover when their arterial pressure is beginning to mount beyond standard limits, and to adopt measures of precaution by which in the majority of cases life can probably be prolonged.

The Standard Measure of Arterial Pressure

The generally accepted standard is the height in millimetres of a column of mercury, or an equivalent measure in instruments calibrated from that source.

The Standard Width of the Compression Armlet

The distensible elastic rubber cuff, encircling the arm and surrounded by an external inexpansible fabric, should have

a width of not less than 12 cm. when used for auditory determination of arterial pressure in adults, and of 9 cm. for children. If the cuff be narrower than the standard width, all readings will be too high. For infants and small children it is often convenient to measure the pressure in the femoral artery.

THE FIVE METHODS OF ESTIMATING ARTERIAL PRESSURE

All the methods of estimating arterial pressure fall under five main heads :—

1. The Auditory Method.
2. The Tactile Method.
3. The Vibratory Method.
4. The Oscillatory Method.
5. The Graphic Method.

1. The Auditory or Auscultatory Method

Since 1905, when Korotkow [8] suggested the estimation of arterial pressure by auscultation, this method, by reason of its simplicity, quickness and accuracy, has come to be largely adopted as a routine measure.

The Auscultatory Arterial Pressure Phenomenon.—On placing the bell of a binaural stethoscope (or an auditory tambour) over the brachial artery at the bend of the elbow and just below the zone of compression exerted by a circular pneumatic armlet, on gradually lowering the pressure within the armlet, a series of sounds becomes audible. These sounds, while presenting similar general characteristics, nevertheless, in individual cases, show variations from which useful deductions in respect of diagnosis may be drawn.

THE FIVE POINTS AND PHASES OF SOUND IN THE NORMAL AUDITORY CURVE

At certain points the character of sounds changes, the duration of successive zones of sound between any two successive points, as measured in millimetres of mercury, being termed phases.

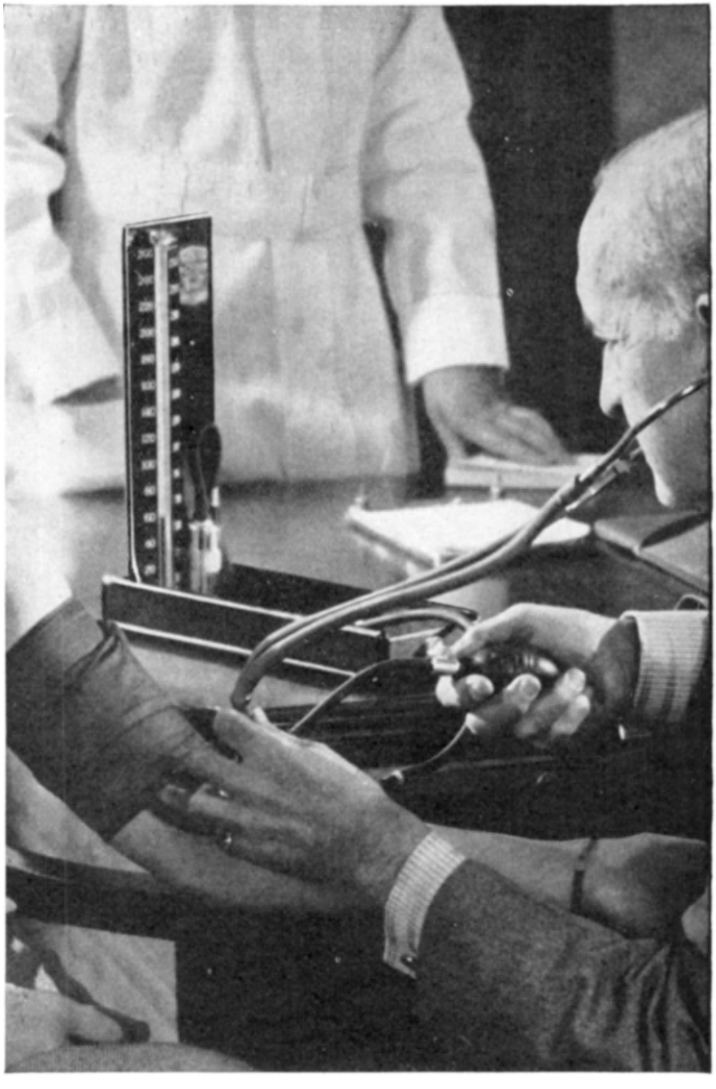


FIG. 1.—The auditory method of estimation of arterial pressure (pp. 7–17 and 25–29). The instrument figured is the latest form of desk model Baumanometer, which reads from 0 to 300 mm., and is the one most generally applicable to consulting-room and hospital work. Size $2\frac{1}{8} \times 4\frac{1}{2} \times 13\frac{3}{4}$ in.

As the armlet pressure is lowered, the five phases of sound [9] (Fig. 2) occur in the following order :—

1. Clicks.
2. Murmurs.
3. Thuds.
4. Dull Sounds.
5. Silence.

The Auditory Sequence of Sounds

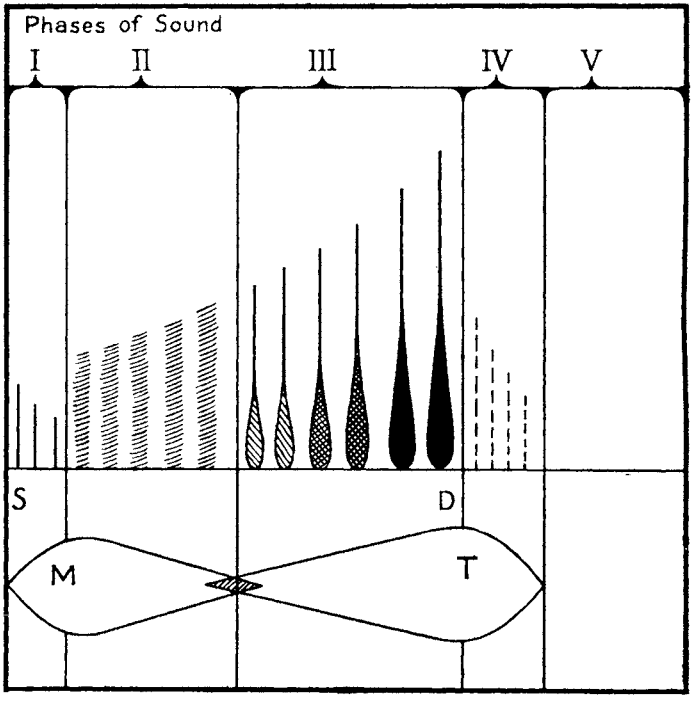


FIG. 2.—The five phases of sound of the auditory sequence. I, clicks; II, murmurs; III, thuds; IV, dull sounds; V, silence. M, zone of murmurs, caused by eddies in the blood current; T, zone of thuds, caused by vibrations of the arterial wall. S, level of systolic pressure; D, level of diastolic pressure.

1. The First Point.—The first point coincides with the appearance of the first sound or click. In reality, it measures the sum of the systolic pressure head, velocity head and such increment as may be due to water hammer.[10]

These clicks together constitute the first phase, which is usually only of a few millimetres' duration.

2. The Second Point.—The second point ushers in the second phase, and is recognisable by either the addition of a soft blowing murmur to the clear sound, or its entire replacement by the murmur. This phase is usually longer than the first one.

3. The Third Point.—The third point occurs when the murmur disappears and gives place to a zone of sounds somewhat similar to those of the first phase but louder, and becoming gradually more accentuated and throbbing in character. This crescendo sequence of clear thudding sounds constitutes the third phase of sound.

4. The Fourth Point.—The fourth point is reached at the instant when the terminal loudest thuds of the third phase abruptly lose their special quality and are succeeded by a zone, either short or long, of muffled and dull sounds, tailing off into silence. These weaker sounds represent the fourth phase, the *beginning* of which, *i.e.*, the first dull sound following the last thud, is the auditory index of the minimal (diastolic) pressure.

5. The Fifth Point.—The fifth point registers the disappearance of all sound.

Analysis of the Five Phases of Sound

Experimental and clinical researches on the production of the five phases of sound have been made by numerous workers,[10, 11, 12, 13, 14] in particular by Gallavardin [15, 16] and his pupil Barbier,[17] to whose writings the reader is referred for fuller details.

These observers have established the presence of *two zones* in the curve of sound: *one* occupying the upper half of the curve with its maximum near S, *caused by eddies in the* **blood current** partaking ordinarily of the character of murmurs; *another* occupying the lower half of the curve with its maximum near D, *composed of sounds originating in the* **vessel wall,** and whose intensity is clearly related to the state of excitability of the periarterial sympathetic system. Normally these two zones overlap in the middle

of the auditory curve which is the synthesis of these two zones of differing origin (Fig. 2).

Detailed investigation of each sound phase [17] reveals the following characteristics (Fig. 3) :—

First Phase.—The sounds are small and light. Frequently the first click is clearer and sharper than the few which immediately follow it. Very often the sounds speedily take on a murmuring tone, which relegates them to the second phase, the first phase then being much abbreviated.

The sounds are light because it is only the crest of the systolic wave which causes the walls of the artery slightly to separate with the least amount of vibration. The duration of opening of the artery is very brief, and the amount of blood in movement minimal, both of which conditions are unfavourable for the production of any considerable blood murmur. If the first click is a little stronger than the following ones, the reason is because it has to open up the obliterated artery, while succeeding waves have not to overcome a like inertia, since they come upon an arterial door which does not so readily shut.

The first sharp click, which denotes the beginning of the first phase, is the auditory index of the maximal (systolic) pressure.

Second Phase.—This zone of murmurs, with its phenomena more defined and easier to follow, is due to eddies in the blood current as it traverses the arterial constriction and drops from a raised pressure to a lower one on entering the uncompressed portion of the brachial below the armlet. Thus the murmurs of the second phase are related with a lack of pressure balance between the segment of the artery above the armlet and the segment distal to it, which is in a state of relative hypotension.

That these murmurs really originate in the blood is proved by the following observations : (*a*) An Esmarch's bandage applied to the middle third of the forearm induces an increase of pressure at the level of the distal arterial segment, and so has the effect of suppressing the murmurs.[10, 11, 12] (*b*) The murmur phenomenon is much more obvious with gradual decompression than with a rising external pressure. In the former, the first blood waves find the artery almost

empty, hence the murmurs ; in the latter, the progressive compression exercised by the armlet has from the outset hindered the escape of venous blood, whilst the arteries remain full ; the arterial segment distal to the armlet is embarrassed, its pressure is kept high, and murmurs are, therefore, absent. (*c*) The most important proof, however,

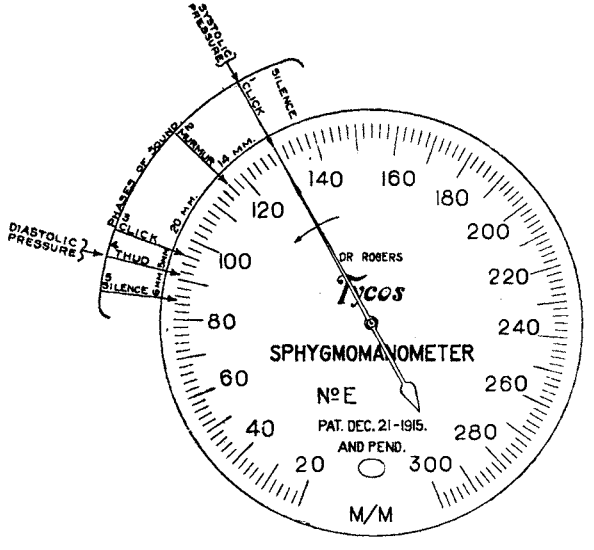


FIG. 3.—Diagram to illustrate the characteristic change of tone and approximate length of the phases of sound heard through the stethoscope.

1. The first sound. A sharp click. The index of systolic pressure. In the above diagram this phase covers 14 mm.

2. A zone of murmurs resembling heart murmurs. Is pictured here as lasting 20 mm.

3. A zone of clear thuds somewhat like the first sound, but generally louder, and continuing for about 4 mm. of the scale reading.

4. Dull sounds. The beginning of this fourth phase is the index of diastolic pressure. Of variable duration from 3 mm. even up to 55 mm.

lies in the fact that the murmur is a transitory phenomenon. After prolonged compression for twenty to thirty beats, the murmur lessens and then disappears, the armlet acting like an Esmarch's tourniquet in impeding venous outflow.[17] (*d*) The murmur can be brought back by promoting evacuation of the distal segment by light massage of the superficial

veins of the forearm or by movements of the hand. (*e*) The murmur zone corresponds to a period during which oscillometry registers only feeble expansions of the vessel wall.

Arterial tonus, nevertheless, can come into play even though the phenomenon be related to the blood much more than to the vessel wall.

Third Phase.—The sounds of the third phase assume more or less quickly a special quality. They become loud and intense, sounding "like the throbbing of a drum or the beat of a gong,"[16] and, after reaching a maximum, abruptly lose their special character. It is interesting to note that this series of thuds coincides exactly with the phase of brachial vibration, whilst the oscillometer needle during this period also manifests a definitely abrupt swing (p. 47).

During this phase the artery does not undergo complete collapse, but, opened by each larger systolic wave, resembles a door closed by a gradually relaxing spring and opening with a sudden gust of wind. Compression of the tissues caused by the armlet represents the spring closure, whilst the systolic wave represents the sudden gust of wind. The sounds produced in the wall of the artery as a result of this brusque opening are directly proportional to the forces of opening and closing. If the artery, feebly closed by an insufficient force, is violently opened by a strong wave, a loud vibrant tone is produced. The variable resistance against which the door closes is the armlet, which, adapted to the artery, with each expansion sustains an impact.[17] "A loud tone may be produced by a stiff artery and a slow stream, or by an elastic artery and a rapid stream."[18]

"The third sound phase is thus directly related to the tonus of the artery under investigation; normal or increased tonus will give for the same blood wave an arterial sound vibration much more thudding and loud than will an artery with flaccid walls, and it is for arteries with normal or heightened tonus that the simile of the tap of the drum can be employed with greatest accuracy. The sympathetic intervenes as the tightener of the arterial drum, regulating at each instant the sonorous properties of its wall."[17] This sympathetic action, evoked by the constriction of the armlet,

varies considerably in different persons, even under like conditions of blood pressure and heart rate. The proofs of this are that (1) normally the arterial sounds are more intense at the first than at subsequent readings. With repeated compression they diminish from minute to minute; the sympathetic, which at the outset has put forth its maximum action, flags, and the artery little by little loses its sonorous properties. (2) Rapid compression up to 300 mm. always induces a sudden increase in sound, which is transitory and followed by rapid diminution. Strong compression provokes energetic vaso-constriction, but the sympathetic tires more quickly in response to a single violent effort. (3) Application of local cold to the artery, as by a compress soaked in ether introduced between the arm and armlet, brings about marked reinforcement of the sounds,[19] for cold acts as a potent vaso-constrictor. (4) Injections of adrenalin or pituitrin cause also a temporary reinforcement. (5) Excess or lack of sympathetic tone finds in the arterial sounds a faithful mirror which renders the auditory method a simple means of investigating sympathetic action.

In irregular hearts there is marked difference in intensity between successive sounds ("tonal arrhythmia" of Goodman and Howell).[20] Loud sounds occur with rapid blood flow; and it is interesting to note that in anæmic states the sounds are also loud and clear, although here they bear no true relation to the cardiac energy. This paradox is due to loss of vasomotor tone in association with atrophy and lack of nutrition of the muscular coats. "In polycythæmia the sounds have a curious, dull, sticky character, and cannot be differentiated into phases. In not all cases can all phases be made out. It is usually the fourth phase which fails to be heard. In such cases the loud third tone almost immediately passes to the fifth phase."[18]

Fourth Phase.—*The change from loud thuds to dull sounds, which marks the beginning of the fourth phase, is the auditory index of the minimal (diastolic) pressure.* "It is the appearance of dull sounds and not the disappearance of all sound that indicates the diastolic pressure."[19]

The sounds are inconstant, dull, weak and sometimes barely perceptible, being proportional to the suddenness and

amplitude of the arterial oscillations. This phase is due to change from an external pressure sufficient to cause distortion of the circular tube to one insufficient to cause any flattening, *i.e.*, an external pressure equal to the internal diastolic pressure.

The duration of the fourth phase varies from 3 mm. even up to 55 mm. Hg, a short phase being usually associated with high pressures, whilst a long phase is often met with in normal or low pressures. The average length is much greater than formerly believed, and there is no constant relation to variations in pulse rate, systolic pressure or differential pressure.[21]

Fifth Phase.—The fifth phase of silence ensues when the artery is relieved from the compression exercised by the armlet.

Although the auditory series of sounds follows a broad general type, shown in Fig. 5, denoting good functional equilibrium between heart and arteries, yet notable individual variations may occur.

Anomalous Auditory Curves

The physiological theory advanced by Barbier[17] of a "mixed" blood and vessel wall origin of the sounds of the auditory curve explains the frequent variations and gives to each form of curve a diagnostic value.

I. **Variations in Intensity of Sounds produced in the Arterial Wall.**—A long and loud third phase betokens vigorous cardiac systole or a moderate degree of arteriosclerosis, but if the latter be of high grade or attended with calcification, the intensity of the sounds is much diminished. The sympathetic plays an important part also in arterial tonality, and specially in the genesis of vibration, which strengthens with heightened sympathetic excitability (hypersympatheticotonus), as in larval or developed Graves' disease, and weakens (*a*) in diminished sympathetic excitability (hyposympathetic tonus), after stimulation by cold or adrenalin ; (*b*) in all valvular or circulatory insufficiencies, including chronic heart-muscle weakness ; and (*c*) in arterial hypotonia.

Abnormal Persistence of Sound Production.—There may be persistence of sound through the normally silent fifth phase.

(1) In cases of collapsing pulse, even in the absence of compression by an armlet, sound vibrations are set up in the arterial wall. Their production depends upon reduction of diastolic distension combined with a considerable or sudden systolic increase of pressure head. Hence the loud "pistol-shot" sounds of aortic insufficiency in the presence of hyperpiesis, caused by vibrations resulting from sudden distension of the arterial wall—a water-hammer effect.

(2) With increase in sympathetic excitability causing arterial constriction, as in hyperthyroidism, and in slight arterial hypertonus.

(3) Sometimes, in the absence of disease, distinct, though weak, thuds may persist far below the diastolic level. Under certain conditions there may be persistence of sound after the cuff pressure has fallen below the pressure necessary to cause any flattening of the artery, but is still sufficient to diminish the arterial calibre from diastolic size as compared with the artery proximal and distal to the length of tube compressed.[10]

II. **Variations in Intensity of Sounds produced in the Blood Current.**—Prolongation of the second phase of murmurs caused by the blood current is due to (*a*) anæmic states, which constitute the chief cause. Sometimes, in extreme cases, a true thrill, accompanying the murmur, may be felt by the finger placed over the brachial artery, which thrill disappears along with the vibration at diastolic level. Exaggeration of the murmur zone may also be due to (*b*) sympathetic influence producing a localised arterial hypertonus; (*c*) modification of the arterial pulse of the nature of anacrotism. The murmurs may be entirely absent in hyperpiesis or aortic stenosis, the rate of arterial blood flow not being rapid enough to cause a murmur during the second phase. An *auditory gap* results, since there is nothing to supplement or replace the arterial sound which during that phase weakens or disappears.

In general terms it may be stated that modifications affecting the whole of the auditory curve are almost always

due to a central cause, cardiac or aortic, while modifications of a portion only of the curve are usually of peripheral origin, arterial in kind. Further, the auditory curve may be deficient at one or other extremity, or in the middle may present a complete gap, no sound being audible over a range usually of about 25 mm. in the position where one would normally expect the second phase. Instead of a gap only faint sounds may be appreciable.

2. The Tactile or Palpatory Method

In determining systolic pressure according to the principle of Riva-Rocci the usual manner of applying the tactile

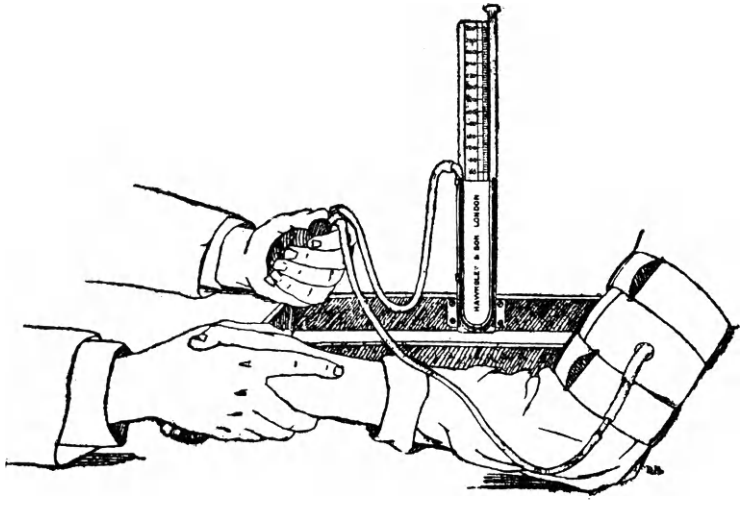


FIG. 4.—The tactile method of estimating arterial pressure by palpation of the radial artery.

or palpatory method is by means of the pressure bag gradually to compress one artery—the brachial in the arm—while noting the disappearance of the pulse by palpation over a totally different artery—the radial in the forearm. The pulse disappears at the moment when the blood stream no longer possesses enough energy to overcome the circular resistance opposed to it at the level of the compressing armlet, *i.e.*, when the pressure within the armlet is equal to the maximal arterial pressure at the given point. *On*

progressive decompression, appearance of the pulse is the tactile index of the maximal pressure. In order to avoid extra error the only *correct* way of radial palpation is to allow the patient's forearm to rest in a position midway between pronation and supination while the observer lightly grasps the patient's hand, at the same time palpating the radial artery with the extended index finger, as shown in Fig. 4.

Since the systolic pressure is gradually falling and the diastolic pressure is gradually rising between the brachial artery and the periphery, as confirmed by numerous comparative experiments which I have performed with different types of arterial pressure instrument,[22] the tactile method, *as usually practised*, has never seemed to me to give *exact* indications of either maximal or minimal pressures, and thus, for practical purposes, in the main has rightly been superseded by the auditory method.

Notable differences, which are not, however, necessarily constant, are at times found between arm and forearm readings (as also between the two arms, or between arm and leg readings). Such are apparently caused by local conditions of unequal contraction or hypermyotrophy. "Diminution in the calibre of an artery, if extreme, may influence the propagation of the systolic wave, but under the ordinary conditions of blood-pressure estimation in the arm this factor is a negligible one." [23]

Apart from local influences due to the state of the arterial wall, systolic pressure cannot be higher in the forearm than in the arm, and although there is no evidence of any *important* loss in transmission of the pulse wave from brachial to radial, since normal arteries, even under widely differing conditions of tone, possess good power of conductance of the systolic wave, palpation of the radial artery usually gives a reading *from about 2 to 10 mm. lower than the brachial systolic pressure, which is the pressure that in reality we wish to determine.*

I have laid stress upon this point because Barbier [17] believes that because of the anomalous auditory curves, which are apt to present difficulties, it is indispensable to supplement the information obtained by the auditory

method as to the exact situation of the systolic pressure by radial palpation, whilst MacWilliam and Melvin [6] use it as a check in the routine application of the auditory method. They state that the auditory reading should always be *at least as high* as the tactile (radial) reading, though very often it is somewhat higher. An auditory reading lower than the tactile one shows that the conditions are such as to invalidate the accuracy of both systolic and diastolic auditory indices, a result which is often due to faulty application of the tambour over the artery, anatomical variation of the latter, etc., and may be remediable.

Whenever possible I prefer a more exact way of assessing the normal relation between auditory and tactile systolic and diastolic indices by placing the finger below the bell of the stethoscope or auditory tambour and palpating the *brachial* artery, when *the first pulsation will be found closely to correspond with the first audible click*, which denotes the level of the maximal pressure, whilst the last notable vibration coincides with the last loud thud, which marks the level of the minimal pressure. Unfortunately, however, the brachial artery usually lies too deeply or is too embedded in fat for this procedure to be applicable, in which events recourse must perforce be had to radial palpation, which, although not as accurate, gives approximate and comparable readings. An addition of 5 mm. to the reading obtained by palpation of the radial artery will give a fairly accurate result.

With an intelligent patient it is often possible to check readings by the patient's own sensations. At the moment of the first click, a slight sensation may be felt beneath the armlet as well as an alteration in intensity of the throb at the beginning of the fourth phase.

Cases of nervous disorder attended by extreme tremor present difficulties in estimation by means of auditory and tactile methods, and it is not always possible to do more than approximate to an exact reading. Light hypnosis has been suggested, but possesses the disadvantage of cutting out the psychic factor, the degree of which at the onset it may be useful to determine, although for precise estimation, elimination of the psychic factor is desirable, and may largely be attained by determination of the *residual* pressure (p. 27).

3. The Vibratory Method

This method is really a modification for a special purpose of the tactile one, and is employed chiefly for the purpose of determining the diastolic pressure by palpating the brachial artery just below the armlet during decompression, when the finger perceives at first simple beats increasing in

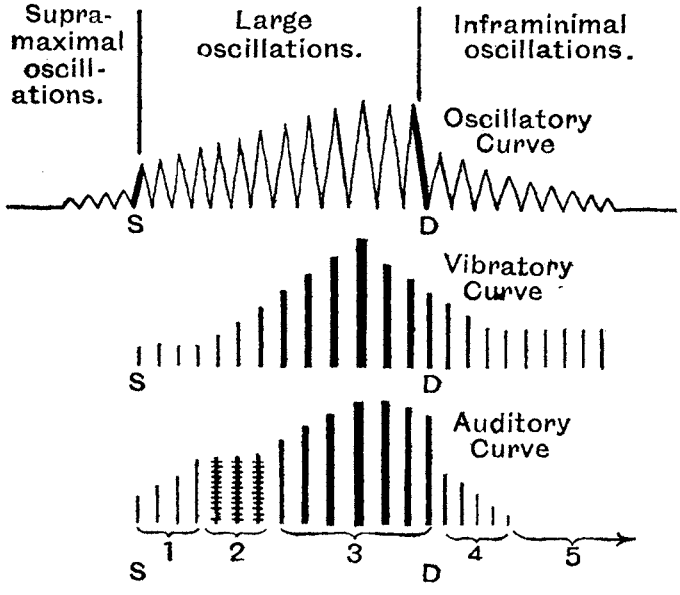


FIG. 5.—Diagrammatic comparison of curves obtained by the use of the oscillatory, palpato-vibratory and auditory methods of estimating arterial pressure. Curves to be read from left to right, showing vertical correspondences between the levels of S, the systolic pressure, and of D, the diastolic pressure. The figures below the auditory curve refer to the five phases of sound: 1 = clicks, 2 = murmurs, 3 = thuds, 4 = dull sounds, 5 = no sound. In comparison with Fig. 2, the third phase shows slight lessening in intensity of sounds towards the end, but the fourth point is well defined.

intensity shortly followed by a series of vibrations which, after attaining a maximum, decrease and then vanish. *This point of transition from vibration to no vibration marks* the change from the third to the fourth phase of sound and has been strongly advocated by Gallavardin [16] as a reliable means of estimating with accuracy *the diastolic index*. It

can also be employed as an additional check on the auditory method, especially in difficult or doubtful cases, or if the observer's hearing be not good. Observations in my wards showed that the diastolic pressure could readily be determined by this method in about 60 per cent. of cases where the ear was unable to gauge the point at which loud thuds gave place to dull sounds.

4. The Oscillatory Method

The oscillatory method is founded upon the original observation of Marey [24] (1876) upon the amplitude of pulsations of a segment of a limb subjected to decreasing circular compression. It is only satisfactory with clinical instruments of visual type possessing a large dial and a long delicate registering needle, such as the Tycos " Clinic Pattern " (which, however, does not register higher readings than 300 mm. Hg), or preferably with Boulitte's Universal Oscillometer, which operates with maximal sensitiveness, and is thus one of the most accurate aneroid instruments yet available. The oscillations of the little needle in the ordinary Tycos aneroid and other instruments of similar type are so tiny that it is difficult to differentiate the systolic oscillation from those which precede and follow it.

5. The Graphic Method

By the systematic use of this method many additional data of fresh value and significance have been afforded. In comparison with the foregoing, this latest advance possesses three important advantages in that it yields (1) automatic, (2) graphic and (3) permanent records of arterial pressures, as well as of individual pulse characters.

Apart from laboratory instruments of older date, such as those of Gibson or Erlanger (*vide* Chapter XV.), various types of clinical instrument have recently been evolved, namely the Tonoscillograph of Plesch, the Arterial Oscillograph and the Portable Registering Oscillometer of Boulitte, and the Tycos Sphygmotonograph.

Conclusions

Parallel in principle, the five methods of estimating arterial pressure are akin in their results.[17, 25, 26, 27]. For the large majority of cases—about 90 per cent.—the first four methods can readily be employed, even by a novice. In the remaining 10 per cent. sometimes they are difficult, and at other times, though rarely, impossible.

Each method naturally has its strong advocates, but in the cases which are really doubtful seldom is any one method found to possess distinct advantages over the others.

As a rule, the auditory method suffices, but although it is the most convenient and accurate yet discovered, nevertheless when used by itself to determine the systolic pressure it has some limitations which militate against its universal application.

The best procedure is that which is liable to the fewest errors, and since unqualified adhesion to the auditory method may mislead, I am in agreement in recommending for unusual cases **the employment of a combined auditory-tactile method,** which presents no added difficulties and possesses many and great advantages.

The True Index of Diastolic Pressure

Uncertainty has been felt in regard to the essential question as to what point in the series of auditory phenomena is to be taken as a true indication of diastolic or minimum pressure.

Korotkow [8] gave as his criterion the "end tone," *i.e.*, change from loud to dull sounds (Figs. 2, 3, and 5), which occurs at the beginning of the fourth sound-phase. Certain of the earlier observers who followed him regarded nevertheless the lower limit or extinction of sound (fifth phase) as the index, since it has been stated to call for the simplest technique, and to be more easily determined by unskilled observers than any other point. Many others again have considered that the true diastolic point must be somewhat higher, and that it should be placed just where the clear, loud note becomes suddenly dull and muffled or distant.

As the result of the investigations of Lang and Manswetowa,[28] Fischer,[29] Warfield,[18] Erlanger,[30] Taussig and Cook,[31] Weysse and Lutz,[32] supported by Oliver [33] in the third edition of his book, and more recently by Gallavardin,[16] any doubt in accepting the beginning of the fourth phase as the index of diastolic pressure has been swept away. The admirable and exhaustive critical studies of Professor MacWilliam, Spencer Melvin and Murray have further succeeded in establishing the correctness of this criterion on a firm and unassailable basis. From experimental observations (1) with a circulatory schema,[5] (2) with animals of sufficient size, such as the sheep,[7] and (3) on man,[2, 21] using concurrently the auditory, the tactile and the graphic method with Erlanger's apparatus, and comparing these with visual results obtained by employing the Pachon sphygmo-oscillometer with the brachial armlet, they find that "weakening and dulling coincides with the point at which the arterial tube just ceases to be flattened by the external pressure between the pulse-beats; . . . vibration associated with the sudden change in the shape of the tube is evidently responsible for the character of the sound," and that this point proves "a very accurate guide to the intra-arterial diastolic pressure as shown by the minimum manometer." They are satisfied that all the evidence as to diastolic pressure gained by other methods, and much more also, can be better obtained by the quick and simple auditory method.[6] Later the substantial correctness of systolic and diastolic criteria was verified by Erlanger [30] in a series of photographic records of movements of points on the exposed artery of the dog whilst undergoing decompression similar to that in clinical arterial pressure determination in man. He stated that the beginning of the first sound phase develops shortly after the compressing pressure falls below the systolic point, and that the change from sounds of sharp quality to dull sounds fairly accurately indicates the moment at which the compressing pressure falls below the diastolic.

Notwithstanding this volume of conclusive evidence (to which the reader in search of fuller information is referred) as to what constitutes the true criterion of diastolic pressure, in

the interests of statistical uniformity it is regrettable to find that certain workers, knowing the exact index, deliberately prefer the much less exact. Notably is this the case with certain American life assurance offices, all of whose medical examiners have been trained to take the diastolic pressure at the very end of the fourth phase, just before the beginning of silence, for the alleged reason that this point calls for the simplest technique, and is more easily determined than the end of the third phase.[34, 35] That such procedure is incorrect is amply proved by Melvin and Murray,[36] who have demonstrated that *the length of the fourth phase is subject to very considerable variation. It may be only a few millimetres, or may extend to* 55 *mm.* In the latter case, as they state, utterly fallacious results would be obtained by taking the lower limit of the sound as the diastolic index.

Personally I think that objections on the ground of expediency to acknowledging the beginning of the fourth phase as the true index of diastolic pressure are more fanciful than real, for, given average auditory acuity and reasonable care and attention, no difficulty is experienced in noting the exact transition from loud to dull sounds in a normal auditory curve. Practically the only cases which are likely to mislead the unwary are those in which the third phase is weak or absent as a result of insufficient blood flow through the brachial either from local congestion distal to the armlet or from loss of cardiac strength, and in aortic regurgitation, where the sound is often continued over the fifth phase. Often, too, in aortic regurgitation the familiar loud systolic thud and less frequently the double murmur of Duroziez are audible over the arteries not subjected to compression. The presence of such sound does not, however, as a rule prevent recognition of the diastolic index, the sudden weakening and dulling being usually very well marked.[6] In such cases the tactile systolic index should be employed simultaneously as a check.

The True Index of Systolic Pressure

If, whilst auscultating the brachial artery, we are able simultaneously to palpate it, we shall discover that the first

audible sound (the click) exactly corresponds to the first brachial pulsation. In this way we are enabled definitely to establish the first sound perceived as the criterion of systolic pressure. Failing this, there is a correspondence nearly, although not quite, as exact between occurrence of the first sound and of pulsation in the radial artery, the return of the pulse wave in the latter being from a few millimetres up to 10 mm. or so later than the appearance of the first sound. Hence the tactile method constitutes a useful check upon the accuracy of the auditory method.

TECHNIQUE OF ARTERIAL PRESSURE ESTIMATION BY THE AUDITORY METHOD

For any reading of arterial pressure to be accurate and trustworthy it is essential that the subject should be at rest, which implies both cessation from physical motion and from psychical disturbance. Such desiderata are best attained by not attempting to measure the blood pressure until sufficient time has elapsed for the patient to have calmed down from any excitement or apprehension incidental to the medical examination and to feel perfectly at ease.

At the first consultation it will be found most convenient to take the observation after the history but before the remainder of the clinical examination.

The following constitutes a description of the auditory method of estimating arterial pressure (vide *Fig.* 1), *which I recommend as the quickest, simplest and most accurate yet devised. It is applicable to any modern type of instrument :—*

1. The armlet should be smoothly and evenly adjusted as high up the arm as possible, the upper margin touching the axillary fold. The lower margin is thus brought well above the bend of the elbow. Care should be taken that the middle of the bag is towards the inner side of the arm, so as to ensure effective compression of the brachial artery. The armlet must not be applied too tightly. This matter is easily arranged with the older form of stiff outer cuff ; with the newer soft bandage-like armlet the folds should gradually be brought down so as to enwrap the whole

width of the pressure bag, and the last 2 inches of the tail neatly tucked under the preceding turn.

2. The patient's arm, which is bared, should be allowed comfortably to lie, with all its muscles completely relaxed, upon a support of such height that the armlet is brought to the same level as that of the heart.

3. At this juncture it is well to distract the patient's attention by determining with the finger the rate and characteristics of the pulse, since both arterial pressures and pulse rate should be taken under the same conditions.

4. Now explain to the patient that the band round the arm will tighten for a minute or so, but that this temporary pressure is quite harmless. Apprehensiveness on the part of a sensitive patient may be further allayed by slight inflation of the bag for a few seconds in order to accustom him to the novel sensation produced by compression of the limb.

5. Rapidly inflate the air-bag of the armlet to a pressure of about 110 mm. Hg.

6. Adjust the bell of a stethoscope, or an auditory tambour with rubber band for keeping it in place, below the armlet and over the brachial artery just above the bend of the elbow to the inner side of the biceps tendon, when in the majority of all cases, whether in health or disease, at or about a pressure of 110 mm. Hg, successive clear and loud thudding sounds will be audible as the blood passes through that portion of the artery which is partially constricted by the armlet.*

7. Quickly raise the pressure still further until all sound disappears, and the brachial and radial pulses are completely extinguished.

8. Slowly open the release valve, thus gradually lowering the external air pressure, and, according to the kind of instrument in use, note accurately either the height of the mercury column or the figure reached by the hand on the dial at which the first definite click is heard. **The first audible click on decompression following obliteration indicates the systolic pressure.** This point is a few millimetres

* A practical way of determining whether the tambour is in the correct position is to apply local increase of pressure, when, if the tambour is directly over the brachial artery, the radial pulse will be felt to lessen or disappear.

higher than the return of the pulse to the finger placed over the radial artery at the wrist.

9. As the pressure continues gently to fall, the clear, sharp click gives place to a soft murmur of variable duration, which is succeeded by a longer phase of clear, vibrant and sonorous thuds, which soon reach a maximum, finally becoming dull and muffled before total disappearance. **The point at which transformation of the clear, loud thud into a dull sound takes place indicates the diastolic pressure.** This change is usually sudden or rapid, but occasionally may be more gradual.

10. The mercury in the manometer is now allowed to return to zero by completely emptying the pressure bag of air and leaving it so for a few moments in order to permit the venous stasis to disappear and the circulation in the upper limb below the armlet to return to normal.

11. Discard the result of the first estimation, which should always be regarded as a rough approximation, and concentrate closely on the next two or even three readings, in order to determine the basic or residual pressure of the patient at the time of estimation by eliminating, through compression of the brachial, any element of hypertonia that may be present. Readings subsequent to the first are very often lower. Especially is this true of the systolic pressure, for the reasons given on pp. 29–32. Each complete estimation should not take longer than one minute. Continue until a constant level is reached, *the residual arterial pressure*. If there is no drop in pressure within three minutes, and the high readings are thought to be accidental, without telling the patient that the pressure appears too high, get him to return and again estimate the pressure levels on a second visit.

12. *The residual pressure, which is what one desires to record, is the lowest constant pressure reading.*

This should forthwith be entered in the notes of the case according to the author's formula, which constitutes **The Complete Arterial Pressure Picture (Fig 6).**

The complete arterial pressure picture includes :—

(1) The figure for the maximal pressure.

(2) The figure for the minimal pressure.

(3) The figure for the differential (pulse) pressure.
(4) The rate and characters of the pulse.
(5) The figure obtained by multiplying the differential pressure by the pulse rate.

A simple and compact formula is thus obtained, which can be expressed with rapidity and accuracy as

$$\frac{S.}{D.}\ D.P. : \text{pulse rate and characters} : D.P. \times P.R.$$

e.g., $\frac{165}{100}$ 65 : 78, small, irregular, wall thickened and tortuous : 5070.

In grouping large numbers of cases for statistical purposes into high, medium and low pressure classes, the graphic method of representation shown in Fig. 6 has been found to be of practical utility.

All comparative observations should be carefully made in a comfortably warmed and silent room, with the patient's limb in the same relative position of rest at the level of the heart, *as far as possible at the same time of day*, preferably midway between meals, *on the same limb and with the same type of instrument, all psychical disturbance being brought to the irreducible minimum.* The first observation is apt to be higher than the subsequent ones, and should always be corrected by others taken before the end of the first interview, when it will usually be found that the later readings tally very closely. The author's practice is to take at least three readings, and to record in the case-notes the results of the third or subsequent constant reading which denotes the required residual pressure.

I have seen so many erroneous deductions drawn through failure to comply with these few simple precautions that it cannot be too strongly urged upon every observer that reliable comparisons can only be instituted between blood pressure tests in the same and in different individuals when such are made under similar conditions. If the type of instrument be changed, this fact should always be noted, since all the older forms with narrow armlet, etc., give readings which are far too high.

Although the question cannot be regarded as definitely

settled, the consensus of opinion points to maximal and minimal pressures being a few millimetres higher in the standing than in the sitting position, and in the sitting position a few millimetres higher than when lying down.

Fallacies in Estimation of Arterial Pressure by the Auditory Method

A. **Sources of Error affecting both Systolic and Diastolic Pressures.**—1. Serious error may be introduced by unnecessarily prolonged compression during estimation, thus causing stasis of blood in the limb.

2. Error may be caused by the bell of the stethoscope or the auditory tambour not being placed directly over the artery, or by the bell being tilted at one edge, or by pressure upon it being excessive.

3. Error may be caused by the existence of an artery of small and insufficient size.

Unavoidable difficulties through any of the above causes should be of extreme rarity. If the chance of error be suspected, a combination of the tactile with the auditory method serves as a useful check in the determination of the correct indices.

The results which follow are entirely concerned with the estimation of systolic pressure, since *the diastolic reading is little, if at all, influenced by the chief sources of fallacy* regarding local conditions of altered conduction, resistance of arterial wall, or reflections from the periphery, *which may affect the systolic reading.*

B. **Sources of Error causing Over-estimation of the Systolic Pressure.**—These depend upon—

(*a*) *Factors External to the Artery under Compression.*

1. Considerable adiposity of the limb.

2. Œdema of the limb. These conditions are infrequent, and invariably prevent accurate readings.

3. Cyanosis of the limb. For general cyanosis no immediate remedy is applicable. Cyanosis due to local congestion induced by too lengthy compression of the armlet can be obviated by (α) rapid filling of the bag with air ; (β) taking care that decompression is not too prolonged ;

(γ) allowing sufficient time between each reading for any cyanosis to disappear.

4. Active contraction, tonic or clonic spasmodic states, such as cramp or tetany of the muscles of the limb. This source of error can be avoided by never taking the blood-pressure until and unless the limb is completely passive with its muscles at rest. In certain disorders of the nervous system, however, complete muscular relaxation is impossible, when perhaps one of the other limbs may not be affected and can be utilised for purposes of estimation.

(*b*) *Factors due to Variations in Resistance of the Arterial Wall.*

1. **Transitory Arterial Hypertonic Spasm** may be due to excitement and apprehension consequent upon the unaccustomed constriction of the armlet and painful feelings of swelling of the limb. These emotional effects are manifested by quickened pulse rate, and can be removed by calming and reassuring the patient as well as by shortening the time of estimation.

2. **Continued Hypertonic Contraction of the Arterial Muscle** definitely influences compressibility, but in the brachial artery a degree of contraction sufficient to cause an error of more than 30 mm. Hg is improbable in adults, and is usually considerably less than this, whilst in children it is negligible.

In common with other observers, W. Russell [37] has laid great stress on the state of the media, and has repeatedly pointed out the effect of arterial contraction on clinical readings. He believes that arterial hypertonus rather than hyperpiesis is mainly what is measured by the obliteration method. The results of his experimental work with rubber tubes and with arteries treated with formalin, etc., are, nevertheless, inadmissible in the case of living arteries, since the conditions are too dissimilar for comparison. Neither can his observations with vaso-dilators (erythrol tetranitrate, etc.) be accepted, since he appears to ascribe the reduction of the readings to relaxation of the wall of the brachial artery whilst ignoring the effect in lowering of systolic pressure due to opening up of the peripheral vessels by the drug.

3. **Calcification of the Arterial Wall.**—The common idea of calcification, wherever in the body it occurs, is that the process results in a condition of stony hardness. But MacCordick [38] has proved that *during life calcified arteries are not rigid.* At operation and at autopsies immediately following death such arteries are found to be pliable and to cut readily.

In the "pipe-stem" radial, for example, the calcareous matter is deposited, not in the intima, but in the media in a state which is identical with that of unset mortar. Mortar sets by conversion of calcium hydroxide into calcium carbonate by absorption of carbon dioxide from the air. Kept in alkaline media, or in bulk away from the air, mortar remains unset, but sets when placed in an acid medium. Identical changes take place within the body. During life the reaction of the body tissues and fluids is alkaline, but becomes acid shortly after death, and the gas which charges the blood within the arteries after death is mainly carbon dioxide. Hence "atheromatous ulceration and saucer-like plaques with rupture of the surrounding endothelium are *post-mortem* products. Calcified areas in the intima become set and rigid before muscular contraction of the aorta gives way, and the tube undergoes dilatation. The result is that tension upon the endothelium at the edges of the plaque induces rupture of the lining."

It is to the valuable work of Professor MacWilliam,[39] E. I. Kesson [4] and G. Spencer Melvin [2] that we are indebted for the most exact and useful knowledge we possess both as to verification of the criterion of diastolic pressure and influence of the arterial wall on blood pressure measurement. These observers, working on broader lines than their predecessors, have undertaken a series of clinical studies in the light of careful experimental data derived from investigation of excised living arteries in different conditions of contraction and relaxation, and of abnormal and thickened arteries as well as normal ones. From their clinical study they draw the following conclusions:—

"Estimations of systolic blood pressure by the obliteration method, when made with suitable precautions, give substantially correct results in ordinary conditions of normal

health and also in the great majority of cases of illness. Even when the disease affects the vascular system with thickened arteries, etc., the indications are in the majority of cases approximately correct, the readings ranging from moderate or low to very high values. It is only in a minority of cases that serious error is liable to occur, in the direction of over-estimation.

"In some such cases the influences of local conditions may be very important, especially the presence of abnormal resistance in the arterial wall, depending mainly at least on contraction of the muscular coat. In such conditions very different readings may be obtained from the same person on the same occasion according to the limbs or parts of limbs examined, the using of first or later readings, etc.

"Continued or repeated compression, with comparison of the two sides, etc., affords a valuable method of detecting the presence of such error, though not invariably decisive; in some instances considerable disturbances of blood pressure may occur. Results, both positive and negative, obtained by this method show a striking parallelism to those yielded by excised surviving arteries." [4]

Thickening of the arterial wall may be held to include atheroma, sclerosis of intima, and often of adventitia, or increase of media—the hypermyotrophy described by Russell,[37] Savill,[40] Dickenson and Rolleston[41] and others.

Upon compressibility arterial thickening has no appreciable effect, and calcification only a slight effect (p. 31); it is contraction that tells. Hence the whole matter may be summed up by the statement that *in the vast majority of arterial pressure determinations the state of the arterial wall is inconsiderable, whether in healthy or diseased conditions, but that, in some abnormally resistant arteries heightened readings of systolic pressure may obtain.* In these cases the method of preliminary digital compression of the brachial artery to remove resistance, as recommended by the above observers, is, in my experience, of the greatest value in minimising the error of over-estimation.

Method of Relaxation of Contracted and Thickened Arteries to secure a Corrected Reading of Systolic Arterial Pressure (*MacWilliam and Melvin* [6])

If resistance is experienced on palpation of the brachial artery, having taken the first reading, the brachial artery is subjected to repeated compression (twenty times), or, preferably, closed by digital pressure with two fingers for three minutes at the middle of the arm. Resistance of the arterial wall, if present, is thus almost entirely overcome, as shown by the second corrected reading, which is now made, being reduced to the level found in a non-resistant artery, but not below that level; further compression causes no further reduction.

This method does not equalise the arm-leg differences in reading associated with cases of well-marked aortic incompetence,[6] in which there is no correspondence between the degree of pulse pressure and the arm-leg difference.[42]

The Differential Blood Pressure Sign.—This term has been applied by Dr. E. F. Cyriax [43] to differences between the systolic blood pressure readings in the two arms. Such differences may be—

(*a*) *Anatomical,* where the arteries of the two limbs differ in size and distribution.

(*b*) *Physiological,* where the first measurement is heightened by psychic or physical causes, the effects of which have passed away by the time that the other arm is examined. If the patient lies on one side, the maximum pressure in the arm lying beneath is usually slightly higher than that in the arm above.[44]

(*c*) *Pathological,* where the two sides manifest differences as the result of aortic or other aneurysm, intrathoracic new growth, arteriosclerosis involving especially that portion of the aortic arch from which arise the innominate and left subclavian arteries, hemiplegia, or a cervical rib. In unilateral war traumatisms E. F. Cyriax [43] has shown that the systolic pressures in the upper limbs are not always identical, and that the pressure in one arm may be higher than in the other on one day and lower a few days later. In 819 observations on the blood pressure in the two arms

in seventy-three cases of unilateral or bilaterally unequal conditions produced by trauma and operation, together with a few of bilaterally equal operations and operations in the middle line, the differential blood pressure sign is nearly always present. For maximum pressures differences of 10 mm. were found in 83 per cent., and of 20 mm. or over in 12 per cent., of all cases ; for minimum pressures similar differences were found in about 80 per cent. and 20 per cent. In a second similar investigation, comprising 1,897 observations on 128 cases, in all the differential blood pressure sign was present.

E. F. Cyriax[45] believes that heightened blood pressure is often due to such irritative states of the spinal extensor muscles as hypertonicity, diffuse fibrosis or fibrositis and venous congestion, which are stated to produce a continuous series of sensory stimuli to the posterior spinal nerves, *i.e.*, a never-ending series of pressor effects which can be successfully treated by appropriate muscle kneading and other movements.

The results of 160 bilateral readings taken by Dr. R. J. Cyriax[46] in twelve patients with pulmonary tuberculosis are comparable, and will be found mentioned on p. 227.

When due to abnormal local conditions of the arterial wall, in a minority of cases the higher readings may be notably reduced by continued local compression, as already described, and in this way divergent readings may be harmonised, the same readings being then obtained from all the limbs. Comparison with a second reading from the other arm, which has not been subjected to repeated or continued compression, is essential, as the pressure may have altered during the period of estimation. No reduction in pressure is found in the absence of abnormality in the arteries.[2]

CHAPTER III

Present-day Instruments Employed in Estimation of Arterial Pressure

BLOOD PRESSURE INSTRUMENTS TO BE RECOMMENDED

The medical man who is about to purchase a sphygmomanometer naturally is inclined to ask what instrument is the most practical and will give him the best return for his outlay.

In Great Britain and America two types of instrument are in common use: A. mercurial, B. dial aneroid. On the Continent oscillometric aneroids C. are largely employed.

A. Mercurial Sphygmomanometers

No matter what claims are put forward by enthusiastic inventors or manufacturers of aneroid apparatus, it is certain that a properly constructed mercurial manometer is capable of registering pressures over long periods of time with greater accuracy and reliability than any instrument that depends upon the elasticity of metal. This does not mean, however, that the *average* mercury manometer used in practice is necessarily more accurate than an aneroid. The majority of mercurial sphygmomanometers in daily use till recent times have a narrow U-tube, along the distal arm of which is fixed a scale bearing equal divisions intended to read in millimetres of mercury. These scales, being stamped by a machine, make no allowance for variations which occur in the calibre of the glass tube at various levels. For the reason that it is a matter of great difficulty to blow a fine glass tube of uniform bore, no two U-tubes are likely to give similar readings on a machine-made scale. This is the first source of inaccuracy. A second

is found in capillary action of the mercury column. When tubes of fine bore are employed, the mercury, especially if at all dirty, tends to adhere to the glass, and gives readings which are inconsistent. A third possible source of error arises from the smallness of the intervals between the divisions on the measuring scale, which thus becomes difficult to read with precision, except in a clear light and by those who possess good eyesight.

The first of the above disadvantages can be overcome by individual calibration of each manometer tube against a standard, or by obtaining after repeated trials a tube of even and uniform bore throughout. The second and third disadvantages are alike obviated by making one arm of the U-tube shorter and of considerably larger diameter than the other, thus forming a reservoir. In this event the ratio between the diameter of the reservoir and that of the small arm of the tube must be accurately determined, and the scale attached to the latter must be compensated for any fall in level of the mercury in the reservoir. Manometers made on this principle are superior to those formerly employed in that glass tubing of three or four times larger diameter can be utilised, with the result that a much more open and clear scale becomes necessary to register the greater amplitude of range of the mercury column through given changes of pressure. The graduations of the measuring scale are thus larger and much easier to read, and because of the wider bore of the tube clean mercury no longer sticks.

The Baumanometer (Figs. 1, 7, 8).—This is the only mercurial sphygmomanometer within the author's experience which comprises all the foregoing mechanical advantages. In simplicity of construction and accuracy of registration it constitutes a distinct advance upon previous types of instrument, from which it differs in the size of the long arm of the U-tube, whose calibre is about four times larger. The shorter arm of the U-tube is expanded into a large standardised steel reservoir, the ratio of its bore to that of the long arm being accurately known. A scale, measuring in calibrated millimetres, each small division of which reads to 2 mm., and the larger divisions in tens, is engraved by hand directly on each accurately interchangeable glass

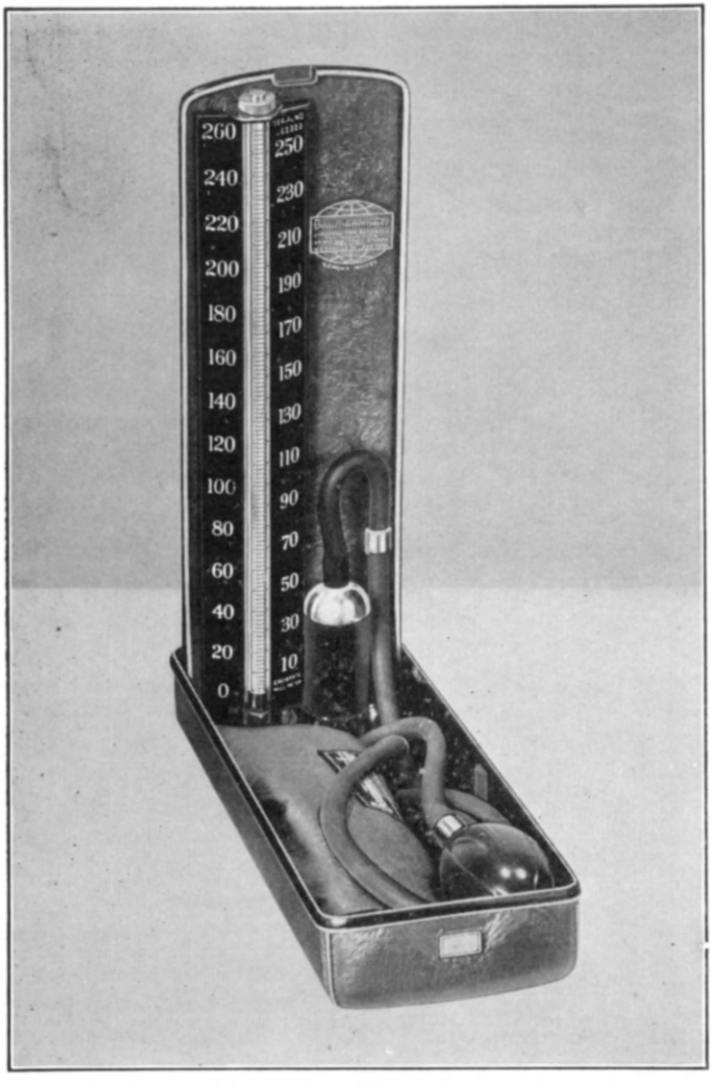


FIG. 7.—The Baumanometer (" Kompak " model) reading from 0 to 260 mm. On the grounds of accuracy, simplicity, permanence, portability and strength of construction this form meets the requirements of most general practitioners. The case is of cast duralamin. Size $1\frac{5}{8} \times 3\frac{7}{8} \times 11\frac{5}{8}$ in. Weight 30 oz.

tube, the figures in tens corresponding to the calibrations being placed alternately in white on a black background on each side of the readily cleanable tube. The scale reads from 0 to 300 mm. in the full-sized models, and is adjusted so that the level of the mercury in the reservoir and in the long arm of the U-tube when at rest stands at 0.

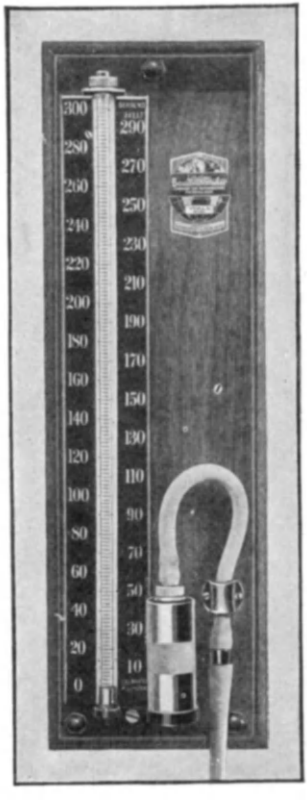


FIG. 8.—The Baumanometer. Wall model suitable for use in hospital or consulting-room (replacing the former cabinet model) reading from 0 to 300 mm. Size 4½ × 14 in., with 6 ft. of tubing.

The makers claim that every instrument is individually calibrated against a standard mercurial manometer, the accuracy of which has, in turn, been checked against the U.S. Bureau of Standards manometer at Washington, *i.e.*, that the scale of each Baumanometer is graduated by hand, so as exactly to compensate for any inaccuracies of the particular glass tube to which it is fitted.

Description of the Instrument (Figs. 7, 8).—To the open end of the long arm of the U-tube is screwed a small metal cap perforated with three small holes and containing a diaphragm. This device, whilst ensuring that the mercury cannot spill, allows air to pass into the tube, so that an even air pressure is maintained; otherwise the instrument would act as a compressed-air manometer and the mercury readings would be too low. To prevent the mercury escaping from the reservoir, a fine glass tube is sealed into its upper end, which is drawn out into a tube for attachment of the heavy walled pure rubber tube leading to the pressure bag. The latter is encased within a satin armlet and has

connected to it a shorter length of similar tubing, which ends in a rubber bulb for purposes of inflation fitted with a sensitive release-valve.

The whole apparatus is attached to the spring-lid of the case, and can be inverted without risk of the mercury being split.

Method of Employment.—1. Turn the milled knob on the outside of the case in a clockwise direction. With the other hand resting against the lid, allow it to swing gently upright. *Do not let the lid fly up suddenly.*

2. Hold the glass tube near the top with one hand, and unscrew the small metal cap with the other, but do not detach the diaphragm from the inside of the cap.

3. Carefully pour the mercury from the bottle sent with the instrument into the U-tube by means of a glass or paper funnel. Should any mercury be lost, place the Baumanometer on a level surface and add enough mercury to bring the rim of the meniscus even with the figure " 0 " on the scale. To judge this correctly, the eye must be on the same level as the " 0."

4. Screw the metal cap firmly in place. The Baumanometer is now ready for use.

5. In order to obtain greatest accuracy, it is important to tap the instrument, so as to ensure that the mercury takes up its proper position, and to stand the instrument on a level base. Errors due to incorrect levelling may be checked by examining the zero reading before putting the sphygmomanometer under pressure.

6. The further procedure is given in detail on pp. 25 to 29 (Fig. 1).

The above description refers to the standard desk-model. Larger and smaller sizes are also made. One defect common to the full-sized instruments as well as to other makes of mercurial sphygmomanometer is that they register only up to 300 mm. of mercury. The "Kompak" and pocket Baumanometers are still more restricted in range, reading as they do only up to 260 and 200 mm. respectively.

The latter instrument, however, is designed for the special purpose of examinations for life assurance, any patient whose arterial pressure reaches the 200 mm. limit being at once rejected.

Whilst it is true that pressures of over 300 mm. are very rare, nevertheless these do occur, and for purposes of general utility one should be able to measure them accurately, which is impossible until manufacturers of arterial pressure instruments realise that a limited scale, while covering ordinary ranges of pressure, is not capable of assessing extremes. At present, utility is subordinated to portability.

B. Dial Sphygmomanometers (Aneroid)

Of portable aneroid instruments suited for clinical work the improved Lauder Brunton apparatus and the Tycos are largely used in this country and in America. Various other forms of dial instruments with single broad inelastic armlet are employed in France, each of which possesses individual features. An early model was the Vaquez-Laubry sphygmotonoscope.[47] In current use are Boulitte's sphygmomanometer, the sphygmophone of Boulitte-Korotkow, and the arteriotensiometer of Donzelot,[48] all designed to register maximal and minimal pressures.

In the use of any of these instruments care should be taken that they are not inclined, since owing to lack of any interior balancing mechanism different readings will be given if the aneroid is sloped.

1. The Brunton Sphygmomanometer (Fig. 9).—This apparatus, suggested by the late Sir T. Lauder Brunton, F.R.S., comprises (α) a pressure gauge resembling an aneroid barometer, the dial of which (only 2 inches in diameter) is graduated in millimetres of mercury by comparison with a "standard" mercurial manometer, and registers from zero to 300 mm.; (β) a broad armlet, the rubber bag of which measures $13\frac{1}{2}$ inches by $4\frac{1}{2}$ inches; (γ) a metal inflating pump with release screw.

In modernised form, the dial gauge made by Jaquet of Basle possesses considerable accuracy. Great sensitivity is imparted to the movement by a double-acting pressure chamber and a specially constructed transmission.

Directions for Use.—Apply the armlet evenly to the arm. See that the screw of the T-valve is close home. Slowly raise the pressure in the armlet by the pump until the radial pulse

can no longer be felt. Then allow the air to escape gradually by reversing the T-valve screw, and observe the pressure

FIG. 9.—The Improved Brunton sphygmomanometer.

indicated on the scale at the time the pulse returns. This indicates the systolic pressure. The dial gauge, having a movable scale, can always be brought to the zero point before use.

2. The Boulitte Sphygmomanometer (Fig. 10) is the French equivalent of the American Tycos, over which it possesses

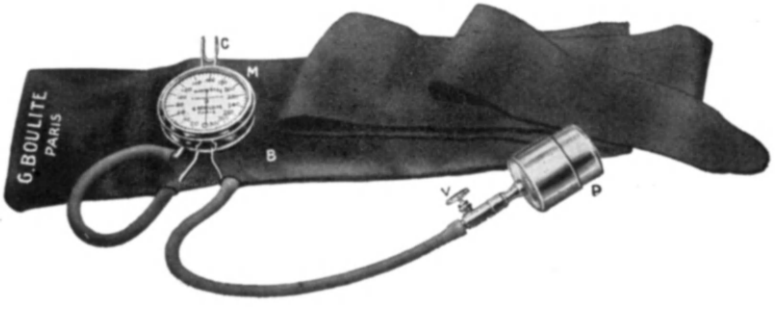


FIG. 10.—The Boulitte sphygmomanometer.

several advantages. These are a metallic pump; an easily adaptable silk sleeve of considerable length, which can be

wrapped several times round the arm ; and a dial-needle which, not being checked, permits independent control. The instrument is very light and handy. It is provided with a leather case which goes easily into the pocket.

3. The Tycos Sphygmomanometer.—This instrument,

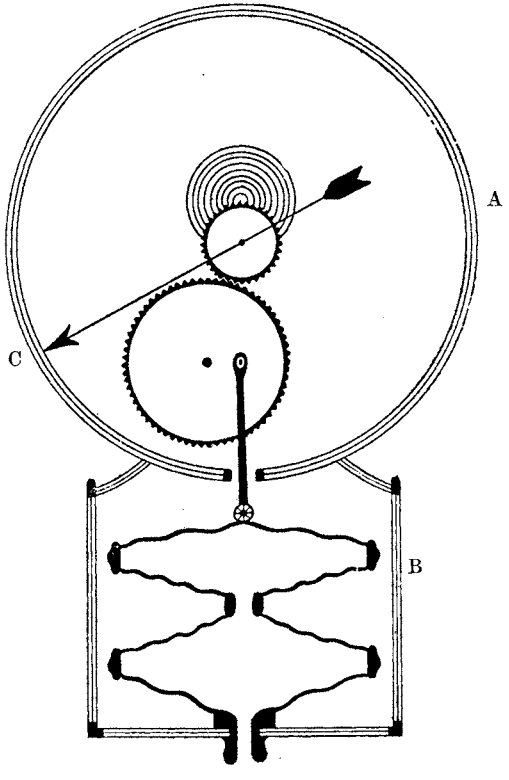


FIG. 11.—Diagram showing the internal mechanism of the Tycos aneroid sphygmomanometer (Norris). A. Dial case. B. Metal case beneath dial containing aneroid chamber composed of two metal discs connected by a lever affixed to a cog-wheel, which engages with a smaller cog-wheel and so causes movement of the dial needle, C.

originally devised by Dr. Oscar H. Rogers, is popular with the general practitioner chiefly because of its compactness and portability (Figs. 11, 12, 13). Two accurately ground metal discs are attached at their margins to the metal case beneath the dial. These discs constitute an aneroid

chamber, into which air can be pumped by the bulb, thus slightly separating them ; with the highest pressures only 2 to 3 mm. of expansion occurs, the measure of resistance of the chambers being accurately determined. A lever affixed to a cog, in turn connected with the dial needle, greatly magnifies the slightest expansion of the discs (Fig. 11). Each dial is made by hand, and the scale divisions on it are graduated against a standard mercury manometer, so that no two dials are exactly alike as regards spacing of the

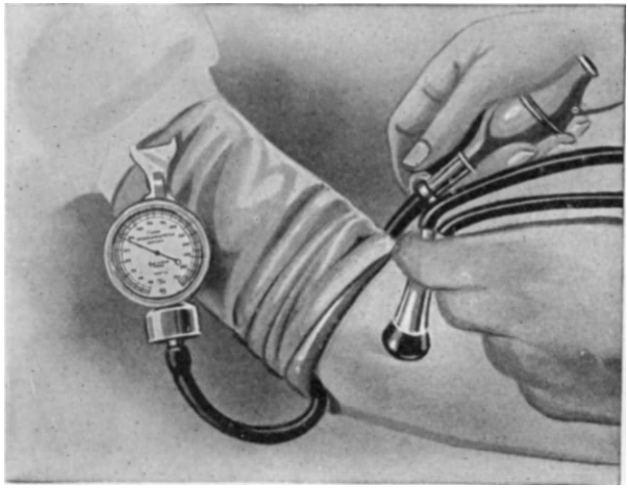


FIG. 12.—The Tycos aneroid sphygmomanometer employed in combination with the auditory method.

divisions. The relation of the needle to a zero point in the middle of an oval on the dial furnishes a check on the accuracy of the instrument. The dial is graduated up to 300 mm. Hg, and the 20-mm. spaces on it are accurately subdivided into ten equal scale divisions, each representing 2 mm. of pressure. The needle travels round the dial, so that its movements can readily be followed. In releasing the pressure these movements are visible before the sound can be heard, and are to be disregarded. They are simply due to beating of the blood against the upper edge of the armlet.

The instrument is unaffected by changes in temperature and barometric pressure, and, with reasonable care and occasional cleaning, should last for several years. At the end of every two years it should always be tested against a standard mercury manometer, since by this time it often

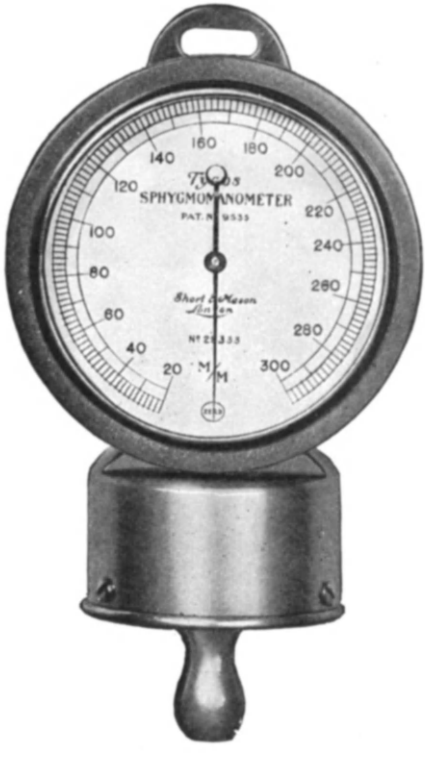


FIG. 13.—The Tycos sphygmomanometer, exact size.

needs readjustment, and is then liable to give readings which are too low in the case of high arterial pressures.

4. The Arteriotensiometer of Donzelot[48] (Fig. 14), designed for rapid, easy and precise measurement of arterial pressure, is well adapted for modern work. The cuff is inextensible, and is readily adjusted by means of two bands of webbing, each of which passes through a metal fastener which clips and secures them tightly. The rubber air-bag

is covered with silk, which produces no sensations of cold to the arm of the subject. The manometer is strongly made, and practically indestructible. It is graduated in millimetres of mercury, and allows estimation of maximal and minimal pressures to within 2 mm. No check is placed on the needle, which, at rest, freely returns to the centre of the oval, denoting zero, so that precision can thus be assured. By reason of this, the manometer is self-controlled, whereas, in ordinary manometers, a check being applied to the needle before its

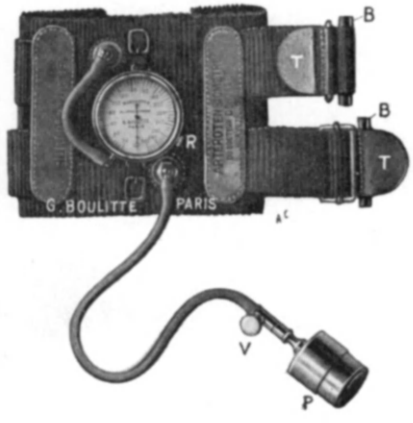


FIG. 14.—The arteriotensiometer of Donzelot.

position of rest may mask an error in the apparatus. A regulating button is added, by which any faulty position of the needle can be regulated as easily as the hands of a watch. The pump is of metal, and, because of its durability, superior to indiarubber bulbs. The escape valve is wedge-shaped, without intervention of leather or rubber, which ensures accuracy and sensitiveness, while a safety-catch prevents the screw from being opened too wide and thus lost. The stethoscope supplied with the instrument is of modern construction.

C. Oscillometric Sphygmomanometers (Aneroid)

1. The Sphygmo-oscillometer of Pachon.[49, 50]—The sphygmo-oscillometer (Fig. 15) consists of a cylindrical air-

tight metallic box which contains an aneroid capsule of aluminium. The rigid box, the contained aneroid chamber and the armlet communicate directly with one another by means of a three-way channel. The pressure within this system at any given moment is indicated on the dial of a small manometer graduated from 0 to 35 cm. of mercury. A separator key, on being depressed, closes the transverse limb of the three-way channel, and so allows pulsations set

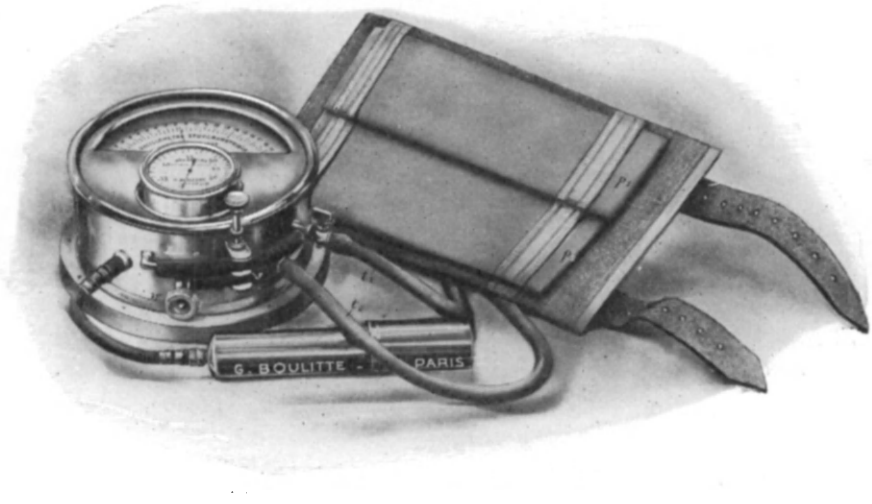


FIG. 15.—The Pachon sphygmometric oscillometer (new model) showing Gallavardin's armlet with two independent pressure bags p^1 and p^2, one of which overlaps the other by about one-third of its diameter; t^1 and t^2 = pressure tubing connecting the oscillometer box with the pressure bags p^1 and p^2 respectively; r = release tap to p^1; s = separator key; v = screw valve for release of external pressure.

up in the armlet to be transmitted direct to the interior of the aneroid chamber, and from this to the delicate registering needle. In use the instrument is connected, by means of a single narrow armlet with the *wrist* (Pachon), or by means of a broad double brachial armlet with the arm (Gallavardin). The pressure throughout the instrument is raised by means of the pump to a point above the maximal pressure of the subject. The separator key is then depressed and the registering needle watched. If no movement of

the needle occurs, the pressure should then be gradually lowered by opening the screw valve.

Note.—The separator key must never be touched while the screw valve is open, lest sudden decompression should injure the aluminium capsule, so that, in order to avoid the possibility of accident, it is advisable to use the right hand only to manipulate escape valve and separator key alternately.

The first of a series of **increasing** *oscillations of the registering needle indicates the maximal pressure*, S (Fig. 5). Before this point is reached small deflections of the needle, differing

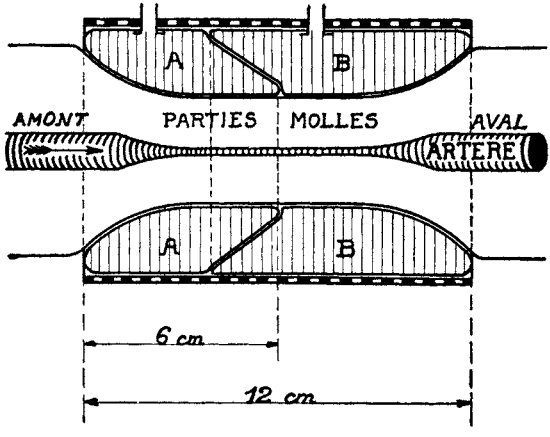


FIG. 16.—Represents diagrammatically in section Gallavardin's armlet inflated upon a limb. The artery is compressed uniformly as a result of the overlapping of the two bags.

little from each other, not infrequently occur (supramaximal oscillations, Fig. 5), especially in high pressure cases, but these are to be disregarded, and only the larger one, which constitutes the beginning of the zone of increasing oscillations, is to be noted. The pulsation which precedes this point and the one which follows it serve as controls.

As the pressure is further lowered the oscillations gradually increase in size, thus constituting a second zone distinct from the first. These large oscillations pass progressively to a point at which they attain their greatest amplitude,[25, 26, 27] the index of the "efficacious" or mean pressure,[56] afterwards decreasing in regular or irregular fashion to terminate

in a third zone of oscillations, only slightly differentiated from each other, the onset of which zone corresponds with the minimal pressure D (Fig. 5).

During the phase of gradually increasing oscillations, by watching the excursions of the needle, one can study the rate, rhythm, form and amplitude of the pulse. This in itself constitutes one of the distinct advantages of the oscillometer, since by its use arrhythmias of respiratory or juvenile type, premature contractions, paroxysmal tachycardia, and auricular fibrillation, etc., can readily be observed. The number of divisions on the oscillometer scale traversed by

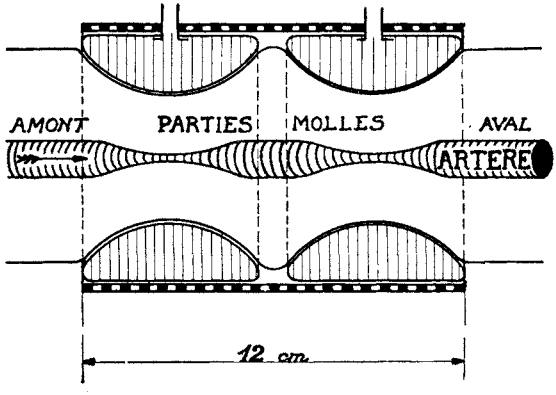


FIG. 17.—Represents diagrammatically in section how the artery is subjected to discontinuous double compression by two inflated bags placed side by side in the same armlet, but without overlapping. (Gallavardin.)

the needle in its widest excursion gives the pulse wave; *e.g.*, if the needle makes its greatest swing between 4 and 10, this represents six divisions on the scale.

Pachon's model has a pressure bag of 7·8 cm. in width, which is applied just above the styloid process of the radius. In consequence of this narrow armlet the readings are always higher than those of the standard mercurial manometer, the auscultatory readings of maximal pressure being from 10 to 20 mm. higher, but the minimal much the same.

Following Gallavardin's [51] suggestions, these higher readings, which, however, Pachon still holds to represent correct pressure values, have been brought into line with other instrumental records in later forms of oscillometer by

the use of two separate pressure bags in the same cuff, which overlap by about one-third of their width, and have a combined width of 12 cm. (Figs. 16, 17, 18). It can easily be demonstrated that these two bags actually do exercise uniform compression upon an artery by inflating the armlet over an empty bottle and seeing how the whole inner surface of the two bags constitutes a perfect cylinder with a very narrow circumferential line of demarcation between them. Each is connected with the oscillometer by pressure tubing, an air release being inserted in the circuit of the upper pressure bag. This arrangement allows

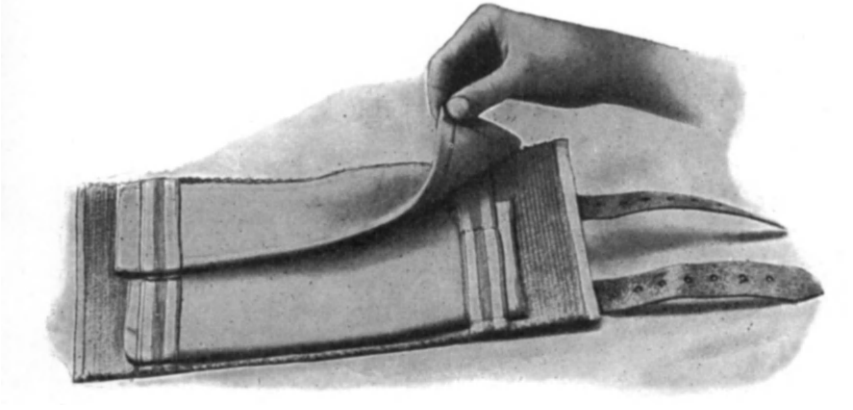


FIG. 18.—Gallavardin's armlet with two independent pressure bags, which overlap by about one-third of their width and have a combined width of 12 cm.

the observer to record separately the oscillations which are produced under the central portion and under the peripheral portion of the same armlet. The blood pressure is estimated in the brachial artery, instead of at the wrist with Pachon's former model of instrument, and gives readings comparable with those of the standard mercurial manometer.

2. Boulitte's Universal Oscillometer (Fig. 19).—This apparatus, an improved form of the older Pachon, is highly to be recommended both for its mechanical and physical qualities as well as for its clinical utility. It fulfils all requirements for precise estimation of minimal, maximal and mean pressures, is of much reduced size and weight, strongly made,

easy to manipulate, and difficult to derange. The needles of the manometer and of the oscillometer are each provided with a regulating screw, so that in the event of either needle assuming a faulty position, as the result of a fall, for example, it is not necessary to send the apparatus back to the makers. Friction is reduced to a minimum, and lag is absent. Hence the needle traces with fidelity the pulsations communicated to it, the amplitude of these under different conditions being exactly proportional to the strength of beat. Each instrument being of equivalent sensitiveness, accurate determina-

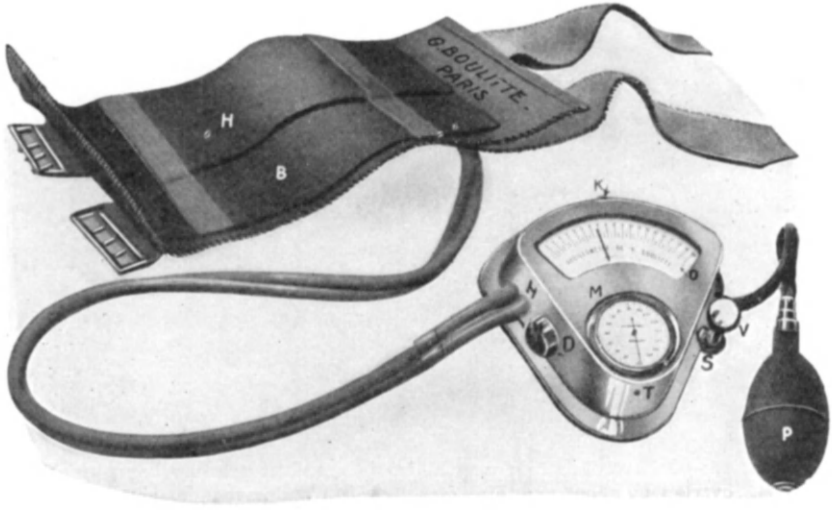


Fig. 19.—Boulitte's universal oscillometer with Gallavardin's armlet.

tions and true comparisons can be made of arterial pressures in the investigation of arterial permeability and elasticity. The apparatus is universal in that it can be applied to measurement of arterial pressure by any of the ordinary methods—oscillatory, auditory, vibratory or tactile. It is carried in small compass within a neat oblong leather case, without detaching the rubber tubes, so that it is always ready for service. In short, it is a good sphygmomanometer with many supplementary advantages.

3. The Kymometer of Vaquez, Gley and Gomez (Fig. 20).—These authors attach considerable importance to determination of the mean dynamic pressure in clinical medicine.[52]

They have, therefore, devised this special oscillometer furnished with a new contrivance by which the oscillometric needle always starts from the same fixed point. Thus in reading amplitudes the point marking the *extent* of the excursion is all that matters.

With this apparatus the reading of the greatest oscillation (oscillometric index) characterising the level of the mean pressure is rendered very simple.

Internal Mechanism.—The kymometer, in common with all the other oscillometers (universal, portable and arterial) made by Boulitte, consists of an aneroid capsule C enclosed

Fig. 20.—The Kymometer of Vaquez, Gley and Gomez.

within a rigid and airtight metallic box. The capsule is put into communication with the box by means of a valve S, so arranged that air can pass from box to capsule, but cannot pass from capsule to box (Fig. 21). Further, the capsule is connected with the cuff B by a two-way tube, one limb of which carries the escape screw V. A metallic pump P allows air to be driven into the box, the pressure within which, as well as that within the capsule, is indicated by the manometer M. This equality of pressure constitutes the chief feature of the apparatus.

When air is pumped into the box, this air pressure opens the valve S and gains the capsule. If, on the other hand, air is allowed to escape from the capsule by the escape screw V, the valve again opens and the equilibrium of pressure between the box and capsule is maintained. When the

escape screw is shut, and arterial pulsations distend the capsule, the valve then remains shut and the pulsations are transmitted to the delicate oscillometric needle. The valve, being extremely light but very solid, acts as an automatic separator (replacing the somewhat troublesome separator key in the older Pachon). At whatever moment V is shut

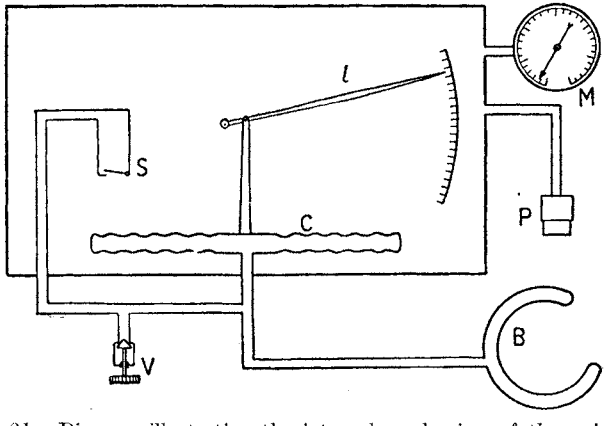


FIG. 21.—Diagram illustrating the internal mechanism of the universal and other sphygmo-oscillometers. (By courtesy of M. G. Boulitte.)

The rigid and airtight metal box contains an aneroid capsule, C. The cylindrical metal box, the aneroid chamber C, and the armlet B communicate directly with one another by the three-way channel.

Any desired pressure can be obtained through the combined system by means of the pump P, the pressure reading at any given moment being indicated on the manometer dial M, which is graduated in centimetres of mercury. An escape valve, V, allows the pressure to be released at will. The one-way valve, S, closes when the pressure becomes equal inside and outside the aneroid chamber, C, thus allowing pulsations set up in the armlet to be transmitted direct to the interior of the aneroid chamber and thus to the delicate registering needle *l*, the free end of which swings over a divided scale and so gives the amplitude of the pulse wave.

in order to read the amplitude, the box is in equilibrium with the lowest pressure existing in the capsule, *i.e.*, corresponding to the diastolic : the capsule is then at rest, the needle at zero and oscillations begin always from this point.

After the requisite pressure has been introduced, air is allowed to escape centimetre by centimetre while successively opening and closing the escape screw.

Records can be made simply and quickly of (*a*) maximal

blood pressure ; (*b*) minimal pressure ; (*c*) amplitude of pulse wave ; (*d*) pulse rate and characteristics, expressed in four sets of figures, or by means of a chart which furnishes a record of the amplitude of oscillation at different pressure levels.

The Oscillometric Curve

By an oscillometric curve is meant a diagram of pulsations of a limb or arterial segment subjected to a variable range of progressively increasing or decreasing external counter pressures. The construction of such a curve is simple. Upon squared paper two straight lines at right angles to each other are drawn, of which one (abscissa) corresponds with the figures for the counter pressure, and the other (ordinate) to the values of amplitude of oscillation read on the scale of the apparatus. In order to allow greater amplitude to the curve, the oscillations are noted on a scale double to that of the pressures. For each degree of counter pressure, upon the squared paper is marked a point situated at the intersection of the vertical line passing through the value of the counter pressure at the moment, and of the horizontal line passing through the figures of the ordinates equal to the size of the oscillations. All the points are then joined by a broken line, which forms the oscillometric curve.*

D. Self-Recording Sphygmomanometers

I. Indirect Clinical.

1. The Tonoscillograph of Plesch.—(*a*) *The Apparatus.* For many years it has been possible to obtain tracings of blood pressure by means of a kymograph. In clinical medicine, however, through lack of any satisfactory objective mode of registration, a numerical formula has had to represent " the complete arterial pressure picture " (p. 27). A distinct advance has now been made by Professor J.

* For further information on this and allied subjects the reader is referred to Pachon and Fabre, " Clinical Investigation of Cardiovascular Function." English translation by J. F. Halls Dally ; Kegan Paul, London, 1934.

Plesch,[53] of Berlin, who, after several years' experiment, has succeeded in perfecting the tonoscillograph (Fig. 22), which, in estimating arterial pressure,[54, 55] affords many substantial advantages as compared with the ordinary types of apparatus. I have done much work with this instrument since 1929, and prefer it to any other recording machine, since it is of such delicacy and mechanical perfection that the tracings obtained suffer no deformation.

Tonoscillograms produced by this apparatus enable

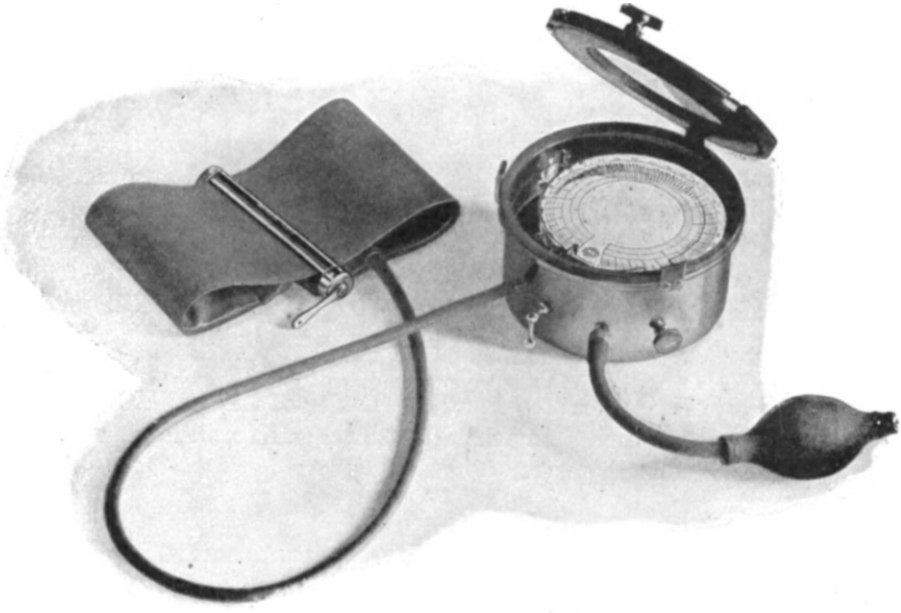


FIG. 22.—The Tonoscillograph of Plesch.

variations in the arterial pressure of any individual to be accurately traced under different conditions. The curves not only yield graphic and permanent records of arterial pressures and of individual pulse characters, but also enable the state of the vascular system to be diagnosed and the oscillations to be measured with a higher degree of accuracy than is possible by any other indirect method.

The taking of an objective tonoscillogram does not occupy more time than is required in the ordinary subjective method

of determining the blood pressure, while the record obtained forms a valuable addition to the clinical chart.

The apparatus is constructed of metal throughout and, being operated automatically by increase or decrease in pressure without any special driving mechanism, is reliable in working and practically free from error.

Like most instruments of great precision, however, the tonoscillograph is delicate in adjustment and requires aptitude in manipulation. Hence, although small and light enough to be portable, it is more applicable for static use in

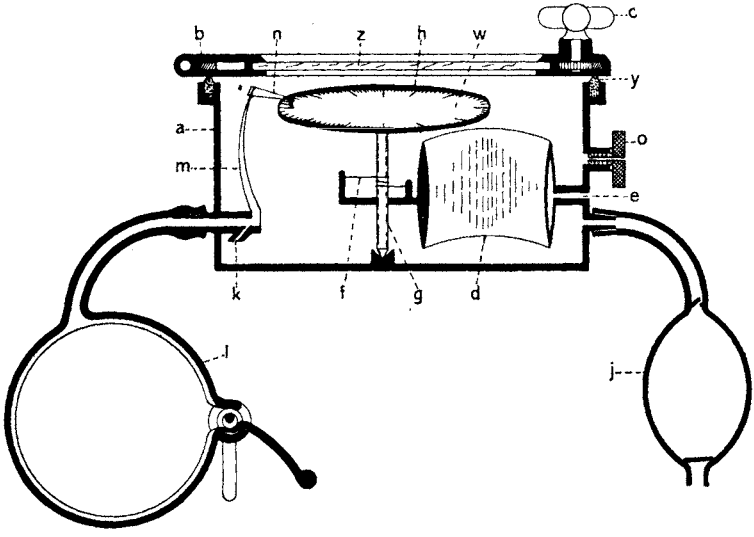


FIG. 23.—The tonoscillograph in vertical section (diagrammatic).

the consulting rooms of those who make a special study of circulatory phenomena than for daily transport on a round of visits to patients.

Fig. 23 is a diagram of the apparatus in vertical section. The outer metal case (*a*) is made airtight by the hinged lid (*b*), in which is set a glass window, and is fastened by means of the device (*c*). Inside the case (*a*) are two manometer systems. One of the manometers (*d*) communicates through the aperture (*e*) with the outer air, so that the interior of this manometer remains constantly under atmospheric pressure. When the pressure within (*a*) is increased by the

rubber inflating bulb (*y*) the manometer (*d*) becomes bent, thus causing the thread (*f*) to be unwound, thereby rotating the table (*h*) fixed to the spindle (*g*). The other manometer (*m*) has a capillary opening (*k*) into the case (*a*), and is in direct communication with the armlet (*l*). By pumping air into the case, the armlet simultaneously becomes inflated through the capillary opening (*k*). In this way the thin-walled and highly sensitive manometer tube (*m*) is protected from over-distension. Nevertheless, by means of the capillary opening, equalisation of pressure is secured, so that the external and internal pressures remain the same.

Attached to the manometer tube (*m*) is the writing pen (*n*), which records any movement directly on a graduated paper disc set on the table (*h*). Any increase in pressure caused by the pulse will, therefore, be recorded by this pen before the difference can be equalised through the capillary opening (*k*). The size of this aperture can be regulated by a lever ; it will readily be seen that the stronger the pulse and the smaller the capillary opening the larger will be the oscillations recorded by the pen. Should these oscillations become unduly large in the case of powerfully beating pulses, they can be damped by increasing the size of the capillary aperture. The valve (*o*) facilitates even reduction of pressure in order that the disc may return to the zero position. During the time that the pressure is falling the pen (*n*) automatically, and in true accordance with the pulse volume, traces impulses, communicated by means of the armlet, on the disc in the form of a curve, the tonoscillogram.

Fig. 24 shows the apparatus viewed from above. At the top is the hinge upon which the circular metal lid surrounding the glass window turns in opening and closing. Opposite to this at the foot is the capillary lever mentioned above ; to the left of this is a nozzle for the rubber tube connecting with the armlet, to the right an attachment for the inflating bulb ; still further to the right a screw-valve for securing even release of internal pressure. The paper disc (25), graduated from 0 to 300 mm. Hg, rests upon a rotating aluminium table (23), the edges of this being represented in the diagram by broken lines. Tiny projections around its circumference hold the paper disc flat. Opposite (0) is the

writing pen with angled supports by which it can be raised or lowered on a transverse bridge (31) actuated by the capillary lever. To the left of the bridge is a spring (30) for arresting movements of the table.

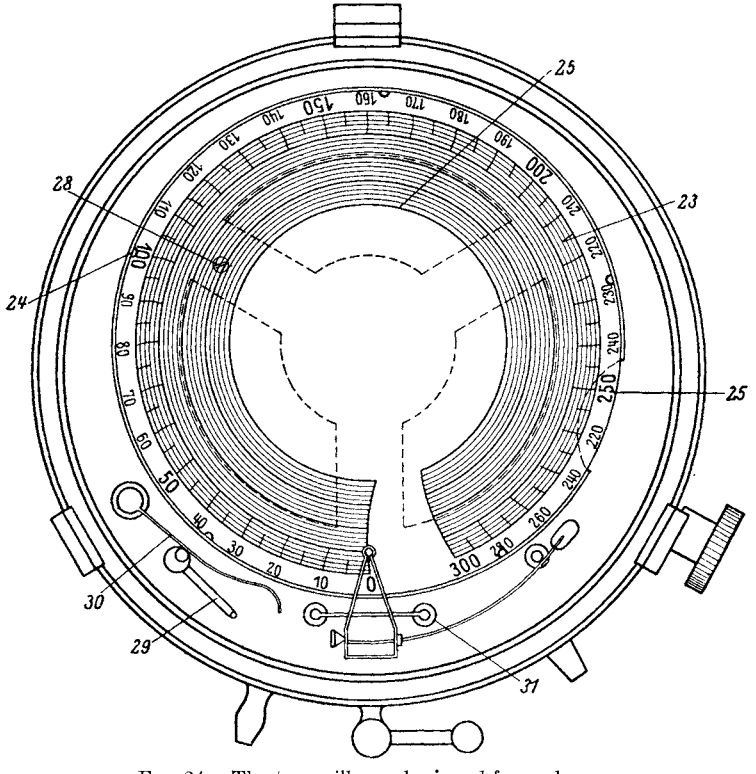


FIG. 24.—The tonoscillograph viewed from above.

Fig. 25 is an analysis of the curve of arterial pressure and volume obtainable with the instrument; the general form of the curve closely resembles those illustrating a paper of mine in 1911 on the sphygmo-oscillometric method of Pachon.[50] As the external pressure is allowed gradually to fall, an approximately even series of small undulations (A) appears as each successive pulse wave impinges against the upper point of arterial constriction. The point for the systolic pressure (S) is marked by the first of a series of slowly increasing waves which, after a varying stage (B) at

the point (P) suddenly become almost double the size of the preceding waves. From this point up to the largest oscillation (C) the increase is much more gradual. Thence onwards the curve is flatter in type, the oscillations slowly diminishing to the point of diastolic pressure (D), at which they undergo

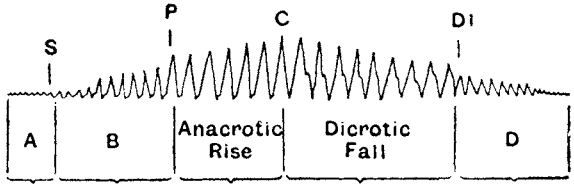


FIG. 25.—The Arterial Pressure Curve, showing successive stages of approach (A), development (B), oscillations (P to Di), and pulsations (D). S = systolic pressure; Di = diastolic pressure; P = point at which pulse waves increase to nearly double the height of the preceding pulse waves; C = mean pressure, *i.e.*, the greatest amplitude of oscillation (Oscillometric Index of Pachon).

a sudden reduction by half. Throughout the whole of the ascending portion of the curve each oscillation is seen to be separated by a flat interval, which becomes less as the highest oscillation is approached. Thereafter the oscillations assume a peak-like ascent. During the ascending portion of the

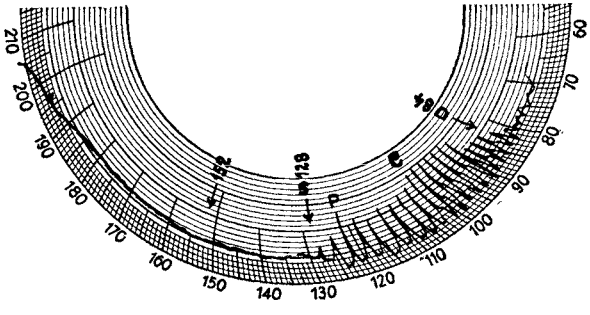


FIG. 26.—Tonoscillogram of healthy man aged 47.

curve the oscillations are anacrotic in form. During the descending portion they are dicrotic, but only as far as the point of minimal pressure. Beyond this point, the external and internal pressures being equalised, the pulse waves are represented by the stage of pulsations.

The foregoing stages are exemplified in the accompanying curves of arterial pressure and pulse volume taken from cases under my care (Frontispiece and Figs. 26, 27, 28, 29).

In the case of a healthy man, aged 47 years (Fig. 26), the stage of approach extends from 152 mm. Hg. to the point of systolic pressure (S) at 128 mm. Next comes a short developmental stage, denoting healthy tone of arterial walls, which ends at 124 mm. (P), from which point the stage of full pulse oscillations with flat anacrotic waves is continued, the intervals becoming less spaced and flattened at the base as they approach C. Following the ascending portion of

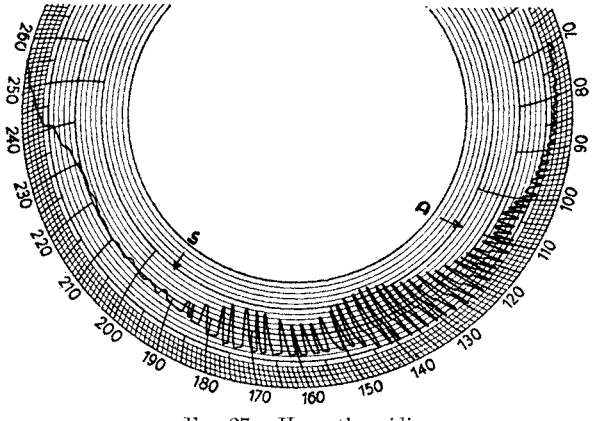


FIG. 27.—Hyperthyroidism.

the curve ending at C, the waves become more uniform in amplitude (descending portion) with evident dicrotism, this ceasing abruptly at 84 mm., the point of diastolic pressure (D). The stage of pulsations completes the curve-analysis.

Fig. 26 is a case of early but typical hyperthyroidism in a lady aged 36, seen in consultation with Dr. R. Tudor Edwards, of Wembley. Herein the various stages of the arterial curve are clearly recognisable.

Fig. 28 is the tracing of a man aged 60 years, 6 feet in height and weighing 15 stone, a teetotaller and non-smoker, athletic in his earlier years and fond of mountain climbing. The chief complaints were of " thumping in the head while in bed," and of pressure feelings referred to the vertex.

The heart showed considerable left ventricular preponderance and a systolic murmur was audible over the whole præcordium with ringing aortic second sound—a case of arteriosclerosis. S is at 194 mm. Anacrotic ascent ends at 150 mm. Descent is gradual to D at 104, where dicrotism suddenly ends. Well-marked pulsations follow this point.

The Frontispiece clearly illustrates high arterial pressure in combination with *Pulsus alternans*. The tracing is of a man aged 47, whose systolic pressure was 269 millimetres of

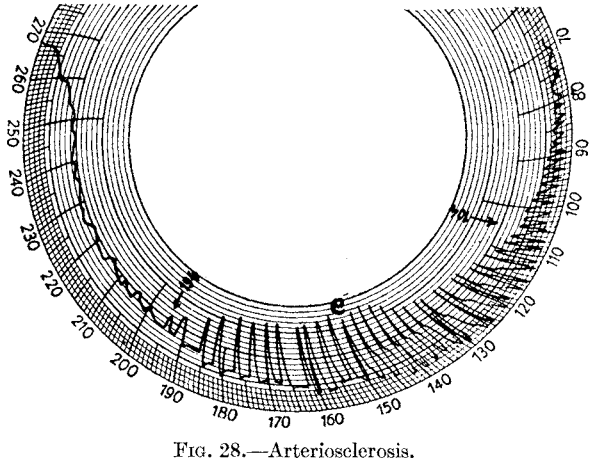


FIG. 28.—Arteriosclerosis.

mercury, diastolic pressure was 151 and mean pressure 204.

In striking contrast to all other curves that I have yet had the opportunity of taking comes Fig. 29, that of a hospital patient, aged 67, housewife, whom I showed in 1925 at the Royal Society of Medicine [56] as a case of hyperpiesia, since established to be of gouty origin.

The later clinical picture was one of cardiac dilatation consequent upon hypertrophy, and changes in the vessels arising independently of renal changes such as occur in the usual forms of Bright's disease. In February, 1930, hæmorrhage occurred in the left retina with impairment of vision two days later. One month after this blurring of speech was noticed, which persisted. The arterial curve is of

unusually bold character, despite the fact that the capillary lever was turned to the full extent so as to damp the amplitude of oscillations. Each pulse beat is seen to be enormous, corresponding clinically with a pulse of full volume and high tension. At every third beat is a regular sequence of premature contractions. The systolic pressure is at approximately 306 mm., but cannot be precisely fixed because this

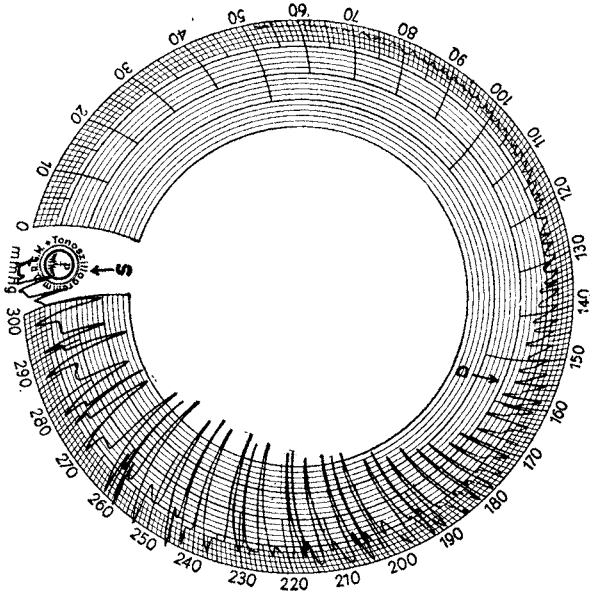


FIG. 29.—Hyperpiesia with secondary changes in heart and vessels.

sphygmomanometer, like the majority of others, has been constructed on the assumption that pressures above 300 mm. Hg cannot occur. This patient's pressure, however, when first seen was 320 mm. systolic, and I have had other cases in my own practice with maximal pressures well above 300 mm. The diastolic pressure is reached at 155 mm., giving a differential pressure of about 150 mm.

2. Boulitte's Portable Recording Oscillometer (Fig. 30).—To Boulitte's Universal Oscillometer (K) is added an oscillographic capsule (C) provided with a pen (J), writing in ink and sensitive enough to record even the weakest pulse. A

clockwork movement (N), regulated by a button (B), propels in a straight line horizontally at requisite speed a roll of paper (E), squared in millimetres.

The record is made in two portions : the first by setting the distributor key at Mn, thus placing in communication the two pockets of the Gallavardin double cuff. This permits estimation of the mean and minimal pressures (Fig. 31) ; the second, by cutting out the upper pocket of the cuff,

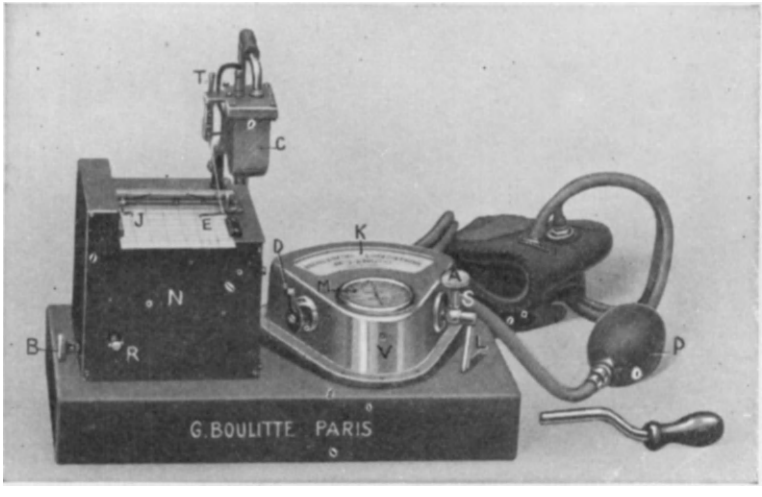


Fig. 30.—Boulitte's portable recording oscillometer.

which permits estimation of the maximal pressure (Fig. 32). If it is desired to save time, the three arterial pressures may be recorded on the same tracing by setting the distributor (D) first at Mx, and next as soon as this pressure is recorded at Mn, when the mean pressure (My on the tracings) is also registered.

3. Boulitte's Arterial Oscillograph.—This is a more elaborate and non-portable form of instrument, mounted on a light table with rubber wheels for use in nursing home or hospital. It yields a continuous and clearer tracing of the oscillometric curve.

4. The Tycos Recording Sphygmotonograph (Fig. 33).—This is useful for routine record of diastolic and systolic

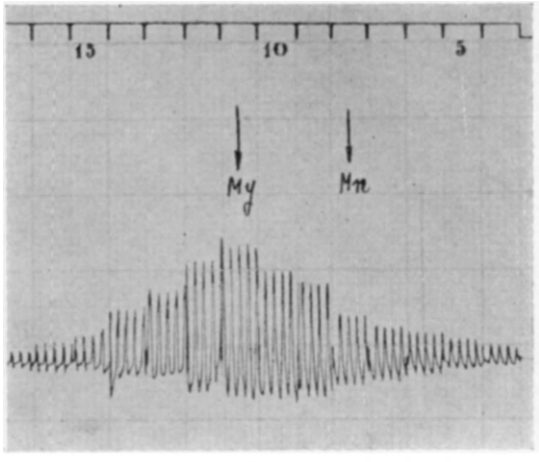


FIG. 31.

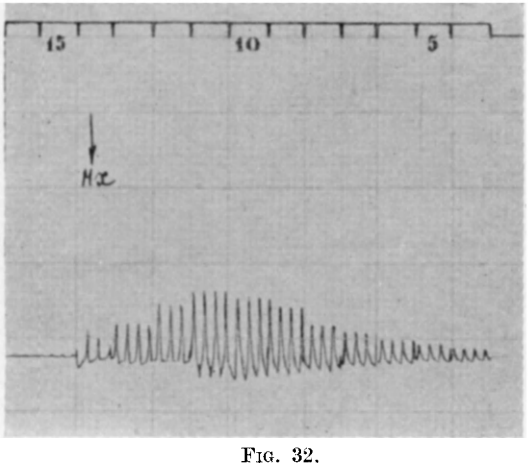


FIG. 32.

Figs. 31 and 32.—Tracings taken on millimetre-squared paper with Boulitte's portable registering oscillometer. The scale at the top registers in centimetres of mercury. Mn = diastolic pressure and My = mean pressure, obtained by adjusting a distributor valve so as to utilise both pockets of the armlet; Mx = systolic pressure, obtained by putting into action only the upper air pocket of the armlet.

pressures, which appear on the tracings with a fair degree of clarity, but since there is a damping down of each pulsation the upper portion of the successive pulse waves appear as straight lines, thus altering their essential characters

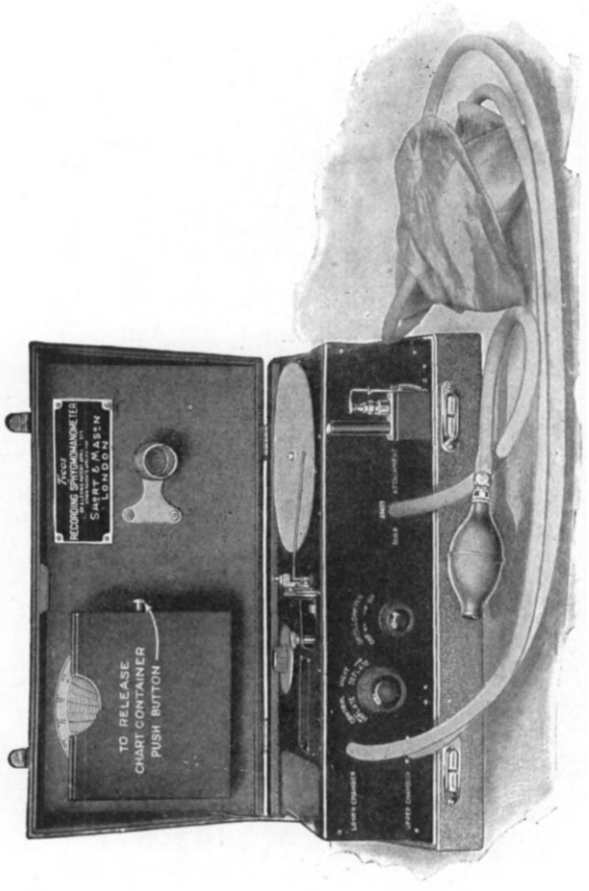


FIG. 33.—The Tycos recording sphygmotonograph. (By courtesy of Messrs. Short & Mason, Ltd.)

(Fig. 34). Moreover, the mean pressure cannot in most instances be determined.

Unless compensated by a device such as that of Gallavardin's double armlet, or by an internal adjustment of the apparatus itself, the graphic method of recording arterial pressure, as in Plesch's tonoscillograph, yields a systolic

reading several millimetres higher than afforded by the auditory method. To avoid confusion, the Tycos sphygmotonograph is compensated by adjusting the width of the compressing armlet so that the reading on the chart is the same as that obtained by the auditory method.

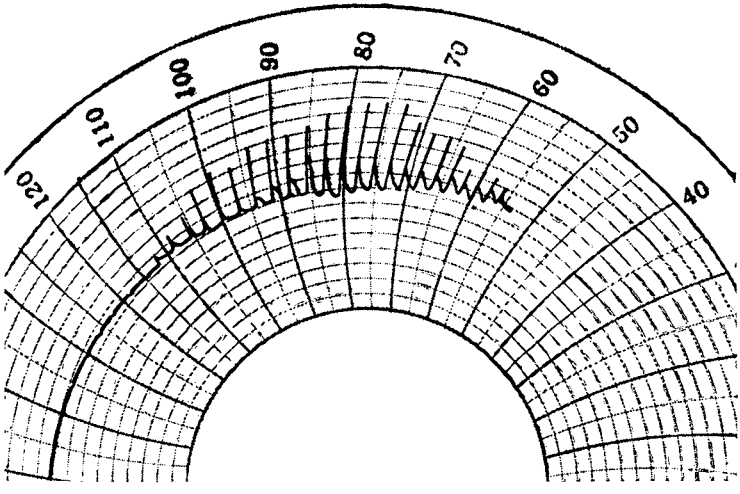


FIG. 34.—Sphygmotonogram of normal arterial pressure. Systolic = 111. Diastolic = 70. (By courtesy of Messrs. Short & Mason, Ltd.)

II. Direct Experimental.

The Boulittograph (Fig. 35).—This latest invention is comparable with the electrocardiograph in that by means of an optical arrangement a ray of light from an electric lamp is projected through lenses on to a roll of photographic paper travelling at a known rate of speed. A time marker is interposed so that the distance between two vertical lines on the paper corresponds to $\frac{1}{25}$ of a second, the speed being either 50 or 25 mm. per second. The horizontal lines indicate absolute pressure valves, each measuring 1 cm. of mercury (Fig. 36).

The *modus operandi* is as follows : the required limb is bared over the artery (femoral for choice, brachial, radial, etc.) to be investigated. A fine sharp needle with flexible lead tube attached is filled with an anticoagulant solution

and the artery is punctured. While the needle is in the lumen of the artery the oscillations due to intra-arterial pressure are observed, as the registering photographic band

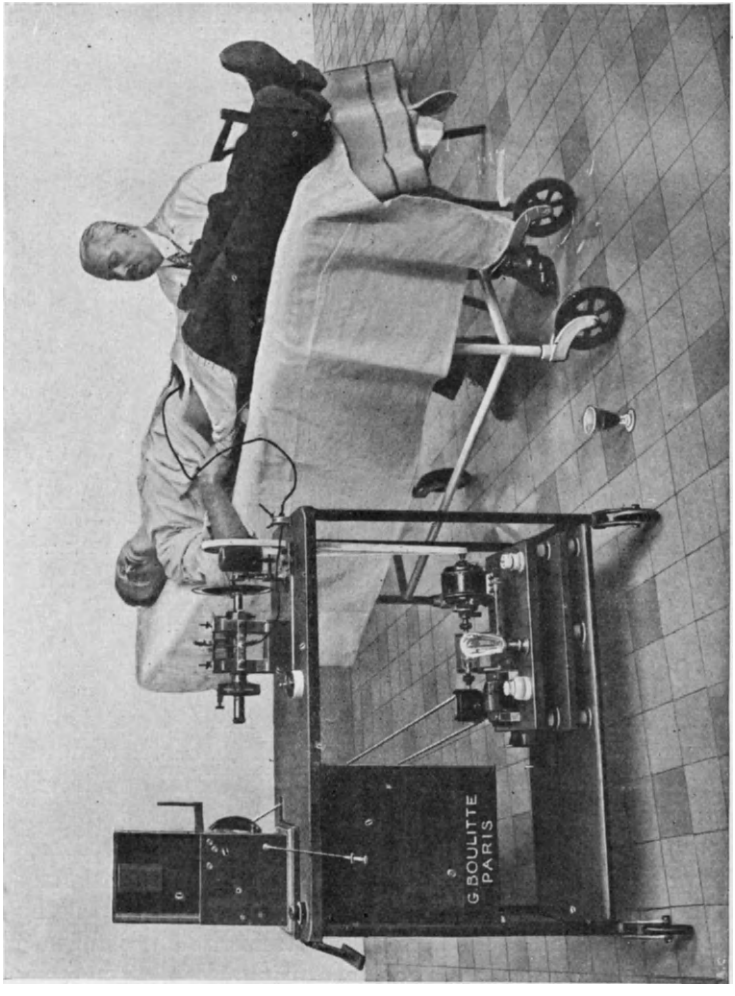


FIG. 35.—The Boulittograph.

of paper is allowed to unrol, through intervention of an index attached to a manometer connected with the blood-containing capsule.

In this way the actual intra-arterial pressures are recorded

for all their values during a series of cardiac cycles for as long a time continuously as the operator desires.

While of considerable interest to a physiological laboratory

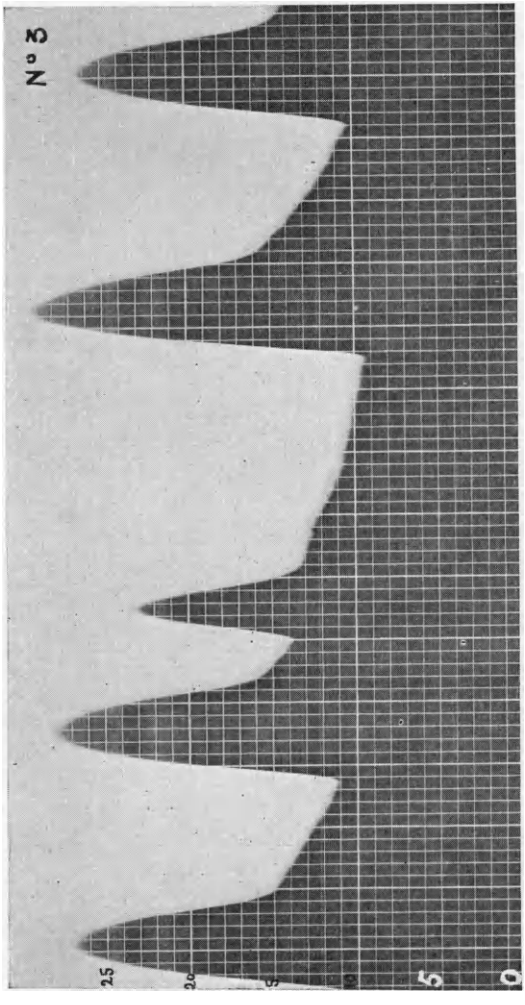


FIG. 36.—Record from femoral artery with the Boulittograph. Hyperpiesia with left ventricular enlargement. Speed 50 mm. per second.

or hospital research unit, this method of direct puncture of one of the larger arteries would probably make considerably less appeal to the majority of private patients.

Blood Pressure Instruments to be Avoided

(A) *Mercurial.*—(*a*) All the older forms of instrument in which the mercury is apt to be spilt.

(*b*) Where the scale is not a true measure of the mercury displacement.

(*c*) Where friction results from oxidation of the mercury on the walls of narrow-calibre glass tubes, resulting in air-pockets.

(*d*) Where the mercury column shows considerable oscillation.

(*e*) Where the long arm of a U-tube has a defective joint.

(*f*) Where metal clips obscure some of the scale divisions.

(*g*) Where there are movable parts and mechanical devices subject to wear and failure of adjustment.

(*h*) Where the apparatus and box are too heavy or bulky for ease in transport.

(B) *Aneroid.*—(*a*) Where the mechanism depends on a spring diaphragm likely soon to get out of order or to wear out, which causes faulty position of the needle. The chief disability from which most aneroids suffer is that alternate expansion and contraction weakens their elasticity till no dependence can be placed upon the reaction of the diaphragm, which tends to undergo permanent distortion when frequently used for the determination of high pressures. Such distortion is usually indicated by failure of the needle to return to the zero point.

(*b*) Where the mechanism is difficult to repair.

(*c*) Where the needle moves in a jerky manner by reason of friction.

(*d*) Where there is an adjustable dial which may allow the zero point to coincide with a wrong position of the needle. The greatest errors are due to this cause and to the aneroid being tested at only two or three points on the scale, on the assumption that intermediate graduations should be uniform.

(*e*) Where a stop is fixed to the dial so that the needle always registers zero under atmospheric pressure alone.

(*f*) Where the apparatus is cheaply constructed with dial markings stamped by machinery and not individually

marked by calibration against a standard mercury manometer.

(*g*) Where the release valve is of trigger pattern, permitting a sudden excessive lowering of air pressure instead of a gradual one.

Other factors, not inherent in any particular make of instrument, which cause inaccurate readings are :—

(*a*) Where the instrument is subjected to variable temperature and barometric conditions, which induce contraction and expansion of the metal.

(*b*) Where the delicate adjustments become shaken or jarred as the result of a blow or fall.

(*c*) Where dust and grit work in to the bearings and cause faulty movements.

(*d*) Where the compression cuff is less than standard width. Too narrow a cuff may import an error reaching to + 40 per cent.

CHAPTER IV

Fundamental Physiological and Physical Factors in Blood Pressure

"A man's life may be said to be a gift of his blood pressure, just as Egypt is a gift of the Nile." Osler.

The physiological aspects of blood pressure are wide and far-reaching, necessitating, as they should, deep and intensive investigation of correlated physical and psychical phenomena.

It is, however, far beyond the scope of this manual to enter into a detailed consideration of all the principles and data involved. I will, therefore, limit myself to a synopsis of the fundamental factors concerned in arterial pressure, which will serve as an introduction to its clinical study.

In this chapter I propose briefly to discuss the leading physiological and physical factors on which clinical variations of pressure depend, and from this groundwork to trace in succeeding chapters the origins of departures from standard levels which constitute either physiologically or pathologically high or low arterial pressures.

Significance of Blood Pressure

First of all, what meaning should the term "blood pressure" convey?

(*a*) *In the physical sense* blood pressure may be defined as that pressure which the blood exerts at a given instant upon a given point in the circulatory system.

(*b*) *In the physiological sense* the term includes pressures which may be intra-auricular, intra-ventricular, arterial, capillary or venous.

Arterial blood pressure is a force *originated* by ventricular contraction, *maintained* by the reaction to distension (resiliency) of the arterial walls, and *regulated* by the degree of resistance in the terminal portion of the arterial system.[16]

The five main factors concerned in its variations are two fundamental and three subsidiary factors, namely :—

Fundamental Factors

1. The energy of the heart, as measured by unit output (an intermittent force).
2. The peripheral resistance (a continuous force).

Subsidiary Factors

3. The resiliency of the arterial walls.
4. The volume of the circulating blood.
5. The viscosity of the blood.

In physics there are only two sources of rise of pressure, **increased force or increased load.** From the physical standpoint the human circulatory system consists of a **central**

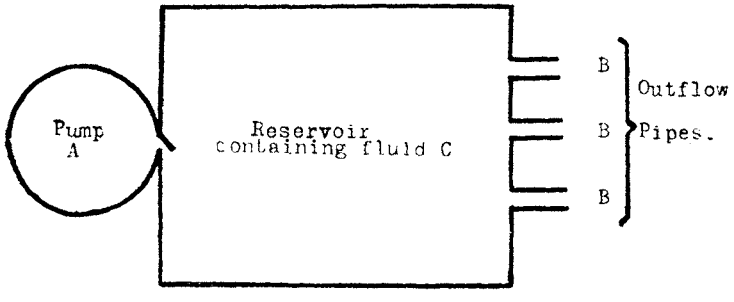


FIG. 37.—Schema of pressure system (diagrammatic).

pump system, an intermediate reservoir, and a peripheral outflow system comprising the series of pipes through which the circulating fluid is distributed throughout the body. In other words, an intermittently acting pump (heart) forces a liquid (blood) into a system of large elastic tubes (aorta and its branches, which act as a second heart), which divide (smaller arteries) and sub-divide (arterioles) to a terminal network (capillaries), from which the liquid is returned through another set of tubes (veins) to the inlet of the pump.

Pressure is varied by the force of the pump A (Fig. 37), the size of the distributing vessels B, and the character of the

circulating fluid C. Variations in the pressure can only be produced by an alteration in one or more of these conditions.

1. If the force of the pump A be increased, and B and C remain the same, the pressure is augmented. If the force of the pump be decreased, the pressure is diminished.

2. If the force of the pump A remains the same, and C is unchanged, then, if the diameter of the pipes B is reduced so as to obstruct the outflow, the pressure is again raised, and if the diameter of B be increased, the pressure falls. In physical parlance this peripheral resistance is termed the load. Hence, if C remains constant, *i.e.*, if the condition of the circulating fluid remains the same, a rise of pressure must be caused either by an increase of the central pump force or by an increase of the peripheral resistance.

The central system may be affected in two ways : primarily by direct increase of pump force ; or, secondarily, increase in peripheral resistance may demand increased action on the part of the pump, so that the increased resistance may be overcome by an augmented central pressure.

The peripheral resistance, or load, may also be affected in two ways : primarily, when there is a direct alteration in the size of the peripheral vessels, or a change in their resiliency in the direction of increased rigidity ; and, secondarily, where these alterations take place in order to compensate the augmented dynamic action of the central pump which necessitates contraction or rigidity of the peripheral vessels.

Hence the primary causes of increased pressure are only two in number : either (1) a central dynamic cause due to increased power of the heart and great vessels, or (2) a peripheral load increase due to increased pressure, vascular thickening or rigidity of the vessels. Any secondary cause is only compensatory to the primary. An expression of these physical terms in equivalent terminology will be found in Table VII., p. 102.

It should be borne in mind that the greater part of the cardiac energy is expended, not in driving the blood through the vessels, but in distending the walls of the arteries.

The more efficient is the circulatory mechanism, the lower is the pressure required to circulate a given volume of blood.

If the individual cardiac contraction becomes quicker and more powerful, though the volume of blood put out with each beat is unchanged, the systolic pressure will rise and the diastolic remain stationary ; if the peripheral arteries contract, and their resistance to blood flow becomes increased, both systolic and diastolic pressures will be elevated. A drop in the systolic and diastolic pressures will occur if each heart-beat becomes prolonged or less forceful and if the arterioles relax. In blood pressure changes with rest or emotional stimuli both these factors are concerned.[57]

The systemic arterial pressure far exceeds that in the arteries of the pulmonary circuit. The reason is that a highly variable physiological resistance exists in the systemic arterioles, whereas in the pulmonary circuit no such resistance occurs, since in the lungs there is no necessity for one inflated portion to receive more blood from the pulmonary artery than another, for all portions fulfil the same function, *i.e.*, aeration of the blood. When an organ demands an increased blood supply, all that is needed is for its arterial system (notably the arterioles) to dilate, when the blood is driven under a high pressure through the corresponding capillaries. These vessels are capable also of active contraction and dilatation, these phases probably coinciding actively with corresponding arteriolar phases in response to the tissue needs of the moment.

In the clinical sense " blood pressure " should be held to include diastolic, mean and systolic pressures.

The common acceptation of the term as denoting only systolic arterial pressure, which when stated as a number —*e.g.*, 160—is usually taken to denote " systolic pressure in the brachial artery at the time of investigation measured in millimetres of mercury," is too narrow, since it represents only a portion of the whole which it should portray. At each estimation the observer should be satisfied with nothing less than a record of the systolic pressure, the diastolic pressure, the differential pressure, the pulse rate and the figures for the latter two multiplied together. These five essentials together constitute what I have termed " the complete arterial pressure picture " (p. 27). If oscillometric methods be used, the mean pressure can also be recorded.

Minimal (Diastolic), Maximal (Systolic) and Mean Arterial Pressures

Because the driving force of the left ventricle is not continuous, but intermittent and pump-like, the output

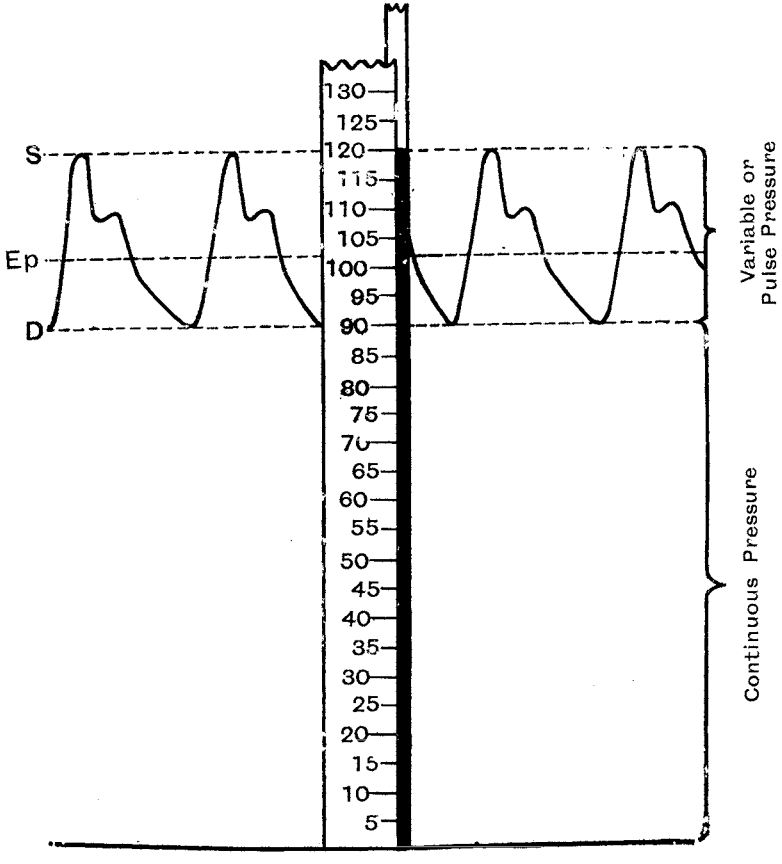


FIG. 38.—Diagram to illustrate the continuous pressure which the arteries have to sustain below the diastolic level D and the superadded pressure which occurs during systole. The variable or pulse pressure, in this example 30 mm. Hg, is the difference between the systolic level S at 120 mm. and the diastolic level D at 90 mm. Ep = level of mean pressure (" efficacious pressure ").

of blood is sent forward in waves, which rhythmically distend the walls of the arteries and so produce the pulse, the base of each wave corresponding with the least, or

minimal, pressure, whilst the crest of each wave corresponds with the greatest, or maximal, pressure (Fig. 38).

Since the minimal pressure occurs between successive ventricular contractions, *i.e.*, during the resting time of the heart (diastole), such pressure is also termed " diastolic," whilst to the maximal pressure the term " systolic " is applied, because it happens during ventricular systole, *i.e.*, that portion of the cardiac cycle during which the ventricles are in process of contraction.

Hence it is incorrect to speak of *arterial blood pressure* as a fixed and definite entity for any individual. In reality it is an amount which *is constantly fluctuating between certain minimum and maximum values characteristic for each subject under like conditions of observation.* At the outset, therefore, it is of the greatest importance to remember that, of the two pressures, **the minimal pressure** is the more valuable in that it **is a measure at the moment of a variable burden which,** during the life of the individual, **the arteries and aortic valves must continuously bear, and from which there is no escape, whilst the maximal pressure indicates only** an **intermittent and superadded load.**

Differential (Pulse) Pressure.—Differential pressure is the difference between the diastolic and systolic pressures ; *e.g.*, a diastolic pressure of 86 and a systolic pressure of 130 will yield a differential or so-called " pulse " pressure of 44.

Mean Pressure.—In laboratory experiments on animals the mean pressure has long been recorded, but in clinical medicine no importance was attached to it until the observations of Pachon in 1921. Since that time a series of publications by the French school of medicine, notably by Astier, Fontan, Dodel, R. Roger, Chevallereau, Escaich, Vaquez, Kisthinios, Gley and Gomez,[52] have assigned to this measurement a value which can only be regarded as somewhat artificial in comparison with the real and vital value of the diastolic pressure. The mean is not, as commonly supposed, the arithmetical mean between the diastolic and systolic pressures, but is the average pressure at a given point which, in a pulse tracing, appears at a level often, but not necessarily, nearer the diastolic than the systolic pressure. The shape of the curve being, however, very variable, there

is no fixed or absolute relationship between the mean pressure and the minimal and maximal extremes. The above points are clearly shown in Fig. 37.

In practice, **the mean pressure is that pressure within the armlet which corresponds with the oscillometric index, that is to say, the largest oscillation measured during the course of an oscillometric investigation.** This largest oscillation follows immediately upon complete decompression and elastic distension of a previously constricted artery under the influence of a wave of pressure corresponding with cardiac systole. Hence **two basic factors enter into its composition,** (*a*) **the force of the cardiac impulse,** (*b*) **the degree of arterial constriction or relaxation in the vessels under investigation.** Both these factors are variables, so that unless one is known, or remains constant during observation, the other cannot be inferred. Thus, although variations of the index in the same subject is helpful in symptomatology, a comparison of respective values cannot directly be made in different subjects.

Under varying mechanical, chemical or physical conditions the same mean pressure will not yield the same circulatory output. Hence consideration *solely* of this pressure does not suffice in assessment of circulatory efficiency or inefficiency. This conclusion has been reached by Pachon and Fabre [58] and confirmed by Lian.[59]

From the foregoing it would appear that no solid grounds exist for attributing to the mean pressure the "great importance" which by the French School, under the influence of Pachon and Vaquez, it is stated to possess. All arterial pressures are "dynamic," hence the adjective should not be limited solely to the mean pressure, nor are the terms "efficient" or "efficacious" any the more applicable in view of the conclusions mentioned in the preceding paragraph.

The Aim of Blood Pressure.—The aim of blood pressure is the maintenance of blood flow, and the strong elastic recoil after distension of the great aortic reservoir is one of the leading factors by which an efficient circulation is sustained, since in recoil the aortic reservoir functions like a second heart.[16]

The Peripheral Resistance.—The peripheral resistance is a combination of several elements, including viscosity of the blood and variation in calibre of the smaller arterioles and capillaries under the influence of the vasomotor system and the external pressure exerted upon them by the tissues.

The Sympathetic Vasomotor Nervous System.—This system consists of a vasomotor centre in the medulla, with subsidiary centres in the spinal cord. The paths for vasomotor reflexes are receptor (afferent) and excitor (efferent). The receptor paths are (i.) pressor and (ii.) depressor. "All sensory nerves are pressor in their action, causing the vasomotor centre to throw out increased constrictor impulses, particularly to the splanchnic area. This tends to produce a rise of pressure. . . . The only pure depressor nerve is the depressor branch of the vagus. It provides a way of escape for the heart when labouring against too high a blood pressure. But depressor fibres may also be demonstrated in sensory nerves."[60] Stimulation of these brings about a fall of pressure. The excitor channels are also of two kinds: (i.) constrictor and (ii.) dilator. Of these the vasoconstrictor are by far the more numerous and important in controlling blood pressure.

The vasomotor system as a whole is concerned with the regulation both of general blood pressure and local blood supply, and, as we have already seen (pp. 13, 15), when stimulated by the pressure of the armlet modifies arterial tonus as expressed in the vibrant and sonorous qualities of the arterial wall. As Barbier[17] has well said, "le sympathique est le tendeur du tambour artériel."

Tone, Tension and Viscosity

Not infrequently confusion is apt to arise as a result of lax use of the terms "tone," "tension," and "viscosity," and since each of these has a distinct bearing on the subject, it is necessary to outline their several significations.

Tone.—During life the arteries, large and small, may be much constricted as a result of contraction, or much dilated as a result of relaxation of their muscular fibres; or, again, during long periods they may be kept in a state

of moderate contraction (tonus). Such variations in calibre are brought about by activity of the vasomotor nervous system, the first and third states being caused by stimulation of vasoconstrictor fibres, the effects being both local and general, whilst the second occurs from stimulation of vasodilator fibres, the effects being limited to special vascular areas, in response to local requirements of organs or tissues.

Tonus (or tone) is thus an active state of the smooth muscle of the arterial wall which determines and regulates its kinetic function. Muscle tone depends on muscle consistency, which latter has to do with stratification of the muscle cells. With increased tonicity the vessel becomes harder, and the difference is appreciable to the touch. "Tone in a vessel is that which preserves its mean diameter, the due proportion between the extremes of dilatation and recoil, and has furthermore the somewhat different virtue of keeping the vessel wall well home upon its contents." [61]

Arterial tone as a component of blood pressure cannot be ignored, but at times its importance is apt to be magnified, and in assessing the quality of the pulse, as I have already said, the finger of itself is often insufficient, since the calibre of any artery can undergo considerable alterations.

Tension.—"Tension" is frequently used, especially by the French and American schools, as synonymous with "pressure," *e.g.*, "pulse tension," "arterial hypertension." Tension, however, implies a *pull*, while pressure implies a *push*. In any event, "supertension" should be read for "hypertension." With reference to blood pressure, however, it is more correct and less obscure to drop the latter hybrid term altogether. "To the coats of an artery only can the word 'tension' apply. The 'blood' cannot be tense in any but an abstruse mathematical sense. . . . Tension is the stress which tends to split the artery longitudinally or transversely, and such stress is at more advantage when the vessel is relaxed." [62] In his last publication Allbutt observed that "hypertension" (or, to speak more grammatically, hyper*tonus* or *super*tension) denotes too narrow a meaning: "tension" can be predicated only of

the proximal distended vessels, and in them is not the essential factor; the essential factor is the rise of blood pressure." [63] The effects of tension are best seen in an advanced case of aortic regurgitation, but "hypertension," as applied to a blood pressure which is raised, can only be regarded as a misused term.

Density, Specific Gravity, Viscosity.—Density is the proportion of mass to volume or bulk. Specific gravity is the weight of any given substance as compared with the weight of an equal volume or bulk of water or other standard substance at the same temperature and pressure, and is a function of density. Viscosity is the property of a liquid whereby it resists the relative motion of its parts. This property is analogous to that of friction. For example, water flows easily on itself or on other surfaces, whereas treacle or some of the heavier oils do so with greater difficulty. On the other hand, the specific gravity of water is often higher than that of a treacly oil.

Viscosity, therefore, is *not* specific gravity, for high specific gravity may co-exist with low viscosity. Viscosity connotes increased resistance to the flow of a liquid, so that, if we accept Allbutt's view that "nearly 200 times more of the heart's energy is expended in overcoming friction than in direct transference of velocity to the stream," "we shall scrutinise most carefully any degrees, however small, in the stickiness of the circulating fluid." [60]

Blood viscosity depends on two main elements :—

(*a*) Colloid, the degree of viscosity varying directly with the size of the individual colloid molecules contained in the liquid, the albumin molecule being larger than the globulin molecule. Thus an increased ratio of albumin to globulin points to an increased blood viscosity.

(*b*) Crystalloid. Blood viscosity is also capable of being increased by a preponderant content of heavy metallic ions, such as ionised calcium.

Blood density curves closely agree with those for percentage of hæmoglobin and number of erythrocytes. Hence in true splenomegalic polycythæmia (erythræmia) the blood viscosity, as measured outside the body, is greatly increased. In spite of this, however, arterial pressure ranges

only slightly, if at all, above the average, and the left ventricle is not usually hypertrophied. Variations in density which are readily balanced by increase or decrease of water content absorbed from the tissues do not largely affect arterial pressure. So that we may conclude that, although density has its share in maintenance of blood pressure, yet changes in consistence and viscosity can readily be balanced by the tissues.

Investigations now proceeding all over the world into the biochemical composition of the blood and further elucidation of colloid and crystalloid phenomena which take place therein may possibly afford assistance in solving the problem of the causation of high arterial pressures, and in particular of the diastolic pressure, although up to now no direct relationship between blood viscosity and blood pressure has been established.

CHAPTER V

Physiological Variations in Arterial Pressure

"We are apt to conceive too uniform a notion of blood pressures, to regard them as moving with a piston-like action on reciprocating planes; whereas a better comparison would be with the waves of the sea, or with the wafting undulation of a large bird."

Allbutt: *System of Medicine.*

Once the fundamental factors (outlined in Chapter IV.) on which blood pressure depends are clearly grasped, the reader will find little difficulty in tracing the processes of physiological variation, and, from a comprehension of these, can logically follow the sequence of events which occur in disordered and diseased states.

From the way in which "blood pressure" is often spoken of, it would appear that a certain blood pressure is assumed by many to be as characteristic of an individual as are his physical peculiarities. But this is not so, for though usually confined within certain general limits, yet such pressure is not constant from month to month or from day to day, but, from respiratory, psychical or other causes, may fluctuate widely and rapidly both in health and in disease, even during the brief time of investigation. Hence the maximal and minimal bounds between which arterial pressure normally rises and falls are the important criteria which in every case we must definitely determine.[64]

Significance and Importance of the Diastolic Pressure

Since the methods by which diastolic pressure can readily and accurately be determined have been described only within recent times, and because earlier workers had perforce to be content with registering systolic pressures alone, it is, perhaps, not altogether surprising, though most

unfortunate, that even in certain modern books the authors have contented themselves with recording only systolic pressures, thus hampering greatly the usefulness of their otherwise admirable work.

A few there are who still think that registration of the diastolic pressure is an irksome and needless refinement, and that for all practical purposes a record of the systolic pressure suffices.

To record the maximum pressure whilst ignoring the minimum not only affords no indication of what the mean pressure is likely to be, but resembles attempting to solve a complicated problem of which only one factor is given. "No fact regarding the blood pressure is better established than its wide range of variation in any individual. It is, therefore, impossible to speak of a normal value for blood pressure, but only of certain normal upper and lower limits."[65]

In short, records of systolic pressure alone are of comparatively little value, because the knowledge which such limited data afford is vastly inferior to that which is obtained from a consideration of the complete arterial pressure picture of systolic pressure in relation to diastolic and pulse pressures and pulse rate.

The reasons which cause me to reaffirm that the diastolic pressure is of paramount importance are as follow :—

1. Diastolic pressure is the measure both of peripheral resistance and of vasomotor nervous tone. Since the degree of peripheral resistance can thus be inferred, the diastolic pressure may be considered to be a better index of high arterial pressure than is the systolic pressure.

2. In relation to systolic pressure the diastolic pressure is the more important in that it is Marey's[66] "constant" element in arterial pressure, transitory systolic elevations which form the pulse representing only an intermittent and superadded load.

3. The diastolic pressure is the measure of the load which throughout life the arterial walls have continuously to support (Fig. 38), and is significant not only from the effect produced in them by mechanical tension but "with regard

to the influence of an excessively high diastolic pressure upon their blood supply, especially in the vessels of the lower extremity, where, in the erect posture, the diastolic pressure is greatly augmented by the influence of gravity." [18]

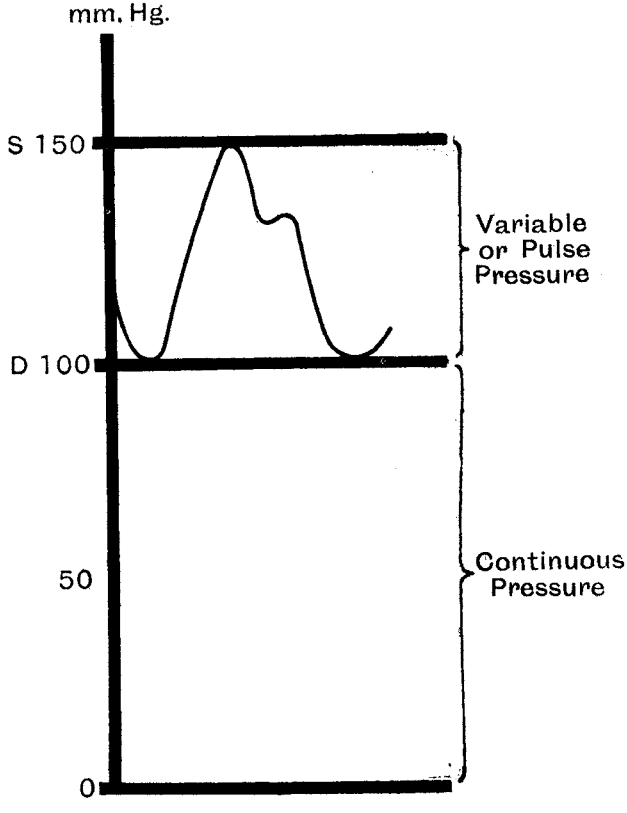


FIG. 39.—Diagram showing the relation of the diastolic to the systolic pressure. In this example the figures for the respective pressures are arbitrary.

4. The diastolic pressure is the measure of resistance in the aorta which has to be overcome by the blood stream in separating the aortic valves during the initial stages of left ventricular contraction (Fig. 40).

5. It is one of the indices of the driving force of the heart and of the eliminative capacity of the body.

6. In relation to systolic pressure it constitutes the additional necessary factor in the equation for determining the differential pressure (Fig. 41).

7. The systolic, diastolic and differential pressures, when conjoined with the pulse rate, and the product of the figures for the two latter together make up "the complete arterial pressure picture."

8. The diastolic pressure is *increased* by causes which produce vasoconstriction and so heighten the peripheral resistance, *e.g.*, aortic stenosis or lowered temperature; at times by increased rapidity of cardiac action; by increased intracranial tension, *e.g.*, cerebral tumour, meningitis; and in certain cases after exercise.

9. Gradual increase of diastolic pressure means harder

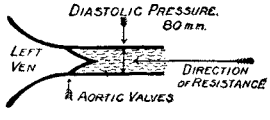


FIG. 40.—Diagram showing the left ventricle and arterial system during diastole. The diastolic pressure is 80 mm. and represents a resistance borne by the aortic valves and arterial walls and directed contrary to cardiac force. The power furnished by the heart must be sufficient to equalise this resistance and enough in excess to render its work potential in the arterial wall.

work for the heart to supply the parts of the body with blood. As compared with the transient systolic elevation at each heart beat, the significance of the diastolic pressure in relation to bursting of a degenerated cerebral artery is great. "This is illustrated by the comparative rarity of cerebral hæmorrhage in cases of aortic regurgitation, with its high systolic but low diastolic pressures." [67] In arteriosclerosis, increased diastolic pressure is accompanied by increased differential pressure and increased size of the left ventricle temporarily (exercise) or permanently.[18]

10. The diastolic pressure is *lowered* by causes which produce vasodilatation, and so lower the peripheral resistance, *e.g.*, fevers, certain cases of hyperthyroidism and neurasthenia; sometimes by conditions which slow the heart rate, *e.g.*, heart block; by diminished resiliency of the

arterial walls in the absence of increased peripheral resistance; and by aortic insufficiency.

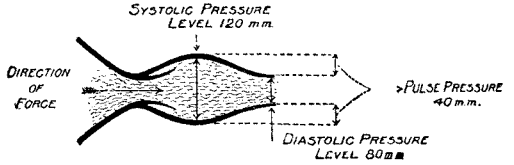


Fig. 41.—Diagram showing left ventricle and arterial system during systole. The diastolic resistance (peripheral resistance) of 80 mm. has been overcome by a systolic force of 120 mm. The difference between these two pressures, the pulse pressure, is 40 mm., and represents the efficient work of the heart in maintaining the circulation. (Figs. 40 and 41 by courtesy of Messrs. Short & Mason, Ltd.)

Significance of the Systolic Pressure

Systolic pressure indicates the maximum cardiac energy at a given moment, together with the degree of peripheral resistance. It fluctuates within wide physiological limits in response to the various activities and needs of the body, being modified under conditions of sleep, rest, posture, food, exercise, emotion, etc. (pp. 94 *et seq.*), the intermittent and more or less regular transitory elevations of systolic pressure above the more stable diastolic level constituting the pulse wave.

Significance of the Differential (Pulse) Pressure

Differential or pulse pressure is not a **direct** measure, like the minimal and maximal pressures, but only the **difference** between these two variables, upon which its attributes depend.

Clinically, the differential pressure represents the load of the heart. Systolic and differential pressures yield myocardial values, whilst diastolic pressure indicates the degree of arterial and arteriolar resistance.

Wide variations in the differential pressure from standard readings at various ages are possible, the coefficient of variation for differential pressures being more than twice as large as the systolic in some age periods, and nearly twice as

large as the diastolic coefficients. Standard averages remain about 44 mm. between ages twenty-five and fifty-five, after which they show considerable increase because of the faster rise in the systolic than in the diastolic pressure.

In adult life the normal range of differential pressure is from 30 to 55 mm. Expressed otherwise, under conditions of normal pressure balance differential pressure is about 40 per cent. of the average systolic reading and 65 per cent. of the average diastolic. A differential pressure reading below 30 mm. suggests an error of observation, except in a young child.

The higher the maximal pressure the larger usually is the differential pressure. Differential pressure also depends on pulse rate. With a slow pulse the blood stream has more time to flow through the arteries in diastole. Hence diastolic pressure will be lowered and differential pressure increased. In general, the slower the pulse rate the larger is the differential pressure ; the more rapid the pulse rate the smaller is the differential pressure.

Significance of the Mean Pressure

In direct contrast to systolic pressure, mean pressure is a physiological constant in the same subject, the standard range being from 80 to 100 mm., with an average of 90 mm. Its physical equivalent is the oscillometric index (p. 76), which bears to the minimal pressure a ratio normally of about one-third. This ratio is linked with the heart rate, decreasing with increased systolic effort and with increased peripheral resistance. Any figure for mean pressure exceeding 110 mm. Hg. may be regarded as pathological. At times the onset of hyperpiesis is signalised by rise of the mean pressure alone. Wide variations of extremes of pressure are not dangerous provided mean pressure remains relatively stable, whereas variations of mean pressure induce grave circulatory disturbances.[68]

Average Normal Arterial Pressures

I. **In Children.**—Arterial pressures gradually rise from birth, at which the systolic pressure varies between 20 and

60 mm. Hg ; in infants up to two years it averages 80 mm. The diastolic pressure is relatively higher, and the differential pressure is about 10 mm. less than in adults.[69] The systolic pressure shows a slight but gradual rise from three to ten years, after which the increase is more abrupt, with a rapid elevation in the fourteenth year during adolescence.

The following table of average arterial pressures from ages three to fifteen is based on 2,300 observations on well-nourished schoolchildren of both sexes by Judson and Nicholson,[70] who employed the auscultatory method checked by a modification of Erlanger's instrument.

TABLE I

Average Arterial Pressures in Children

Age, years.	Width of cuff in centi-metres.	No. of observa-tions.	Systolic pressure, millimetres of Hg.	Diastolic pressure in milli-metres of Hg, at beginning of 4th phase.	Differen-tial (pulse) pressure.	Pulse Rate.	D.P. × P.R.
3	9	24	92·0	58·4	33·6	92	3,091
4	9	95	92·6	61·7	30·9	99	3,059
5	9	69	91·6	60·0	31·6	93	2,939
6	9	110	93·8	63·5	30·3	95	2,878
7	9	145	87·9	64 2	22·7	87	1,975
8	13	128	93 0	59·6	33·4	88	2,939
9	13	149	91·7	62·2	29·5	84	2,478
10	13	203	99·0	64·6	34·4	87	2,993
11	13	169	95·8	62·3	33·5	87	2,914
12	13	94	99·9	59·6	40·3	89	3,587
13	13	80	104·0	63·2	30·8	96	2,957
14	13	43	105·8	63·7	42·1	84	3,536
15	13	35	99·6	61·8	37·7	84	3,167

In boys and girls of from four to sixteen years of age, Faber and James [71] have stated that the mean systolic pressure shows no significant differences between the sexes, whereas the mean diastolic and mean differential pressures show significant sex differences. Standard deviations are

greater for girls, indicating a normally greater variability in females, especially during adolescence.

In British boys Stocks and Karn [72] find lower systolic averages at age 5 than those recorded by the two sets of American observers just quoted but a considerably higher level at age 14 (115 mm.)—a rise of 30 mm., nearly twice that found by the latter authors. Stocks and Karn find the accelerated rise between ages 13 and 17 to amount to 16 mm.

Melvin and Murray 36 found the auscultatory method easily applicable to twelve healthy children of ages between eight and fourteen, who gave an average systolic value of 107 mm. and a diastolic value of 74 mm., with an average pulse pressure of 33 mm.

"The rising pressure of childhood undergoes some acceleration about puberty and reaches the normal adult level somewhere in the age period of seventeen to twenty; there is some evidence of a slight lowering in the early adult years. The pressure then alters little up to the age of about forty, after which a more definite progressive rise becomes manifest." [67]

II. **In Adults.**—Both for children and adults reliable British figures are few. American figures both for healthy children and adults are lower than those usually found in this country. Whether the explanation lies in differences in environment, including climate and diet, or along other lines, is uncertain.

Readings by Continental and American observers under various conditions of observation place the level of the diastolic pressure at between 65 and 110 mm. for adults, which accords with the figures given by Janeway.[65] This upper limit, based on observations with the older types of instruments, is far too high, and it is in accord with later experience that a diastolic pressure persistently above 90 mm. is to be regarded with suspicion, whilst one of 95 mm. or over is definitely pathological.

All the following figures are stated on the assumption that the blood pressure is estimated in the absence of psychical and physical disturbing factors, the subject being seated with muscles relaxed. My own series of averages I have set out as follows :—

TABLE II

Standard Arterial Pressures for Adults at all Ages

—	Systolic Pressure.	Diastolic Pressure.	Differential Pressure.
Men . .	127	83	44
Women .	123	81	42

Further observations with modern instruments on standard normal arterial pressures at various ages, especially as to the diastolic level, are still required, since one can only place reliance on those which have been collected during the past few years for four chief reasons: (1) Pioneers of blood pressure investigation dealt only with small numbers, which are insufficient to obviate experimental errors due

TABLE III

Systolic Pressure—Men Only

Age.	Symonds.	Mackenzie.	Rogers and Hunter.	Average Totals.
15—19	121·2	119	120	120·06
20—24	123·4	122	122	122·5
25—29	123·9	123	123	123·3
30—34	123·8	124	124	123·9
35—39	124·9	126	125	125·3
40—44	126·5	127	127	126·8
45—49	128·4	129	129	128·8
50—54	130·9	132	133	131·9
55—59	133·9	135	134	134·3
60 and over	135·2	137	—	136·1
Entrants .	150,419	18,637	62,000	231,056 Total Entrants

to personal equation and to fulfil the law of averages. (2) All earlier records and, unfortunately, many later ones are concerned solely with systolic pressure. With old apparatus this deficiency is comprehensible, for diastolic pressure could not be registered with any accuracy, and therefore was so variable as to be of little or no importance. (3) Early types of armlet, which were all too narrow, necessarily caused readings to be too high. (4) All the American assurance figures are vitiated for clinical purposes, because of the arbitrary choice of the very end of the fourth phase, just before silence, as the criterion of diastolic pressure, a point which is always too low, and not infrequently far too low.

Notwithstanding this defect, which is in some cases serious, it is to American life insurance that we are indebted for the only data of arterial pressure which have to do with vast numbers of healthy subjects. Five comprehensive

TABLE IV

Average Systolic and Diastolic Pressures (Symonds)

Age.	Men.		Women.	
	Systolic.	Diastolic.	Systolic.	Diastolic.
15—19	121·2	77·7	119·2	77·2
20—24	123·4	79·6	120·6	78·2
25—29	123·9	80·5	120·9	78·8
30—34	123·8	81·5	121·7	80·3
35—39	124·9	82·7	123·3	81·8
40—44	126·5	83·8	126·0	83·4
45—49	128·4	84·9	128·2	85·0
50—54	130·9	86·3	130·9	87·1
55—59	133·9	87·0	134·8	87·8
60 and over	135·2	86·6	135·5	89·8
Entrants .	150,419	60,733	11.937	5,276

reports are to hand, namely, those of Fisher (1914),[73] Mackenzie (1915),[74] Rogers and Hunter (1919),[75] Goepp (1919),[76] and Symonds (1923).[35]

Table III. has been compiled from the records of three series of observations taken under similar conditions and age groups.

Fisher deals with an additional 19,339 cases, the average systolic pressure for all ages being 128·91, and Goepp with 9,996, his average for all ages being 123·1, but both take slightly different age groups, which prevent inclusion in the above table. In Fisher's report only 13 per cent. of entrants are younger than thirty-six, so that his general average is high. In Mackenzie's and Goepp's tables the largest number of entrants was about age thirty, so that their general averages are low. Goepp's examinations were made in 1918 under war conditions, particularly in the matter of eating and the

TABLE V

Average Differential Pressures—Men only (Symonds)

Ages.	Average Totals.
15—19 . . .	42·9
20—24 . . .	43·0
25—29 . . .	43·0
30—34 . . .	41·8
35—39 . . .	42·2
40—44 . . .	42·3
45—49 . . .	43·1
50—54 . . .	45·2
55—59 . . .	46·7
60 and over . .	49·2
6,071 Entrants.	42·8 All ages.

use of alcohol, which may have lowered the general and individual age period averages.

By reviewing these tabulated figures with large numbers of my own records I have drawn up the following theoretical table corrected to represent standard arterial pressures at various ages for men of medium physique (Table VI.).

TABLE VI

*Theoretical Standard Arterial Pressures in Males of Medium Physique at various Ages**

Age in Years.	Systolic Pressure.	Diastolic Pressure.	Differential Pressure.
At birth	20—60 (average 40)	?	?
Up to 2	80	48	32
5	88	55	33
10	100	64	36
15	110	70	40
20	122	80	42
25	124	81	43
30	126	82	44
35	127	83	44
40	128	84	44
45	129	85	44
50	130	86	44
55	132	87	45
60	135	89	46
65	140	90	50
70	143	92	51
75	147	93	54
80	150	94	56
Over 80	All pressures tend to fall.		

* Pressures estimated by auscultatory method with Riva-Rocci or Tycos types of instrument. In children under the age of seven years the diastolic point is difficult to determine.

Athletes often give readings below standard, owing to the effect of training in promoting a vigorous peripheral circulation. At times the systolic pressure may be as low as 100 to 105 mm. As a result of greater vasodilatation, low pressures are alleged to be the rule in tropical countries.[77]

Tables, though handy for reference, are hard to memorise. Hence, whenever one comes across a case in which the blood pressure is out of the ordinary, it is desirable to be able to apply some easy rule for finding out what is approximately the standard pressure for a given age. The popular idea that the systolic pressure of any adult can be arrived at by adding 100 to the age in years is erroneous and most inaccurate, particularly for all ages over forty years. It is negatived by *post-mortem* evidence as to the weight of the heart in hyperpiesis as compared with records of the arterial pressure during life, for, in both sexes, greatest increase in heart weight does not coincide with the greatest age (p. 105).

A closer approximation is afforded by taking standard systolic pressure as 100 plus half the age, and standard diastolic pressure as 75 plus one-quarter of the age.

It must, nevertheless, be realised that no mechanical formula is likely to be as exact as the above theoretical table. The two rules which the author finds best and most serviceable in practice are here appended :—

1. **Simple Rule for calculating Standard * Diastolic Arterial Pressure** (Halls Dally).—*Taking standard diastolic pressure at age twenty as* 80 *mm. Hg, for each five years above twenty up to and including age sixty add* 1 *mm. Hg, and for each five years above sixty up to and including age eighty add* 2 *mm. Hg.*

2. **Simple Rule for calculating Standard * Systolic Arterial Pressure** (Halls Dally).—*For ages twenty up to sixty, standard systolic pressure equals* 120 *plus one-fifth of the age. At age sixty standard systolic pressure is* 135, *and for each year above this up to and including age eighty add* 1 *mm. Hg.*

Standard differential pressure between ages fifteen and twenty-five is 43 mm. Hg ; between ages twenty-five and fifty, 44 mm. Hg.

* Standard = " normal," *i.e.*, non-pathological.

Limits of Normal Arterial Pressure Variation

Systolic arterial pressure may vary 15 mm., and diastolic pressure 10 mm., *above or below* the figures given in Table VI. for various ages without such deviations being regarded as necessarily abnormal.

The lowest normal limit of systolic pressure may be regarded as—in young children, 70 mm. Hg ; in adolescents, 85 mm. Hg ; in adults, 105 mm. Hg.

For working purposes it may be said that "normal" arterial pressures for young and healthy adults are as follow : the systolic pressure may vary between 105 and 125 ; the diastolic level is usually between 65 and 80 mm., and the differential pressure 42 to 44 mm. The figures usually met with under normal conditions from middle age onwards range from 130 to 145 mm. Hg.

Influence of various Physiological Factors on Arterial Pressure

Body Weight, Age, Height and Sex.—Arterial pressure varies directly with weight and height, as a rule increasing with increasing weight, and *vice versâ*. Height has a similar, but less, influence. Larimore[78] found that in 417 factory hands the sthenic habitus was accompanied by a higher arterial pressure than the asthenic. The pressure in the hyposthenic habitus was intermediate.

Examination of 200,000 males, whose lives were insured between 1887 and 1908, and whose histories were followed till 1921 or prior to termination of the policy, has shown that danger to life increases directly with the degree of overweight. The relation between age and weight is also noteworthy. After age forty-five excess mortality among overweights becomes of great importance. Those who are more than 25 per cent. overweight have a mortality rate double the normal (L. I. Dublin). Nevertheless, the normal rise in arterial pressure during the second half of life, as evidenced by mass statistics, does not amount to more than 15 mm. Hg.

In women up to the age of forty years, standard diastolic and systolic pressures are a few millimetres lower than the

above figures for men. After this age in women, standard systolic pressure becomes equal or higher, while diastolic pressure for the next ten years remains about the same. At the age of fifty, standard diastolic pressure increases rapidly, and afterwards is substantially higher than that for men. Perhaps menstruation is responsible for the lower range of pressures in women below forty, but it does not seem reasonable to assign the menopause as the cause of higher pressures after fifty. Note that systolic and diastolic pressures in both men and women show a definite increase with weight as well as with age.

Sleep.—During quiet sleep there is a fall of from 10 to 20 mm. or even more in the systolic pressure, with a fall also in the diastolic, both as a result of lessened irritability of the nervous system, muscular relaxation, and vascular dilatation, the pulse rate being slowed by twenty beats per minute. The pressures are lowest during the first few hours, and gradually rise again towards the hour of waking. During disturbed sleep the systolic pressure may rise from 50 to 70 mm., and the diastolic by 30 mm. Hg, which accounts for the incidence of cerebral hæmorrhage during sleep in about 20 per cent. of hyperpietics.[79]

Rest.—The combination of muscular relaxation and mental quietude produces similar effects to those observed during sleep, though to a less degree.

Posture.—Mortensen [80] found on examination of several hundred adults of both sexes that passive postural change from the reclining to the standard position caused slight decrease in systolic pressure and slightly greater rise in diastolic pressure, thus cutting the differential pressure at both extremes. There was also a uniform increase in pulse rate. In very few cases was the systolic pressure higher in the erect posture, and in no case was the diastolic lower. The findings of Max Ellis [81] in the main correspond with these results.

Food.—The influence of food intake on blood pressure is not yet definitely established. Probably immediately after meals a slight rise which results from gastric filling occurs in systolic pressure, accompanied by a constant fall in diastolic pressure, even up to 20 mm., independently of the time of

day. This is speedily succeeded by a drop in systolic pressure to below standard level, coincident with the period of active stomach secretion. Finally, there is another rise probably due to intestinal distension with food.

Fasting.—Fasting induces a gradual fall in both pressures which rapidly return to normal when the fast is broken.

Alcohol.—(1) Experiments show that, if alcohol raises blood pressure at all, such rise is transient, depending on temporary reflex vasoconstriction in the splanchnic area through the afferent fibres of the gastro-intestinal surface, and that, as soon as alcohol reaches the blood stream, peripheral vasodilatation results, and the pressures fall. Intravenous injection of alcohol causes a drop in blood pressures without preliminary rise.

(2) Donnison's [82] observations on African natives support the view that hyperpiesis and arteriosclerosis are evils connected with civilisation and mental stress, since in 1,800 natives investigated by him no clinical case of raised arterial pressure nor of arteriosclerosis was found. Yet, to my knowledge, there are few races, however primitive, that do not brew some form of alcoholic beverage.

(3) Hyperpiesis and arteriosclerosis are by no means necessarily coincident, *i.e.*, in only about 50 per cent. of cases of established arteriosclerosis are arterial pressures above normal.

(4) Of 16,662 policy holders of the New York Metropolitan Life Office, 19·8 per cent. manifested abnormal arterial pressures, approximately 16 per cent. being high. But the percentage of high pressures in excess alcoholics was little if any higher than for the others.

(5) In habitual drunkards the incidence of arteriosclerosis has repeatedly been found to be not more than normal, and Ruffer,[83] who performed over 800 autopsies on Mohammedan pilgrims who had never imbibed alcohol, found in them arterial disease as frequent and as early as in individuals who practise no such abstinence.

(6) The actual amount of alcoholic consumption per diem must always be taken into consideration when assessing the possible effects. The issue is often confused by those who class together light wines and beer, which contain very

little alcohol, with heavy beers, wines, spirits and liqueurs, whose alcoholic content is far heavier.

Hence there is no direct evidence to connect alcohol either with the production of hyperpiesia or arteriosclerosis.

In this connection a tale told by the late Sir James Mackenzie is illustrative. An elderly brewer's agent consulted him because of symptoms of slight angina pectoris. Hyperpiesis of 210 mm. systolic, with a very hard pulse and thickened arteries, were found. Sir James said to the patient, "You must give up beer and spirits." The reply was, "I'm a teetotaller." "Well then, you must eat less butcher's meat." "I'm a vegetarian," was the answer!

Tobacco.—Like alcohol, the immediate effect of smoking is a pressor one, which raises both pressures, the maximal more than the minimal, and quickens the pulse rate by about twelve beats per minute. In respect of both alcohol and tobacco it should, however, be remembered that experimental dosage is much greater than the amounts habitually absorbed by man, and that in drinks the percentage of alcohol varies enormously.

Tobacco smoke contains pyridine bases and nicotine, the latter being the main toxic product. The composition of tobacco smoke varies greatly both with the kind of tobacco and the manner of smoking. Most of the nicotine is usually burnt up, and that which reaches the mouth is volatilised by the hot gases in passing over the unburnt area. A thick cigar has the worst effect, since it acts as a chimney for the gases which in a thin cigar or cigarette escape into the surrounding air. In long-stemmed pipes much of the nicotine condenses before reaching the mouth. Inhaling the smoke, however, more than compensates for this difference in combustion.[84]

In non-smokers the immediate effect of smoking is to send up systolic pressure by 10 to 30 mm., the diastolic being much less influenced. Within five minutes or so systolic pressure drops often by 50 mm., with palpitation, pallor, sweating, colicky abdominal pains, *muscæ volitantes* and weak pulse.

In those more habituated to the use of tobacco subacute symptoms occur only from over-indulgence, or from smoking

a kind of tobacco to which they are unaccustomed. In seasoned smokers little or no rise in systolic pressure results.

Generally speaking, smoking at first increases systolic, diastolic and differential pressures, though later these return to normal. Excessive smoking lowers the systolic pressure. Middle-aged smokers are liable to precordial stabbing pains and angina pectoris, which disappear on giving up the habit. In the elderly, secondary arteriosclerosis and myocardial changes may be induced.[18]

Cornwall [85] has made a series of observations upon the effect of chronic tobacco poisoning on blood pressure. Patients with symptoms of tobacco heart had subnormal blood pressures, the systolic ranging between 85 and 115 mm. Hg, the diastolic between 60 and 75 mm. Hg; the pulse rate was not necessarily rapid, notwithstanding the presence of cardiac symptoms. The systolic pressure was generally lowest when the subjective symptoms were most pronounced, whilst brisk exercise caused a uniform fall in both systolic and diastolic pressures. A systolic pressure of less than 100 mm. Hg in an otherwise healthy man is almost certainly due to excessive smoking. If there is no evidence of this, careful search should be made for symptoms and physical signs of active fibro-caseous pulmonary tuberculosis.[86] As age advances, and as arteriosclerosis becomes more frequent, the tolerance for tobacco becomes less.[87] Cigar-smokers are more prone to arteriosclerosis than cigarette or pipe-smokers. Excessive cigarette smoking causes cardiac irritability, but for most people *moderate* use of tobacco does not appear to be injurious.

Exercise.—In healthy subjects, increase in blood pressure accompanying physical exertion is in direct proportion to voluntary effort required, and to amount of work done. The rise in pressure mainly results from (*a*) increased cardiac energy, (*b*) mental concentration. Unaccustomed kinds of exertion which involve mental effort produce a much greater rise than is caused by an equivalent amount of routine work, as, for example, the flushed face of one learning to cycle, or of a young barrister conducting his first important case. Both psychical and

physical factors influence systolic pressure more than diastolic.

Mild muscular exercise, such as walking, tends to lower the minimal pressure whilst augmenting the blood flow, pulse rate and differential pressure. Foot racing in healthy athletes causes a considerable reduction in both systolic and diastolic pressures, the differential remaining almost contant. More strenuous exertion increases all of these, and, if very severe, the initial rise of pressure may be extreme, particularly in those unused to manual labour, through lack of automatic adaptation to such efforts. Diastolic pressure may be raised by even as much as 30 mm. temporarily, and systolic pressure by as much as 70 mm. In men given to arduous muscular work, arterial pressures, tested during the intervals of rest, range habitually below the average.[61]

Altitude.—The effect of altitudes of 4,000 feet or above on the pulse rate of normal individuals is to cause an initial and temporary acceleration lasting for about ten days, after which the pulse rate gradually falls. Those acclimatised to high altitudes manifest pulse rates which are slow, readings of from 54 to 60 beats per minute being commonly met with.

Fatigue.—If exertion be so prolonged and excessive as to trench upon the reserve power of the heart, fatigue ensues. After a transitory rise of pressure, mainly systolic, due to an effort on the part of the vasoconstrictor centre to maintain the pressure head, the centre becomes depressed, with resultant fall of both maximal and minimal pressures. The pulse rate diminishes, and dyspnœa and other subjective symptoms appear.

Psychical Stimuli.—Fear, anger, emotion, worry, anxiety, pain and mental activity exert through vasoconstriction a pressor effect, and bring about sudden changes in systolic pressure. Under stress of emotional crises there may be an extraordinary and rapid rise in association with notable quickening of the heart rate. Nervous individuals are more susceptible than those of placid temperament. In normal persons it is usually only the systolic pressure that varies, whilst in the arteriosclerotic the diastolic fluctuates to a similar extent.

The following history, told me by a general practitioner,

is of psychological interest, since it illustrates the dramatic rise which can occur in systolic pressure as the result of sudden and strong emotion, as well as emphasises the importance of not being content with one observation of blood pressure only :—

The patient was a man aged fifty-seven, whose shipping business necessitated his working at high pressure and making frequent long railway journeys. On arrival at Paddington one evening, feeling tired out, he went to the refreshment room and had two glasses of brandy-and-soda. He then took a train to his home, some eighteen miles from London, and on arrival went straight to bed and sent for the doctor.

The doctor found his patient restless and excited, with flushed face and complaining of throbbing headache, the pulse of 90 per minute being full and bounding. The average of three blood pressure readings was 265 mm. systolic. The urine was found to be normal. A strong dose of calomel was given, and cooling applications made to the head.

On the following morning, to the doctor's surprise, the patient appeared calm, cheerful, and free from all symptoms.

The patient, noting the doctor's astonishment, asked the reason, and was told of the rapid fall in blood pressure since the night before. Having again found the urine normal, the doctor suggested that a "sudden shock" might have caused the condition, whereupon the patient told him that he had married late in life a wife thirty years younger than himself. Previous to marriage he had been a hard liver and heavy drinker, but after a long and terrible fight became a total abstainer. From that day no alcohol passed his lips until the evening before. On letting himself into the house, feeling still tired out, he was met by his wife, who asked : "What's the matter ? " and detecting the odour of spirit, exclaimed : "At your old drinking habits again, are you ? "

The memory of the agony through which he had won and the realisation, which then became acute, that he had made a mistake in marrying a wife so much younger than himself, together with the tone in which she spoke, just seemed the breaking point. His head felt as if it would burst ; he clung to the side of the stairs to prevent himself falling, and sent for his doctor.

Normal Puberty, Menstruation, Pregnancy, Labour and the Menopause.—(*a*) *Puberty.*—From very early ages there is a progressive steady rise of arterial pressure up to the onset of puberty, then a quickening of the rise up to the adult level somewhere between the ages of seventeen to twenty years.

(*b*) *Menstruation.*—During the seven to nine days preceding the menstrual flow both pressures rise. With the onset of the period they rapidly fall to reach their lowest

levels at its end. During the next fortnight pressures gradually return to their previous standard levels.

(*c*) *Normal Pregnancy.*—In primiparæ below thirty years of age as a rule one does not expect to find systolic pressures of more than 95 to 115 mm. Hg, with diastolic pressures respectively of from 50 to 75. In multiparæ, probably as a result of increased age, pressures range somewhat higher, from 115 to 125 mm. systolic, and 60 to 80 diastolic respectively. About 60 per cent. of all cases fall within a maximum of 125 systolic. Thus, as a result of general experience, one may say that, during the whole term of normal pregnancy, arterial pressures are within standard limits, having regard to the fact that the range of arterial pressures in women is lower than that for men of corresponding age. Initial lower ranges of pressure gradually give place to higher ones. During the last three months, however, there is usually a slight and gradual rise of arterial pressure in conjunction with a definite increase in basal metabolic rate. This latter is in all probability not wholly due to growth of the fœtus, but also to enhanced thyroid activity.

(*d*) *Normal Labour.*—Arterial pressures should be estimated before, during and after delivery. Any rise at the beginning of and during normal labour is brought about solely by pain, or by voluntary and involuntary muscular exertions due to labour. On subsequent relaxation, pressures gradually drop during convalescence to normal levels.

(*e*) *Normal Menopause.*—The normal menopause should be unassociated with arterial pressure variations, but in the majority of all cases there is a rise in the maximal and differential pressures (*vide* p. 180).

CHAPTER VI

HYPERPIESIS : HYPERPIESIA : HYPERTONIA

"In primary contracted kidney hyperpiesis is but an incidental coefficient, while in hyperpiesia on the contrary it would seem that it is the high systolic and diastolic pressures themselves that do the mischief, or most of it."

ALLBUTT : *Arteriosclerosis*, 1925.

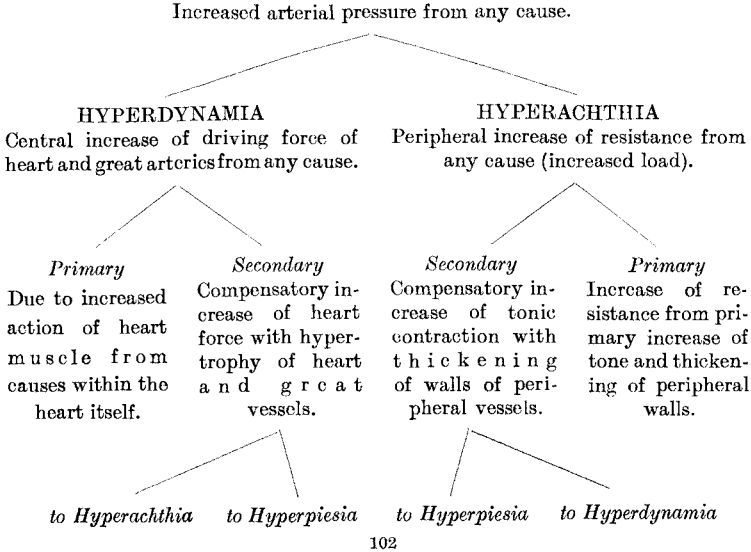
I. Hyperpiesis

HYPERPIESIS (Gr. ὑπέρ, πίεσις = over-pressure), in its application to the arterial system, means high arterial pressure, *i.e.*, a rise of pressure within the arteries from any cause whatever, whether of renal or non-renal origin, temporary or permanent, physiological or pathological.

Starting with this definition, we are thus enabled to translate the physical principles of the circulation (p. 71) into their equivalents and so formulate a classification according to Table VII.

TABLE VII

Classification of Hyperpiesis



In physics all pressures are expressed in terms of force and load, so that, in order to avoid the repetition of long and cumbersome phrases, I have introduced as corresponding physical expressions two new words, hyperdynamia (ὑπέρ, δύναμις = over-action) and hyperachthia (ὑπέρ, ἄχθος = over-load). Hyperpiesia is maintained in its original signification.

On this classification hyperpiesis constitutes the chief heading. This includes two subsidiary divisions, hyperdynamia and hyperachthia.

Primary hyperdynamia results from any cause which necessitates the driving power of the heart and great arteries being augmented, and in time leads to hypertrophy.

Secondary hyperdynamia results from hyperachthia, *i.e.*, increased load, which causes the heart and great vessels to hypertrophy, *e.g.*, a damaged kidney produces secondary hyperdynamia in the rest of the circulation. Similarly arteriosclerosis causes secondary increase in the force of the pump because of the greater difficulty met with in driving the blood through sclerosed arteries—again a secondary hyperdynamia.

Primary hyperachthia, or increase of load, results from any cause which increases peripheral resistance, and leads to hypertonia, *i.e.*, an increased contractility of vessel walls, which in turn, if long continued, induces vascular hyperplasia.

Secondary hyperachthia occurs in response to a primary or secondary hyperdynamia, as in the case of a primary arteriosclerosis.

Secondary hyperdynamia and hyperachthia respectively result from hyperpiesia acting either centrally or peripherally. Even over long periods of time it is surprising to find that little change may have occurred in the heart, this organ being affected solely by increased load. In the majority of established cases of true hyperpiesia, nevertheless, the heart becomes overworked and hypertrophies to meet the pressure, as evidenced clinically by some degree of enlargement, which can be confirmed by X-ray investigation, by more rapid action (sometimes amounting to tachycardia) and by lessened response to effort. Still later, the

vessels of the periphery become thickened for a similar reason in a combined process of degeneration and repair.

Thus the circle is completed, as shown in Table VII., which includes all varieties of hyperpiesis, and can readily be adapted for low pressure states by the substitution of "hypo" for "hyper."

Range of Pressures in Hyperpiesis.—Any diastolic pressure of 105 mm. or over, and any systolic pressure of 160 mm. or over, may be regarded as hyperpiesis, which, as stated previously in other words, is not *essentially* a pathological condition.

General Frequency.—Of the total deaths from all causes in England and Wales, diseases of the cardio-vascular system account for more than one-third. In this group approximately one-fifth show evidences of high arterial pressure.

Age Incidence.—Hyperpiesis may become manifest at any age, most frequently between the ages of fifty-five and seventy years. Below the age of forty it is relatively uncommon.

Sex Incidence.—Women are affected in slightly less degree than men except at the climacteric.

Operative Risks.—As a rule hyperpictic patients stand operation well. The opposite is often the case with subjects of low arterial pressure.

Variability.—One striking fact concerning hyperpiesis is its extreme variability. Apart from the action of drugs, within a few days it is capable of dropping from a great height to a point only slightly above standard. The opposite phenomenon equally may obtain. The association of arteriosclerosis, which is a constant and stable condition, cannot be invoked to explain these interesting fluctuations, but a variable amount of circulating toxin might readily account for changes in the media as well as the small-cell proliferation in the intima.

Characters.—In well-established hyperpiesis, "whether renal or not, the vessels are tightened with blood dammed back. The heart and main vessels are so full that the output of the ventricle against the resistance is less and less, while distension of the arteries is pushed towards the outward limit of their elasticity; thus their further function, as an

elastic reservoir between heart and capillaries, of forwarding the blood during cardiac diastole is checked, and diastolic pressure rises." [88]

The Heart-weight in Hyperpiesis.—From an analysis of 7,000 *post-mortem* cases occurring in twenty consecutive years at St. Bartholomew's Hospital, and from a further series of thirty-two cases,[89] Dr. Geoffrey Evans finds that a heart-weight of 15 oz. or over has some definite relation to chronic interstitial nephritis or cerebral hæmorrhage, whereas a heart-weight of from 12 to 14 oz. may occur in a great variety of other non-related conditions. "Idiopathic" cardiac hypertrophy, *i.e.*, not due to disease of heart and lung, of 15 oz. *post-mortem* is thus a direct indication of diffuse hyperplastic sclerosis, evidenced clinically by a systolic pressure of 180 mm. or more. In this group within the limits imposed by certain factors, *e.g.*, size of body, physique and adiposity, the measure of the degree of hypertonia may be stated as an increase of 1 oz. in heart-weight for each 10 mm. rise in arterial pressure.

For the reason that the walls of the remaining three chambers of the heart may hypertrophy, especially if, as a result of leakage of the mitral valve, slowing of blood flow occurs in the pulmonary circuit, Batty Shaw [90] is of opinion that left-sided ventricular hypertrophy is a better gauge of the direct effect of hyperpiesis upon the heart than the total weight of the organ. He gives a series of cases, twenty-nine men and six women, with hyperpietic hypertrophy of the left ventricle uncomplicated by valvular endocarditis, the average weight of the whole heart for the men being 18 oz. (normal 11 oz.), and for the women 14 oz. (normal 9 oz.). Greatest increase of cardiac weight was not found at the greatest ages in either sex. This is explained by the weakening of all muscles, together with lessening of tissue resistance, which coincide with advancing age.

II. Hyperpiesia

Hyperpiesia.—From *hyperpiesis*, which includes high arterial pressure arising from any cause, it is useful to separate out *hyperpiesia*, a condition synonymous with the

so-called "essential arterial hypertension," a cumbrous and hybrid term now unfortunately employed at home as well as abroad (p. 78).

Nature of Hyperpiesia.—As described by Allbutt, hyperpiesia is a clinical morbid series characterised by raised arterial pressure in association with hypertrophy of the heart and changes in the vessels, distinct from the recognised forms of Bright's disease.

It has been suggested that one of the primary causes of hyperpiesia may be found in a heightened viscosity of the blood, resulting either from a primary albuminæmia—a colloid phenomenon—or from mixed colloid and crystalloid phenomena differing in degree in individual cases. Heightened blood viscosity is the rule in polycythæmia, but of 66 out of 189 collected cases of this malady investigated by Lucas,[91] in which records of arterial pressure had been preserved, in 21 (approximately one-third) the systolic pressure was under 140 mm. Hence hyperpiesia appears neither to be caused by polycythæmia nor by increased viscosity of the blood.

Associations with other Conditions

Other accompaniments of hyperpiesia include proteinuria and ocular changes. There are also associations with such widely differing conditions as exophthalmic goitre, asthma, gout, lead poisoning, infections of and hæmorrhages into various organs and tissues, paralysis, and other conditions too numerous to mention.

Batty Shaw's [90] exhaustive study of forty-seven cases of hyperpiesia compels him to believe that hyperpiesia and eclampsia are closely allied toxic states, that both may be associated with renal disease and retinal changes, and that both may be temporary and recovered from, or may be permanent and fatal. The early symptomatology suggests that the primary causes of hyperpiesia are either psychical or toxic.[92, 93, 94, 95] Endocrine imbalance and vasomotor instability are thus probably secondary, an over-responsive endocrine-autonomic system with attendant vasomotor instability in the direction of vaso-constriction being due in

many cases to overaction of the posterior pituitary as a result of long-continued psychical stress or sudden and violent emotional strain.

Clinical Features.—Hyperpiesia constitutes *a state of primary high arterial pressure* in association with a distinct train of clinical and pathological events, recurring in many persons with such frequency of association and consistency of course as to constitute a disease, which, arising independently of arteriocapillary, renal, cerebral or other demonstrable morbid change, after a variable period of latency, to which no definite term can be assigned, clinically manifests individual characters, the chief of which are (1) high diastolic and systolic pressures, persistent but not necessarily permanent; (2) left ventricular hypertrophy. On X-ray investigation the heart is found to be enlarged, and an electrocardiogram shows left ventricular preponderance.

Hyperpiesia is among the commonest of diseases. It is wont to occur in subjects above middle age in both sexes, more often in men, and mostly between the years from fifty to seventy. The onset is insidious, for in most cases no date can be assigned to the initial changes, which may have begun at least twice as long ago as the time at which the condition was first recognised. For variable periods the subjects may have fair or even robust health, slight symptoms either having been ignored, or more usually ascribed to causes other than the actual, so that by the time that the patient seeks medical advice, the condition is already well established.

Types of Hyperpiesia.—Two forms are recognisable—labile and stable. The early labile form [96] presents a curve of wide daily fluctuations in pressure, though the general average level is definitely high. The lowest levels, often to normal values, occurs only during sleep. For a true picture of this condition, an instrument recording continuous pressures is necessary. This notable instability is more characteristic of the labile form of hyperpiesia than the actual heights of pressure recorded. The later stable form, as the name implies, shows high levels with much slighter variations, and few symptoms, whereas the labile form has many symptoms.

The majority of cases have an abnormally low sugar tolerance, which may depend upon psychical disturbance leading to a lack of endocrine balance, glycosuria, which is at times associated, depending upon this and not upon disturbance of pancreatic function.

In pure hyperpiesia, moreover, there is no evidence of blood stasis in the capillary circulation.

Among certain authors there has been a tendency to divide hyperpiesia into various types, such as paroxysmal, benign and malignant. The paroxysmal type is characterised by temporary pressure elevations above a constant high level; the "malignant" is probably a more rapid and severe form of the same pathological picture that occurs in the "benign," usually terminating by uræmia as a result of renal failure.

Irritative influences, which upset circulatory balance, whether of toxæmic origin or otherwise, tend to induce a state of ill-health, increasing the cardiac load and thus leading to cardiac failure, while the production of arterial thickening and subsequent degeneration disposes to apoplexy, renal involvement and other structural changes.

Pathology.—A. The Vasomotor Centre in the Medulla.—Most observers are in agreement that the blood pressure raising stimulus, however induced, has to do with the controlling vasomotor centre in the medulla oblongata. Such stimulus has been attributed to a heightened sensitiveness,[67] or alternatively, to arteriolar proliferation, claudication, or toxic damage of its capillaries, or interference with oxidation,[97] or to arteriolosclerosis of the medulla.[98] Arguments have been advanced, however, against both the latter suppositions.[99, 100, 101]

B. The Autonomic-Endocrine System.—Under the direction and control of the medullary vasomotor centre, regulation of the circulation is largely vested in the autonomic-endocrine system. The autonomic (vegetative) nervous system consists of two physiologically antagonistic divisions; namely, the sympathetic system (katabolic), and the parasympathetic or extended vagus system (anabolic).

The sympathetic acts with the pituitary, thyroid, adrenals and gonads, exerting also a considerable influence upon

metabolism in general, while the parasympathetic has more limited associations with the parathyroids, and with the cell-islets of the pancreas over which direct vagus control has been proved.

In turn the endocrine glands activate or depress the circulatory mechanisms *viâ* the sympathetic nervous system by means of their chemical messengers (hormones), a pressor influence being exerted by vasopressin (posterior lobe of pituitary) and by adrenaline, while the cell-islets of the pancreas furnish the depressor hormone, vagotonine.

C. Influence of the Pituitary Gland.—For many years the thyroid has been regarded as the master gland and activator of the endocrine series. Recent experiments on amphibia and mammals, however, go to prove that the differentiation and continued functioning of the thyroid depends on a thyroidotropic hormone of the anterior lobe of the pituitary, and the functions of the pituitary so far discovered suggest that it should now be held to supersede the thyroid as the dominant member of the ductless chain in bringing to structural and physiological fruition the thyroid, parathyroid suprarenals and gonads, thus exerting general effects upon the body, and through the thyroid influencing metabolic rate.

This new and striking conception is strongly supported by the recent work of Harvey Cushing.[102] From this it appears that the central representation of both sympathetic and parasympathetic systems lies in the hypothalamus, between which and the neighbouring posterior lobe of the pituitary (neurohypophysis) there is a definite nervous connection. Cushing states that the posterior pituitary lobe possesses an active principle or secretion capable, among other properties, of raising arterial pressure and of diminishing renal secretion. This active principle is derived from the pars intermedia, whose cells, when ripened, become basophilic, invade the pars nervosa and become transformed into hyaline bodies which make their way towards the infundibular cavity and extrude themselves into the third ventricles, in the fluid of which this secretion of the posterior lobe may eventually be detectable. In other words, the basophile cells normally present in the anterior lobe and

pars intermedia of the pituitary migrate into the posterior lobe and thence to the third ventricle.

In hyperpiesia, and in certain toxæmic states, *e.g.*, eclampsia, characterised by raised arterial pressure, a substance indistinguishable in its effects from posterior lobe extract has been found in the blood stream, but not detectable therein under normal conditions (Anselmino [103]). The same or a similar substance has been found in the blood of certain patients with hyperpiesia (Irvine Page).

From the above, Cushing derives the conclusion that *infiltrative basophilia of the pars nervosa of the pituitary gland is an expression of functional overactivation of the posterior lobe, and represents the pathological basis of these hyperpietic disorders.*

D. Influence of the Adrenal Glands.—The next step is to determine as far as possible how this stimulus to pituitary overaction is brought about.

Excessive secretion of adrenaline has been invoked by Vaquez,[104] Josué [105] and Paul [106] as a cause of raised arterial pressure. In support of this belief various reasons may be adduced :—

(*a*) " Preganglionic sympathetic fibres end in the adrenal gland, whose medullary cells *are* the sympathetic ganglion cells, and we have here an interesting example of a nervous structure assuming secretory properties. It is of striking interest to find, as Langley showed, that their secretion, adrenaline, has the same effect on any part as stimulation of the sympathetic nerves to that part." [107]

Thus the adrenal glands are closely related to the sympathetic, and the effect of adrenaline on any part is to heighten the sensitiveness of response to the sympathetic in the same way as by stimulating its sympathetic nerve, which in turn increases the flow of adrenaline into the circulation. Reaction to infection and to elementary emotional experiences has been explained as an emergency adrenaline response of protective character. The reciprocal associations of the thyroid with the sympathetic are similar, but katabolic. Many of the high pressures which I saw in soldiers invalided for " disordered action of the heart " were of this type, sustained endocrine-sympathetic reaction to

the fear sense being evidenced by the staring eyes, sweating, quick pulse and tremors, resembling the acute stage of hyperthyroidism.

(*b*) Healthy human adrenal glands together contain approximately 8 mg. adrenaline, an amount which in high pressure states is increased in conjunction with hyperplasia of the adrenal medulla.[108] Bru[109] injected 10 c.c. anti-medullary serum intravenously into dogs, and found within five minutes a rise in arterial pressure which lasted for over ten minutes.

(*c*) Although in hyperpiesia the presence of adrenaline in the blood has not been established, it is, nevertheless, probable that a flow of adrenaline into the blood from the cells of chromaffin sympathetic tissue in the adrenals and elsewhere exerts an influence upon the adjacent sympathetic system. An analogy exists in the autonomic system in which an "autonomin," vagotonine, has been isolated by Santenoise[110] in pure form from the internal secretory portion of the pancreas.

(*d*) Primary tumours of the adrenal cortex have been found occasionally attended by a permanent hyperpiesis, and the rare adrenaline-secreting adrenal medullary tumours give rise to paroxysmal or permanent hyperpiesis. The subjects of chromaffin tumours have manifested paroxysmal hyperpietic crises.

Discrepancies noted by numerous observers between the extent of pathological changes in the adrenal glands and the clinical appearance of hyperadrenalæmia, giving rise to so much controversy and for which no satisfactory explanation has been forthcoming, may, however, be rationalised if it be remembered that *secretion of adrenaline is not limited merely to the adrenal tissue, but is shared by other islets of chromaffin sympathetic tissue scattered throughout the body.*[111]

On the other hand, Cushing[102] has adduced more recent evidence that certain reported cases of cortico-adrenal hyperpiesis are examples of basophile adenoma of the anterior lobe of the pituitary gland. Out of eighty patients with various forms of hyperpiesis treated by Abrami, Santenoise and Bernal[112] with vagotonine, usually by

hypodermic injection, thirty derived permanent benefit, the best results being obtained in those with paroxysmal crises of hyperpiesis, while several patients did not react. "These results, taken in conjunction with the paroxysmal crises of high blood pressure in patients with chromaffin-celled tumours of the adrenals, lend support to the view that essential hypertension is not due to excess of adrenaline in the blood." [113]

E. Other Pressor Substances.—Considerable endeavours have been made to isolate other pressor substances in the blood, due to altered metabolism or to inadequate excretion, particularly of protein derivatives (*vide* Chapter VIII).

Since 1924 Major and Stephenson [114] have studied the probable *rôle* of guanidine compounds in the production of raised arterial pressures, and have determined the daily output of guanidine bases during the period of falling arterial pressure. This fall coincided with a gradual rise in output of dimethyl guanidine, the rise being maintained for several days after the arterial pressure decline, thus suggesting that increased diuresis eliminates an excess of pressor substance which has been retained in the body.

In about half the number of hyperpietics in a large series investigated, Major [115] found an increase in the blood of a substance with reactions similar to those of guanidine. These results have received confirmation from Pfiffner and Myers.[116]

Other non-protein nitrogenous substances such as creatine and creatinine appear to have no effect in raising arterial pressures.

Since tyramine may be produced *in vitro* from tyrosine by the action of fæcal bacteria, it is not unreasonable to suppose that the presence of this substance in the large bowel and its absorption therefrom may promote pathological conditions of which heightened arterial pressures constitute a prominent symptom. As in the case of adrenaline, lengthy administration of tyramine induces renal and vascular lesions akin to those which so generally accompany persistent hyperpiesis in man. Further, it is of interest to note that there is a relationship between the amino-acid group, the blood nitrogen urea, and the diastolic pressure, so that, if

one can control the diastolic pressure by checking absorption of pressor amines, systolic variations are of less moment.

TABLE VIII

Differential Diagnosis between Hyperpiesia and Chronic Glomerular Nephritis

	Hyperplesia.	Chronic Glomerular Nephritis.
History	No definite antecedents.	Preceded by subacute nephritis or long latent stage.
Appearance	Robust; often plethoric.	Variable; sometimes pallid.
Age	50–70 years. Rare under 40.	Usually under 40 years.
Arterial pressure	May be very high, 250 + Labile: often falls with rest.	Moderate or high. Stable.
Retinitis	Uncommon.	Frequent.
Renal function tests	Normal, or slight changes.	Reveal inadequacy.
Urinary findings:		
Volume	Augmented.	Augmented; diminishing at the last.
Specific gravity	Low: may be fixed.	Low and fixed.
Albumin	Absent or slight trace.	Slight in early stage: abundant in late.
Casts	Absent usually.	Scanty, hyaline or fatty.
Red blood cells	Absent.	Absent without acute exacerbation.
Nycturia	Absent.	Present.
Blood findings:		
Nitrogen retention	Absent.	Present.
Anæmia	Absent or slight.	Present.
Uræmia	In about 8 per cent. of cases.	Frequent.

III. Hypertonia

Hypertonia (Angiospasm) represents an *active* state of the smooth muscle in the walls of arteries and veins, in contradistinction to high arterial pressure (hyperpiesis) which indicates the lateral pressure exerted by the blood upon the vessel wall.[117]

Normal arteries are barely palpable, but in hypertonia both qualitative and quantitative changes in hardness occur. Arterial hypertonicity, in larger or smaller areas, is met with in a host of disorders. In some the hardness persists; in others it may lessen with time. To realise that

long-hardened arteries may suddenly become soft, one has only to study the vessels of the paralysed side in hemiplegia. Since hardening may coexist with weak cardiac action, there can be no necessary connection between hardening and increase of blood pressure, though, in general, increased tonus is linked with increased pressure, and is compensatory.

Owing to the presence of smooth muscle in the walls of the veins, hypertonia, and even hypertrophy, may also occur in these.

Arteries may vary considerably in calibre (*vide* "Tonus," p. 77). Narrow rather than wide is the rule in hypertonic vessels. A narrow hypertonic artery feels very different from a wide one, yet the former is found in secondary contracted kidney cases, while the latter may at times be met with in association with arteriosclerotic kidneys.

To some extent raised arterial pressure may be differentiated from arterial hypertonus by instrumental methods, yet the varying degrees of each cannot be measured independently (p. 115, B, *b*).

Associations of Hyperpiesis, Hyperpiesia and Hypertonia (Vascular Hypertonus), with Arteriosclerosis and Renal States.

Persistently raised blood pressures do not *necessarily* connote anatomically altered arteries, though presumably the muscle elements manifest some increase in number and size. That hypertrophy of the vessel wall can, however, occur in order to keep up a high blood pressure has been proved by Alexis Carrel[118] and by Fischer and Schmieden,[119] who showed that sections of a vein transplanted into an artery not only did not dilate in consequence of the increased pressure, but actually became thickened with narrowing of their lumina.

The following schema will be found helpful in contrasting and correlating these conditions, any one of which may, and often does, blend with the others :—

A. **Primary (Essential or Benign) High Arterial Pressure or Hyperpiesia.**

(*a*) Temporary, at times over long periods.

(*b*) Persistent.

B. **Primary (Essential) Arterial Hypertonus, or Hypertonia.**

(*a*) Temporary.

(*b*) Persistent—a "functional arteriosclerosis." Of unknown origin, it may arise independently, and, whether connected with granular kidney or not, is at times associated with excess of erythrocytes in the blood, constituting the condition known as "hypertonia polycythæmica." At first latent, subsequently it tends to cardiac hypertrophy, with its congestive states of headache, irritability, insomnia, and the like. In the large arteries no foci of atheroma are discoverable. Increased blood viscosity is a constant accompaniment of polycythæmia rubra, but neither probably accounts for the rise in arterial pressure which may be due to the onset of congestive heart failure in late stages.

Functional hardness is related to calcification: in both the media is affected, and the condition is general and not focal. Often, however, differential diagnosis is impossible.

C. **Arteriosclerosis.**

(i.) Diffuse hyperplastic sclerosis.
(ii.) Mönckeberg's sclerosis.
(iii.) Senile sclerosis.

D. **Renal States.**—The pathogenetic classification of these is as follows:—

Degenerative—nephrosis.

Inflammatory—nephritis—acute, subacute or chronic.

Chronic nephritis may be:

(i.) Parenchymatous.
(ii.) Interstitial.

(i) *Chronic Nephritis, General or Parenchymatous.*—Glomerulitis and inflammatory new fibrous tissue are distributed throughout the kidneys, with a tendency to association with cardiac hypertrophy and diffuse hyperplastic sclerosis.

Clinically this form is characterised by defective elimination of salt, with resultant production of œdema and ascites.

(ii) *Chronic Interstitial Nephritis.* (*a*) *Partial.*—"The lesion is limited to wedge-shaped areas of fibrosis containing damaged renal parenchyma and more or less small-cell infil-

tration. The intervening tissue is relatively normal, and the parenchyma may be hypertrophied." [89]

True Chronic Interstitial Nephritis. (*b*) *General.*—There is histological evidence of active inflammation, and the most marked cases of diffuse hyperplastic sclerosis are found in this sub-group.

Clinically this form is characterised by defective elimination of waste products of nitrogenous metabolism, which accumulate in the blood, and is associated with cardiac hypertrophy and excessively high and continuous blood pressures.

(*c*) *True Sclerosis.*—This form may again be sub-divided into two, according as the vascular lesion is of the type of—

(α) The arteriolosclerotic kidney.

(β) The arteriosclerotic kidney.

In (α), hyaline material is laid down immediately beneath the endothelium, increasing in amount with progress of the disease. This hyalinisation or sclerosis involves the smaller arterioles of the afferent or efferent vessels, but there is never a true arteritis. It is probable that this damage to the arterioles of the kidney is pathognomonic of hyperpiesia.

In (β), there is a diffuse, elastic hyperplasia affecting the intima, and causing thickening thereof, which may extend only as far as the afferent glomerular arterioles. This is the senile form, unless of severe degree at younger ages, when it points to hyperpiesis. It is of little clinical import, as it practically never causes renal insufficiency.

So much confusion still exists as to the precise meaning to be attached to the terms hyperpiesis, hyperpiesia, and hypertonia, and these terms are so loosely used by many writers, that the author has deemed it of urgent necessity to clarify, so far as lies in his power, the current obscurity which enwraps the various aspects of high arterial pressure, and to indicate the essential differences between them by means of the above descriptions of each condition and by Table VII.

CHAPTER VII

ARTERIOSCLEROSIS

" Our age is to be reckoned by the state of our arteries."
SAMUEL GEE: *Medical Lectures.*

To Lobstein (1833) we owe the term " arteriosclerosis."

Since the publication of Allbutt's well-known views, this complicated problem has been further investigated by an able group of contributors under the editorship of Dr. E. V. Cowdry in " Arteriosclerosis; A Survey of the Problem " (Macmillan, 1933). The following description is a brief synopsis of the work of these and other writers.

Definition

Arteriosclerosis may be looked upon as " a chronic disturbance of the vessels which manifests itself by deposits of the most varied kinds in the vascular walls and which becomes irreversible on reaching its climax in vessels impaired by changes attending the process of ageing with resulting deformation of the lumen and brittleness of the vascular walls " (Aschoff).

Causation of Arteriosclerosis

As to the ætiology of arteriosclerosis, little definite is yet known. In production there appear to be two main elements, (1) changes due to advancing age resulting in distension, dilatation and eventual tortuosity of the arteries along with trophic disturbances resulting in deposition of waste material, (2) pathological changes, which represent the essential nature of the malady.

The various suppositions may now be reviewed in greater detail :—

(*a*) Age changes do not constitute the whole story. Arteriosclerosis in most instances is a senile malady of the

vessels, seldom seen below the age of fifty years and rare above the age of seventy years—the "decrescent" form of Allbutt—independent of known toxin or rise of arterial pressure, but possibly induced by increasing deviation of the acid-base equilibrium to the acid side, a metabolic phenomenon of advancing years, leading to greater difficulties in elimination of waste products.

(*b*) Physico-mechanical effects of wear and tear over long periods of time.

(*c*) Psychical effects of long-continued stress and strain.

(*d*) Trophic disturbances.

(*e*) Changes in composition of the blood plasma, and thus of the vascular lymph, leading to absorption, infiltration of vessel walls and precipitation therein.

(*f*) Faulty regulation of metabolism by hormones and vitamins; by unbalanced diets, especially in the direction of prolonged and excessive intake of proteins and lipoids; and by somatic errors of metabolism, as gout, obesity, diabetes.

(*g*) Specific poisons, such as those of syphilis, lead and nicotine; hormonic over-production of pituitrin, adrenaline, thyroxin.

(*h*) Heredity. Osler's [120] references to good or bad quality of the "vital rubber" inherited are well known, and numerous writers [121, 122] have emphasised the part played by heredity in determining vascular disease.

(*i*) Habits. Sedentary habits are thought to dispose. Active animals are found to be less liable to arterial changes than those that are more passive.

(*j*) Cosmic factors, such as damp, cold, heat, light, climate and atmosphere, have been thought to exert some influence, but evidence as to these is vague.

(*k*) Acute and chronic infections, as well as intestinal toxæmia, have been invoked, but probably have no effect.

(*l*) Alcohol. In the past, alcohol received frequent mention as a cause of arteriosclerosis. The balance of evidence, nevertheless, is entirely against alcohol of itself as an ætiological factor. Out of 283 cases of cirrhosis of the liver associated with excessive alcoholism in persons under fifty years of age, Cabot [123] found that only 6 per cent. had

arteriosclerosis. In another group of 656 arteriosclerotic subjects, only 95 (14·5 per cent.) were under fifty years of age, and of those ninety-five cases only 17 per cent. took alcohol. Mohammedan pilgrims, who never take alcohol, frequently develop arteriosclerosis.[83]

(*m*) Environment. Much discussion has arisen as to the effect of different industrial surroundings on the health of workers. Apart from its special dangers, coal-mining has been as healthy an occupation up to the age of fifty-five years as that of any manual labour. After that age, mortality among miners rises rapidly, mainly because of arteriosclerosis, which was found by Dickson [124] in 91 per cent. of patients, a noteworthy feature being its great frequency in youths of twenty years or under. Arterial pressure was rarely increased. Experts agreed that the physical work done by miners does not exceed that of any class of ordinary labourer. Hence the cause was attributed to the situation of the work, either by reason of lack of sunlight or of the composition of the air breathed. Thus it would appear that sunshine and fresh air are as requisite for middle and later ages as for childhood.

Pathology

The arterial system is composed of three different kinds of vessels: elastic arteries of supply (aorta and its main branches), smaller muscular arteries of various sizes in muscles, organs and tissues (*e.g.*, radial), and smallest arteries (arterioles). Manifestations of reaction to different injuries, though having a broad similarity, differ in individual qualities and degrees.

Probably the intima is first affected. The internal elastic lamina begins to split. This is succeeded by progressive formation of elastic tissue, alterations in the composition of which form the chief feature of the arteriosclerotic process.

The arterial walls may themselves be divided into three layers—intima, media and adventitia. Arteriosclerosis is characterised by involvement of the entire vessel wall, the relative proportions of intimal and medial affection depending solely upon the nature and position of the artery. In

these several layers chemical changes occur resulting in deposition of lipoids, calcium and various proteins. Such chemical changes in the intima—atheromatosis, atherosclerosis, arteriolosclerosis—should be differentiated from calcinosis or calcification of the media, to which the term "arteriosclerosis" is most often limited.

Fibrous tissue and elastic fibres may undergo a further change into hyalinisation. Like changes may occur in muscular arteries, but are then secondary to hypertrophy, and perhaps also to hyperplasia, of the medial muscle.

From this pathological consideration one may proceed to separate out for discussion four main varieties, though, by reason of the many gaps in our present knowledge, neither this nor any other category can be regarded as comprehensive or final :—

I. Diffuse hyperplastic sclerosis, including arteriolosclerosis ; atherosclerosis ; atheroma.
II. Mönckeberg's sclerosis.
III. Senile arteriosclerosis.
IV. Syphilitic arteritis.

I. (A) "Diffuse" Hyperplastic Sclerosis.

Disease of the tunica intima has been variously termed "diffuse hyperplastic sclerosis" (detailed by Jorès,[125] Aschoff,[126] Gaskell [127] and G. Evans [89]), "arteriocapillary fibrosis" (Gull and Sutton),[128] "atherosclerosis" (Marchand, 1904) or in the larger vessels "atheroma."

1. *Morbid Anatomy.*—(*a*) Small arteries and arterioles, especially of the kidneys and spleen, are thickened, and protrude like quills from the cut surface. When arterial pressure has been raised during life, the larger arteries are also thickened and the hypertrophied media appears as transverse ridges on the inner surface of the vessel wall. This is a primary hypertonia, which induces secondary changes in the larger arteries.

(*b*) The heart shows idiopathic hypertrophy, particularly of the left ventricle, a secondary hyperpiesia to overcome a primary hypertonia.

2. *Morbid Histology.*—The essential lesion is a thickening of the intima in the smallest arteries, largely due to intense

tissue activity, evidenced by swelling and proliferation of the endothelial cells, *i.e.*, a primary hypertonia.[200] Secondary fatty degeneration of these cells ensues, leading finally to occlusion of the lumina of the terminal arterioles in the kidney and of the smallest vessels in other organs.

The general distribution of the sclerosis is fairly constant, the kidney being most often affected (a primary hypertonia), and the spleen being the best indicator of the degree and extent of the lesion in the body as a whole. The splenic condition records a secondary hyperpiesia, a second site in which we may estimate the degree of hypertonia, the spleen tissue being soft and reacting readily.

3. *Clinical Associations.*—(*a*) High blood pressure and hyperglycæmia frequently, sometimes obesity.

(*b*) Idiopathic cardiac hypertrophy.

(*c*) " It is found in children with renal disease, in adults with chronic diffuse nephritis, and as an incident when death has resulted from some other not related cause." [89]

Christian [129] and Wiseman [130] have drawn attention to the close relation which exists between atherosclerosis, chronic nephritis and certain degenerative changes in the heart, which may be unified as the late stage of an " essential " high pressure condition which began many years before.

Up to, but not after, age thirty, association with chronic nephritis is frequent.

(B) **Atheroma.**

Atheroma is a patch of " diffuse " hyperplastic sclerosis in the intima of large vessels, but there is no histological difference between patchy and diffuse atherosclerosis, this term well expressing the essential identity of the two varieties.

1. *Morbid Anatomy.*—Distributed haphazard over the aorta and its branches, especially around the orifices of the larger vessels, are nodules of various size which form slightly raised yellowish-white plaques. In the abdominal aorta these tend to be largest and most numerous.

2. *Morbid Histology.*—Similarly to the diffuse hyperplastic form, the essential and initial change is *a thickening of the intima* due to proliferation of the superficial cells and

deposition of lipoids, confined to the intima save where the deeper layers are of loose mesh (aorta, carotids, coronaries) when the media also becomes involved. Later the thickened intima is permeated by elastic fibrils as an essential compensatory change. These lesions may develop into nodular sclerosis or may heal. In certain cases the intimal thickening may be compensatory to a primary affection of the media.

II. **Mönckeberg's Sclerosis.**

A primary medial calcification, especially favouring the limb vessels, not necessarily associated with intimal changes nor preceded by lipoid change—a primary calcification of the muscle fibres and the surrounding collagen fibres, leading ultimately to necrosis. The process begins in the layer immediately adjacent to the elastic layer, and results in the formation of annular calcareous plaques. There is little or no intimal proliferation, and the not infrequent incidence in comparatively young subjects seems to point to some toxic or infective factor in causation over and above simple wear and tear.

The blood has a potent share in the causation of these vascular affections, for all diseases associated with a large increase in blood cholesterol, such as hypertonia, cardiac disease and diabetes, lead to augmented deposition of lipoid.

For the sake of completeness, one may add to the above—

III. **Senile Arteriosclerosis.**

1. *Morbid Anatomy.*—A diffuse change, which in established cases is often indistinguishable from I.

2. *Morbid Histology.*—It is *primarily a medial degeneration*, beginning with fatty degeneration and atrophy of the muscle fibres and affecting both larger and smaller vessels (Klotz). This is followed by replacement fibrosis and deposition in the media of lime salts, which, during life, are present in a state similar to that of unset mortar (p. 31), solidifying only after death. Intimal thickening, either diffuse, focal or both combined, is generally an accompaniment. It differs from diffuse hyperplastic sclerosis both in respect of site and the characters of its initial lesion.

3. *Clinical Associations.*—In this form cardiac hyper-

trophy and raised blood pressure are both entirely absent. Senile arteriosclerosis is essentially metabolic in character, being probably due to an error in the calcium balance of contractile tissues, but may arise from a potash deficiency caused through lime excess. In other instances it may be primarily toxic. It is doubtful whether purely mechanical factors are responsible for its ætiology. In elderly people cerebral arteriosclerosis may be mistaken for neurasthenia because of symptoms of readily induced mental and physical fatigue, lapses of memory and a lack of mental clarity.[167]

IV. **Syphilitic Arteritis.**

When recent, this constitutes a distinct type, but later becomes indistinguishable from ordinary arteriosclerosis. The intima is first affected, but all three coats are prone to involvement, and active proliferation, largely perivascular, may be noted in the adventitia.

Hyperpiesis and Arteriosclerosis

Since pathological changes due to arteriosclerosis may occur in childhood, or before the advent of any notable rise in arterial pressure, there is no general agreement as to whether such changes are dependent upon hyperpiesis.

Evidence is accumulating to suggest that hyperpiesis is responsible for arteriolar, but probably not for arterial, thickening. My own view is that intermittent hyperpiesis in the form of angiospasm resulting from sympathetic over-action tends to induce a state of hypertonia, and that hypertonia, maintained over increasingly long periods of time, gives rise to arteriolar constriction and may eventually lead to actual thickening of the arteriolar walls (*vide* Chapter VIII, section 8). On this view arteriolar thickening is secondary and not primary to hyperpiesis.

The position may be thus summarised :—

1. Hyperpiesis can run its course independently of arteriosclerosis.

2. Arteriosclerosis can and does run its course in more than half the total number of cases independently of hyperpiesis.

3. Persistent hyperpiesis *per se* is considered by some not to favour the development of arteriosclerosis.[201] Others are strongly of opinion that it does. At present the matter should be regarded as not finally settled.

Hyperpiesis is independent of Arteriosclerosis

No support can be found for the view, so long held, that the heart has a selective action which enables it to overcome a general or local resistance due to arterial occlusion [131, 132] nor for the deduction that hyperpiesis is consequent upon arteriosclerosis for which age changes have been held responsible, in view of the fact that sphygmomanometric findings have established both the presence of hyperpiesis in the absence of any factors which induce arterial narrowing and, conversely, the absence of hyperpiesis in healthy and vigorous old men, whose pressures remain constantly below 150 mm. Hg. Indeed, it may now be taken as amply proved that arteriosclerosis without high arterial pressure is a clinical and pathological entity.[133, 134, 135]

Hyperpiesis and Atheroma

The causal relation between hyperpiesis and atheroma is supported by considerable evidence. The readiness with which blood serum penetrates the walls of arteries is highly significant, and this depends both upon their structure and upon the height of the arterial pressure. Certain diets are known to produce atheroma, which leads to the hope that a diet capable of staving off the onset of this disease may eventually be discovered.

CHAPTER VIII

The Causation and Significance of High Arterial Pressure

"High pressure is an attempt of the organism to maintain the equilibrium of its circulation." Allbutt: *Diseases of Arteries.*

The primary cause or causes of pathologically high arterial pressures are not yet definitely established.

Hence this fundamental difficulty confronts us as soon as we attempt to get back to first principles. Much confusion, too, exists by reason of the many different names employed by numerous authors to describe conditions which are identical or overlapping, some attacking the problem from biological and clinical standpoints, others from a groundwork of morbid anatomy and pathology.

While we are tentatively assured of certain factors being associated with the origin of hyperpiesis, we are unable to point to any one with a definite conviction of being right. Yet the time is probably not far distant when the real causes will be brought to light, and measures taken that will prevent the development of conditions which to-day are responsible for a mortality that is far too high.

Transitory variations from physiological standards for age and weight capable of wide modification to meet the varying needs of daily life have been dealt with in Chapter V. We now have to consider temporary or permanent variations within still wider ranges due to some underlying cause or causes of pathological origin.

CAUSAL FACTORS

In the light of our present knowledge the following considerations lend support to a provisional attempt to tabulate the causes of raised arterial pressure as follow :—

1. Compensatory.

2. Mechanical—the effect of " wear and tear " upon the cardio-vascular system.
3. Hereditary and familial influences.
4. Arteriolar constriction.
5. Metabolic influences.
6. Deficient elimination.
7. Toxæmic influences. (*a*) Bacterial, (*b*) chemical.
8. Psychical influences.
9. Endocrino-sympathetic influences.
10. Allergy.
11. From the interaction of two or more of the above causes.

1. **Compensatory Influences** to maintain an efficient circulation.

2. **Mechanical Influences.**—It has been suggested that in hyperpiesis associated with arterial degeneration, the effects are due to " wear and tear " upon the cardio-vascular system, the initial qualities of the " vital rubber " conditioning the degree of arterial resistance to stresses and strains.

The main mechanical cause of high arterial pressures is increased peripheral resistance, *i.e.*, " an augmented frictional resistance to the onward passage of blood from the arteries, dependent on narrowing of the outlet by active contraction of the arterioles or by structural change " [67] (*vide* 4, *infra*).

3. **Hereditary and Familial Influences.**—These undoubtedly play a part, as is seen in cases of congenital narrowing of the blood vessels, and in those which tend to early arterial degeneration by reason of the poor quality of elastic tissue of which their walls are composed. Again, the quality may vary in different branches of the arterial tree. A family tendency is detectable in more than one-third of all histories.[137]

4. **Arteriolar Constriction.**—Formerly it was believed that, with a constant cardiac output, the amount of blood flowing through the capillary circulation was regulated primarily by the arterioles from which they originate, and secondarily by the venous pressure ; in other words, that passive dilatation of the capillaries with rise in intracapillary pressure occurred when the arterioles dilated, while, when

these latter contracted, the capillaries by reason of their elasticity resumed their state of tonic contraction.

As regards ætiology, diagnosis and treatment, however, in 1919 Krogh [138] opened up a new and valuable line of thought by bringing forward evidence, which has been corroborated by Lewis,[139] Dale [140] and other observers,[141, 142] that the arterial and capillary systems are regulated by their own special mechanisms, and thus react and function independently. Krogh has proved that not only do the capillaries contract and dilate apart from the influence of the arterioles, but that their calibre when the tissue they supply is at rest is considerably less than when the tissue is active, showing that the capillaries exist in a state of slight constrictor tone.

Two Probable Groups of High Arterial Pressure.—A. Without high capillary pressures.

That active constriction of the arterioles precedes the onset of œdema and of hæmorrhage into the retina can be demonstrated most readily in cases of toxæmia of pregnancy. From this and other evidence it seems obvious that spastic constriction of the arterioles is a significant factor in the causation of hyperpiesis itself and of many of the symptoms and complications. Experimental results further show that in renal arteriosclerosis the arterioles alone are involved, the capillary pressure remaining normal.

B. With high capillary pressures. In chronic interstitial nephritis the capillary system is associated with, even if not the actual cause of, the raised pressure. It is probable that the more severe and rapidly progressive types of hyperpiesis have their earliest manifestations in a diffuse lesion of the vascular system (diffuse hyperplastic sclerosis) of which involvement of the kidney is only a part, and not, as previously supposed, the cause.

In contrast to extensive fluctuations which occur in states of simple high arterial pressure, the pressure in the capillaries is usually normal or even below normal. Thus, a systemic pressure of 200 mm. in the brachial artery becomes broken down to one of 20 mm. in the capillaries of the fingers with corresponding obliteration of the differential pressure.[143] This damping down of pressure must necessarily take place

in the arterioles, which act as transformers, and has a greater effect in regulating capillary circulation than the arterial pressure itself.[144] It is the resistance in the terminal vascular bed which not only reduces high to low pressure but converts the intermittent, pulsating arterial flow into an even, continuous capillary current. Hence the real seat of rise in systemic arterial pressure lies in increased tone of the tiny arterioles, their smooth muscle cells being the anatomical tissues directly concerned.

Since the blood mass cannot fill more than half the vascular bed, and since local vasoconstriction is readily balanced by vasodilatation elsewhere, hyperpiesis " can only arise when the machinery of regulation is damaged or the pressor stimuli are too powerful and overcome the depressor mechanism." [131]

For such elevation of pressure to be induced, increase in tone of the muscle elements of the arterioles must be widespread. Constriction limited to the vessels of the kidney, to the splanchnic area or even to the extremities is insufficient to bring about hyperpiesis.[145]

" In the healthy person at rest the arterial pressure which is normal for that individual is remarkably constant within narrow limits, apart from ascertainable disturbing conditions; the regulating nervous mechanism (medullary centres) is very efficient. But in the high-pressure subject this regulating mechanism has ceased to be adequate; the pressure remains at an abnormally high level, and is often subject to sudden and extensive variations, sometimes in the absence of any recognisable cause or from slight exciting influences that would have little effect in the normal individual. The normal regulating mechanism includes the depressing or inhibitory effect on the vasomotor centre of high blood pressure acting (1) directly on the centre, and (2) through afferent impulses from the heart, aorta, sinus caroticus, etc. In addition, there is the controlling influence of the vagus centre on the cardiac pump, this centre being also influenced directly by the blood pressure in it and reflexly by afferent impulses from the heart, aorta and sinus caroticus." [67]

What, then, are the underlying causes that in high

pressure states bring about either temporary arteriolar spasm or more permanent vasoconstriction ?

5. **Metabolic Influences.**—The precise nature of the neuro-humoral impulse that primarily induces neuro-hypophysial basophilia, and thus secondarily activates the vasomotor centre in the medulla, has yet to be determined. It may be that ischæmia of the centre is brought about by a selective constrictor effect upon its arterioles exerted by an autogenous toxic substance, itself arising as a result of disordered metabolism.

Up to now no one chemical substance circulating in the blood can be invoked as the primary cause. From the knowledge afforded by recent biochemical investigations into the chemistry of the body, it would not be difficult to assume that all hyperpiesis begins as a disturbance, whether as a result of toxæmia or otherwise, of the body metabolism, a tissue-resistance problem involving cellular, lymphatic and arteriolo-capillary balance which varies enormously with age and with deviation of the acid-base balance in the direction either of acidity or alkalinity.

6. **Deficient Elimination.**—Deficient elimination through the kidneys, skin and lungs is another important factor, since the body becomes thus unable to get rid of its waste material.

Attempts have also been made to show that, apart from the rise in blood pressure associated with efforts on the part of the kidneys to secure effective elimination of concentrated urine, salt or protein, heightened blood pressure may be due to endeavours of the kidney to secure adequate elimination of acid waste products when their production is in excess of normal, or when there is some defect, functional or structural, in the renal mechanism. Much further evidence on the association of acidosis with high blood pressure is, however, still required.

7. **Toxæmic Influences.**—In the cases with normal kidneys some prerenal causal factor must be sought for hyperpiesis, which is often afforded by *chronic toxæmia*, induced by :—

(*a*) *Bacterial Intoxication.*—This arises from septic foci, *e.g.*, tonsils, gums, teeth, gall bladder, appendix, etc. The intestine is the main source, Bacillus coli being largely

responsible. Streptoccal or pneumococcal infection of the upper air passages or lungs causes destruction of red blood cells with resultant lessening of processes of oxidation in the blood. B. coli may then become deprived of its free oxygen, and so have to alter its mode of living. Organisms swallowed in the saliva also find their way into the intestine, so that the latter becomes a nidus for organisms which begin their activities in the mouth or respiratory system. These can usually be isolated from the fæces. After some time the liver becomes unable to carry on detoxication adequately enough to rid the body of abnormal products of metabolism set up by these organisms, the evidence being a slow rise and fall in the blood sugar tolerance curve, the opposite of the normal picture. It is probable that direct effects upon the blood vessels may be caused by bacterial toxins in like manner with syphilis, or that indirect influences may be exerted through damage to the kidney during excretion of live bacteria.

Under the influence of infections proteinogenous substances may be formed in the blood, capable of inducing spasm, similar to those which culminate in the eclampsia of pregnancy. Under ordinary circumstances they are dealt with by the liver, but when the liver suffers as the result of infection, these substances accumulate in the blood, and high blood pressure results from spasm of the arterioles. The effects of toxæmia are most apparent in the kidneys by reason of their vital importance, the manifestations produced thus forming only one symptom of the general poisoning.[146]

(*b*) *Chemical Intoxication.*—(i.) Arising within the body. Digestive troubles of the nature of spasm and atony of stomach and bowels with impaired secretion of digestive juices may arise as a result of sympathetic nervous dysfunction. Some of these toxins have been identified; others are only surmised. They are capable of producing states of high and low pressure, and originate most frequently in the course of intestinal stasis with resultant fermentation, putrefaction and absorption of poisonous products of incomplete digestion into the blood stream. Putrefaction arises most often from a high protein diet, split products of protein digestion being formed through faulty metabolism, thus giving rise to

chemical poisoning which appears to cause endocrine stimulation sufficient in amount to produce irritability of the vasomotor system leading to initial transitory changes in arterial pressure, which later become permanent.

Biochemistry has shown that chemical bodies of the nature of amine bases are formed during putrefaction, *e.g.*, skatol, and even during prolonged peptic digestion, the various symptoms depending upon pathological absorption from the gastro-intestinal tract of different amine bodies, some of which are pressor whilst others are not. " Designed as an intensive preparation for action or defence, the sympathetic response may be so dissociated, perverted or prolonged as to disorganise digestion by exciting spasm and atony in stomach and bowels and inhibiting the secretion of digestive juices ; it may keep blood pressure at a level which is inappropriate for the task of the heart and the arteries. These effects are not necessarily distinct. Thus intestinal stasis from sympathetic inhibition causes poisons of putrefactive origin to be absorbed, which in their turn lead to vasoconstriction, and hence an unduly raised blood pressure." [60] According to Robertson,[147] the aliphatic monamines exert a physiological action akin to that produced by stimulation of the sympathetic nervous system, the most active of the monamines derived from the amino-acid cleavage products of protein being tyramine, which on intravenous injection causes a rapid and pronounced rise in arterial pressure about one-twentieth of that exerted by adrenaline but more prolonged.

The proof of the existence of intestinal auto-toxæmia is the presence in the urine of indican, when constant and in undue amount, but in hyperpietic states this is seldom found, and injections of indol are without effect upon arterial pressures. At the same time, it is probable that arterial degeneration in various kinds and degrees results from absorption into the blood stream of certain toxic substances. The respiratory tract is also another source of chronic toxæmia.

Hence, although we cannot point to any one factor as the definite source of origin of supernormal arterial pressure—and it is highly probable that more than one source exists—

a mass of evidence has been brought forward supporting the hypothesis that the majority of cases of hyperpiesis are in reality due to circulatory toxins arising from infective or autogenic sources. The results of treatment are strongly in favour of this view.

(ii.) Arising outside the body. Of these the best known is chronic poisoning by means of lead, which directly induces arterial sclerosis.

Whilst for the sake of clearness I have classified the above causative factors into psychical, endocrine-sympathetic and toxæmic groups, it should be remembered that these often interpenetrate and overlap, so that the clinical picture may be a composite one, necessitating evaluation of the due proportions of its several constituents.

8. **Psychical Influences.**—Mind has a potent influence over matter. If one reviews cases of hyperpiesis affecting the greater circulation, one cannot fail to be struck by the fact that many originate from psychical causes, and it is only in late stages that processes of disease become manifest. Numerous instances are found as the result of long-continued anxiety states, or of repeated emotional shocks, which send up arterial pressure and induce angiospasm. The stress of life due to faulty methods of living and repeated worries contribute largely to arterial changes. This hypertonia of vessel walls, at first transitory, may pass into a condition of permanent contraction, and finally end in true thickening of the muscular coats.

When daily anxieties are carried over to be rehearsed during the night, the pressure remains at high level and the vasoconstrictors are working overtime. High peripheral resistance thus becomes a constant factor, cardiac energy is augmented and arterial degeneration takes place at the weakest points, which may be in the kidney (renal arteriosclerosis), but more often is in the brain (cerebral arteriosclerosis) with risk of apoplexy.

Moschcowitz,[148] of New York, has studied this aspect, and states that, in his experience, most hyperpietics conform to a certain type, which he thus describes :—

" Physically, these people are soft-muscled, short-necked, ungraceful, non-athletic and over-weight. Psychically, they

are the opposite of the child. They do not play. They have no illusions. They are tense and irritable, with 'single-track' minds. While their mental horizon is narrow, within this range they are terribly concentrated, and pursue their aims with grim desperation.

"Many belong to the class of the successful, if by 'success' one refers to accumulation of wealth or power that is not always accompanied by a spiritual or ethical uplift. One can readily grasp why such individuals are successful, for they throw everything in life aside, especially play, that does not directly contribute to their purpose. One of the commonest and most pathetic experiences in medical practice is the tragedy of the 'successful' man.

"On the other hand, it is not always the desire for 'success' that causes mental conflict. Those who have fallen by the wayside, the poor and the meek, acquire hypertension as well. Most have lived what may be called a 'hard' life. The struggle for existence has begun early, and their life represents little else than a desperate battle.

"They have no time for play. Here again the child that is in all of us goes early, and once gone rarely heeds the summons to return.

"There is no question in my mind that such constitutions are acquired and not inborn. One sees occasionally hypertensive disease and its consequences common in certain families. This is the result of the imitative tendency of children."

The above graphic picture, though highly coloured, delineates a characteristic sub-group of hyperpiesis. Two of this author's points, however, are open to question. The first is that which relegates such type of constitution entirely to acquired characteristics, and ignores the effects of heredity. The second is that which assigns to a certain fixity of mental processes a corresponding bodily form. In this country at any rate one sees hyperpiesis of psychical (or acquired) origin in the tall as well as in the short; in the nervous, thin and emaciated as well as in the obese. Nevertheless, some cases, including "captains of industry," do fall within the limits described by Moschcowitz, but I have found the well-rounded contours of such men commonly to

be due to physical causes such as " business lunches," public dinners, big cigars, luxurious motor cars and sedentary occupations rather than solely to a tense mental attitude towards life. Certain it is that in the United States the busy public man lives continuously at extreme high pressure, as I have witnessed, and is quite unable to relax even when playing games. He is out to win every time, and concentrates every faculty on so doing. May we venture to think that our more sport-loving countrymen are wiser in their generation in that they appreciate more fully the advantages of leisure, and are not for ever sitting upon the safety-valve ?

Further research is required as to the biochemical and electrical changes which nerve cells undergo during activity. As to these, we know that wear and tear of nerve cells causes disintegration of the complex protein and albuminoid substances of which they are composed, and we are justified in drawing an analogy between these and the pressor effects due to the breaking down of protein bodies by bacteria. Sudden shocks, mental emotion and excitement are all potent in raising the arterial pressure for the time being to extraordinary heights, whilst continued worry and anxiety conditions have a strong action in promoting vasoconstriction over long periods of time, tending ultimately to permanent thickening of the vessel walls.

Of the somatic neuroses, in general terms we may say that in true anxiety neurosis the blood pressure is often raised, whilst in neurasthenia it is usually low. Pseudo-neurasthenia, nevertheless, presents the symptom complex of neurasthenia, but these symptoms actually originate from hyperpiesis, with or without arteriosclerosis, gastric disturbance or chronic nephritis, and the heightened blood pressure will put us on the right track. I have seen in consultation patients over sixty in whom the presence of cancer was suspected by the local practitioner by reason of the gradual pallor and loss of weight, these symptoms being in reality due to arteriosclerosis.

9. **Endocrine-sympathetic Influences.**—The importance of the sympathetic endocrine group is becoming increasingly recognised. " As both endocrine glands and the sympathetic nervous system became specialised they remained associated.

This association is reciprocal, as not only does the sympathetic nervous system stimulate the secretion of these ductless glands, but their secretion increases in turn the sympathetic response. Thus the sympathetic nervous system, the endocrine glands, and the gonads form a basic tripod, whose relationship is shown in disease as well as in health, and is reflected in many of the neuroses and psychoses." [60]

This brings us to the question as to whether hyperpiesis in its early course is a disturbance of function which leads to changes in structure, or whether it is preceded and produced by changes in structure.

If one desires to study the beginnings of hyperpiesis, it is necessary to do so before the onset of complications. The solution of this problem has been sought through investigations of apparently healthy schoolchildren and adolescents.[136] Results of such study go to prove that hyperpiesis begins as a transient functional phenomenon in subjects with an over-responsive vasomotor system when exposed to physical or psychical stress. " Commonly the blood pressure at rest is above normal, but not always. It might be even a family trait. Usually the activating cause is apprehension, and anxiety for success, the link between the higher centres and the vasomotor centre being too intimate. . . . What is in store depends on circumstances ; whether the temperament is placid, or eager and over-anxious ; and on the nature of the employment. Arterial pressure might remain within the normal until the responsibilities of maturity and the metabolic imperfections of middle life reinforced the tendency." [149] In later life it may be a reaction to the hurry and strain of modern civilisation in the form of a heightened excitability of the vasomotor centre. The chief ductless glands implicated are the pituitary and adrenals (which increase the sensitiveness of response to the sympathetic system under the influence of emotion or of infection), and to a less extent the thyroid gland and gonads.

Cases of heightened arterial pressure associated with hyperthyroidism, adrenal and pituitary over-activity, usually with some evidence of hypertrophy are not uncommon, and, when recognised, adequately explain the rise in pressure and suggest appropriate treatment.

10. **Allergy.**—Bishop [150] has advanced a simple yet wide conception of a general cellular disorder from which few cells of the body are exempt. The heart, blood vessels, liver and kidneys suffer conspicuously because of their importance and activity, and because of the extra strain to which they are subjected by cellular damage. " Cardio-arteriosclerosis " would thus include hardening of the heart and arteries, progressive cardiac failure, Bright's disease and presenility. It is suggested that a reactive irritation is set up against some material with which the cells have to deal in the processes of cellular nutrition, and that this irritation, which Bishop regards as allergic in nature, leads to cell injury with ultimate destruction and replacement by fibrous tissue. Manifestations of this take place in various organs, *e.g.*, in the eye, causing neuroretinitis, in the kidney, causing nephrosclerosis, etc.

The extraneous material is believed to be a protein or protein derivative from ordinary food, or from the bodies of bacteria to which the cells of the patient have become sensitive. Such sensitiveness dates usually from some acute illness of the nature of gall-stones, appendicitis, typhoid, malaria, etc., or from shock or great mental perturbation, or again from acute food poisoning. Strickland Goodall,[151] who examined 2,000 cases under the age of forty, found that the most frequent antecedent of hyperpiesis was scarlet fever. These findings favour the infective origin of certain forms of hyperpiesis and support the view that hyperplastic sclerosis of the intima is the underlying change.

Allergy has been invoked also by Waldbott as a cause of hyperpiesia, but Cohen, Fineberg and Rudolph are in opposition to these conclusions.

Hyperpiesis is not necessarily a Symptom of Cardio-arterio-renal Changes

It used to be thought that persistently increased arterial pressure was in every case symptomatic of vascular, cardiac or renal changes, each bearing its own individual label, as, for example, thickening of arterial walls, cardiac hypertrophy

and impaired renal permeability, these manifestations appearing jointly or severally with individual variations in kind or degree.

One fact is certain, namely, that raised arterial pressure, transient, intermittent or even permanent, sometimes also of high grade, frequently manifests itself in the absence of any evidence of kidney disease.

"That patients with essential hypertension may later in life develop cardiorenal complications affords no justification for the conclusion that the primary cause of the high blood pressure lies in the organ or organs which secondarily show evidence of disease. Study of histories of patients and careful continuous clinical observation will reveal many patients with hypertension of fairly high grade and yet with no evidence of myocardial degeneration, arterial change or disturbance in kidney function."[152]

Cardiac decompensation with marked passive hyperæmia of the kidneys is associated with a moderate retention of non-protein nitrogen in the blood, and the height of the blood pressure bears no relationship to the amount of the non-protein nitrogen substances in the blood. High arterial pressure with normal non-protein nitrogen values in the blood and normal kidney excretion, determined by functional tests and urine examinations, does not justify the clinical diagnosis of chronic nephritis.

In certain cases the rise in pressure is of the nature of a compensatory and protective mechanism induced by factors which have for their object the maintenance of an efficient circulation through the various organs and tissues.

Association of Hyperpiesis with Renal and Myocardial Involvement

Wallgren[153] examined kidneys from forty-four patients with high blood pressure, comparing them with fifty-one controls with normal kidneys. Eleven out of the forty-four cases (hyperpietic) exhibited blood vessels conforming with those in the normal age group. In the remaining thirty-four cases (hypertonic) typical nephrosclerosis was found, the condition of the blood vessels differing quantitatively, but

not qualitatively, from the changes of normal development and ageing.

Clinical and *post-mortem* diagnoses of nephritis do not always tally, and the presence and degree of high pressure bear no absolute relation to the amount of kidney involvement. In fact, one or the other may be entirely absent.[154, 155] Although there are still certain observers, including Lian and Haguenau,[156] who affirm on clinical grounds that acute or chronic nephritis is, in certain cases, the exciting cause of a transitory or permanent rise in arterial pressure, nevertheless cases are recorded by Moschcowitz[148] in which raised arterial pressure and clinical evidences of nephritis were present, although the kidneys showed but slight lesions. His experimental and clinical observations tend strongly to prove that even in cases of definite nephritis high arterial pressure may be the earliest demonstrable symptom, and lend no support to the belief that, when associated with nephritis, it is of renal origin. Rather does he incline to the idea that arteriocapillary fibrosis is merely the localised and prominent manifestation of a generalised vascular affection, which explains the frequency of clinical phenomena referable to other organs, *e.g.*, brain, aorta, heart, pancreas, arteries, etc. On this view arteriocapillary fibrosis (diffuse hyperplastic sclerosis) and arterial disease are contemporaneous reactions to the same hurt. In other words, a hypertonia produces a secondary hyperpiesis.

The end results of a glomerular nephritis with secondary contracted kidney and of a primary contracted kidney are morphologically the same. The causes must be identical. In the former there is a blood pressure which exceeds the normal ; in the latter vascular lesions occur even at times with normal pressures.

"Diffuse hyperplastic sclerosis shares with chronic nephritis and chronic interstitial nephritis a common ætiology, which, so far as is known at present, is due to the action of bacterial toxins, and both the character of the vascular lesion in diffuse hyperplastic sclerosis and the distribution of the lesion in the vascular tree are compatible with the view that it also is caused by a circulating toxin." [88]

Such toxins, however, probably do not act unless an error

of metabolism is present as the fundamental basis producing a condition of hypertonia which may induce a secondary hyperpiesis.

It is in accordance with numerous observations that cases of renal affection are met with in which hyperpiesis, either transitory or permanent, coincides with the bacterial infection or with the intoxication which induces a process of acute or chronic nephritis.

Hence we should rightly regard the kidney lesions in nephrosclerosis as due to injury of the vessels, which arises from the cause or causes which simultaneously produce the high pressure. Moreover, the effects upon the kidney will vary according as to whether arterioles or capillaries are the more directly concerned.

Like causative factors are operative in the production of myocarditis, and in all three conditions—raised blood pressure, nephritis and myocarditis—disturbance in the arterioles constitutes an integral part of the reaction to injury. Rise in blood pressure and œdema are thus earlier indications of kidney affection than protein, casts and erythrocytes in the urine. So, too, certain causal factors give rise to the appearance of œdema from disorganised capillary mechanism akin to its manifestation in stasis from cardiac decompensation.

CHAPTER IX

Symptomatology of High Arterial Pressure

"Supertension has no rigid limits or defined boundaries, and passes gradually, perhaps silently, into states of disease, as manifested, for example, by damaged arteries or defeated hearts."

Lord Dawson of Penn: *Proc. Roy. Soc. Med.*, June, 1926.

A. Symptoms of High Arterial Pressure

In its earlier stages, and over a period of time varying with the individual, apart from transient bleeding, usually from the nose, simple and uncomplicated high arterial pressure has no symptoms. Indeed, the subjects are often conscious of no disability, and feel particularly hale and hearty. Many of them up to middle life exhibit a high degree of mental and physical activity, the condition being discovered only on medical examination for life assurance or for some minor symptoms for which they seek advice. Their metabolic processes are usually in excess, but of this they are blissfully unconscious.

From this pre-sclerotic phase they gradually pass into a state in which vague symptoms and signs afford an often-disregarded warning.

The symptoms of hyperpiesis may be any one or more of the following, most of which are due to disorder of the circulatory mechanism in which the central nervous system is involved, certain of them constituting evidences of congestion, cerebral or otherwise, the result of overfilled blood vessels.

The earliest and most frequent subjective disorder of sensation of which the patient complains is frontal or vertico-occipital *headache*, commonly dull, and aggravated by attempts at mental concentration, less often acute and throbbing. Occasionally headache is associated with *dizziness* on rapid change of position, as in rising after stooping or getting up suddenly from the recumbent posture,

True vertigo is rare. Sensations of *fullness or heaviness in the head* may be experienced. A symptom often more obvious to relatives and friends than to the patient is a *change in temperament.* From being calm and equable, evidences of lack of control become apparent in the shape of *irritability* on slight or no provocation, with sudden and ungovernable fits of anger. *Drowsiness and disinclination* for effort cause social, business and domestic duties to become irksome and neglected. Nervous symptoms, with or without *emotional outbreaks,* may occur, particularly in women, in whom they tend to become more pronounced at or after the menopause. *Memory is increasingly impaired,* along with *incapacity for sustained mental or physical effort,* eventually leading to a state of complete *exhaustion.* Anxiety neurosis with vague apprehensions of impending disaster, *neuralgia, migraine* and *mild psychic exaltation with confusion of ideas* may also supervene. With high pressures arterial pulsation is often perceptible as a throbbing sensation when recumbent, and sometimes prevents sleep.

In established cases the symptoms attributable to hyperpiesis proper begin to be interwoven with those of the precedent, induced or associated morbid conditions. Thus the sallow complexion and loss of weight of certain advanced arteriosclerotic subjects in conjunction with nervous depression, exhaustion and symptoms referred to the gastro-intestinal tract may cause the condition to be mistaken for neurasthenia unless the arterial pressure be taken. In over 50 per cent. of established cases of hyperpiesis, palpitation, flushing, precordial pain and dyspnœa are apt to occur in the absence of primary myocardial or valvular disease, generalised arteriosclerosis, renal disease or hyperthyroidism. Retinal hæmorrhages may be present in about 5 per cent. of all cases. A rude awakening may happen from one of the accidents consequent upon the increasing pressure-load, and with fully developed sclerosis of heart, arteries and kidneys still more dramatic issues impend and may come to pass, often with fatal results.

The high-pressure individual is often the subject of excess metabolism and labours under the strain of acid retention, being unable adequately to eliminate waste products. The

acid-base balance of the tissues is deviated to the acid side, and tissue excess formation, particularly of fat, preponderates over destruction and elimination of waste products.

B. Signs of High Arterial Pressure

The single sign of uncomplicated hyperpiesis is the height of the arterial pressure recorded by the sphygmomanometer.

Effects of Persistently Raised Arterial Pressure on the Heart and Arteries

Protracted high arterial pressure causes the heart to enlarge. If, therefore, in a case of high arterial pressure the heart is found to be of normal size, one may conclude that the heightened pressure has not been of long duration. A persistently raised pressure is bound to lead to general cardio-vascular hypertrophy, with sclerotic changes, especially in the middle coats of muscular arteries and in the left ventricle of the heart. So long as the left ventricle successfully overcomes the resistance by adequate systole, progressive hypertrophy only occurs. Directly it fails to do so, and an excess of residual blood remains after systole, dilatation gradually ensues with degeneration of the muscular tissue. Whether the arteries dilate or not under a persistently raised arterial pressure depends upon their tone. The radial arteries, for example, may have sufficient contractile power to resist heightened pressures for years without becoming dilated and tortuous. Similarly with the aorta, though the brachial arteries tend to become involved at an earlier date. Contracted muscular tissue, while still unimpaired, is elastic and resilient, arterial muscle thus permitting the arteries to yield and to recoil.

C. Effects of High Arterial Pressure

1. *On the Heart.*—Even over long periods it is astonishing to find in many cases little induced change in the heart, this organ being influenced solely by increased load (hyperacthia). Palpitation and tachycardia may be due to forcible cardiac action of nervous origin, or to extrasystoles. In the majority

of established cases, however, the heart undergoes hypertrophy as a result of its overload, manifested clinically by augmented heart rate and lessened response to effort. The slowly progressive left ventricular preponderance can be confirmed by percussion, X-rays and the electrocardiograph.

Subsequently dilatation supervenes, with blowing systolic murmurs, mitral and sometimes aortic, the latter constituting "the cardio-aortic type of hyperpietic heart" described by Vaquez. Myocardial degeneration leads to cardiac failure, of which increasing breathlessness on exertion is a frequent symptom. More advanced cases may suffer from distressing nocturnal attacks of dyspnœa, the so-called "cardiac asthma," with or without Cheyne-Stokes breathing.

The rule is for myocardial failure, whether or not the result of cardiac defeat, to be accompanied by distinctly low pressures, but, every now and again, one comes across cases of arterial pressure which have overtopped the 200 mm. mark, associated with a failing heart and feeble pulse. Such cases present many points of interest. "The first explanation that occurs is that the heart is failing behind the high pressure, but if so, lowering the pressure should relieve the heart, which, as a matter of fact, it fails to do. The next explanation offered was that as the output of the heart diminishes, vasoconstriction must occur to diminish the size of the bed to be filled, and thus to spare the heart. This would account for pressure being maintained at its normal level, but not for its rising as the heart fails. But we now know that the strongest stimulus to a muscular contraction is the previous stretching.[199] Hence the marked hypertrophy of the left ventricle in aortic regurgitation, where the ventricle is filled during diastole, both from the auricle and from the aorta, and so is stimulated to increased work. In the same way we may regard the vasoconstriction produced through the sympathetic as an attempt, by raising blood pressure, to stimulate the flagging heart, for the diastolic stretching will be increased thereby. But when the myocardium is diseased it fails to respond to a remedy which is at best a desperate one—the overstretched muscle fails to respond and dilatation increases." [60]

2. *On the Arteries and Arterioles.*—These vessels may be

unable to withstand the strain of high pressures, the aorta, as the main artery exposed to the propulsive shock of the heart, undergoing atheroma as a sequel. When a more general degeneration exceeds repair and the tiny vessels succumb, the peripheral arterioles share in this dual process, and cerebral hæmorrhage or thrombosis may ensue. Generalised arterial disease, involving the coronary vessels, often leads to substernal oppression or anginal pain, at first on strenuous effort, but later on effort that is only slight.

3. *On the Kidneys.*—Frequently there are no indications of renal involvement. Hyperpiesia may persist for many years, even till death, without a trace of albumen in the urine or evidences of renal inadequacy. Epistaxis, hæmaturia or hæmophysis may occur in established cases. No primary and direct effect is produced as a rule upon the kidney tissue proper. Exceptional changes appear which are difficult clinically to differentiate from chronic nephritis. The usual effect is a secondary one upon the renal vessels—the arteriolo-sclerotic kidney—leading to ischæmic fibrosis with slight albuminuria and a few casts.

4. *On the Brain.*—Recent careful investigations have established that the blood vessels of the pia mater, and probably those within the brain, are effectively controlled by the vasomotor nervous system, this regulator action being attributed to the chemical composition of the blood.

Hyperpietic cerebral attacks, formerly thought to be induced by uræmia, have been discovered to be due not to nitrogen retention but to sudden or rapid increments of arterial pressure causing derangements of the cerebral circulation in the shape of headaches, convulsions or coma. The seizures are of two kinds :—

1. *With Signs of Cerebral Œdema.*—This form is commonest in patients befow the age of forty years. Severe headache is a constant symptom, often with concomitant signs in fundi and urine indicating a generalised circulatory disturbance.

2. *Without Signs of Cerebral Œdema.*—This form is commonest in middle-aged subjects. Convulsions and coma are the usual symptoms—a hyperpietic epilepsy—mainly induced by angiospasm.

CHAPTER X

Diagnosis and Prognosis of High Arterial Pressure

"If I were allowed to have only two instruments of precision for my aid in physical diagnosis, they would be the stethoscope and the blood pressure machine."—Cabot: *Lectures.*

A. DIAGNOSIS OF HIGH ARTERIAL PRESSURE

Clinical Examination of the Patient.—For accurate diagnosis of high pressure conditions, examination of the patient cannot be too careful. I recommend the following order as being the most convenient :—

1. History.—This must be taken carefully, special attention being directed to :—

(*a*) Family history of gout, rheumatism, syphilis or alcohol.

(*b*) Personal history of antecedent infections, especially nephritis and syphilis ; occupations involving contact with lead or involving physical and mental strain ; emotional shocks ; long-continued worry ; too little sleep ; wrong habits, particularly as regards over-eating, errors in diet, hurried meals, abuse of alcohol or tobacco ; habitual constipation ; chronic sepsis, focal or general.

2. Systematic Examination of the Cardiovascular System.—(*a*) Take several readings from both brachial arteries and discard the first. Record the *pulse rate and characteristics.*

(*b*) Note *position of apex beat, nature of cardiac sounds* at apex and both bases, *always with the chest bared, and state of superficial arteries.*

3. General Condition of Patient.—Inspect for presence or absence of œdema. It should be remembered that hydrostatic dropsy is of frequent occurrence in patients over middle age, of sedentary habit and heavy build, and does not imply cardiac failure unless attended by increased general venous pressure. Such rise can readily be determined by gradually

raising the patient's head from the recumbent position, when the vertical distance above the sternum at which the external jugular vein is still seen to be distended marks the degree of venous engorgement.

4. Ophthalmic Examination.—Investigation of the fundus with the ophthalmoscope should never be omitted, since it often furnishes the earliest diagnosis of arteriosclerotic manifestations in the body. The patient should be referred to an ophthalmic surgeon for expert opinion if the practitioner does not feel certain as to the state of the retina. He can, however, always adopt the easier course of examining the conjunctival vessels with the aid of a lens. The vessels appear bluish white, and in high pressure states look like cords. Pathological vessels are readily distinguishable from physiological, for changes appear early, and striking manifestations may occur in the shape of areas of obliteration and small saccular aneurysms.[157]

Since the conjunctival vessels are an index of the cerebral circulation, this simple diagnostic method at times yields information of considerable value.

B. BACTERIOLOGICAL AND HISTOLOGICAL INVESTIGATIONS

Systemic bacterial infection is often a factor in the causation of a raised blood pressure. For the detection of this a thorough bacteriological examination of the *patient*—not of isolated specimens—should be made, combined with histological tests on the blood. In regard to the former, it is essential that laboratory tests should be made for pathogenicity of the organisms isolated. The statement, for instance, that streptococci are present is valueless.

In a histological examination, the differential count accompanied by estimation of the Cooke index—a modification of the Arneth count—affords valuable information. This consists in counting the number of lobes of 500 polynuclear cells and classifying them into one, two, three, four, five or more, and comparing the grouping with that of normal blood. When systemic infection is present, the picture shows a "shift to the left," so that the number of nuclei in

the polynuclear cells is on the whole smaller than normal. The test should be performed by a trained histologist.

C. BIOCHEMICAL INVESTIGATIONS

Tests of Renal Efficiency.—Where disturbance of nitrogenous metabolism is suspected, recourse should be had to biochemical tests, which are divided into those performed on the urine and blood respectively.

By the former we learn what the patient is excreting ; the latter tell us what he is retaining.

As regards clinical utility, the latter factor is of much greater importance than the former.

(a) **Urinary Tests.**—In regard to urinary tests, it must be emphasised that simple estimation of the amount of urea present in the urine, even in a twenty-four hours' specimen, is of little value ; in a " casual " specimen, it is not only useless but misleading.

Many elaborations of urinary methods, such as the urea concentration test and its derivatives (Maclean) [158] and the water elimination tests (Calvert),[159] have been employed, but are all open to the practical objection that in any given case they may mislead, for it is now recognised that at times a person with disorganised kidneys may be found to concentrate urea within normal limits, while, on the other hand, persons in whom no clinical kidney defect is apparent fail to concentrate urea sufficiently.

(b) **Blood Tests.**—" Retention " tests on the blood are now regarded as essential, and should be performed whenever the functioning power of the kidneys is in doubt, as there is no purely clinical method of determining this most important factor.

(α) *The Blood Urea Test.*—The test is performed as follows : not less than 3 c.c. of intravenous blood are withdrawn into a tube coated internally with sodium fluoride. This is treated with urease (a ferment derived from the Soya bean) in the presence of a buffered phosphate solution, which maintains a suitable *p*H in the mixture. The ammonia is then liberated by strong alkali, and is either distilled or aspirated by a current of air into standard acid solution and finally estimated either by

titration against standard alkali or colorimetrically after the addition of Nessler's reagent.

A blood urea content of 40 mgms. per 100 c.c. or under is regarded as normal. In the interpretation of figures above this, it should be remembered that in acute intestinal obstruction high readings may be obtained which are not necessarily indicative of a failing kidney. With this exception, a reading above 50 mgms. should be regarded as conclusive of some degree of nitrogen retention and as a factor in the causation or maintenance of a high blood pressure.

(β) *Blood Calcium and Blood Phosphorus Estimations.*—In any case of hyperpiesis it is always of help to estimate the blood calcium and blood phosphorus content. In regard to calcium, what is desirable to know is the degree of calcium retention ; the degree of calcium excretion is relatively unimportant. In a number of cases of hyperpiesia the blood calcium figure is found to be low. On the other hand, in many cases of hyperpiesis with which arteriosclerosis is associated the blood calcium figure is found to be high (*vide* p. 123).

D. PROGNOSIS OF HIGH ARTERIAL PRESSURE

On accurate diagnosis correct prognosis and treatment alike depend.

In any consideration of prognosis in high arterial pressure, one must take into account the state of the heart, of the arteries and of the kidneys.

The first step is to exclude the presence of syphilis. The next step is to differentiate hyperpiesia from hyperpiesis and hypertonia due to chronic renal disease. In hyperpiesia the prognosis is better than in chronic renal disease, more especially when coming under observation in its earlier stages. When associated with obesity the prognosis in the majority of cases is good.

Determine by repeated tests whether a rise in pressure is temporary or permanent. The systolic transitory type, due to emotion or excitement, is commoner in women than in men, and may be diagnosed by the appearance of the patient and the absence of cardiac hypertrophy, arterio-

sclerosis, proteinuria, retinal changes and other manifestations which accompany the fully developed condition. Part of the rise may be due to arterial spasm. If uncertainty exists, rest for a few days in bed clears up the diagnosis by reducing the unstable nervous pressures to within normal limits.

Favourable Features.—The outlook is best where removal of severe mental stress brings about a cure, or where the condition is revealed fortuitously, as during examination for life assurance, and the patient is leading a full life without symptoms, effort-tolerance being good. With appearance and increase of circulatory or renal defects, the prognosis correspondingly becomes worse. Maintenance of a quiet mode of life, with abandonment of strenuous efforts, frequently brings about prolonged improvement. For this reason the prognosis is better in elderly women than in men of equivalent age.

Unfavourable features are increased pressure at comparatively early ages—the younger the patient, the worse the outlook; family history of vascular disease; a relatively high diastolic pressure; evidences of systemic degeneration in arteries, heart, kidneys and digestive tract; limited effort-tolerance.[160]

Very high pressures are always unfavourable, but pressures of moderate grade have to be considered on their individual merits, and may be requisite for maintenance of the visceral pulse. On the whole, prognosis is far less grave than formerly thought.

Finally, since prognosis largely depends upon whether high arterial pressure is associated or not with clinical evidences of chronic nephritis, before expressing any opinion it is wise to watch the effect of treatment, and to try daily catharsis with sodium and magnesium sulphates. Failure to react to treatment of this kind is practically pathognomonic of the incidence of renal disease.[160]

"In many persons, even in the quite elderly, phases of high pressure occur which prove to be transient; though probably recurrent. Much will depend on the biochemical reports on the blood and the urine, and on the course of diastolic pressures; diastolic pressures round about 100 are

of ill-omen. Response to treatment, on the other hand, is a favourable sign. The peril lies in the bigger incidents ; in cardiac defect, or apoplexy, or pulmonary œdema. If, however, the high pressures prove to have a renal origin, the prognosis is more sinister, both as to suffering and duration of life. Any incidental complications will have to be taken into our reckoning, and these are far more perilous in the renal cases.

"Non-renal hyperpietics bear surgical operation fairly well ; the renal very ill." [161]

The height of the arterial pressure reading is important, though an isolated reading has little more significance than an isolated record of temperature.

The actual pressure-reading on the sphygmomanometer is relatively of little account. What is important is the co-existence of symptoms and physical signs. A subject with a moderately raised arterial pressure may be dangerously ill, while another with a pressure which is excessively high may, nevertheless, have no symptoms and be able to do full work over a period of years.

Thus simple high pressure without unfavourable symptoms, even if it resists attempts at reduction, does not necessarily warrant a bad prognosis. If, however, the pressure rises rapidly or undergoes sudden elevations, the import is less favourable than when the pressure has mounted insensibly and keeps steadily at a fixed maximum. Even if the rise is permanent and associated with organic changes, life may possibly be continued for many years despite absence of treatment.

From time to time patients are seen whose pressure is continuously elevated apart from detectable cardiac, arterial or renal disease. Here the difficulty lies in excluding sclerosis of the cerebral, coronary or splanchnic vessels, since vascular changes may be very localised. Hence the value of expert ophthalmoscopic examination, which may reveal alterations in the fundus which are significant of arteriosclerosis.

Arterial pressure should not be considered as pathologically high unless the systolic pressure is *persistently* above 155 mm. Hg, and the diastolic *persistently* above

100 mm. Hg, but it should always be remembered that, as the result of increased peripheral resistance dependent upon tissue changes, arterial pressure rises more rapidly between sixty and eighty years of age. A moderate elevation of arterial pressure in a middle-aged patient may, *per se*, be disregarded.

TABLE IX

Pathologically High Arterial Pressures

	Systolic.	Diastolic.
Excessively high . .	280—320	165—180
Very high . . .	240—275	140—160
High	155—230	120—130
Suspiciously high if occurring before age 40 . .	145—150	90—110

Range of High Arterial Pressures

Dr. Maurice Campbell [162] has pointed out that there is a regular gradation in the relationship between increasingly high systolic and diastolic pressures. Starting from 160/100

TABLE X

Relationship between High Systolic and Diastolic Pressures

Average Standard.	
Systolic.	Diastolic.
160	100
180	110
200	120
220	130
240	140

for every 10 mm. rise in the diastolic, there is an increase of 20 mm. in the systolic.

Basal Metabolism in Hyperpietic Cases

In patients with hyperpiesia, whose renal function remains normal, the basal metabolic rate taken with Benedict's apparatus is usually normal, or occasionally shows a slight increase, the average being — 6. In most cases of hyperpiesis, however, in which renal function is damaged (primary and secondary contracted kidney), with systolic pressures ranging from 180 to 270 mm. Hg. and definite cardiac hypertrophy and dilatation, the basal metabolic rate is increased, the average being + 18.[163]

Importance of the Heart and Systolic Pressure in Prognosis

In prognosis the state of the heart is an important factor, although in many instances far too much influence is assigned to the heart and far too little to the arteries. The systolic pressure is an index of the maximum cardiac energy, though this may be profoundly modified by the pulse rate. In cases of permanently high pressure the onset of signs of commencing heart failure is of the gravest import. Such signs are dyspnœa, cyanosis, occasional or grouped premature contractions, gallop rhythm, total arrhythmia and continuously accelerated pulse rate. Any combination of these latter signs is indicative of cardiac defeat. "What usually happens is not simply the wearing out of the heart by excessive work, but the defeat of a labouring heart impaired by defective nutrition due to lesions of the coronary circulation, and deficient quality of blood resulting from damaged kidneys and other organs."[161] Evidences of the circulation becoming inefficient are afforded by notable deviation from the more normal 3 : 2 : 1 ratio between systolic, diastolic and pulse pressures.

Arterial pressures may remain high during many years without inducing cerebral hæmorrhage or anginal attacks, and even should these latter be severe or brought on by

exertion, the prognosis is far less grave than was formerly believed.[121]

The influence of heredity is important both in arteriosclerosis and in angina pectoris. In angina, if the pressure is high, the prognosis is better than if low, for in the latter event death often occurs unexpectedly. A rise in differential pressure, however produced, is always of much moment. Warfield[18] states that it is invariably accompanied by increase in size of the left ventricle, dilatation of the aortic arch and increase in the size of all the distributing arteries. The best examples of this syndrome are found in aortic regurgitation.

Importance of the Mean Pressure in Prognosis and Treatment

The mean pressure is a useful guide to prognosis and treatment.[58] A rise above previous levels solely of the mean pressure indicates the onset of high arterial pressure. In established cases, irrespectively of the height of the systolic pressure, the height of the mean pressure affords an indication of the gravity of the affection. So also, where symptoms are present apart from any material rise in systolic pressure, the existence of arterial supertension is suggested by the level of the mean pressure which is always above 120 mm. Hg., and prognosis is correspondingly less favourable. Increase of the mean pressure runs concurrently with increase of left venticular or total cardiac insufficiency. Latent insufficiency may be revealed by the amount and duration of rise of mean pressure consequent upon effort. Under normal conditions the mean pressure is not increased by effort.

Further, the mean pressure is useful as a guide to treatment. Cardiac tonics and rest lessen the mean pressure coincidently with amelioration in the symptoms and signs of cardiac insufficiency. When a drop has occurred, or the level remains stationary at a figure lower than it was before the institution of therapy, this may be discontinued. Failure to reduce the mean pressure indicates a bad outlook.

Importance of the Diastolic Pressure in Diagnosis and Prognosis

The cardinal point to which again I desire to draw special attention is the prime importance of the minimal pressure, which is more instructive as to actual rise in arterial pressure than the height of the systolic, since the minimal pressure is far less liable to temporary fluctuations. It is a more definite indicator of (*a*) the peripheral resistance, (*b*) the amount of continuous strain to which the arteries are subjected, and (*c*) the eliminative capacity of the body. Accurate records are of the greatest value in hypertonic, toxæmic and arteriosclerotic states. If the diastolic pressure is maintained at 135 mm. or higher, the outlook is grave. Lian, Broca and Clement[164] state that of seventy-seven persons with diastolic pressure above 135 mm. Hg observed from 1st August, 1914, to 1st September, 1919, only two were known to be then living, and of thirty-four seen before 1919 twenty-six are known to have died.

To register systolic pressures alone is inadequate and misleading. If the interval between the top of the systolic crest of the pressure wave and the bottom of the diastolic notch were always the same, it would not matter which reading we took. As it is, however, merely to state this assumption is sufficient to show its falsity. We know that the amplitude of the pulse wave varies enormously in different individuals, and indeed in the same individual at different times. In other words, the relation between the systolic maximum and the diastolic minimum varies.[165]

The two following examples will serve to illustrate my meaning: A patient of mine suffering from aortic regurgitation gave as his systolic pressure 210 mm. Hg. Another—a case of granular kidney—gave a systolic pressure of 180. Had I regarded the systolic pressures alone, I should have said that the aortic case had the higher blood pressure, and that his arteries were in a condition of greater strain than those of the man with granular kidney. But records of the diastolic pressure put an entirely different construction on the matter. That of the aortic case was 70 mm. Hg, whilst that of the renal case was 140—that is to say, the renal case

had his arteries constantly kept on the stretch by a minimal pressure of 140, which during systole rose to 180; whilst in the aortic case the diastolic pressure was only half this amount, and it was solely during the brief interval of time represented by the upper part of the sharp systolic crest that the pressure reached a notable elevation. During diastole the arteries were far less stretched than normal. Hence any argument based on systolic readings alone must be received with the very greatest caution.

A high systolic pressure with high diastolic puts out of court aortic regurgitation. If the diastolic rise is proportionately less than the systolic, this shows that a hypertrophied left ventricle is carrying on efficiently. In hyperpiesis a diastolic fall is often of good omen, whilst a rise is of bad import, but this is not invariable.

Hyperpiesis in Various Pathological States

1. Arteriosclerosis.—When heightened arterial pressure and arteriosclerosis are combined, the level of the pressures is no guide to the extent or degree of arteriosclerosis present. Not infrequently, indeed, the highest pressures occur in the absence of detectable arteriosclerosis, which may, however, be a later accompaniment.

Conversely, both clinically and pathologically, extreme forms of *arteriosclerosis may be present with pressures which* not only *are not raised*, but even may be well below the standard level, when there is always the possibility of the onset of thrombosis.

Again, when arteriosclerosis happens in conjunction with raised pressures, prognosis is correspondingly worse owing to the risk of cerebral hæmorrhage, which, nevertheless, may be warded off by regular daily purgation with salines.

The cardinal signs of advanced arteriosclerosis are (*a*) high blood pressure, (*b*) cardiac hypertrophy, (*c*) palpable arteries, and (*d*) accentuated and ringing second sound at the aortic cartilage. At times this ringing sound is best heard over the carotid artery.

2.Chronic Heart Muscle Insufficiency (The High Pressure Heart).—This is often incorrectly termed "chronic myo-

carditis," but in reality is "an overgrown, over-laboured, outfought myocardium, presenting no inflammatory elements, unless it be an exceedingly slow quasi-reparative fibrosis." [161]

A low systolic pressure with a high diastolic frequently suggests cardiac inadequacy induced by the high pressure or myocardial weakness in association with severe pressor factors, such as nephritis or vascular spasm. A continuously high diastolic pressure increases the load of the heart, which has to hypertrophy in order to cope with the increased peripheral resistance, and eventually to dilatation.

A rapid fall of systolic pressure while the diastolic remains high is alarming. In cardiac failure with a moderate or low systolic pressure, a wrong impression may be derived from the sphygmomanometer reading. In many such cases the pressure was originally high, the decline being due to the incidence of heart failure.

A low diastolic and systolic may be the expression of simple low pressure and of diminished reserve force, but not necessarily of a feeble circulation. On the other hand, it may spell cardiac dilatation with lessened peripheral resistance and general lowering of vitality, in which case the differentiate pressure is also decreased. A rapid descent of both systolic and diastolic pressures is a feature of coronary thrombosis. Here again a single pressure-estimation may be misleading. In a patient who has previously manifested systolic pressures of about 220 mm., the pressure may be as high as 140 mm., even after the formation of a coronary clot.

Permanent hyperpiesis is a frequent cause of chronic degeneration of the heart muscle, which is probably due to and is usually accompanied by a high blood uric acid, with infrequent retention of urea in the blood, and therefore no tendency to uræmia. Such cases of primary uricacidæmia, originating from some fundamental alteration in metabolism, suffer notably from the direct consequences of circulatory strain, and can only be controlled by the correction of this metabolic error.

The pathological changes presented by such a heart are slight, histologically speaking, where fatigue is the main element. With the additional advent of chronic or acute

infections or of toxic causes other than these, exudates and hæmorrhages may be seen between the muscle fibres, which themselves are cloudy and studded with minute fat particles. With further failure of an efficient coronary circulation fibrosis is apt to result.

Myocardial involvement is denoted clinically by wide variation in the readings of high pressure cases at various times, together with a greatly reduced differential pressure. My experience coincides closely with that of Lian, Broca and Clement,[164] who state that every hyperpietic seen by them with appreciably diminished amplitude of differential pressure and pulse wave has developed fatal cardio-renal disease, and that no treatment has had any effect on this arterial over-distension or sclerosis.

3. Acute Infections.—In most of the acute infections the systolic pressure is lowered more than the diastolic. In scarlet fever the depression is much less than in diphtheria, influenza and enteric fever. In pneumonia the blood pressure tends to rise in cases which terminate fatally. "In syphilis the blood pressure is not necessarily raised. Secondary syphilitic changes in the endocrine glands and the widespread lesions of congenital syphilis, of which granular kidney may be one, may lead to a high blood pressure. It appears that though recent syphilitic infection does not raise the blood pressure, its protean effects may do so. When this occurs the special arterial lesions will increase the liability to cerebral hæmorrhage." [87]

In certain of the acute infections, notably in scarlet fever and tonsillitis, less commonly in enteric fever, small-pox, diphtheria and measles, also in syphilis and acute tuberculosis, a definite rise in both systolic and diastolic arterial pressures may precede by a few days the appearance of blood chromogens, protein and casts in the urine. By keeping daily charts it may thus be possible to predict and, more important still, to ward off, an attack of acute nephritis.

4. Acute Nephritis.—In acute nephritis increase and decrease of œdema coincide respectively with a moderate rise and fall in each pressure, the curve of which shows a close correspondence, save that the minimal fluctuates less than

the maximal and rarely attains a level of more than 110 mm., whilst the maximal may reach 180 mm. When the pressures are raised, the pulse rate is slow, only to quicken when both pressures drop suddenly or gradually with occasional transitory elevations.

Lian and Haguenau[156] found in seven soldier patients that the maximal arterial pressure was definitely raised in six and the minimal pressure in five. In one of these the systolic arterial pressure was only moderately high—a patient aged twenty years had a maximal pressure of 148·5 mm., with a minimal pressure of 138. Since he was not seen until a month after the beginning of acute nephritis, it is probable that at the onset the systolic pressure was considerably higher and had fallen progressively. None of the above patients manifested any evidences of pre-existing chronic nephritis. In another series of observations on eleven soldiers only three exhibited a raised pressure after the disappearance of acute symptoms. Lian and Haguenau classify these cases in three groups: (*a*) a certain number recover completely; (*b*) in some a "simple" albuminuria persists without hyperpiesis or evidence of renal changes; (*c*) in others chronic nephritis, with or without raised arterial pressure, supervenes, but all authorities agree that this is rare.

In hyperpiesia lowering of systolic pressure is often productive of general improvement. Batty Shaw[90] has shown that temporary falls frequently occur apart from drugs, and may be attributed to temporary relaxation of arterial spasm or weakening of the cardiac over-efficiency, or both. "The theory is an attractive one that the change is due to a falling off and resumption of effectiveness in whatever agency is responsible for the hyperpiesis. In other instances the fall is due to a temporary infection; if remedied, the original high pressure is resumed. If the infection is severe and 'terminal,' there is often a rapid fall to a low level with a pulse which cannot be felt before death."

5. Chronic Nephritis.—A high diastolic pressure, with a small differential pressure, may be found in the incidence of chronic nephritis. If the systolic pressure be raised to 180 or higher, and there is a corresponding dispropor-

tionate rise in the diastolic to a height of from 120 to 150 mm., unless some other cause is discoverable, it is almost certain that some degree of renal inadequacy is present, and the outlook is grave. Diagnosis is materially aided by a previous history of acute nephritis, a urine of low specific gravity with usually a slight amount only of protein therein, nocturnal frequency of micturition, and later, albuminuric retinitis and nocturnal dyspnœa ; also by a high blood urea content, definite failure to concentrate urea in the urine and failure to react to treatment. Neither a normal specific gravity, nor absence of protein and casts in the urine exclude renal disease, but a persistently low blood urea content, in the absence of œdema, does. In early cases high pressures may be only intermittently present, and even when uræmia has supervened, high pressures are not constant. In chronic nephritis the tendency is to death by uræmia or cerebral hæmorrhage.

6. Cerebral Angiospasm.—Spasm of an arteriole within the brain leads to ischæmia of the area of brain substance supplied, with consequent loss of function. This leads to *transient* attacks of dizziness without rotation, faintness, paresis or paralysis. Hemiplegia, monoplegia or aphasia, lack of mental clearness, loss of memory and paræsthesiæ appear suddenly, last for a few hours up to a few days, and then disappear, either completely, as is usual in first attacks, or, in the case of recurrent seizures, leaving various degrees of paresis. Subsequently, a fatal termination may ensue from cerebral hæmorrhage. The complete recovery that so frequently takes place is a bar to believing that these paralyses originate from cerebral thrombosis, and there is support for the assumption that the symptoms are due to angiospasm because in the comparable condition of sensory transient amaurosis the retinal arteries have been seen to be constricted, the constriction passing off with return of vision. The cause of such angiospasm has been proved to be non-toxic.

7. Cerebral Hæmorrhage.—Cerebral hæmorrhage is usually, but not invariably, associated with the years past middle life. It may, however, occur at earlier ages, and rarely may be fatal during adolescence. In fact, *the age of*

cerebral hæmorrhage coincides with that of hyperpiesis, although this statement must be qualified by saying that cerebral hæmorrhage is an accident due to disease of the cerebral arteries from any cause, with or without accompanying high pressure.

The most dramatic feature of high arterial pressure, whether diagnosed or not, which I have often noted, is the feeling of comfort and physical well-being on the part of those about to succumb to cerebral hæmorrhage. If such a subject expresses himself as feeling particularly fit, then is the time to look out for sudden and desperate events.

Nevertheless, arterial pressures which are excessive in the absence of arterial degeneration are insufficient of themselves to provoke the incidence of cerebral hæmorrhage, although they influence its severity, and thus become a notable element in prognosis. Encephalitis plays also an important part. In relatively young subjects considerable hyperpiesis is apt to terminate in apoplexy with extensive extravasation into the ventricles. A more elderly subject, whose arterial pressures are less, may on the contrary survive his lesion, and succumb to intercurrent complications, the hæmorrhage itself remaining limited in extent.

An interesting fact that emerges from my own practice is that the majority of cases of cerebral hæmorrhage occurring over the age of forty years that I have seen in consultation have run arterial pressures well below 200 mm., and most commonly between 145 and 190 mm., which looks as if extremely high pressures are usually met with in arteries capable of withstanding them, whereas, if the arteries have become diseased, they tend to burst at the weakest point under pressures which have not mounted to inordinately high levels. The explanation of this apparently paradoxical phenomenon is that the presence of *superficial* arteries which are thickened, tortuous or even calcareous (generalised arteriosclerosis) bears no relation to the existence of *deep* intracerebral arterioles degenerated by reason of atheroma (nodular and localised atherosclerosis) leading to angio-necrosis, which may be extensive. The pathological processes in the two cases are entirely distinct, and the march of events in generalised arteriosclerosis being slow

and gradual, the arteries have time to accustom themselves to grades of high pressure far higher than would cause cerebral hæmorrhage in arteries locally degenerated. As soon, however, as intracranial pressure becomes higher than systemic pressure, death ensues (Harvey Cushing : " Vaso-motor Reflex ").

Another point to which attention may be directed is the frequent occurrence of hæmorrhages, angina pectoris and sudden death during sleep. It is well known that during quiet sleep the vital functions are considerably reduced. The apparent paradox is explained by the fact that in disturbed sleep with reflex excitations, and especially during terrifying dreams, the rise in arterial pressure " may be both large and steep in ascent, and the danger of rupture of a weakened cerebral artery is facilitated by the recumbent posture." [166]

During and after a sudden cerebral hæmorrhage the arterial pressures should be carefully noted. A definite fall in pressure indicates that the hæmorrhage is grave ; if the height of the pressure does not become re-established, or continues to fall, this denotes that hæmorrhage is progressive and that the patient will lapse into coma with an almost invariably fatal termination. If, on the other hand, the fall is not great, and the pressure recovers its former height without rise of temperature, the patient's chances of recovery are correspondingly increased.

8. Cerebral Thrombosis.—Hyperpiesis is no protection against the formation of thrombi. The vast majority of apoplectic seizures which patients survive are due to thrombosis and not to hæmorrhage.

9. Aortic Regurgitation.—A high systolic with normal diastolic may be due to emotional disturbance or physical exertion apart from arteriosclerosis. Occasionally it may result from peripheral vasodilatation associated with forcible cardiac action. In aortic regurgitation during the early stages the systolic is raised while the diastolic remains steady. Subsequently the diastolic becomes lowered, the systolic keeping high, while later still lowering of the diastolic is out of all proportion to the slight lowering of the systolic. In other words, the support has given way

at each end of the arterial pressure scale. Thus a very high systolic with a very low diastolic and the largest differential pressures usually found constitute the clinical picture of arterial pressure in fully developed aortic regurgitation. When the patient lies down, the systolic is much higher in the leg than in the arm.[168] So characteristic is this difference that it is diagnostic. It is due to an altered condition of the arterial wall, and not to diminished peripheral resistance. A similar but less marked difference is also present in abdominal aortitis,[169] in arteriosclerosis and in hyperthyroidism.

10. Hyperthyroidism.—In early stages of Graves' disease the systolic pressure is usually lowered, coincidently with alteration in the differential pressure, which frequently shows an increase. Later the systolic pressure begins to rise, and with development of cardiac hypertrophy, vascular and renal changes, the case becomes one of typical chronic high pressure. Harris[170] believes that "it is only in thyroidism that there is both an increase in pulse rate and at the same time a high pulse pressure." The averages of twelve cases recorded by him are $\frac{S\ 140}{D\ 78}$ P.P. 62, P.R. 113. This point may prove of utility as a differential diagnostic test, but a larger series of cases will first have to be compared with control cases of tachycardia of non-thyroid source. Nearly always the myocardium is affected. At times in a young subject the maximal pressure will be found raised to 150 mm. or more, when to the finger the pulse appears small and the pressure does not appear to be increased. Such usually turn out to be cases of either hyperthyroidism or chronic nephritis.

11. Bilateral Carotid Pulsation.—In late stages of hyperthyroidism and the arteriosclerotic form of aortic insufficiency, bilateral carotid pulsation is not infrequent in association with a high systolic pressure.

In both diseases, visible and palpable pulsatile dilatation of the carotids occurs together with a collapsing arterial pulse and capillary pulsation. Diastolic pressure remains within standard limits, as contrasted with the endocarditic form of aortic insufficiency in which the diastolic is often low.

Carotid pulsation is most obvious when the patient stands, which differentiates it from venous pulsation, best seen when the patient lies down, and lessening or disappearing in the erect posture.

My experience is that carotid pulsation is not as a rule manifest in cases of hyperpiesia.[171] In arteriosclerosis, though the pulse wave is propagated more rapidly because of the thickened arterial walls, distensibility is lessened to a degree commensurate with that of a normal relaxed artery. In a tortuous brachial artery, for example, visible movements are not due to expansion but to each systolic impact which tends to straighten out the wavy vessel.

12. Ocular Changes.—The following summary of a discussion at the Royal Society of Medicine represents British views [172] on the relationship of vascular and other retinal changes, such as white exudates and papillœdema, to high arterial pressure, arteriosclerosis and renal disease.

Most speakers agreed that there are two main clinical types of retinitis : (1) " Arteriosclerotic," associated with hæmorrhages into the retina. The disc is a deep brick-red, with round, irregular or flame-shaped hæmorrhages scattered about the retina, but mostly near the blood vessels ; arteries glistening, tortuous and perhaps uneven in calibre, whilst the veins are full. (2) " Renal," associated with albuminuria. The disc is pink with feathery edges ; there may be a few scattered hæmorrhages, but the striking feature consists of large or small white exudates, irregular in shape or distribution, or arranged symmetrically about the macula (Leighton Davies). In many instances it is not possible to distinguish absolutely, from ophthalmoscopic appearances, between the one and the other (Fisher).

Foster Moore, however, suggested :—

1. That in a proportion of cases of general arteriosclerosis, as disease of the retinal vessels increases, exudates form in the retinal tissues which are probably dependent upon the local vascular disease.

2. That the ophthalmoscopic appearances resulting are in large measure distinctive.

3. That prognosis as to length of life is uncertain, but

may extend to several years, and that it differs greatly from renal retinitis in this respect.

4. That a large number of these patients die of a gross vascular cerebral lesion (50 per cent. of his cases investigated).

5. That the condition calls for separate recognition, and that the term " arteriosclerotic retinitis " seems appropriate.

Feiling [173] investigated thirty cases, referred to him for failure of vision, from the point of view of the physician. Twenty-five were arteriosclerotic, with average age 63·3 years, fifteen being females. Sixty per cent. had unilateral retinitis, which is against the view that toxæmia is the direct cause of the retinitis. The average systolic pressure was 214, and the diastolic 118. The five renal cases were differentiated from the former group by (*a*) history of nephritis ; (*b*) persistence of large amounts of protein in the urine ; (*c*) well-marked œdema. The average age was 43·8 years, two being females. All had bilateral retinitis. The average systolic pressure was 235 mm., and the diastolic 135. In both groups there was high arterial pressure, well-marked thickening of the accessible arteries, and cardiac hypertrophy.

Twenty cases tested for renal efficiency by A. Ellis and Marrack [68] fell naturally into two groups : one early, with no evidence of inefficient renal function and no excess of blood urea, life being prolonged, and the majority of deaths being due to cerebral hæmorrhage ; the other with evidence of very inefficient kidneys and excessive blood urea, the prognosis being much more grave, and the patients dying within a short time from uræmia.

Batty Shaw [90] invoked a pre-renal toxæmia to explain all retinal manifestations, arteriosclerosis being the first effect and arteriosclerotic retinitis a later effect of a slow toxæmia, minimal in amount, but when in greater intensity producing the " renal " type of retinitis, the changes being to a large extent indistinguishable.

The conclusions were that retinitis may in certain cases be caused by toxæmia (as in pregnancy) of endogenous or exogenous origin, but may also result from vascular causes local in the retina, *e.g.*, the stellate figure accompanying

papillœdema, and that hæmorrhages are due to capillary leakage through diapedesis, akin to the petechiæ seen in certain infective disorders.

The views of American ophthalmologists are expressed in a series of three papers, to which the reader in search of fuller information is referred.[174, 175, 176]

Latency

The length of time taken for raised blood pressure to become permanently established in cases without foregoing renal disease is not absolute. It varies with individual and external factors, and probably in many cases the process is spread over a number of months or years. That acute nephritis may speedily bring about high pressure conditions is well recognised, and certain other infections also leave their permanent impress. High pressure, nevertheless, may long be latent and unaccompanied by no disability, or by signs which are negligible and discoverable only when a test is made. For some time and under certain conditions the process may rightly be regarded as advantageous and in nature protective against or compensatory for disordered function.

By reason of its insidiousness early recognition of raised arterial pressure before secondary and structural changes have had time to evolve is of the highest prophylactic value, since in the early stages prevention, or at any rate delay, of advance, particularly in respect of the incidence of nephritis, is possible.

A spurious latency may be due to lack of opportunity of medical examination in persons who feel well, because they find no necessity to seek advice.

On the other hand some people avoid consulting doctors, and prefer to live in a fool's paradise because they are afraid of being told unpleasant truths, or that their enjoyment of the material pleasures of life may be curtailed. In apprehensive subjects it is advisable for the doctor never to state the exact figures that the arterial pressure has reached, for, in these latter days, the fear of high blood pressure is often as much of a bogy as the fear of cancer. The

truth, but not the whole truth, should be told, so that the patient may be warned and safeguarded, but not alarmed.

If **all** persons over forty years of age could be induced to realise the importance of having their blood pressure accurately taken, say once a year, this wise precaution would be greatly to their advantage. Directions tactfully given by the medical adviser, and carefully followed by the patient, as to general management and mode of life, would avert many premature deaths.

Mortality

In the United States about 10 per cent. of the total annual mortality is due to the consequences of high arterial pressure. This mortality is stated by Fahr [177] to be as great for persons fifty years of age and older as the combined death rates for cancer, all forms of tuberculosis, and of respiratory disease including pneumonia, for the same age category and year, and twice as great as the death rate for cancer. From the Registrar-General's Statistical Review for the year 1932 I have calculated that hyperpiesis accounts for about 7 per cent. of the total mortality in England and Wales, a figure not so high as the American, but one which stresses the fact that hyperpiesis, and particularly hyperpiesia, steadily reduces the expectation of life at ages from fifty years upwards.

The effect is well shown in Table XI, published in 1922 by the North-Western Life Insurance Company of America.

TABLE XI

Effect on Mortality of High Arterial Pressure

Accepted or Rejected.	Average Systolic Blood Pressure.	Approximate Extra Mortality.
Accepted . . .	141 mm.	10 per cent.
,, . . .	146 ,,	35 ,,
,, . . .	153 ,,	60 ,,
Rejected . . .	160 ,,	110 ,,
,, . . .	170 ,,	165 ,,

From these figures it is clear that systolic pressures above 150 mm. become increasingly serious as regards prognosis. The mortality rate is higher in cases in which the pressure ranges above 200 mm. Hg. than in those in which it is below this level, while pressures of 300 mm. or over reduce still further the expectation of life to a few years.

In Cadbury's [178] series of 305 hospital cases, more than half the fatal ones had manifested a systolic pressure of over 200 mm., and 86 per cent. a diastolic pressure of over 100 mm. It is interesting to compare these with Lian's cases (pp. 154 and 158, In general, however, too much importance should not be attached to hospital statistics, since these cover the worst cases admitted for reasons other than their raised blood pressure, and not comparable with the large numbers of people with pressures of equal height who are carrying on their ordinary avocations, frequently without experiencing any need for treatment. Hence for unselected cases the outlook is less gloomy than at first sight would appear.

The duration between onset of first symptoms and death is very variable ; it may be as short as nine months, or as long as twenty, or even thirty, years.

The most frequent causes of death are circulatory failure (approximately 45 per cent.), cerebral hæmorrhage or thrombosis (30 per cent.), while angina, as a symptom of coronary thrombosis (15 per cent.), and terminal infections—notably pericarditis, pleurisy and pneumonia—2 per cent., account for the remainder. These figures are in direct contrast to the cause of death in chronic glomerular nephritis, which is nearly always uræmia due to renal insufficiency (8 per cent.). Death is most frequent between the ages of forty and sixty, the underlying condition being either chronic heart-muscle disease, chronic nephritis or both combined. Seventy per cent. of all chronic myocardial disease not associated with primary valve defect or luetic aortitis is consequent upon long-continued high blood pressure.[177]

CHAPTER XI

Prevention of High Arterial Pressure

Having realised that potent causes of this condition are worry and anxiety, excess or chronic poisoning of some kind, the solution is to guard against these and other causes as far as possible, and, if present, to deal with them on appropriate lines.

The daily life of a person who wishes to avoid high arterial pressure or who has already become the subject of it, may conveniently be divided into two portions, the hours of activity and the hours of rest.

1. The Hours of Activity

The waking hours should be pervaded by a philosophical and balanced outlook, the issue of a quiet mind. The mental attitude of the ancient Stoics, who were neither unduly overjoyed nor cast down by any human affairs, is the philosophy at which to aim. This does not necessarily imply that the individual who desires to maintain a normal level of arterial pressure should lead a dull, unimaginative and phlegmatic existence, but that harmonious attributes, which can be cultivated by those in whom they are not inborn, are likely to check those frequent excesses which occur in persons of unbalanced temperament. Such excesses may be either psychical or physical. In the lives of many people psychical influences induce greater upheavals and cause more destructive effects upon the organism than those which are physical. "With such persons discipline must be attained by spending day after day in drill, in gaining self-control, in repressing volatility. In this precaution there is nothing false to a man's best self: it is the way to get the most work out of himself before he dies. . . . Due vigilance may be exercised without the encouragement of hypochondria; as someone well put the rule: find out

what you can do and do it, find out what you cannot do and never do it (Allbutt)."

1. Environment.—In working out these principles, consideration should first be given to environment, which is of the greatest importance. By this I do not mean solely bricks and mortar, although the immediate environment should be made as hygienic and as attractive as possible, but I refer rather to the daily round in its triple aspects—spiritual, mental and physical. There may be a psychical environment of love, or one of hate; an environment of trust and confidence, or one of jealousy and suspicion; an environment of calm and repose, or one of uncertainty and anger. Individuals create their own psychical atmosphere, and though it is usually impossible to escape from the material surroundings imposed by circumstances, yet it may be possible to change the moral atmosphere so as to render environment in general less irksome and more harmonious. The counsel of perfection, therefore, is at all times to cultivate a cheerful disposition, and to minimise as far as may be any disadvantages, always remembering that true happiness comes from within.

2. Occupation.—An occupation of steady routine is to be preferred to one of constant anxieties and sudden hazards, even though the latter may appear more lucrative. A sufficiency of holidays should be taken, so that work, when resumed, is entered upon with renewed zest. These vacations should be spent in quiet, sunshiny and pleasant surroundings remote from the noise, hustle, excitement, late hours and nervous stress imposed by our modern super-civilisation.

3. Habits. (*a*) *Regularity.* Habits should be regular and methodical, fixed times being set for meals, for retiring to rest and for daily evacuation of the bowels. Especial care should be taken by provision of a suitable diet, with the addition of drugs only if necessary, to prevent constipation, and so to ensure an easy action each day. *Straining at stool is particularly harmful in that it sends up the level of blood pressure*, and, in the case of those in whom the pressure is already high, may precipitate a sudden fatality in the shape of apoplexy.

(*b*) *Moderation.*—More people are killed by over-eating

than by over-drinking, so that all the pleasures of the table should be enjoyed in strict moderation. With advancing age the total quantity of food at each meal should be lessened. Many in the latter half of life dig their graves with their dentures, which instead of a help become a handicap. In old age the diet should approximate to that of the child, for " a man should learn to go out of life as he comes into it."

(*c*) *Tobacco.*—Tobacco in any form is an insidious poison with effects upon the arterial walls and upon digestion, both of which may easily suffer in susceptible individuals. " Chain smoking " of cigarettes, to which many women as well as men are prone, is also bad for the nervous system, and makes its victims " jumpy " and irritable, besides drying the tongue and thus directly facilitating the consumption of alcohol. From the blood pressure point of view this habit possesses no recommendations.

4. Diet.—(*a*) *Solids.* Meals should be varied in kind, and should be balanced, special attention being directed to inclusion of those vital principles in foodstuffs known as vitamins, which are found in *fresh* fruit and fruit juices, vegetables, milk, cream and butter. My own opinion is that more benefit results from lessening *total* food intake to the minimum needs of the individual than by mere protein restriction. Excess of fat is inadvisable, especially when food is fried in oil or other fatty substances, though olive oil in the natural state may be taken well mixed with fresh salads. Similarly with carbohydrates, excess causes flatulence and digestive disturbances, thus tending to augment arterial pressure by inducing obesity. The quantity of protein, including all forms of meat, bacon and fish, taken at any one meal should be limited. It is best to avoid protein more than once or at most twice a day, the quantity of cooked meat not exceeding a daily total of 8 oz., or preferably less than this amount in accordance with the body weight. The dietary should consist mainly of milk, vegetables, eggs and fruit. In general, boiled meats, chicken and fish are preferable to roast joints. Eggs are usually admissible, and a light breakfast of wholemeal bread and butter, or toast and marmalade, followed by fresh fruit, such as apples,

pears, peaches, oranges, melons, grape fruit or bananas, is better than heavy dishes of fish, or bacon, sausages and eggs. The American custom of taking a glass of orange juice before breakfast is excellent.

The chief points at which to aim are :—

(i.) To diminish the bulk and number of meals.

(ii.) To reduce the amounts of carbohydrates and fats, with still stricter protein limitation. Seven grains of protein per pound of body weight may be adequate.

(iii.) To secure sufficient vitamin supply and prevent acidosis.

Immediate benefit from an altered dietary of this character is not apparent, for some time must necessarily elapse before the full effects of lessened intake become manifest.

(*b*) *Fluids.* (i.) *Water and Other Fluids.*—Water is the best, and should be taken on an empty stomach an hour before meals and at bedtime. Where the water is hard, as in London, it may with advantage be softened by installation of a water-softening plant, or, if only individual supply is needed, small machines are now made for softening a tumblerful at a time. Pure English table waters are Stretton, Malvern, Braceborough and still Salutaris, which latter is distilled water passed over forest-burnt wood charcoal in order to render it palatable. Of the French mineralised waters, Evian, Vittel and Contrexéville enjoy a deserved reputation in flushing out the kidneys. If there are tendencies to acidosis, uric acid retention and congestion of the liver, Vichy water is the most alkaline of all table waters, and is of great efficacy. To any of these, lemon juice, one quarter, with three-quarters of orange juice or citrate of potash, may be added if desired.

In addition to other drinks, one pint of water for each five stones of body weight imbibed during the twenty-four hours is a good working rule.

Other drinks should be taken sparingly. China tea freshly made is to be preferred to Indian blends, and tea in any form should be freshly infused, taken weak and not allowed to stand. Coffee should be eschewed, or if a craving for this is experienced, taken only in the form of H.A.G. coffee, which contains only a small amount of caffein.

(ii.) *Alcohol.*—Alcohol, except in the smallest quantities and well diluted, is best avoided. The subjects of high arterial pressure are well advised "to go on the water waggon" and to leave alcohol alone. Cocktails and liqueurs are the most insidious and most dangerous forms of alcohol because of their mixed and high alcoholic strength within small compass. It is thought by some that spirits, such as whisky and brandy, are rendered comparatively harmless by addition of soda water, but, even so, there is a tendency to repeat the dose, and when this happens on an empty stomach, the alcohol is rapidly taken into the blood stream and exerts its effects. In cardiac subjects, brandy should only be taken under medical direction, and its results in increasing heart rate and tone are capable of substitution by other diffusible stimulants, such as ether, chloroform and ammonia in appropriate formulæ. Individuals vary enormously in their reactions to alcohol, so that, without in any way advocating teetotalism for mankind in general unless inclination and habit point in this direction, for the subjects of arterial degeneration and of high pressure the general counsel of safety should be enjoined that they will do far better without alcohol at all, and are likely to live longer for such abstinence.

5. Fasting.—Particularly in cases of metabolic excess, where the patient is obese and aften plethoric, a fast day once a week at the outset, later diminished to once a fortnight or three weeks, is of the greatest benefit in aiding high arterial pressure to become reduced by natural means. Less body weight means less work for the already overburdened heart to do in the performance of any necessary exertion, and since this organ always shares in the general fatty change and thus becomes essentially hampered, by gradual weight reduction a return to more normal tone of the heart and other muscles is facilitated. Added to which, a slim person can achieve more in the way of exertion, and so help to keep the body weight at a more normal level, without running grave risks, provided always that exercise is kept within due limits with avoidance of physical fatigue. Some there are, nevertheless, who find that their weight tends to increase in spite of exercise. These people may be

the subjects of water retention in the tissues, and are dealt with on the lines of exclusion of table salt, dry meals, limitation of fluid which should be taken in the form of hot water—half a pint sipped on rising, at mid-day and at bedtime—and total abstinence from alcohol.

On the days of fasting, nothing should be taken during twenty-four hours save water, hot or cold as preferred, weak tea or orange juice at three-hourly intervals.

6. Exercise.—Individuals of the "John Bull" type manifesting high arterial pressures will in general be wise to take regular exercise of a steady kind, but to avoid over-strain and excitement. Exercise within the limits of fatigue may be taken on foot, on horseback or in the form of games and sports. The middle-aged and elderly as a general rule should not vie with their juniors in games involving sudden and violent exertion, or in those that demand long-continued efforts of endurance. Motoring, but not at high speeds, suits most, but not all. Golf should be preferred to tennis for those in whom age changes have begun to be manifest.

2. The Hours of Rest

Enough rest and repose is essential. The exact amount varies with the individual. One hour's recumbent rest after the mid-day meal is advantageous for nervous and excitable subjects. In general, a minimum of eight hours should be spent in bed. Many require more than eight hours, the young needing longer hours of rest than adults. So far as is possible this time in bed should be devoted to quiet sleep.

The above rule of life, which of course has to be modified in accordance with individual conditions and circumstances, is a counsel of prevention. Put in the shortest formula, the keynote in avoidance, as well as in control, of high arterial pressure is to be found in four words—MODERATION IN ALL THINGS. This precept applies equally to any person at any age, and is based on the dictates of common sense.

CHAPTER XII

Control of High Arterial Pressure

"We are apt to forget that high arterial pressure is not infrequently a necessary evil, and should sometimes be regarded as a compensatory effort, or as one of the natural defences of the body."—Oliver: *Studies in Blood Pressure.*

Given a patient with raised arterial pressure, since any deviation from standard level can only be regarded as symptomatic, the first requisite is to find out what we can of the clinical factors concerned.

Although, as already stated, the prime causes of pathologically raised blood pressure can only be inferred, yet numerous allied and secondary factors are known, to which attention should justly be directed. To regard blood pressure as an entity is a mistake. The resultant of the interplay of the *total* forces involved forms the clinical picture, and on recognition of these forces successful management rests. In many cases some degree of high pressure may be compensatory, its existence, without associated symptoms, not calling for attempts at reduction, the aim rather being to prevent further rise.[179]

There are still many gaps in our knowledge. What the practitioner wants to know is under what conditions and within what limits it is justifiable to reduce a high or to increase a low pressure, and how this is best brought about.

As to such procedure, it is difficult to lay down hard and fast lines. Each case must, of necessity, be dealt with on its merits, for no two are alike, and each presents its own problems, which cannot be solved until one has learned to pick out, differentiate and evaluate the many components of the symptom-complex.

The varying proportions and degrees, for example, in which fundamental physical characters of the circulation may be altered must always be taken into account, for pathological alterations in these are set in motion by operation of the underlying cause or causes. Biochemical studies

of the blood and results of experimental work based thereon may enable us to solve the problem which up to now has been so entirely baffling, namely, that of therapeutic regulation of the diastolic pressure.

In management of high arterial pressures the first salient point to realise is that each person possesses an individual range of pressure, *i.e.*, within which limits he or she feels most comfortable. Should the pressure rise much above or fall much below these limits, subjective sensations of discomfort are likely to occur. A second salient point is that immediate reduction of arterial pressure after the institution of treatment is not to be looked for. Even in the most favourable cases results worth securing are gradual, and restoration to normal levels of an elevated arterial pressure is not usually attainable within a brief period of time. Patience and perseverance are thus requisite on the part of patient and doctor alike.

Hyperpiesia, even of high grade, may accidentally be discovered during the course of examinations for physical fitness or for life insurance in numerous apparently healthy subjects, who admit no serious illnesses and confess to no or few symptoms. In such the liability to cerebral hæmorrhage is slight, and they will speedily be rendered miserable if misguided endeavours are made to depress their heightened pressures. In other cases it may be that the raised pressures are needful to carry on renal function in the face of some degree of organic defect.

Having completed the investigation of the patient as detailed in Chapter X., we are now in a position to determine the precise directions which should be given.

These will vary according as the case falls into one or other of the following clinical categories :—

1. The simple high pressure (hyperpietic) group.
2. The cardiovascular (arteriosclerotic group).
3. The renal (nephrosclerotic) group.

1. The Simple High Pressure Group

A survey of my cases indicates that during the first years of increasing arterial pressures the majority of hyperpietic

subjects are totally unconscious that they are otherwise than in a perfect state of health.[180] For a time, indeed, owing to greater supply of blood to the brain, their energies are enhanced, and they enjoy life to the full. Their ruddy complexions appear to them and to their friends as visible evidences of robust and vigorous well-being. Their activities are unimpaired, and they tend to vie with those of younger age in games and sports. And yet it is just during these early years that the need for timely and preventive management passes unrecognised. The time at which these subjects are first led to consult a physician is usually one at which symptoms of the malady have become apparent, and chances of permanent amelioration have thus become correspondingly diminished.

Hyperpiesis, in its initial stages, before diastolic pressure has become permanently elevated, and while recovery is still feasible, is best controlled by regulation of diet, rest and exercise. Such procedure often has extraordinarily good effects in improving the general health and in correcting metabolic errors to which the disorder of pressure may be due.

In its temporary stages hyperpiesis, even if not seen during its initial stage, can generally be relieved by calming the patient's fears, whether about the height of the individual arterial pressure itself or about more general affairs, by clearing up infective processes or toxic agencies, general, respiratory or alimentary. Acute infections should be checked, and chronic foci of infection sought for and removed. The respiratory and alimentary tracts as a whole should be investigated, particular attention being paid to the state of the gums, teeth, tonsils, gall-bladder, appendix and cæcum, not forgetting other possible sources of pressor toxins, such as the nasal sinuses and antra, the auditory apparatus, and the pelvic viscera.

It is useful to bear in mind the three pathological stages that occur when arterial pressure becomes high : (1) hypertonia of the muscular coats of arterioles and capillaries ; (2) progressive hypertrophy of the muscular tissues of the cardiovascular system ; (3) lessened muscular efficiency by reason of progressive fibrosis, causing vascular narrowing.

Hypertrophy is not limited to the heart, but is shared by the entire vascular system. Only the first stage is curable.

Psychical Influences, promoting and maintaining a rise in arterial pressure, should receive due attention (pp. 99,132, 168), and any lack of endocrine balance discoverable should be rectified. All sources of irritation, mental or physical, in the daily life should be sought for and as far as possible removed. Such are domestic or occupational conditions giving rise to anxiety or worry, improper and excessive food, alcohol, tobacco, etc. It is wise not to set up an added source of fear by too frequent arterial pressure estimations, and particularly not to give detailed and comparative information to the patient as to the heights of the pressures recorded. Habitual dietary excess in protein tends to cause a hypersensitive nervous system and reflex high pressure.

Longer hours of rest in the intervals of work often do wonders. A busy practitioner of my acquaintance in advancing years checks a tendency to temporary rises of systolic pressure by retiring to bed every night for a week at 9 p.m. with satisfactory results. Work itself may have to be curtailed, in time, in amount or both, but to advise complete retirement is unsound. Certain transient nervous forms clear up with a few days' rest in bed, which is essential at the outset of treatment for fully developed cases associated with anæmia, dyspnœa, fœtid breath, vomiting, diarrhœa, dropsy and other systemic disturbances.

Hypertonic Arterial Spasm (*Angiospasm*).—Tendencies to vascular spasm inducing hypertonic pressures are alleviated by mental and physical repose, light diet, relaxation and other exercises, walking, riding and sport in moderation, psycho- and hydrotherapy. When angiospasm is set up and maintained by excess of acid in the blood, the best treatment is to administer alkalis. In such cases I have found 20- to 30-grain doses of sodium bicarbonate thrice daily in a simple digestive mixture to be of considerable service.

Becchini [181] states that in persons between thirty and forty years of age, five to ten hypodermic injections of 2–5 c.c. of

Trunecek's solution * is sufficient to banish spasm more or less completely. In the elderly, larger doses may be necessary. The tendency usually persists, and may need an occasional small dose. Personal experience of the results of these injections in certain cases has been satisfactory.

Of the newer remedies, the most successful have been (i.) padutin, a pancreatic extract, ½ to 1 c.c. intramuscularly, or three tablets orally, in daily dosage, and (ii.) pacyl, a choline derivative, two tablets by mouth three times a day, beginning with small doses, increasing if necessary, and diminishing after three weeks to one or two tablets daily.

Endocrine Influences.—The inter-relationships of the endocrine glands are so manifold, and so influenced by numerous other factors, that from the scientific standpoint it has been asserted with conviction that endocrinology needs a leadership willing to admit the magnitude of its shortcomings. In the past, organotherapy has severely suffered from the unbalanced urge of enthusiastic empiricists; let us hope that, for the future, zeal may be tempered with discretion.

Granted that the endocrine glands are concerned in certain disturbances of arterial pressure, the problem of its control along the lines of organotherapy is complex. Collected observations, though largely empirical, nevertheless lead to the inference that therapeutic efforts are most likely to succeed in control of hyperpiesis without organic changes, when due to (*a*) temporary over-stimulation of the pituitary or adrenal glands by absorption into their blood supply of irritating substances derived from infective foci or toxæmic origins; (*b*) functional inefficiency of thyroid metabolism with consequent accumulation of waste products; (*c*) disturbances of internal secretion in the gonads, particularly the ovaries.

Clinical evidence shows that hyperpituitarism or hyper-

* *Note.*—Trunecek's solution consists of—

Sodium chloride	4·92 gm.
Sodium sulphate	0·44 gm.
Sodium bicarbonate	0·21 gm.
Potassium sulphate	0·40 gm.
Distilled water to	100·0 c.c.

adrenia may successfully be checked by administration of pancreatic hormones, whilst hypothyroidic cellular infiltration of the walls of the precapillary arterioles may be removed, and detoxication promoted, by dry thyroid in $\frac{1}{4}$- to 2-grain doses thrice daily in cases apart from arteriosclerosis or heart disease, especially when obesity is an accompaniment.*

Kylin [182] believes that hyperpiesia results from disturbance of the autonomic nervous system, and that control of this is the primary indication. He regards great instability of arterial pressure as more characteristic of essential high arterial pressure than its actual height. The majority of the patients have an abnormally low sugar tolerance, which may be dependent upon an upset of nervous regulation differing from that of the pancreatic form. A rest cure should be tried first, since this often gives good results, though when the patient returns to his former occupation the pathological condition may reappear. Kylin treats hyperpiesia by calcium salts and atropine given by the mouth, and is of opinion that improvement observed in cases so treated warrants further trial of this method. Theoretical grounds for this treatment are predominance of the parasympathetic over the sympathetic nervous system in essential hypertonus (as indicated by the adrenaline reaction) and the view that calcium ions stimulate the sympathetic system. It has been shown also that in essential hypertonus the calcium of the blood is diminished. Atropine is employed in the endeavour to restore the balance between the vagal and sympathetic nervous systems.

Hyperpiesia and Asthma.—Kerppola [183] found that among 200 cases of primary hypertonia there were ten in which characteristic signs of bronchial asthma were present. Most of these had a normal or subnormal blood pressure in the intervals between attacks. In 20 cases he measured the blood pressure during the attacks of asthma, and in 5 of these found no appreciable rise. In the remaining 15 cases during an attack there was an appreciable rise, the blood

* *Note.*—Dry thyroid, B.P. 1932, is standardised to contain 0·1 per cent. iodine in combination as thyroxine, and 3/10 gr. of this represents gr. j fresh healthy gland substance.

pressure in 12 of these 15 cases having been normal or subnormal before the attack. In these 12 cases the rise of blood pressure ranged from 25 to 100 mm. of mercury ; in the remaining 3 cases, in which the pressure was abnormally high before an attack, the rise during an attack ranged from only 15 to 25 mm. of mercury. As a rule, the more severe the attack of asthma the greater was the rise of blood pressure. In 10 cases in which an attack of asthma was stopped by an injection of adrenaline, with or without pituitrin, the blood pressure began to fall during the first minutes following the injection, the fall being very rapid during the subsequent ten to fifteen minutes. The author suggests that the relief obtained in asthma with adrenaline is due to its paralysing effect on vasoconstriction, this effect being achieved only when a comparatively large dose of adrenaline, such as 0·5 mg. by intramuscular injection, or 0·1 mg. by intravenous injection, is given.

Similarly with asthma, A. Francis [184] has claimed reduction of arterial pressure, permanent in a fair percentage of cases of pure hyperpiesia, by cauterising the nasal septum as high as possible. He believes the effect to be due to inhibition of vasoconstrictor action of sympathetic fibres running in the mucous membrane.

Climacteric Hyperpiesia.—At or about the climacteric great fluctuations in arterial pressure are met with, connected with instability of the vascular innervation. Hence the hot flushes and other vasomotor disturbances. A benign form of hyperpiesia occurs,[185] most frequently in multiparæ inclined to obesity and constipation, the condition being aggravated by worry and anxiety. In many of these cases the rise, which is said to precede the cessation, is transient ; or it abides at a moderate, and perhaps harmless, level, such as 150–160 ; and the pulse, even if the arteries be a little stretched, keeps fairly soft. There is sometimes a considerable elevation of the systolic pressure, which is increased out of proportion to the rise of diastolic pressure, a fact which materially suggests a favourable prognosis. In other cases, however, the heart is large and fatty, perchance with symptoms of substernal oppression, and peripheral vasoconstriction is the rule. Faulty endocrine balance

almost certainly plays a large part in causation, instability of the pressor mechanism being connected with pituitary and ovarian insufficiency, and, maybe, also with adrenal over-activity. Lack of endocrine harmony may last for a few months up to two years or so, but the ultimate prognosis, in most cases, is good. A substance has been extracted from the ovaries which lowers arterial pressure. It is possible that, during the term of ovarian activity, this substance acts as a depressor, and that at the menopause, when it ceases to circulate in the blood stream, the pressure rises and the body has to readjust its metabolism to the altered conditions.[186]

Kisch,[187] nevertheless, believes that if hyperpiesis occurs at or about the menopause, it is usually due to other associated causes for which search should be made, since he does not think that the rise in pressure depends always upon reduction of ovarian secretion.

Extract of corpus luteum has been advocated in treatment, but I have seen no benefit from its oral administration. On the other hand, many cases derive help from a combination of whole gland pituitary, pancreas, thyroid and ovarian glandular substances, with calcium and magnesium phosphates, calcium glycerophosphate and sodium bicarbonate. For men, spermin is used instead of ovarian substance. Adrenal gland therapy is contra-indicated.

For the manifold subjective symptoms referred to the heart, triple valerianates in combination with ammonium bromide gr. v. and tincture of sumbul ℥x. are often efficacious.

Hyperpiesis and the Toxæmias of Pregnancy.—Though pregnancy and its complications are the most frequent causes of chronic nephritis in adult women during the child-bearing age, yet many patients in whom this disease is suspected are in reality the subjects of chronic hyperpiesis, the kidneys having been either not primarily affected, or, if affected, recovery had since taken place. In such cases, a fast day once a week is of considerable service in combination with detoxication therapy.

Hyperpiesis and Fibroid Diseases of the Uterus.—Examination of 416 patients suffering from uterine fibroids showed no effect on the blood pressure in young women; myo-

matous subjects who have high blood pressure are usually over forty or show signs of renal or cardio-vascular disease.[188]

Hyperpiesis, Gout, Cardiac Œdema and Obesity.—An exclusively milk diet (Karell, 1866) is of service during the first two or three weeks in treating obesity, whether or not of endocrine origin, associated with hyperpiesis and other circulatory disorders. By reason of its calcium content, and especially because of its low sodium chloride (1·6 gm. per litre), milk improves cardiac muscular tone, lessens waste energy and promotes diuresis, thus reducing cardiac œdema. Milk forms a perfect diet in virtue of its balanced proportions of protein, fat, carbohydrate and vitamin A. For adult use it is too voluminous for lengthy administration, so that after two weeks or so it should be replaced by a limited mixed diet containing 2 gm. of sodium chloride per diem, equivalent to one-eighth of the normal intake. Œdema in heart disease and in acute and chronic nephritis is partly dependent upon a disturbed water balance of the tissues, a hydræmic plethora. Of itself, this does not induce fluid retention, but, as soon as the circulatory or renal organs suffer damage, it rapidly leads to œdema. The effects of digitalis are at times evident only when sodium chloride is limited in amount. In gouty or obese patients over forty-five years of age, small doses of thyroid gland, with or without a little digitalis, according to circumstances, at each meal, will often modify raised arterial pressure and improve the general health. It may be given over long periods under supervision, but should be stopped if any symptoms of palpitation or cardiac irregularity supervene. Gout, however, may occur with standard arterial pressure, and is not necessarily associated with one that is raised.

Plethoric Hyperpiesis.—In plethoric persons blood pressure may approach the upper limit of the normal range, or may exceed it. This phenomenon is closely related to body weight, higher pressures corresponding as a rule with heavier weights, and *vice versâ*, though athletes, by reason of peripheral dilatation and vigorous circulation induced by training, are an exception.

When plethora coexists with polycythæmia or erythræmia and supernormal pressure, reduce excess in diet and fluids,

quantitatively by lessening the intake, and qualitatively by substitution of green vegetables and fresh fruit juices for protein and alcohol. Such a diet provides some of the alkali needed, which can be supplemented by mineral waters, such as Vichy or Evian, and administration of alkaline salts. As a further measure of depletion, venesection may from time to time be necessary.

A word of caution is necessary to avoid mistaking a physiological blood pressure for one that is pathological. A full-blooded, robust and muscular country girl, for example, leading a healthy, active life in the open differs not only constitutionally from a thin anæmic and weakly town-dweller, but also in the standard of blood pressure, which, though much higher, is still physiological.

2. The Cardiovascular Group

A. The Heart.

In this group the systolic pressure is very high, usually well over 200 mm., while the diastolic is proportionately less raised. Exercise should be moderate and over-exertion avoided. Dietetic restriction and eliminative treatment are not here of the prime importance that they assume in the nephrosclerotic class.

In cases of progressive myocardial insufficiency of congestive nature, a rapid fall in arterial pressure often follows administration of (*a*) diuretics and cardiac tonics, such as digitalis, theobromine sodium salicylate, iod-calcium diuretin or scillaren. Such fall is all the more striking when preceded by a marked diuresis; (*b*) depletives—mercurials by mouth, salines, leeches and venesection; (*c*) oxygen inhalations for dyspnœa. If œdema persists, salyrgan if the kidneys are sound, with fluid restriction to 2 pints in the twenty-four hours and a salt-free diet.

For cardiac failure with angina and hyperpiesis, bromides, iodine, hot drinks with diffusible stimulants, and counter-irritants. For the more severe cases, atropine or morphine may be required.

Never should drastic attempts be made by vasodilator drugs to cause a *sudden* drop in an unduly high pressure, any more than one should bring down a high temperature

by the use of powerful antipyretics, for grave and at times irreparable harm may be inflicted on the patient, whose blood pressure has become raised for some definite reason. Indeed in some persons a pressure of 200 mm. Hg. or over is requisite to ensure the necessary flow of blood through the capillary network.

"As high pressures creep on stealthily, the system as silently adapts itself to them; so that blindly to try to reduce excessive pressures permanently to the normal would be as harmful in practice as fallacious in reason." [166] But, although in certain cases high pressure may be compensatory, yet it is entirely erroneous to suppose that attempts should not be made to ascertain whether high pressures cannot be lowered judiciously and gradually with benefit to the patient. Sometimes symptoms abate or disappear even though the original height of the pressure head remains little if at all altered. It is first needful, therefore, to find out why the rise has come to pass.

Many persons there are in whom visceral blood supply depends on maintenance of a sufficiently high pressure, and if the result of treatment is to bring about a pronounced fall, unpleasant results will follow for patient and doctor alike, since the heart, and other organs and tissues, through lack of adequate nutrition will fail.

In this group management should be so directed as to keep up the high pressure by maintaining cardiac energy at an efficient pitch whenever heart failure threatens.

B. The Arteries.

By reason of the irreversible character of the arteriosclerotic process, little prospect of radical cure in the present state of our knowledge can be advanced. Associated symptoms, nevertheless, may often be relieved by appropriate therapy. A careful watch should be kept upon the heart, and acute complications such as œdema of the lungs or anginal seizures should be relieved.

In *arteriosclerosis*, drugging, save for relief of symptoms and to aid elimination if faulty, is futile, for no remedies are known which have the power to alter the condition of the thickened intima. The only times when one can do good are

those in which the peripheral circulation is affected by toxic causes (p. 118).

The above group is best managed by moderate restriction of diet, general measures of hygiene, shunning of sudden exertion and strong emotions, and promotion of eliminatory processes by ½ to 1 grain of calomel, or 3 to 5 grains blue pill once or twice a week followed next morning by a saline draught of equal parts of mixed sodium and magnesium sulphates.

For arteriosclerotic subjects with cardiac enfeeblement and persistent arhythmical ventricular premature contractions, Matthew Baillie's well-known pill may be regarded as a sheet-anchor. Alternatively, Pil. Hydrargyri et Digitalis Co. of the St. Bartholomew's Hospital Pharmacopœia, which in composition is somewhat similar, I have used for many years with most gratifying results. It consists of :—

Pil. hydrargyri . . .	1 grain.
Digitalis	1 ,,
Scillæ	1 ,,
Ext. hyoscyami . . .	2 grains.

Sufferers from angina pectoris should be cautioned against climbing hills or going upstairs rapidly, and against any sudden access of strong emotion, especially anger. Flatulent distension of stomach or colon is to be remedied by administration of wood charcoal in combination with magnesium carbonate and betanaphthol.

Hyperpietic cerebral attacks with cerebral œdema.—For these free venesection followed by intravenous injection of hypertonic saline solution will lessen the severe headache. Lumbar puncture has also the dual recommendations of gauging the height of the increased intracranial pressure, and secondarily for therapeusis. If the cerebrospinal fluid be under a moderate pressure of 200 to 250 mm. of water, 10 to 20 c.c. of fluid may safely be removed. If, however, the cerebrospinal pressure is excessive, great caution should be exercised to avoid a " pressure cone " which may prove fatal.[189] Supervention of more intense headache is an indication to cease spinal drainage. For patients without cerebral œdema, puncture and salines are valueless. Venesection is again of use, with inhalation of amyl nitrite ℥ v,

followed by erythrol tetranitrate gr. ½ by mouth three-hourly until the arterial pressure falls. Liability to further attacks may be diminished by vasodilators in combination with prominal gr. iii, one to two tablets daily by mouth.

Cerebral hæmorrhage should be treated by rest in bed, care of the respiratory airway, emptying the bladder and free purgation. Inhalation of amyl nitrite rapidly reduces blood pressure and causes general dilatation of the superficial vessels while diminishing the circulation through the brain. Lumbar puncture and venesection have their appropriate uses.

In *cerebral thrombosis*, rest, posture and avoidance of effort are important. Drastic purgatives are unnecessary and harmful. The following is a mixture designed to prevent relapse of thrombosis in the sluggish circulation :—

Potassii citratis . . .	gr.xv
Tincturæ nucis vomicæ . .	ℿv
Tincturæ digitalis (standardised)	ℿv
Aquam chloroformi . . .	ℨss. t.d.s. inter cibos.

3. The Renal Group

is differentiated by a history of nephritis—acute, sub-acute or chronic—either preceding the raised pressure, or deducible from evidence derived from tests of renal efficiency or ophthalmic examinations.

Acute Diffuse Nephritis, happening in the course of one of the acute specific infections, notably scarlet fever, or as the result of irritant poisoning, is linked with raised blood pressure due to toxic spasm of the tiny arterioles of the kidney.

During the course of scarlet fever an imminent attack of acute nephritis may be kept at bay by prophylactic active purgation with salines. In severe cases of acute diffuse nephritis, however caused, it is of vital importance to lighten the excretory task of the highly inflamed kidneys as far as possible by promoting the action of the skin and bowels, notably by abstinence from both solids and liquids for the space of a few days, in order to give the kidneys a chance to recover. Thirst can be assuaged by sips of fruit juices or scraps of ice. Should the pressure still persist,

two or three pints of water or weak tea may be sipped in the course of half an hour. Spasm is likely to be subdued by the spate which results from this water test, and coincidentally with the flood of urine the raised pressure falls. If otherwise, recourse may be had to digoxin, lumbar puncture or venesection. The latter two measures should not, however, be lightly undertaken, and sufficient time should always be given to permit the nephritis to subside.

Chronic Nephritis.—In like manner as the volatile acid carried by the blood is eliminated by the lungs as carbonic acid, so the more stable acids are eliminated by the kidneys.

Acid is excreted by the kidney in two forms :—

1. Free—chiefly as phosphoric acid ;

2. Combined—as acid phosphates in combination with ammonia.

The amount of free acid in a given specimen of urine can be estimated directly by titration in terms of decinormal soda. The combined acid is next released by adding formalin, and then the amount can be estimated similarly as free acid.

The results of these two estimations give figures from which can be determined the ratio of (1) to (2), *i.e.*, the acid ammonia ratio, which varies as 1 : 1 (acid) to 1 : 3 or over (alkaline), the normal ratio being as 1 : $1\frac{1}{2}$ for men and 1 : 2 for women.

This ratio is of the greatest importance in assessing the nature of the constitution of any individual, and when combined with a detailed biochemical investigation of the urinary excretion and of the blood yields information of the highest value as to the metabolism of the individual at the time of examination.

Simplified laboratory methods of determining these biochemical reactions have been published by Dr. Henry Ellis,[190] who has advanced the proposition that " acid elimination is one of the main controllers of renal function," and that as such it is an essential determining factor in controlling the height of arterial pressure. On this view metabolism and arterial pressure are co-ordinated and co-related terms. Arterial pressure rises as the need for acid elimination increases. Diastolic pressure thus represents the measure

of tissue tone and blood requirements, whilst systolic pressure indicates the cardiac effort necessary to maintain that tone and to supply the pressure level which the tissues demand. It is important to bear in mind, nevertheless, that renal insufficiency does not ensue until three-quarters of the functional activity of the kidneys has become seriously impaired. As the kidneys fail, waste products accumulate, and increasing effort to rid the system of these necessitates a rising arterial pressure. Thus a vicious circle is created which at first is only temporary, but tends rapidly to become permanent, when the heart hypertrophies, the arteries thicken to meet the increasing hypertonic strain, nycturia and a low specific gravity urine reflect the same kidney strain, and arterial pressure assumes a continuously high level. The heightened arterial pressure due to hypertonia of the kidneys induces a condition of secondary hyperpiesis in relation to the other tissues. This results in the production of arteriosclerosis with the possible risk of hæmorrhage from the weakened vessels of the brain. This hyperpiesis in relation to the cerebral vessels is of secondary origin, hypertonia of the kidneys being primary ; whether arteriosclerosis is due to primary acidosis or to intoxications is uncertain. Probably both these factors take part in its causation.[182, 191, 192] Under such conditions the only way of reducing hyperpiesis is by promoting detoxication, aiding elimination through increasing the functions of the skin and intestines, diminishing the total food-intake, and limiting effort, which favours production of acid, with the object of reducing anabolism. Experience agrees with these procedures.

In hypertonia the presence of a little protein in the urine is not uncommon, and, if associated with a reasonable blood urea, will improve on a diet which is largely vegetarian. Where the presence of chronic nephritis has been established, and the degree of failure to excrete nitrogenous waste products determined by renal efficiency tests, it is not only useless to attempt to lower the indispensably raised pressure by means of vasodilator drugs, but also injurious. In association with nephrosclerosis the adrenals are frequently hypertrophied.[193, 194] Hence endeavours should be made to

diminish adrenal activity and to increase thyroid activity.

General management consists in a quiet life, with avoidance of sources of worry or over-exertion, and residence in a warm house in an equable climate. Clothing should be of silk and wool, and diet light and unstimulating, extractives being forbidden. The cardio-arterio-renal subject should smoke only in the strictest moderation, if at all. Elimination should be fostered by encouraging the action of the skin, bowels and kidneys. The bowels should be kept open by salines, 1 to 2 drachms of equal parts of these sulphates of magnesium and sodium being efficacious, or by hydragogue purgatives, of which jalap in the form of pulv. jalapæ co. is especially serviceable for patients with Bright's disease or uræmia. Perspiration should be encouraged by hot-air or water baths. Particularly injurious is the routine employment of vasodilators, such as the nitrites, and drug treatment in general should be merely symptomatic and of the simplest character.

The patients suffer from the most varied symptoms, which, not being referred by them to the true source, lead them first to consult specialists other than cardiological. The diagnosis is often made first by the ophthalmic surgeon, whilst pulmonary complaints drive them to the lung specialist, and cerebral symptoms to the neurologist.

Hæmorrhages are not infrequent. After middle age epistaxis may prove both serious and severe. In the lesser grades it acts as a safety valve to the embarrassed circulation. Apart from headache of migraine type, giddiness or dyspepsia, it may be the first symptom which causes a patient to seek advice. There is seldom any immediate danger from a first hæmorrhage, even if profuse, since it can usually be stopped, if necessary, by an inflated bag or some form of packing accurately inserted into the nasal fossæ, but occurrence of epistaxis should always lead to the blood pressure being taken, when this will invariably be found in such cases to be very high.

Uræmia results from retention in the blood of waste products usually excreted by the kidneys, and does not supervene until approximately three-quarters of the renal excretory function has been put out of action.

If danger threatens from uræmic coma, catharsis, pilocarpine and hot vapour baths should be prescribed. For uræmic convulsions, inhalations or chloroform, lumbar puncture or venesection are indicated. Delirium and restlessness are best combated by morphia, which is also of special service in the dyspnœa and Cheyne-Stokes breathing of advanced cases.

Control of Eclampsia and Pre-eclamptic States

The mean arterial pressure always increases considerably before and after the attack, even when the maximal and minimal pressures remain normal. A dehydration method advocated by Arnold and Fay [195] in order to control the basic disturbance of water balance leading to cerebral œdema in acute cases of this syndrome has attained considerable success. Bright Banister [196] recommends perseverance along medical lines of treatment in a patient in whom arterial pressure remains steady under 170 mm. systolic, whose output of urine remains good, and whose skin continues to act well even when albumen does not diminish or œdema disappear.

Paroxysmal Hyperpiesis following Eclampsia.—Heitz [197] notes that after eclampsia there may occur every stage between permanent vascular hypertonia and typical "hypertonic crises" in which the blood pressure rises abruptly and returns to normal at the end of the crisis. The majority of such crises spring from a more or less definite condition of spasm which frequently coincides with some attack of severe pain, particularly angina pectoris. Several French and English authors have reported variations of blood pressure, sometimes amounting to 60–80 mm., coinciding with the crisis and terminating with it, and H. Paillard has recorded eight cases of this kind in the course of renal colic. Paroxysmal hypertonia has also been observed in biliary colic, and in the gastric crises of tabes dorsalis. In pregnancy associated with albuminuria or toxæmia, eclampsia is always preceded by a sudden rise of blood pressure. If the patient survives, the blood pressure falls to normal more or less rapidly in the majority of cases. The patients should be kept under observation, however,

since it is often found that ten or fifteen years afterwards there is permanent hyperpiesis with or without albuminuria and evidence of renal disease. It is not so well known that patients who have survived eclampsia, and who exhibit symptoms of hyperpiesis after apparent cure, remain subject to sudden hypertonic crises resembling those of chronic lead poisoning in their sudden onset and the co-existence of secondary cerebral lesions.

Psychical Measures

The modern trend of thought is to ascribe far more importance to the psychical and less to the physical factors in blood pressure. Under the influence of emotion, as we have already noted (pp. 99, 132), most sudden and dramatic variations in the maximal pressure are wont to occur. Patients of nervous and excitable temperament have unstable arterial pressures which sway with every chance wind of circumstance. Such patients should be told about the height of their blood pressure only in general terms, and actual figures should not be stated.

In addition to fluctuations due to momentary excitement, and wide fluctuations which are known to occur in high pressure cases with slight or no impairment of renal function, spastic vascular hypertonia not infrequently is set up by states of worry and apprehension, which lead to establishment of a vicious circle, for not only do continued anxiety conditions tend to the induction of persistently high pressures, albeit with occasional remissions, but a conviction that the pressure is unduly high affords the patient an ever-present source of mental perturbation.

In management of such cases much depends on the personality of the physician. The first step is to allay the patient's fears and to inspire confidence. Reassurance, if accepted by the patient, is of itself a valuable therapeutic measure. A sympathetic talk may have the effect of bringing to light half-conscious worries and anxieties that the patient intuitively keeps to himself, but a special knowledge of psychotherapy is necessary thoroughly to elucidate those hidden complexes which constitute the origins of mental conflicts and resultant strong repression.

Once repressions and anxiety states have been traced to their true causes and these have been removed, a spirit of cheery optimism on the part of the doctor goes a long way towards helping the patient to recover. Psycho-analysis, suggestion and re-education are of service, according to individual needs. Mental and physical repose is to be aimed at, and the environment of personal association should never be lost sight of. It is hardly necessary to point out the far-reaching effects of antagonistic personal association in promoting functional disturbances in general, and sudden leaps in the height of arterial pressure in particular. John Hunter said that his life was in the hands of any rascal who chose to annoy him, and this is also true of many less distinguished individuals.

For certain cases at the beginning rest in bed may be necessary ; for others it is inadvisable. At times drugs are indicated ; more often the patient is better without. A nerve tonic and sedative which I have found useful to allay hyperexcitability has the following composition: ℞ Ammon. brom. gr. v.–viii., Ext. cinchonæ liq. ℥x, Syr. aurantii ʒi, Aq. chlorof. ad ʒss. Sig : ʒss. t.i.d.a.c. If there is a tendency to depression, Tr. nucis vom. up to ℥x may be added.

There is a high pressure group over the age of fifty, liable to periods of anxiety, restlessness and depression, sometimes amounting to melancholia, with a tendency to apoplexy. Both sexes are equally represented.[198] These patients do best on appropriate diet, tepid baths, free daily purgation, and nitroglycerine if more urgent symptoms threaten, though the latter should not be resorted to without the fullest consideration.

As a rule, it is well for nervous patients to have some definite occupation, and, although it may be a good thing to advise them to diminish their total activities, particularly if excessive and productive of worry and strain, complete retirement from business is seldom wise.

Diet

By appropriate diet, proper care of the skin and bowels, and regulated habits of life in hygienic surroundings we can attenuate and in some measure control tendencies to super-

normal pressure. The points to take into consideration are (1) vital energy; (2) nitrogen balance; (3) proper ratio of carbohydrate to fat; (4) vitamins; (5) inorganic salts, and (6) fluid.[143]

Influence of Protein Intake on High Arterial Pressure.—All the most recent work [202, 203, 204] goes to prove that arterial pressure is not raised by the ingestion of moderate amounts of protein. Careful experiments by Strouse and Kelman [152] show that the wide variations that occur in arterial pressure as the direct result of vasomotor disturbances bear no relation to intake of protein food. In such cases no damage to renal function and no increase in non-protein nitrogen or urea nitrogen of the blood was found to follow protein feeding up to 150 gm. daily. In three such cases strong stock soup and coffee given daily did not increase blood pressure. Chronic proteinuria is unaltered by restriction of protein in the diet, so that such patients may be allowed to take eggs, except in acute nephritis, which presents a totally different problem, and in which adoption of von Noorden's method of giving fresh fruit juices and sugar during the first few days is attended with good results. In cases of progressive nephritis with high pressure, diminution of protein intake, sufficient to lower the figures for blood non-protein nitrogen and urea, did not cause lowering of the blood pressure. These experiments add further evidence to that already accumulated to prove the existence of a clinical entity characterised by a primary rise in arterial pressure.[129, 205, 206, 207, 208]

Where the kidneys, however, find difficulty in eliminating waste nitrogenous products, butcher's meat, spiced and seasoned dishes, together with meat soups are injurious, partly because of the tendency to increase intestinal putrefaction with resultant absorption of pressor toxins, and partly by cardiovascular stimulation through the contained extractives. In these respects boiled meats served without the broth are preferable to roast, since boiling to a large extent does away with these principles. Although Newburgh [209] was able to show experimentally that large amounts of meat damaged the kidneys, he found that no blood pressure changes took place.

Experiments of other observers on rabbits have demonstrated that administration of large quantities of protein are not injurious, provided always that a small amount of green vegetables be added to the dietary.

Excess of any kind of food appears to be more harmful than a moderate amount of protein in a well-balanced diet.[152] Flatulence and digestive disturbances often result from substitution of carbohydrate and fat, and these may indirectly augment blood pressure by causing obesity.

"A mixed diet such as the patient is accustomed to, reduced to simplicity and to a quantity adequate to maintain weight and no more (1,500 to 2,000 calories, according to the exercise taken) is best." [210]

Influence of Salt Intake on High Arterial Pressure.—In simple hyperpiesis tedious dietary restrictions are needless, and salt may be taken without fear of raising the pressure to further heights ; if cardiac disease with œdema, renal or cerebral complications are present, salt is then injurious, particularly in large quantities.[211] It should be remembered, however, that many articles of food, *e.g.*, bread, contain sodium chloride, therefore it is hardly ever worth while to prescribe a totally chloride-free diet, which soon becomes very irksome.[212]

Certain patients, often elderly, exhibit marked craving for salt with their meals, and for these a non-pressor table salt may be ordered. Such an one is : ammonium hippurate, one part ; potassium nitrate, four parts ; potassium chloride, twenty-five parts.[33]

Touching diet of high pressure cases in general, my own view is that *far more benefit accrues from lessening the total food intake to the minimum metabolic need than by protein restriction.*

In prevention and treatment of chronic nephritis I have not found dietic measures to be of much avail, and where organic changes—notably renal—exist, it is not wise to hope for too much from a restricted diet, although at times one is agreeably surprised. In less complicated cases, nevertheless, within a few weeks it may be possible to lower a systolic pressure of over 200 mm. to some point below 150 mm., with corresponding improvement in the patient's general condition. In any event, apart from rest in bed,

some time must of necessity elapse before the full effects of lessened intake become apparent.

The following dietary, on lines suggested by Fontaine,[211] is useful in beginning treatment, and has the additional advantage of reducing weight in constitutional obesity :—

Breakfast.—One orange, or one glass of orange juice, or half a grape fruit with a little sugar, or half a melon, one baked apple or two fried bananas, two or three slices of crisp bacon, two thin slices of buttered toast or a rusk, cocoa or a very weak tea or coffee, with a tablespoonful of milk and one lump of sugar.

Lunch.—Fruit only. One apple, or two bananas, or grapes, or one orange, or half a grape fruit.

Dinner.—One moderate helping of lean meat or fish, no potatoes, but two other well-cooked vegetables vegetable or fruit salad, one slice of buttered toast or rusk, no dessert except fruit or a thin slice of sponge cake, a little cream cheese with a biscuit if desired, weak coffee with one lump of sugar and a tablespoonful of milk.

This dietary allows for 0·8 gm. protein per kilo. of body weight for a person of average build, and contains approximately 1,500 calories.

" After a patient has been on a restricted diet until his weight is about normal for his age and height, and has maintained it at that level for five or six weeks, his weight becomes, in a measure, standardised. He may then carefully increase the quantity of each article of food at breakfast and dinner until his total intake is about 2,000 to 2,500 calories without taking on more weight."

While agreeing that restriction of foodstuffs may be useful, it should be borne in mind that the condition of the patient must be the measure of the suitability of the diet prescribed, so that it would be unwise slavishly to adhere to so limited a diet because in selected cases it has been successful. Under-nutrition is as injudicious as overfeeding, because it leads to depressed vitality and secondary anæmia. Any treatment which produces a sense of discomfort in a patient will frequently be more injurious than beneficial, and there are not many who are able to perform much physical or mental effort on a diet yielding only 1,500 calories.

Biochemical Aspects of Diet.—The chief base-forming articles of diet are potatoes, oranges, apples, raisins and bananas, and, since most high pressure cases manifest biochemically a deviation of the acid-base equilibrium to the acid side, these are to be recommended. On the other hand, the chief acid-forming foods are rice, wholemeal bread, meat and eggs, which should all be moderately restricted in hyperpiesis and in chronic intestitial nephritis.

Fluids.—Contrary to the generally accepted opinion that fluids should be restricted because of alleged extra strain upon the kidneys, recent experiments on normal subjects and on persons with high arterial pressures have shown that addition of from 2 to 4 pints of water to the usual daily consumption is followed by a distinct fall in both pressures. Neither rate nor force of heart beat is increased, and the decreased pressures are maintained for some time after the extra water has been excreted.[213] Definite results have been obtained in a large proportion of high pressure subjects, reduction from excessively high systolic pressures to normal, maintained over several months, having taken place presumably through washing out of intermediate metabolites which may be of protein origin. This is in accordance with results obtained in spa treatment, where several litres of water are imbibed daily, with the result of flushing out the kidneys, and so of getting rid of the pressor bodies that cause vasoconstriction and heightened pressures.

PHYSICAL MEASURES

1. Rest

The value of absolute rest, both mental and physical, in effecting sure and speedy improvement can hardly be overestimated. Apart from other treatment, rest in bed at the outset for two to three weeks under conditions of complete relaxation and low diet will often suffice to bring down an exalted pressure to a level which, if not normal, is, at any rate, less hazardous. At the same time untoward symptoms are relieved. Subsequently, it is advisable for ten to twelve hours out of the twenty-four to be spent in bed, with half a

day on Sunday. In severe cases rest may advantageously be combined with purgation, daily warm baths of ten to fifteen minutes' duration at 93° to 98° F., massage and passive movements. Absolute recumbency has a sedative action on the over-driven and irritable left ventricle, which is largely responsible for keeping up pressures which are unduly high. The length of time to be spent in bed may vary from a day or so a week to a month or six weeks at a time, after which the daily affairs of life may be *gradually* resumed and continued in the absence of symptoms, though frequently with the caution that for the future it will be necessary to lessen the totality of work previously performed, and to be content to live on a lower plane of activity than before. Patients who lead active and busy lives are not always amenable to bed treatment, particularly if conscious of no symptoms and anxious about their livelihood, so that, as ever, one has to be guided by individual circumstances and advise accordingly.

Sometimes rest fails to lower the pressure, although it usually alleviates the symptoms.[214] This point, as has already been stated, is helpful in diagnosing between a simple high pressure and one that is complicated.

2. Exercise

Such kinds of exercise as permit of mental relaxation and repose should be prescribed. Thus a moderate amount of regular and daily walking on the level is beneficial. One may also encourage gardening, gentle cycling on low gradients, golf, and, in suitable cases, riding, in accordance with Lord Palmerston's aphorism that "the best thing for the inside of a man is the outside of a horse."

The quantity of exercise should be graded according to the type of high blood pressure present, care being taken always to avoid over-exertion and fatigue. Patients who manifest hyperpiesia in conjunction with alimentary toxæmia or acidosis have a tendency towards inaction, lethargy, flabbiness and effort dyspnœa, and should be roused to take moderate daily exercise, which directly helps to lower pressure by preventing accumulation of pressor substances

in the blood, and by promoting peripheral dilatation and perspiration. On the other hand, many arteriosclerotics, particularly in advancing years, are stimulated by their high pressures to disproportionate feelings of fitness, sense of power and readiness for activity on both physical and mental planes. In contradistinction to the lethargic type, these patients are restless and irritable, always wanting to be " up and doing," and possessing a more or less obvious strain of wilfulness and obstinacy which hinders them from following advice which in their inner consciousness they know to be salutary. Such persons not infrequently provide striking object-lessons in the shape of dramatic events, such as bleeding from ruptured vessels, sudden cardiac failure or sudden death during slight operations.

Many fatalities have occurred through cerebral hæmorrhage induced by straining at stool. Hence, all sudden or severe muscular exertion should be forbidden, such as running for trains, jumping on moving vehicles and the like. Stair-climbing should also be reduced to a minimum.

As regards exertion, as a general rule, when a man reaches forty years of age he should begin to be cautious ; by the time he has reached sixty years of age it behoves him to be still more cautious ; and, if between or over these ages, he has a pressure which is rising, it is wise for him to forego all games or pursuits which necessitate strenuous or prolonged effort, and to confine himself to such as are within his limits.

So many men who have been deprived of the pleasures of sport and pastime during their sedentary and hard-working years, when released from the tide of business affairs, enter with keener zest upon the games habitually played by younger men, and in stress of competition are apt to overstep the bounds set by the changes of involution and chronic inflammation in their tissues. Warning signals are anginal pains and effort dyspnœa.

3. Deep Breathing Exercises

As I have frequently pointed out elsewhere, in order of importance among the muscles of the body the diaphragm ranks only second to the heart. It is the chief and most

powerful muscle of respiration. Even during deep inspiration the curvature of its domes hardly flattens in descent, for it contracts downwards and forwards after the fashion of a true piston.[215]

The aspiratory force put into action by enlargement of the thorax, extension of the vertebral column and descent of the diaphragm is of considerable moment; in fact, so great is the effect produced that the pumping action of the diaphragm has been likened to that of a second heart. In addition to favouring the flow of blood through the heart, the diaphragm, by its action as compressor and tractor, exercises a powerful influence upon the venous and lymphatic circulations, thus materially aiding the processes of metabolism and elimination.[216]

Supernormal arterial pressures are reduced, and symptoms alleviated by deep breathing exercises, "the mechanism of the natural complete breath" being practised slowly and fully in the recumbent posture, special attention being paid to securing efficient expansion of the chest in all three diameters. In reduction of pressure the full inspiratory phase is important, the effect being largely mechanical, and due to suction by reason of the negative pressure induced. The arteries and heart become compressed, and arterial pressure is thereby lowered with each inspiration.[217] Biochemical changes also result by displacement of carbon dioxide by oxygen, thus lessening cyanosis and dyspnœa and promoting peripheral vasodilatation.*

4. Relaxation Exercises and Passive Movements

In arterial hypertonus with spasm, respiratory exercises may advantageously be combined with relaxation and passive movements. After seeing that the clothing is loose, and that all sources of irritation are eliminated, the patient is instructed to recline comfortably at full length, muscular hypertension and spasm being gradually overcome by passive

* The expiratory phase is of equal value in reducing high pressures through elimination of respiratory and gastro-intestinal toxins by induction of efficient contraction of the anterior abdominal wall and lower ribs, followed by contraction of the upper ribs, and alternating with full inspiratory lateral expansion of the chest, especially at the apices—a "concertina-like" action.

movements, beginning with the fingers, which are alternately flexed and extended passively, then taking wrist, elbow and shoulder joints in order, the patient being instructed to allow the limbs to remain lax. Next the lower limbs are similarly moved, and then the neck and trunk, so that finally the patient acquires the power and habit of muscular relaxation which he can daily practise at will. When much nervous hyper-excitability coexists, three or four sittings may be required to achieve complete relaxation. If the blood pressure is taken before and after the exercises, a fall will be seen to occur in both pressures, which will by degrees attain a lower level owing to inhibition of the psychic impulses which tend to vasoconstriction.[217, 218, 219]

5. Resistance Exercises

In supernormal blood pressure associated with cardiac insufficiency, especially the rheumatic or gouty mitral types, regulated resistance exercises after the "Schott" method form a useful adjunct to Nauheim baths, if care be taken gradually to increase the resistance while noting the pulse rate, and decreasing the resistance and the number of exercises if the patient begins to exhibit signals of distress, which are rapid pulse and breathing, sweating or cyanosis.

6. Massage

over considerable areas at first raises arterial pressure; then by redistribution of the blood and increasing velocity the pressures fall to normal or less. Norris [220] recommends good general massage combined with Swedish movements as *one of the most useful measures* in the treatment of raised arterial pressure. To a less extent, vibratory massage is also beneficial, especially if followed by rest and relaxation for an hour afterwards.

Vigorous massage of the abdomen is, however, contra-indicated in cases of hyperpiesis associated with toxic absorption from intestinal stasis, lest the massed toxins thrown into the circulation by such procedure promote a fatal issue in an already debilitated or elderly individual.

7. Diaphoresis

Diaphoresis is conveniently attained by the use of the *foam sweating bath*, made by adding to 1½ oz. of foam-producing extract 4 inches of water at a temperature of 116° F. Compressed air is introduced through a distributor, which produces a gradually rising mass of white foam. When the bath is half filled with foam, the temperature of the water is reduced to 102° F. while that of the foam is about 98° F. The patient, recumbent in the bath, is covered with dense foam up to the chin. When the bath becomes moderately full of foam, the air compressor is switched off, while the temperature of the water can be raised (or lowered) as desired. Profuse perspiration soon occurs, the patient remaining in the bath up to half an hour. The foam acts as an insulator, so that body-heat is pent up, metabolism is stimulated, oxygen intake increased, and dilatation of superficial blood vessels occurs, causing arterial pressure to fall and the heart-load to become much reduced. Hence foam baths are of value when hyperpiesis is combined with arteriosclerosis or myocardial defect of not too advanced grade.

Diaphoresis is also of value in hydræmic cases, which derive benefit from the judicious employment of hot air, foam or vapour baths. Sufficient sodium chloride is thus excreted through the skin to ease the retention of salt in the tissues, and so to promote disappearance of œdema.

8. High Frequency and Diathermic Currents

For several years the author[221] has advocated electrical treatment carried out by recognised experts for cases of hyperpiesia, and of hyperpiesis in which no evidence exists of cardiac defect, fully developed arteriosclerosis or chronic nephritis. The effects are variable; in some patients symptoms abate without permanent reduction of arterial pressure, in others reduction, with or without relief of symptoms, persists after termination of the course of treatment. It may be that the original cause is not even influenced by the electric currents, and that only contributory causes are thereby minimised. Be this as it may,

in the words of Allbutt: "d'Arsonvalisation is the most valuable immediate aid we possess for hyperpiesia," and this experience is confirmed by those who practise this ancillary branch of Medicine.

Especially in early stages of hyperpiesia with gradually rising pressures will this procedure be found useful in causing relief or disappearance of the indefinite but frequently troublesome symptoms associated with disturbance of vasomotor equilibrium and eliminatory difficulties due to defective metabolism.[221, 222, 223]

Dr. Howard Humphris[224] believes that "the treatment will check the degenerative processes that are going on and will ward off inevitable consequences, such as cerebral hæmorrhages, renal affections, and the myriad ills which follow in the train of arteriosclerosis. Though electricity may be no cure, it will keep a patient for years in a condition of safety, and, indeed, in a state of comparative comfort."

High frequency currents are applied generally, either by induction or condensation (d'Arsonvalisation), or locally to the central nervous system—diathermy.

They are unsuited to cases with gross arterial, cardiac or renal involvement, in which there is failure to respond to electrical excitation, and arterial pressure stays high. They are, however, specially indicated in simple hypertonia with defective metabolism, where they relieve vascular spasm through their action in diminishing peripheral resistance and stimulating cardiac energy. Hence, to attain the best results, the arteries must not be too thickened, the heart muscle must be fairly efficient, and the kidneys reasonably permeable. Provided that these conditions obtain, high frequency currents often produce an initial fall of 10 to 20 mm. Hg with increased urea output, followed by increased excretion of total nitrogen. The pressures again rise, but not to the same height, and, after several treatments, may fall gradually to a lower and more permanent level. Daily applications are advisable until no further reduction is effected. Subsequently treatment should be given at such intervals as will maintain the pressures at this low level. Diathermy causes a fall in both pressures, and increase in leucocytes, urea and ammonia nitrogen.[225]

9. Ultra-Violet Rays

The author's experience of the effects of ultra-violet irradiation leads him to believe this method to be efficacious in many cases of pure hyperpiesia and of hyperpiesis.[226] Not all hyperpietic pressures are, however, reduced by this treatment, although disappearance of symptoms and general improvement in health frequently ensue. Beneficial results which follow in appropriately selected cases are due to penetration of the rays to the blood-capillary layer of the skin. Oxidative effects are thereby produced upon the circulation and upon special kinds of metabolism. With accurately adjusted doses these effects are tonic in character and the patient experiences general sensations of elation and well-being. The rays are usually applied by means of the carbon arc with gradually increasing exposures. For systemic effects the author has found the carbon arc lamp of greater value than either the mercury or tungsten arcs. Dr. Percy Hall [227] recommends a combination of diathermy with ultra-violet radiation, and claims reduction of both diastolic and systolic pressures by this means, symptoms disappearing after about six weeks' therapy, and not tending to relapse, provided that the patient leads a healthy life and avoids excess of food and alcohol.

10. X-Rays

X-rays lower arterial pressure, especially in high pressure cases, the reduction being proportional to the dosage.

Decrease is practically constant immediately after exposure, and persists for several days, sometimes even for weeks. In a case of hyperpiesis a reduction of 30 mm. was manifest after two months. In this country the method has attracted little attention.

11. Lumbar Puncture

Withdrawal of cerebrospinal fluid by lumbar puncture is promptly efficacious in warding off or relieving uræmic con-

vulsions or severe headache. The fluid should be allowed to escape until the rate of drip is about half that of the pulse rate.

In cerebral hæmorrhage lumbar puncture may be tried, but patients nearly always die within three days, sometimes within a few hours. If survival takes place, the case is almost certainly one of cerebral thrombosis or angiospasm.

12. Venesection and Fasting

In refractory cases venesection constitutes an important addition to our armamentarium. In crises it affords speedy and direct relief to an embarrassed circulation and dilated left or right heart by withdrawal of fluid. Section of the vein further causes a reflex action in the shape of dilatation of the vessels, especially those of the kidneys. Venesection can be repeated with advantage in all forms of renal disease associated with raised blood pressure, and in all conditions of venous stasis and acute pulmonary œdema. To ensure reduction of pressure the blood must be drawn *rapidly* in sufficient quantity. "Rapid venesection, amounting to from 300 to 500 c.c., will generally reduce blood pressure from 5 to 30 mm. . . . The symptomatic relief afforded is sometimes immediate, even before an appreciable quantity of blood has been withdrawn." [228]

Venesection of itself constitutes merely a temporary remedy, the volume of the blood being soon afterwards restored to its original level. A more lasting effect in reduction of arterial pressure and in circulatory relief is attained by a combination of blood-letting with starvation for twenty-four to forty-eight hours or more. The length of time should be assessed by the general and circulatory condition of the patient, age and other factors being duly taken into account. During the fasting period, nothing should be taken except water, weak tea or fresh fruit juices, four-hourly if required. Return to solid food should be gradual. In cerebral hæmorrhage, venesection, by reducing systemic pressure, is harmful, whereas lumbar puncture, by reducing intracranial pressure, has a theoretical utility.

13. Drug Therapy

Detoxication Treatment of Intestinal Toxæmia

A long and considerable experience of hyperpiesis disposes me to believe that the majority of cases are largely dependent upon, and symptomatic of, toxæmia, especially of intestinal origin, and are in most cases amenable to properly instituted therapy. If the intestines are at fault, the procedure should be as follows :—

Deal with any *intestinal stasis* and eliminate indigestion and flatulence from this source by regulating the diet (*q.v.*), by aperients—in the milder cases senna pods, taxol or liquid paraffin; in the more obstinate ones by petrolagar with phenolphthalein or agarol.

Purgatives.—*Calomel.*—At the outset, but not for prolonged administration, calomel may be employed in doses of 1 to 2 grains at bedtime twice weekly for its eliminative effect, or in tiny doses of $\frac{1}{20}$ to $\frac{1}{10}$ grain twice or thrice repeated for its alleged antiseptic effect, followed next morning by $\frac{1}{2}$ drachm each of the sulphates of sodium and magnesium. Probably the effects of calomel are due to (1) its ability to cause relaxation of the arterial walls, (2) its stimulating effect upon the intestinal mucosa, and (3) its indirect cholagogue properties in reflexly causing the gall bladder to contract and so release an increased amount of bile. "Though the bile salts are weak antiseptics, the bile itself is readily putrescible, and the power it has of diminishing putrefaction in the intestine is due chiefly to the fact that by increasing absorption it lessens the amount of putrescible matter in the bowel." [229]

To promote depletion of the vascular system, and to rid the bowel of toxic products of fermentation and putrefaction, when the pressure is very high, daily catharsis is necessary, with calomel or elaterium and/or sufficient morning doses of magnesium and sodium sulphates in equal proportions. In less severe cases smaller doses are indicated, and daily administration may not always be required. In purely toxæmic cases great benefit is usually experienced from this mode of treatment, since absorption of intestinal toxins is prevented, and blood pressure falls gradually

by 30 to 50 mm. or more, with corresponding subjective feelings of buoyancy and rejuvenation.

For the *insomnia* due to raised arterial pressure associated with advancing years and indigestion or constipation, 2 to 3 grains of calomel followed by a saline aperient next morning, is usually far more beneficial than hypnotic drugs.

For *intestinal catarrh and bacillary disturbances*, the iodine-containing yatren-105, orally in pills (0·25 gm. 1 to 4 daily), or by bowel lavage, is of distinct utility. For *sphincteric spasm* due to catarrh, tincture of belladonna, ℥5 may be given three times a day. Colloidal kaolin and the various preparations of wood-charcoal are of additional value in counteracting *putrefactive processes* within the intestinal canal. If the lower ileum is loaded with septic material, it can be cleaned by saline aperients. Should investigation reveal the presence of pathogenic organisms, such as serum-resisting streptococci or serum-resisting *B. coli*, an autogenous vaccine may be given.

For reducing arterial pressure in cases of gastro-intestinal stasis and catarrhal conditions, such as mucous colitis, rectal injections, administered after the method of detoxication advocated by Dr. H. W. Nott,[230] daily for three weeks, followed by oral administration of cachets of potassium permanganate gr. $\frac{1}{8}$ to $\frac{1}{4}$, combined with desiccated thyroid, gr. $\frac{1}{4}$ to $\frac{1}{2}$, are frequently efficacious.

The solution of potassium permanganate is prepared by crushing to powder in an earthenware basin a 1-grain tablet of pure fresh potassium permanganate ; cold water is poured on to it so that it will just dissolve, and hot water added to make $1\frac{1}{2}$ pints of standard solution.

To begin with, about $\frac{1}{2}$ pint of this solution is injected very slowly by means of a rectal tube and funnel, the patient lying on the left side. Should the bowels not have acted sufficiently beforehand, the first $\frac{1}{2}$ pint is to be treated as a wash-out and a second administered, the whole object of the procedure being that the permanganate solution should be retained. The above is the average amount for an adult, but, according to age, from 3 oz. to a pint may be put in on each occasion and the strength of the solution doubled if necessary. Even if, as occasionally happens, the height

of arterial pressure shows no reduction, any troublesome symptoms present often abate or disappear.

Another good local method is found in Plombières lavage, which at a spa may be conveniently employed in combination with hydro- and balneo-therapy, particularly for cases of mucous colitis with nervous symptoms.

Intestinal Antiseptics.—The lack of a trustworthy intestinal disinfectant has long been felt. For many years doubt has been expressed as to the possibility of disinfecting the interior of the alimentary tract without at the same time injuring either the mucosa or the myriad flora which carry on the normal processes of food disintegration. Unhappily, most of the so-called intestinal antiseptics and disinfectants fail in accomplishing what they are supposed to do, and of the numerous ones which I have tried in cases of high pressure associated with long-standing intestinal intoxication, there are very few which are of any practical value. These are (*a*) thymol, for short administration by mouth or rectum, (*b*) potassium permanganate, which can be continued for as long as is required.

Thymol, extracted from oil of thyme, is of most service in cases attended by much putrefaction in the large intestine, with formation of pressor amines, and in which the stools possess a very offensive odour. Given in pill form in doses up to 2 grains thrice daily or in a single massive dose of 5 grains, as preferred, together with elimination from the dietary of meat proteins which give rise to putrefaction within the intestine, this drug will cause the odour to disappear often within twenty-four hours with marked relief of symptoms. Administration should only be in short courses.

Note.—Since thymol is freely soluble in alcohol, during its administration it is imperative strictly to warn the patient to avoid alcohol in any shape or form, otherwise symptoms similar to those produced by phenol poisoning will ensue. Apart from this, in doses such as suggested it has no ill-effects.

Dimol, a phenol derivative, in 1 grain tablets, two to four with water thrice daily after meals, in certain cases of hyperpiesis associated with intestinal toxæmia, appears to exercise a depressor effect.

Garlic.—A depressor in the form of fresh cloves of garlic, well peeled and macerated in spirit, has been advocated.[251]

It possesses also cardiotonic properties. Twenty to thirty drops are administered in a single dose for three to four days, with five days' interval. Preparations of oil of garlic, best given in the form of pills, are sometimes efficacious in hyperpiesia originating from intestinal stasis with absorption of pressor amines.

Benzyl benzoate has little action on arterial pressure in normal subjects. In hyperpiesis a systolic fall of from 10 to 20 mm., and a diastolic fall up to 10 mm., occur usually within an hour after administration of 25 drops of a 1–20 solution, night and morning, but the result is usually transient, and does not exceed forty-eight hours even if dosage be maintained. No deleterious effects have been observed even when given three or four times daily in doses of 10 to 30 minims in water, or as an emulsion over long periods. At the end of a week a continued fall in pressure justifies reduction of the dose by a third or a quarter. Headache, giddiness, numbness and vascular pains are speedily relieved. Soon after discontinuance of the drug, however, the pressure returns to its original height. In simple hyperpiesis with systolic pressure of 250 mm. or more, benzyl benzoate in large doses, up to 120 drops a day, has a more definite, and sometimes a lasting, effect, causing a fall up to 50 mm. in six days. In angina, benzyl benzoate is less efficacious than amyl nitrite or trinitrin, and is inoperative in high pressures complicated by aortitis, nephritis or confirmed insufficiency of the left ventricle.

Respecting other drugs which are stated to exercise antiseptic action in the alimentary tract, "their name is legion, for they are many." A large number of them are too caustic, toxic or unpleasant to be used in doses sufficient to secure the desired internal disinfectant action, while the remainder are liable to decomposition by the digestive juices and are therefore inert.

Vasodilators

The Nitrite Group.—Routine and unintelligent attempts at lowering supernormal pressures by means of the nitrites are strongly to be deprecated. Their habitual employment

cannot be defended as either radical or rational, since in action they are evanescent, and in removal of underlying causes of high pressure they are futile.

All the members of the group have a family likeness, differing only in the rapidity, amount and duration of their effects. Hence they should be used for a short time in full doses, so as to produce their appropriate action in emergencies. As temporary measures the ones in most common use are amyl nitrite, ℥ iii.–v., in capsules for inhalation to relieve the pain of angina or to ward off threatened apoplexy or heart failure from excessive arterial pressure. Nitroglycerine gr. $\frac{1}{50}$ may be prescribed for internal administration on like occasions. As a general rule it is inadvisable to employ vasodilator drugs in early and mild cases. Amyl nitrite and nitroglycerine both cause a sudden but transitory reduction of pressure. If a slower, gentler and more sustained action is desired, erythrol nitrate gr. $\frac{1}{4}$ or sodium nitrite gr. $\frac{1}{2}$–3 may be given thrice daily either alone, or in a simple digestive mixture, or in combination with diaphoretics or diuretics, in order to lessen the circulatory load. Where raised arterial pressure is associated with arteriosclerosis or chronic interstitial nephritis, a combination of nitroglycerine gr. $\frac{1}{200}$ with sodium nitrite gr. i. and lithium hippurate gr. ii. is often successful in bringing about a speedy reduction. This gives time for the various general measures detailed above to be adopted. The most efficacious preparation is one suggested by the late Dr. George Oliver :—

℞.

Sodii nitritis	gr. ss.
Erythrol nitratis . . .	gr. $\frac{1}{8}$
Mannitol nitratis . . .	gr. $\frac{1}{4}$
Ammonium hippuratis . .	gr. i.

M. Ft. tabellæ. Sig. One or more tablets thrice daily.

In certain cases erythrol nitrate causes severe headache, so that it is wise not to increase the dose too rapidly. A favourite formula on the part of some is to combine sodium nitrite gr. i with sodium iodide gr. iii, or with liq. trinitrini and sp. æth. in the case of children, who are very susceptible to nitrites.

Substantial lowering of pressure by the nitrite group, up to 60 mm., or by venesection does not impair the urea-

concentrating function of the kidney, nor in most instances is the total excretion of urea lessened.[231] Compare with this Major's [232, 233] observations on guanidine, and those of Gruber, Shackelford and Ecklund [234] on phenobarbital and phenolsulpho-naphthalein excretion.

The Halogens

The Chloride Group.—The action of this group depends mainly on the chlorine ion, which, on liberation from a chloride, unites with the blood alkali. The alkaline reserve of the blood is thus diminished, so that where acidosis prevails, as in advanced cardio-arterio-renal patients, all of whom exhibit an acid-alkali ratio of 1 : 1 or less, this group should not be employed.

With the lesser grades of hyperpiesis I have obtained encouraging results by administration of 2-drachm doses of potassium chloride, made up with ½-oz. each of the syrups of orange and tolu. This should be given half an hour after meals, in a tumblerful of water, thrice daily. The potassium ion is helpful in reduction of œdema, and the chlorine ion administered in sufficiently large doses will often bring down an excessively raised arterial pressure by 20 to 40 mm. S. and by 5 to 15 mm. D. Calcium and potassium chlorides have been reported upon favourably by Addison and Clarke,[235, 236] who treated forty-five cases of hyperpiesis of all grades. Only those cases were considered to react in which a continuous lowering of S by 30 or more mm. and of D by 12 or more mm. was observed. Of the forty-five cases, twenty-six (57·7 per cent.) reacted with calcium chloride, and six (13 per cent.) with potassium chloride. Potassium chloride was only substituted for calcium chloride in the same doses of 2 to 4 drachms thrice daily where at the end of a month's time no fall in pressure had occurred with the latter salt.

In my experience, however, potassium chloride is to be preferred to either calcium or ammonium chlorides. Calcium chloride has a less range of efficacy owing to its liability after a few days to cause reaction with nausea or troublesome gastro-intestinal irritation and an unpleasant after-taste, which is difficult to disguise or get rid of. Ammonium

chloride produces somewhat similar results, but is not recommended, as it is still more unpleasant to take.

Hyperpietics with a low blood calcium content are best treated by intramuscular injections of calcium gluconate, alternating with parathormone by the same route if necessary. Amelioration of the general condition is the rule, accompanied frequently by reduction in the levels of arterial pressure. In arteriosclerotic hyperpiesis the blood calcium figure is high and should be balanced by oral administration of acid sodium phosphate gr. xx. t.d.s. or diacid potassium phosphate gr. iii. to xv., twice to thrice daily after meals in accordance with the tolerance of the patient. The hypertonic group with continuously raised arterial pressures benefit from administration of theobromine-calcium-salicylate gr. viiss., with or without potassium iodide gr. iss., thrice daily between meals. A rapid drop in arterial pressure frequently takes place, lower levels being maintained with coincident improvement in the general condition. Courses of ten to fourteen days' treatment can be given at two or three weekly intervals over several months. If the renal tissues are too greatly damaged the initial fall of pressure is not maintained, and the continuously high level is resumed as soon as exhibition of the drug is discontinued. Such cases justify an unfavourable prognosis.

The Iodide Group and Iodine.—Though frequently employed, iodides have apparently little direct effect on arterial pressure. When this is raised as the result of plumbism or syphilis they are undoubtedly of benefit, and should be administered over long periods with occasional intervals. Any effect is probably due rather to the potassium ion, in the case of potassium iodide, than to the iodine ion, except in the rheumatic diathesis in which iodides are often of use. It should always be remembered that iodism is more likely to occur in susceptible subjects with small doses than if doses of not less than 10 grains are administered. A course of tinctura iodi sine pot. iod. ℥iii.–v. daily in milk after the mid-day meal, iodo-protein compounds or colloidal iodine in doses of ℥xx.–lx. thrice daily is less liable to cause gastric disturbance or iodism than the iodides themselves.

Tiodine (iodine 47 per cent., sulphur 12 per cent.), 1 c.c.

intramuscularly or subcutaneously on alternate days, may be given with advantage in the earlier phases of arteriosclerosis and myocarditis attended by high arterial pressure.

The Bromide Group.—In arterial spasm, after removal of toxic causes if any, bromides, especially ammonium bromide combined with triple valerianates, are of great service, and are best given in small doses.

Nervous subjects whose high pressures are associated with disturbances of the central nervous system, communicated in turn to the sympathetic system which is in a state of undue excitability, in general find considerable relief from bromides. Of these the ammonium and potassium salts are to be preferred in 5-grain doses thrice daily before meals, either alone, in combination with nerve tonics or with alkaline digestives.

In arterial spasm, after removal of any possible toxic cause, bromides are of much service. Emotional and apprehensive subjects benefit by the addition of triple valerianates to small doses of bromide.

For insomnia of nervous origin, particularly when associated with headache and sensations of vertico-occipital oppression or pain in the nape of the neck, chloral hydrate with potassium bromide aa. gr. xx. to xxx. is strongly to be recommended.

Other Nerve Sedatives.—Where sympathetic instability is evidenced by considerable systolic fluctuation of the mercury column or oscillometric needle at the first, one or two pressure readings, luminal in small doses, ½ to 3 grains daily, has a good sedative effect. By the addition of 5 grains of sodium biborate, the beneficial results of luminal are greatly enhanced. Chloral hydrate, 5 to 15 grains, one to three times daily, is of great value as a sedative.[237, 238]

The Sulphocyanates.—Chemically pure potassium or sodium sulphocyanate is successful in some cases of hyperpiesia,[239, 240] in dosage of 2½ grains with syrup of orange ℥i and water to ½ oz., thrice daily after meals for the first week, twice daily for the second week, and once daily for the third week, but has not justified its early promise. Thereafter the dose is repeated daily, or on alternate days.

Bismuth Subnitrate.—This drug acts by virtue of its property in interrupting the vicious circle of vascular fatigue and excessive irritability,[241] leading to further spasticity and fatigue, and thus promoting physiological rest. It is most valuable where arteriosclerosis is not great. The mode of action is by the breaking down of nitrate into minute amounts of nitrite ions which are continuously absorbed and cause gradual vascular relaxation and reduction of the blood chloride content by causing a chloride diuresis. Average dose 10 to 15 grains.

Misletoe.—This drug in various preparations has been recommended, chiefly by the French school, but I have seen no permanent effects in pressure-reduction from its use. Temporarily to alleviate the cerebral symptoms of hyperpiesis it has been advocated by O'Hare.[242] The extract may be given by intramuscular injection (5 cg. night and morning) or in pills (15 cg. night and morning). Misletoe lowers the systolic pressure by reducing the force of ventricular systole during and for a few days after cessation of treatment, but has little or no effect upon the diastolic pressure. Like the nitrite group, its action is but transient, and the systolic pressure tends to resume its original height within a few days after dosage has been discontinued.

Alkalis and Stomachics.—These are useful in aiding reduction of high pressure by their antacid action in relieving gastric fermentation. "The ideal gastric antacid is a substance which can neutralise hydrochloric acid, but in excess cannot make the stomach, and much less the tissues, alkaline, and which is excreted, unchanged, in the intestines and not in the urine."[243] Such substances are the tribasic phosphates of magnesium and calcium, which can usefully be combined with other buffer salts such as magnesium citrate and sodium diphosphate as a pleasant effervescent powder of considerable value in the treatment of hyperchlorhydria. The free acidity of the gastric contents is depressed with little change in the combined acidity, thus remaining in a very slightly acid state of equilibrium.

An alkaline carminative mixture containing sod. bicarb. gr. 15 to 20 with tinct. card. co., chloroform and gentian, to which a little rhubarb may be added if desired, often works

wonders in keeping down a persistent hyperpiesis of alimentary toxæmia type. Sodium bicarbonate and calomel in powder are also specially serviceable for thinning the bile.

Acids.—In the case of patients who exhibit raised arterial pressures, but who, nevertheless, on investigation of the urine, show a ratio of free to ammonium acid which is greater than 1 : 2, together with clinical manifestations of chronic gastro-intestinal disorder, cautious administration of acids, beginning with fresh fruit juices of orange and lemon, and proceeding to dilute phosphoric acid, is often beneficial. Dilute hydrochloric acid in doses of 1 to 2 minims at the outset, gradually increased if such can be tolerated, or, still better, betaine hydrochloride with pepsin, have the effect of changing into harmless bodies the toxic nitrogenous pressor amines absorbed from the intestinal tract. In this way it is possible to bring down an excessively high blood urea to within normal limits. Great care is, however, requisite in embarking on such lines of treatment, since a proportion of these biochemically "alkaline" gastro-intestinal subjects are acid-sensitive, and may react in alarming fashion even to tiny doses of mineral acids. If acid-sensitiveness can be overcome, which in the majority of cases is possible by careful management, as tissue alkalinity decreases so symptoms disappear and the patient feels better than for a long time previously. Chloride of ammonium in doses of 10 grains thrice daily is a valuable acidifying agent, and is often well tolerated when dilute hydrochloric acid is not.

Organotherapy

Pancreatic Extracts. — *Vagotonine.* — Of substances extracted from the pancreas which have been employed in control of high arterial pressure, the most promising is vagotonine, recently isolated in pure form by Santenoise,[110] of Nancy.

Physiological Properties.—Vagotonine is a true hormone, excreted normally by the pancreas, and poured by its emissary veins into the blood, in which this hormone has been found in an amount physiologically active. It is essentially a regulator of the circulation, as evidenced by the following effects observed by Abrami, Santenoise and Bernal :—[244]

(1) *On Vascular Tonus.*—It augments the reflex excitability and tonus of the parasympathetic (extended vagus) centres, and lessens secondarily and progressively the excitability of the sympathetic system, *i.e.*, while causing an increase of vasodilator tone, it diminishes vasoconstrictor tone.

(2) *On Cardiac Rate.*—By increasing vagal tonus, which is normally cardio-moderator, it slows the heart rate.

(3) *On the Blood.*—Modifications in the physico-chemical state of the blood take place ; (*a*) a rise of *p*H, with diminution of the alkaline reserve, (*b*) changes in the blood content of calcium and potassium, calcium usually diminishing while potassium is increased.

(4) *On Adrenaline.*—Intravenous injection of vagotonine prior to one of adrenaline inhibits the secretion of adrenaline and lessens its activity.

Therapeutic Indications.—By its combined action, vagotonine exerts an important influence both upon control of cardiac rate and vascular calibre. Strictly speaking, it is not a depressor substance, but a hormone which normally controls the functional activity of the arterial pressure-regulating mechanisms. From depressor agents causing reduction even in normal subjects, vagotonine differs by manifesting reduction of arterial pressure only when hyperpiesis is linked with disturbance of regulation corresponding with the amount of pressure rise. Hence vagotonine has a known action, established by numerous experimental proofs and confirmed by clinical studies, which renders it of more definite value than many extracts whose action can only be surmised.

In functional disorders not dependent on regulating mechanism, as well as in conditions which inhibit a response of the nervous centres involved, vagotonine is ineffective.

Administration.—Vagotonine is injected subcutaneously in daily dosage of 0·02 gm. during a period of about three weeks. A more rapid effect is attained by intravenous injection of $\frac{1}{40}$ to $\frac{1}{10}$ mgm. Reduction of arterial pressure is delayed for half an hour or so, gradually increases, is prolonged, and never followed by a hyperpietic reaction, such as may follow other tissue extracts.

In certain cases improvement persists after vagotonine

has been discontinued. Good results have also been obtained in paroxysmal tachycardia, hyperadrenalism and hypersympathicotonia. Out of eighty patients with hyperpiesis of various kinds, Abrami, Santenoise and Bernal [244] found that in thirty a lasting fall of arterial pressure followed a series of injections of vagotonine. It was successful in cases influenced only slightly by bleeding, lumbar puncture, injections of acetylcholine and inhalations of amyl nitrite. Conversely, cases irresponsive to vagotonine reacted to subcutaneous injections of methyl-acetylcholine, which accords with my own clinical experience. If the patient shows no lessening of arterial pressure two hours after the first test injection, it is useless to persevere with this remedy. If only an appreciable fall occurs, vagotonine should be continued, and I have found it of considerable value.

Other Pancreatic Extracts, Depressor in Action.—These are : *angioxyl*, the insulin-free pancreatic extract of P. Gley and Kisthinios,[245] administered subcutaneously in doses of 2 c.c. daily ; *kallikrein* (*padutin*), the " vasomotor hormone " of Frey and Kraut,[246] of complicated structure and very unstable to heat, acids, bases and alcohol, used in cases of arteriolar constriction either by intramuscular injection of 1 unit twice daily for three days, afterwards 2 units twice daily, or orally in doses of 3 to 4 units thrice daily ; *insulin*, given in hyperthyroidism and cardiovascular affections subcutaneously, the dosage being measured in units. A series of chemical and physiological properties differentiates it from vagotonine, and when freed from vagotonine it never augments parasympathetic excitability.

Substances Derived from Various Sources.—*Choline derivatives.* The most active of these is pure stabilised acetylcholine, which may be administered subcutaneously in doses ranging from 0·10 gm. to 0·20 gm. in accordance with individual reaction, but usually once daily. Acetylcholine resembles vagotonine in its action on the parasympathetic, in dilating arteries and arterioles, thus checking arterial spasm, and in regulating the peripheral circulation. It is of special service in checking periodical rises of arterial pressure and may advantageously be combined with venesection.

Time is yet too short to express final conclusions as to the

efficacy of the above remedies ; in selected cases I have had the best and most lasting results with vagotonine, and satisfactory results on the whole with acetylcholine and padutin.

More recently still, Clerc, Sterne and René Paris [247] have recorded their tentative results with *octylic alcohol*, which they claim to combine maximal activity with minimal toxicity. These authors are aware of no contra-indications to treatment by their method, which consists in intravenous injection on alternate days of 10 c.c. of a saturated solution of octylic alcohol (C^8H^9OH), carefully purified and redistilled, 1 in 100,000 parts of distilled water, the patient being recumbent. Later, the amount injected is raised to 20 c.c.

In eighteen patients with high pressures, eight above 250/140 mm., the remainder above 200/90 to 120, they found a progressive and at times lasting reduction of arterial pressure ; diuresis about the fourth day ; blood protein and urea lessened, total serum chlorides slightly lessened ; notable amelioration of symptoms, such as vertigo, headache and noises in the head, disappearing entirely in fifteen out of the eighteen cases, some complicated by cardiorenal inadequacy.

Digitalis.—In all cases of myocardial decompensation, apart from fibroid myocarditis, digitalis is our standby. There is no other drug that is its equivalent, and where cardiac insufficiency arises in the course of arteriosclerosis, angina pectoris or chronic nephritis, digitalis may be safely given. It acts directly on the myocardium, and indirectly by stimulation of the vagus, appearing as a cardiac regulator rather than as a stimulant, with specific effects in auricular fibrillation and flutter. Not only does digitalis not raise the arterial pressure, but under its influence both maximal and minimal pressures are often reduced, while pulse pressure is increased. The fall in diastolic pressure is neither due to diminished cardiac output nor to arterial dilatation, but under digitalis the heart contains at the onset of systole a greater amount of blood than before, and the stream leaves the arterial circulation at a greater rate than under normal conditions. Quickening of the pulse in later stages is due to the fact than in less time than normal the intracardiac pressure is sufficiently high to excite contraction.[243]

If the pulse rate can be kept steady at about 60 beats per minute digitalis is doing good, and is keeping off the symptoms that foreshadow breakdown. In cases where the drug is not well tolerated, or where adequate diuresis cannot be maintained in the presence of œdema of the limbs or anasarca, as well as in those with œdema of the lungs and bronchitis, scillaren in combination with digitalis, or following its administration, will not infrequently give good results.

Strophanthus is a direct stimulant of the heart muscle, differing from digitalis in increasing contractility and in not stimulating the vagus, and resembling digitalis in augmenting excitability and depressing conductivity. Hence in uncompensated valvular as opposed to myocardial disease, as well as in dilatation, strophanthus is often to be preferred if arterial pressure be not already high.

For rapid analeptic stimulation, *cardiazol* 20 drops of a 10 per cent. solution 6 hourly or oftener, is advantageous.

Hepatic Extracts.—The physiological effect of hepatic extracts has been studied by James, Laughton and Macallum,[248] as well as by Macdonald,[249] with reference to the specific nature of the depressor principle contained therein. Major,[250] in a study of forty-two hospital patients, reports that in subjects with normal pressures tested as controls injection of a therapeutic dose of hepatic extract was without effect; but that when the pressure was previously raised by administration of guanidine derivatives, or in cases of hyperpiesis, a prompt though gradual fall of arterial pressure resulted. The younger patients who gave no evidence of renal damage or of arteriosclerosis responded best, but some patients with renal deficiency and general arteriosclerosis were benefited. Out-patients pursuing their usual occupations while under treatment were found to react equally with those who were inmates in hospital. A fall in arterial pressure was as a rule unaccompanied by symptoms; rarely there was some slight dizziness. In some cases lowering of pressure persisted only for a few hours, in others it lasted for one or more days. Six patients proved quite refractory to treatment, one died, and necropsy revealed intense arteriosclerosis of the splanchnic area. Further study has failed to establish that the administration

of liver products in hyperpiesis produces any lasting benefit.

In general, control of high arterial pressure resolves itself into careful individual regulation of the patient's work, rest, sleep, diet, exercise and recreation, with protection against psychical and physical stresses, and special attention to the gastro-intestinal tract, in combination with appropriate external measures, *e.g.*, hydrotherapy, if indicated. Few drugs find much advocacy ; those that are of service I have indicated in this chapter.

APPENDIX

The Treatment of High Arterial Pressure by Baths and Waters

Contributed by R. FORTESCUE FOX, M.D., F.R.C.P.

1. BATHS

Warmth applied to the surface of the body expands the superficial capillaries and increases the flow of blood to the skin. This widening of the circulatory bed should cause a corresponding diminution of pressure but for the simultaneous reflex stimulation of the heart by the impression of heat. In a *hot* bath (say 104° F.) the ventricular action becomes so strong that a temporary rise of pressure takes place. In a *warm* bath (say 100° F.) the pressure usually falls, if the patient is quiet and the bath is prolonged for ten or fifteen minutes. In *tepid* and *cool* baths (from 95° F. downwards), the blood pressure is usually raised, the surface vessels being contracted by the impression of cold and the circulatory bed thereby diminished.

But heat and cold often produce a temporary fall or rise of pressure, followed by a more permanent change of a contrary kind. Thus cold applications cause a temporary rise from surface contraction, followed by the familiar *reaction*, in which the chilled surface becomes flushed and warm and the pressure returns to normal or below it. The effect of a hot bath is similarly complex. A very hot bath (say

105° F.) may send up the systolic pressure 20 to 30 mm., but after the bath the pressure soon falls. This fall of pressure is more obvious when baths are taken in daily succession. In cases of vascular hypertonus a daily warm bath (100°F.) for fifteen minutes after a time loses its immediate hypertonic effect, as the cutaneous and subcutaneous circulation is progressively increased. But to obtain this result it is necessary to adjust the temperature of the bath so as to provoke a minimal initial rise.

Moist heat is much more effective as a depressor agent in reducing high arterial pressure than dry heat, and the vascular dilatation thereby produced is often progressive and lasting.

Vapour baths of a moderate temperature, like those of Monsummano, are of much service in this respect. Many experienced hydrotherapists regard the warm bath or vapour bath, accurately adjusted, as the most effectual means for the reduction of arteriolar spasm and the high pressures associated with it. When given in the summer season at sedative spas, like Strathpeffer, Llandrindod, St. Sauveur, Néris or Schlangenbad, optimum results are obtained.

Prolonged baths, at a temperature a little below blood heat, but above that of the skin (say 93° to 98° F.), have also a notable reducing effect on arterial pressure in many cases—especially in those with a predominating nervous element. The effect of one massive, prolonged, sub-thermal impression upon the surface of the body is very sedative. All nervous excitation is diminished, the heart's action is quieted and slowed, and the balance between the deep and superficial circulation is gradually established, generally with a definite fall of pressure in hypertensive cases. Natural baths of this description are given at Loéche-les-Bains in Switzerland and Néris in France, and have also been used at Strathpeffer, Buxton and Cheltenham. The " sedative pool bath " is of this character.

Effervescent baths (carbonic acid gas and water) have a double action on the circulation corresponding to their duplex character. The temperature of the bath (below blood heat) is *cooling*, but the impression of the gas, owing to its low

capacity for heat in comparison with water, is *thermal* and stimulant. The water-gas bath is vasoconstrictor as regards the water, and vasodilator as regards the gas, causing reddening of the skin, and also through the cutaneous nerves it exerts a reflex vagotonic action, the heart's systole becoming stronger and slower. The actual effect of these baths upon arterial pressure depends upon the balance between these actions. Sometimes a reduction and sometimes an increase of arterial pressure is observed, depending upon the pre-existing state. It should be added that cool effervescent baths like ordinary cool baths stimulate metabolism in persons who can react in this manner. Their effect in hyperpiesis may therefore be partly attributable to an increased oxidation of toxic products.

The cardio-vascular condition for which effervescent baths are primarily indicated is atony with subnormal pressure, the result of lowered peripheral resistance or weakness of the myocardium, or from both these causes, as, for example, in Graves' disease, convalescence from infective illness, heart strain, etc. The heart is usually dilated, and the circulation is maintained by the increased frequency of its contractions. In this semi-paralytic condition the *cool* gas-water bath appears to be doubly tonic. The cutaneous capillaries, which have their own autonomous vasomotor mechanism (see Van Breemen, *Note infra*), recover their contractile power, and the heart's action is slowed and strengthened. The surface action is on the balance vasoconstrictor. Any medical man who will take the trouble to watch the effect upon the pulse in a case of this kind during and after a properly graded effervescent bath can convince himself of these facts. After a certain number of these baths the tone of the entire cardio-vascular system is definitely improved and the blood pressure, if previously subnormal, is raised. Experiment upon normal persons does not, naturally, show the same order of events.

It is also true that there are certain types of raised arterial pressure that respond favourably to the effervescent bath. These include the various forms of "nervous" tension or angiospasm, with or without an element of toxæmia. Contraction of the superficial vessels yields, as we have already

seen, when the body is exposed to a certain degree of heat.* Recent reports have shown that effervescent baths at a somewhat higher temperature may be used with good results, even in angina pectoris. The effect is sedative and on the whole depressor. Guillaume, from his experience with the naturally effervescent baths at Spa, concludes that these baths, properly given, increase the reserve force and the nutrition of the heart by lengthening its diastole and by improving metabolism; that they reduce blood pressure by lowering the peripheral resistance, and are at the same time sedative to the nerve centres.†

Manipulation baths and douches are of much benefit in high arterial pressure cases, and also in low pressure cases with circulatory stasis. They combine moisture, heat and mechanical stimulation, and are indeed very powerful sedative or stimulant agencies according to the technique employed. One of the best methods in hyperpiesis is the Buxton manipulation-douche, given to the patient reclining in flowing sub-thermal water. The warm baths at Bourbon Lancy with subaqueous douche are also of service in such cases, and the low-pressure douches at Aix-les-Bains and Strathpeffer. What is known as the Vichy douche is often too stimulating and ought not to be employed.

Hyper-thermal pediluvia and packs of fango, peat, etc. (sometimes called *revulsive*), are of undoubted value. They relax the peripheral vascular network and so cause a centrifugal movement of the blood. Flowing foot baths have been used from early times at some of the French spas, such as Mont Dore, and are rightly regarded as an important factor in the depressor action. Daily repetitions of the bath with gradation of temperature and duration is of the essence of this treatment. They could, of course, be given at any properly appointed spa or clinic.

* Great importance attaches to the temperature at which the bath is given. Hediger has very properly pointed out that baths at or below 92° F. are contra-indicated in all conditions of high arterial pressure of whatever kind.

† For a description of the foam sweating bath see under Diaphoresis, p. 201.

2. Waters.

In a large proportion of cases of vascular hypertension there is an element of plethora or toxæmia. For this reason *eliminative waters* have a place in the scheme of treatment. In overfed, congestive, sedentary and especially in constipated subects, elimination by the bowel is indicated. In England the waters of Harrogate, Cheltenham and Leamington may be recommended for this purpose, and abroad, Carlsbad and Brides-les-Bains ; or a systematic course of the bottled waters of Rubinat, Friedrichshall or Bilina may be taken at home.

The present writer has sometimes noticed in cases of hyperpiesis that before commencing a course of waters venesection is of much benefit. Spa treatment must be used with some caution in hyperpiesis, for if elimination is very defective, the ingestion of waters may cause a temporary hydræmia, and even cerebral hæmorrhage.

In gout, whether hereditary or acquired, which still survives in a variety of manifestations, *sulphur waters* are generally beneficial, and may be taken at Strathpeffer, Harrogate or Llandrindod. Where the liver shows signs of inadequacy, especially in obese subjects, the patient responds best to *alkaline waters*, such as those of Vichy, or to the alkaline-sulphated waters of Cheltenham (the Pittville source), or of the Bohemian spas, Carlsbad and Marienbad.

In contrast with the congestive, hyperpietic subject, the spa physician also encounters high arterial pressure in pale, nervous persons, often of a spare habit and of gouty parentage. Investigation generally reveals in these people a marked defect of renal elimination. Many careful observations have shown that *diuretic calcium waters*, both cold and thermal, are beneficial in this type of case. In Great Britain the waters of Buxton, Bath, Strathpeffer and Malvern, taken fresh at the source, can all be recommended, and in France, Evian and the Vosges spas, Contrexéville, Martigny and Vittel. The waters should be taken in the early morning, recumbent, in divided doses and elimination should be carefully watched. Sometimes, but not always, it is advisable to

combine an internal (diuretic) and an external (cutaneous) treatment, as already described. But not a few cases respond best to waters alone, or simply to a course of baths, prolonged for three or four weeks, and followed by an "after cure" in a sedative climatic resort.

NOTE.—For further details of the treatment of high arterial pressure by baths and waters, the reader may consult in *Archives of Medical Hydrology*, 1922–25 :— WYBAUW, "The Treatment of Circulatory Trouble by Natural Effervescent Baths"; EDGECOMBE, "Balneotherapy and Hypertension"; DEBIDOUR, "The Effect of the Mont Dore Cure on Arterial Tension"; HEDIGER, "Recent Researches on Natural Carbonic Acid Baths" and "Researches, etc."; GUILLAUME, "Natural Effervescent Baths in the Treatment of Angina Pectoris"; VAN BREEMEN, "The Circulation of the Blood in the Skin"; HEDIGER, "Recherches Expérimentales sur l'action vasomotrice des Eaux carbo-gaseuses." Also R. F. FOX, "The Breakdowns of Middle Life" (*Lancet*, 1923).

CHAPTER XIII

Arterial Pressure in Pulmonary Tuberculosis

"La sphygmomanométrie est aux affections chroniques ce que la thermométrie est aux maladies aigues."—Gallavardin: *La Tension Artérielle en Clinique.*

In clinical interest there are few diseases which approach pulmonary tuberculosis, for few present such a wide field of diverse and varying characteristics. Its aspects are protean, and within the compass of one disease it comprises many. Hence in generalisation it behoves one to be more than ordinarily cautious. An illustration of this is to be found in the prevalence during upwards of thirty years of an almost universal, but erroneous, impression that in chronic pulmonary tuberculosis low arterial pressures are the rule. Like most errors, this impression conveys a partial truth: low pressures *may* occur under certain conditions and in certain grades,[252] but are by no means specifically characteristic of the disease as a whole. The mistake has arisen from the recorded experiences of early workers on blood pressure, who necessarily laboured under serious disadvantages. Only primitive instruments were at their disposal, capable solely of registering systolic pressures; and, owing to the fact that until comparatively recently diagnosis was not possible in such an early stage of the malady as it is to-day, their clinical material consisted of cases all of which were in what we should now term a late stage, and the majority of which actually manifested low pressures for a definite reason.

Observations made over a long time and at frequent intervals show that the arterial pressures of tuberculous patients are subject to considerable fluctuations, the diastolic pressure being less affected than the systolic. In point of fact, for pulmonary tuberculosis my opinion is that the level of arterial pressure gives no direct indication as to *extent* of invasion of the lung by the tuberculous process. Where the disease is latent or where there is very slight activity, the diagnosis being perhaps still in suspense and the general

condition good, low pressures may be entirely absent, whilst normal or even high pressures may be found at the onset of many cases of "open" tuberculosis, as well as in those which are advanced in respect solely of chronicity and extensive changes in the lung tissues. Thus from the point of view of extent of disease but not of intensity, in both slight and severe cases, daily estimations of arterial pressure may demonstrate sometimes a rise and at other times a fall. They are, therefore, of little diagnostic value in incipient disease. The systolic pressure is higher in the fibroid forms, especially in women over forty; this is probably secondary to the menopause. Rarely, high pressures may occur without demonstrable cause, and may then originate from interference with blood pressure regulation by microbic toxins.[253]

The essence of the whole matter is intimately bound up with the problems of specific immunity and allergy (specific hypersensitiveness). Both vary directly with activity of disease, which in turn largely depends on the virulence of the infecting tubercle bacilli. The level of blood pressure closely accords with this variation, or, otherwise expressed, *the degree of decline of arterial pressure in pulmonary tuberculosis is directly dependent upon the amount of absorption into the system of tuberculous toxins.* As toxæmia waxes, so arterial pressure wanes.

Once this basic fact is appreciated, the variations between blood pressure records in cases which present similar clinical characteristics as regards the extent of lung affected will be readily understood. As I have insisted for many years, classification of pulmonary tuberculosis into stages corresponding with the extent of involvement of each lobe is of little value either for prognosis or treatment, the reason being that *extent of tissue involved is of far less practical importance than intensity of infection.* Hence it is not surprising that much confusion has existed as to the meaning of blood pressure determinations in tuberculosis of the lungs, for slight forms of still "closed" tuberculosis, but with toxic absorption into the blood or lymph streams from the tubercles present, may exhibit lowered pressures as well as the gravest "open" cases, though not to the same extent.

In cases of pulmonary tuberculosis the arterial pressure is

notably labile, and the tone of the brachial artery is inclined quickly to lapse. My habit is to determine pressures three times in series with as little fatigue to the patient as possible, but, even so, in many cases the third phase is found either to be materially shortened, or the thuds to have diminished in intensity by the time that the third estimation has been reached. This phenomenon is observable even in patients whose general condition is good, and who present only slight evidences of lung disease.

Barometric variations definitely influence tuberculous labile blood pressures, which will rise on clear, frosty, bracing days with sunshine, and drop on days which are dull, damp and depressing. High altitudes have also an appreciable effect on the level of blood pressure,[254, 255] which is higher, both in normal individuals and in consumptives, at elevations of over 4,000 feet than at sea level, the rise between ages twenty and sixty being approximately 35 mm. Hg, and continued residence tending within limits further to increase the pressure. Thus, apart from the many other advantages of high altitude treatment of pulmonary tuberculosis, the benefit which accrues in toxæmic cases which are not too extreme is manifest.

Touching prognosis, a careful study of arterial pressures is helpful. Low pressures constitute a guide to the amount of toxic absorption at the time of investigation. Should toxæmia diminish or cease, the blood pressure will rise; should toxæmia increase, the pressure will coincidently fall. Hence low pressures, within limits, serve as an indicator of the degree of toxæmia, and if these steadily continue to fall, the outlook is grave. When systolic pressure falls, diastolic pressure also falls, but to a less extent, although not infrequently there is a definite drop, even to a point as low as 60 mm. Hg. With improvement, or if the condition becomes stationary, the pressure gradually rises. Favourable evolution is the rule with normal or raised pressures; with low pressures it is the exception. Dr. R. J. Cyriax[46] took 160 bilateral readings of systolic pressure on twelve patients in Mount Vernon Hospital. Six patients had unilateral, and six bilateral, disease; in all the cases tubercle bacilli were present. In patients with acute spread of disease, stained

sputum or hæmoptysis, the maximum pressure on the affected side was as a rule stated to be higher than on the healthy side, the difference in some cases amounting to 26 mm.

Simultaneous Arterial Pressures (Brachial) in Pulmonary Tuberculosis

In order to test this statement, simultaneous observations with two mercurial sphygmomanometers on corresponding points over the brachial artery of each arm were made at Mount Vernon Hospital. Table XII gives the results in

TABLE XII

Pulmonary Tuberculosis (*T.B.* +)

Simultaneous Arterial Pressures (*Brachial*)

A. MEN

No.	Age in Years.	Arterial Pressure. Right Arm. S.	Right Arm. D.	Left Arm. S.	Left Arm. D.	Pulse Rate per Minute.	Preponderance of Disease. B = Clinically bilateral. U = Clinically unilateral.
1	53	142	83	138	83	80	B : R > L.
2	47	95	60	92	58	100	B : R > L.
3	33	130	79	125	77	76	B : R > L.
4	48	118	79	120	78	80	B : R > L.
5	25	110	58	110	60	72	B : R > L.
6	33	118	82	121	83	80	B : R > L.
7	32	115	88	117	88	70	B : R > L.
8	19	128	88	132	90	95	B : R > L.
9	22	128	69	130	70	96	B : R > L.
10	29	124	80	121	77	95	B : L > R.
11	23	106	78	102	76	115	B : L > R.
12	43	121	81	118	80	98	B : L > R. L. artificial pneumothorax.
13	22	105	79	102	79	72	B : L > R.
14	29	110	80	105	80	76	B : L > R.
15	17	114	78	120	78	108	B : L > R.
16	17	133	73	130	75	104	B : L = R.
17	46	120	80	120	80	84	B : L = R.
18	23	125	79	120	80	102	B : L = R.
19	25	120	78	125	78	84	B : L = R.
20	24	160	105	156	105	90	B : L = R.
21	22	120	72	122	70	76	U : R. R. artificial pneumothorax.
22	26	122	87	118	85	78	U : R.
23	25	140	78	138	76	80	U : R. R. hydropneumothorax.
24	17	93	56	92	54	100	U : L. L. pleural effusion.

twenty-four men, and Table XIII in forty-two women. All the cases had tubercle bacilli in the sputum.

TABLE XIII

Pulmonary Tuberculosis (T.B. +)

Simultaneous Arterial Pressures (Brachial)

B. WOMEN

No.	Age in Years.	Arterial Pressure. Right Arm. S.	Right Arm. D.	Left Arm. S.	Left Arm. D.	Pulse Rate per Minute.	Preponderance of Disease. B = Clinically bilateral. U = Clinically unilateral.
1	27	95	60	95	60	120	B : R > L.
2	20	120	78	118	78	98	B : R > L.
3	24	119	71	120	70	95	B : R > L.
4	29	124	81	128	82	85	B : R > L.
5	32	120	81	120	81	95	B : R > L. R. artificial pneumothorax.
6	23	98	68	98	68	70	B : R > L.
7	16	112	80	112	80	120	B : R > L.
8	20	92	55	98	58	80	B : R > L.
9	29	102	71	104	72	94	B : R > L.
10	32	130	90	128	88	90	B : R > L.
11	19	120	86	118	86	92	B : R > L.
12	26	105	60	105	60	110	B : L > R.
13	27	122	78	122	78	80	B : L > R.
14	20	106	74	110	75	102	B : L > R.
15	20	112	79	110	77	104	B : L > R.
16	20	102	80	102	80	80	B : L > R.
17	19	110	77	110	77	78	B : L > R.
18	22	128	80	120	82	98	B : L > R. L. artificial pneumothorax.
19	24	118	82	120	82	100	B : L > R.
20	24	98	70	98	70	102	B : L > R. L. artificial pneumothorax.
21	32	109	71	106	70	98	B : L > R. L. hydrothorax.
22	21	120	76	120	78	102	B : L > R.
23	45	134	80	136	78	96	B : L > R ; also mitral regurgitation.
24	42	180	120	180	120	96	B : L > R.
25	24	110	68	110	72	105	B : L > R.
26	25	100	56	98	56	98	B : L > R.
27	33	130	80	130	80	94	B : L > R.
28	25	108	70	105	70	95	B : L = R.
29	21	128	64	121	60	100	B : L = R.
30	36	136	99	138	99	74	B : L = R.
31	23	95	45	95	45	98	B : L = R.
32	42	140	100	140	100	76	B : L = R.

TABLE XIII—*continued.*

No.	Age in Years.	Arterial Pressure. Right Arm. S.	Right Arm. D.	Left Arm. S.	Left Arm. D.	Pulse Rate per Minute.	Preponderance of Disease. B = Clinically bilateral. U = Clinically unilateral.
33	30	118	70	116	68	90	U : R. R. artificial pneumothorax.
34	38	121	99	120	99	72	U : R.
35	28	112	92	112	92	84	U : R.
36	21	127	88	128	88	76	U : L.
37	24	122	85	122	85	90	U : L.
38	14	92	59	90	60	100	U : L.
39	21	115	78	116	78	76	U : L.
40	38	119	78	119	78	82	U : L.
41	29	127	92	128	92	104	U : L.
42	26	130	60	136	64	90	U : L.

ANALYSIS OF TABLES XII AND XIII

D = Diastolic pressure ; S = Systolic pressure.

	Men.	Women.	Totals.
Total number of cases of pulmonary tuberculosis investigated	24	42	66
D. on the more affected side was higher than on the less affected side in . . .	3	6	9
D. on the more affected side was lower than on the less affected side in . . .	7	6	13
D. on the more affected side was equal to that on the less affected side in . . .	5	15	20
Where the clinical incidence of disease was approximately equal on the two sides :—			
D. was higher on the right side . .	0	2	2
D. was lower on the right side in . .	2	1	3
D. was equal on the two sides in . .	3	2	5
Where the clinical incidence of disease was unilateral :—			
D. was higher on the affected side in . .	3	3	6
D. was lower on the affected side in . .	1	0	1
D. was equal on the two sides in . .	0	7	7

	Men.	Women.	Totals.
S. on the more affected side was higher than on the less affected side in . . .	4	10	14
S. on the more affected side was lower than on the less affected side in . . .	10	8	18
S. on the more affected side was equal to that on the less affected side in . . .	1	13	14

Where the clinical incidence of disease was approximately equal on the two sides :

S. was higher on the right side in . .	3	2	5
S. was lower on the right side in . .	1	1	2
S. was equal on the two sides in . .	1	2	3

Where the clinical incidence of disease was unilateral :

S. was higher on the affected side in . .	2	6	8
S. was lower on the affected side in . .	2	1	3
S. was equal on the two sides in . .	0	3	3

These figures show no correspondence between height of arterial pressure and preponderance of disease.

Cyriax [46] states that a rise in lesion pressure above standard (systolic) usually accompanies symptoms of activity, whilst a fall is of favourable import, and that the onset of hæmoptysis may sometimes be anticipated when other signs of its imminence are lacking.

I find blood pressure estimations, however, of little avail in predicting the onset of hæmoptysis, nor can I discover any correspondence between height of arterial pressure and frequency or amount of hæmoptysis.

Upon the nervous and endocrine systems tuberculous toxins have a potent depressor effect [256] chiefly in the direction of stimulation of depressor fibres in sensory nerves combined with over-excitation and consequent depletion of the adrenal glands, leading ultimately to grave impairment of function with resultant low arterial pressures, diminished oxidation and elimination, digestive troubles, subnormal temperature and muscular fatigue—a functional Addison's disease. Hyperthyroidism and hypothyroidism may also both occur, the former in early cases and the latter in cases of longer standing where toxæmia has substantially lessened the individual resistance.

Pulmonary tuberculosis and pleurisy alike exercise an early influence upon the inspiratory descent of the diaphragm, inflammation of the lung tissue causing spasm of the powerful crura which receive their innervation through the phrenics. Hesitation in diaphragmatic descent is thus one of the earliest signs of pulmonary tuberculosis,[257] and as the tuberculous process advances this limitation of diaphragmatic movement becomes more and more apparent, the mechanical action being not infrequently still further hampered by the presence of adhesions. Decrease in elasticity of the lung tissue is an added element in lessening the inspiratory excursion.

Combination of all these factors lessens oxygenation, causes relative emptying of arteries and filling of veins, and reduces blood pressure. With vasodilatation the diastolic pressure also falls, the lack of vascular tone being noticeable in the extremities, which consequently exhibit increasing cyanosis.

As regards treatment, rest in bed will produce increase of arterial pressure, if toxæmia be not extreme. Estimations of arterial pressure are also valuable as a check upon the amount of exercise which may safely be prescribed, as well as in assessing the grade of labour for which the patient may be suited. The more the pressure approaches normal, the greater can be the amount of work prescribed. Development of pleural effusion and induction of artificial pneumothorax or phrenic evulsion exert no appreciable or permanent influence on arterial pressure provided that intrathoracic equilibrium is not unduly disturbed. Owing to the inevitable mediastinal displacement, thoracoplasty has a greater effect which subsides with gradual readjustment and recovery. In determining the effect of artificial pneumothorax, estimations of blood pressure are too discrepant to be of any practical value, but in general therapy their psychical effect may be utilised in treatment to foster interest and zeal by direct suggestion. For sympathetic hypoadrenia in tuberculosis, adrenal and thyroid preparations, together with the glycerophosphates, have their appropriate uses in stimulating oxidation and promoting sympathetic vasomotor equilibrium.

CHAPTER XIV

Arterial Pressure in Relation to Life Assurance

"A life insurance examiner has a difficult position to fill. He has four people to satisfy: the applicant, the agent, the medical director and himself. The straight and narrow path of strict honesty is his only salvation."—Warfield; *Arteriosclerosis and Hypertension.*

During recent years, notwithstanding that blood pressure determinations have been put to profitable use by many life offices, the position of blood pressure in relation to life assurance cannot at present be regarded as satisfactorily established.

British offices are beginning to take a serious interest in the subject, and, in the case of elderly lives at all events, it is becoming customary for blood pressure to be included among the particulars asked for at an examination. In my opinion, however, a record of blood pressure in **all** examinations for life assurance is desirable, and by this I mean a complete record, not only of systolic, but of diastolic and pulse, pressures as well, in conjunction with pulse rate.

At a meeting in 1921 of the Assurance Medical Society [258] a report of the discussion following Dr. Parkes Weber's paper showed a very limited appreciation of the value of blood pressure estimations, chiefly for the reason that all the speakers contented themselves mainly with a consideration of systolic pressures, diastolic pressure being barely mentioned, except by Dr. A. T. Davies, who expressed his conviction that the prognostic and other significance of the diastolic pressure "will become more and more important." Dr. de Havilland Hall's views as to systolic pressure, based on 170 cases, were that any systolic pressure exceeding 160 mm. should be regarded with suspicion; if 180 mm., the application should be postponed for a second examination, unless there were a history of apoplexy and of death from degenerative lesions at an early age in the family, when the

application should be declined; a pressure of 200 mm., even if only taken once, is sufficient to exclude the life. My own feeling as regards life assurance is that any systolic pressure above 140 mm. should be looked upon as suspicious, but that the figure, whatever it is, of the systolic pressure should never in assessment be considered by itself.

Reference to the reasons which lead me to believe that diastolic pressure is of even more importance than systolic pressure, and that registration of the latter alone is inadequate and misleading, has already been made on pp. 74, 81, 154, but I would again emphasise this weighty point, since life assurance companies at times lose valuable business because certain applicants are excluded on systolic readings alone who in reality are eligible for acceptance—even though perhaps not as first-class lives—whilst, alternatively, others are accepted, the height of whose diastolic pressure, had it been known at the time, would inevitably have ensured their prompt rejection. I trust, therefore, that the day will soon arrive when all assurance companies will insist upon records of the complete arterial pressure picture as an integral part of the customary examination.

For several years Canadian and American offices have realised the value of sphygmomanometry as applied to life assurance, and some of these require all their medical officers to furnish records of blood pressure as a routine measure irrespective of age of applicant or amount of protection desired.

In Great Britain, nevertheless, it is felt that not all blood pressure records are of equal value by reason of variations in personal equation of examiners and in types of instrument, and there is considerable doubt as to the individual and statistical worth of figures furnished by large numbers of outside examiners of differing capacity and intelligence. To a few practitioners, unhappily, the study of blood pressure is still a sealed book. Perchance the method has been introduced since their student days, and they have never felt any necessity to spend time, trouble or means in acquiring even a working knowledge of a procedure that they consider difficult and superfluous. Although almost incredible, I have known instances where blood pressure has been looked

upon as a kind of specific gravity phenomenon, and the figure for the systolic pressure has been recorded as "1035" or approximately, and have heard of others where almost identical figures have been sent in for each of a large series of cases.

Hence, to ensure accuracy both for statistical tables as well as for separate reports, it would appear essential either for estimations to be made only by capable examiners at head and branch offices or for companies to circulate to each of their medical referees a definite scheme of examination in order to regularise and render comparable the results. Otherwise, it is too much to expect that practitioners will universally take post-graduate courses of instruction in technique and instruments to be employed.

Another practical point also arises as to the degree of weight that should be attached to the results of a single estimation. In a doubtful case several examinations at intervals may be required, and one of the difficulties in life assurance work is to get sufficient opportunity for these. "We know perfectly well that in a large proportion of cases the proposer is not anxious to carry out the business, but is doing so under the persuasion of an agent. He will not go to the doctor three or four times, and the doctor will not go to the patient three or four times without extra fees, which the small cases will not carry."[259] Frequently an applicant snatches a hurried half-hour from business or professional cares, and presents himself for examination in a state of psychical or physical unrest, which does not favourably dispose to discovery of his true normal blood pressure. At the time, therefore, two or three readings should always be taken, lest no further chance of investigation, even if desirable, be afforded. If, during the course of these readings, the systolic pressure drops, it is safe to infer that the first of the series was influenced by transitory excitement or exertion. If readings of both pressures are on the border line or are suspiciously high or low, a number of estimations should be made and reviewed in the further light afforded by calculations of pulse pressure and pulse rate. Experience leads me to believe that a persistent diastolic pressure of 100 mm. is really more

significant of hyperpiesis than is a systolic pressure of 150 mm.

When, in the absence of other signs and symptoms, hyperpiesis alone is discovered on the results of a single examination, I do not regard it as fair to the proposer to load or decline a policy. The matter is otherwise, however, if with the hyperpiesis cardiac hypertrophy, arteriosclerosis, or both are associated. The importance of a ringing and accentuated second sound at the aortic cartilage as one of the four cardinal signs of arteriosclerosis should not be forgotten. In arteriosclerosis also the second sound is not infrequently better heard over the carotid artery, oftener in the right than in the left. In all the above cases it is advisable to try for one or more successive examinations after the proposer has rested for several hours. Should high pressure then prove to be permanent, particularly if conjoined with cardiac hypertrophy or changes in the vessels, an additional premium, postponement, limited endowment, or rejection becomes requisite in accordance with the estimated gravity of the condition.

Local as well as general thickening of vessels may be present without alteration in the height of blood pressure, or with hyperpiesis. Tortuosity of the temporal arteries is frequently met with in men of affairs, particularly such as involve large interests or sudden fluctuations, *e.g.*, in captains of industry and stockbrokers ; but is no more of an index to local vascular changes in the cerebral vessels than is sclerosis of the limb vessels, which may be caused by occupations involving local stress, the vessels of the upper limbs being acted upon by conditions of heavy manual labour, and those of the lower limbs at times manifesting patchy sclerosis in riders and others whose work entails arduous leg movements.

Blood pressures raised disproportionately to age, weight and sex connote diminished expectancy of life. Lead poisoning and renal disease are associated with uniformly high pressures : constitutional debilitated states, congenital or acquired, associated with certain forms of endocrine lack of balance, nerve exhaustion and gastro-intestinal atony, arthritic conditions, and status lymphaticus, together with

the toxæmias of influenza, diphtheria and tuberculosis, are usually associated with low pressures. Finally, no applicant with permanently high blood pressure, palpably thickened arteries or high blood urea content can ever be regarded as a first-class life.

The presence of protein in the urine along with high blood pressure always necessitates extra caution on the part of the examiner in order to distinguish as far as may be between physiological and pathological varieties. As to this, the problem is not yet definitely solved.

For general guidance the following hints may prove of service. Where neither family history nor clinical investigation reveals any other abnormality which compels loading of the policy, adolescent proteinuria, *i.e.*, protein in the urine in cases below age thirty, if absent after a night's rest, may be looked upon as a normal risk, since statistics go to prove that in such cases mortality barely exceeds the standard for healthy lives. Hence an application can safely be accepted at ordinary rates. It is well, however, to note that in the following decade the rate of mortality is appreciably higher.

If, after a night's rest, protein be found in the urine in the absence of hyperpiesis or vascular changes, either loading, shortened endowment or postponement of the assurance is indicated; if combined with hyperpiesis, the application should be declined. If the proposer be over age thirty and have proteinuria which clears up after rest, the policy may be accepted with five years' addition; if the protein persists, a limited endowment may be granted; but if hyperpiesis or cardiovascular changes be present as well, this means rejection of the proposal. Similarly in the case of "albuminuric" neuroretinitis, which should always be regarded as a direct contra-indication to life assurance at any age.[90]

In cases of proteinuria where doubt exists as to the precise significance to be attached to its presence and degree, the best and simplest index of renal efficiency is the blood urea test (p. 147). The import of slight grades of inability to concentrate urea in the urine is no more easy to determine than is the import of the lesser grades of proteinuria.

As a general rule, whenever an extra premium equivalent to more than an addition of five years has been imposed in order to obviate a possibility of the applicant feeling that he has been dealt with too strictly, I would suggest that he be given the option of attending the company's medical officer on any renewal date at his own expense, so that the rating may be revised. If, by that time, the applicant's health has improved, the assessment may then be lowered.

Certain well-known American and South African life assurance offices have adopted a principle, new to this country, of offering free advice and treatment by a staff of consultants and specialists to the lives assured with these companies. By this means it is expected that mortality of the lives assured will be decreased, and duration of life prolonged.

Occasionally doubt is expressed as to the value of any medical examination for life assurance. As against this view Dr. Brandreth Symonds [260] has shown that the influence of reliable medical examinations is conspicuous for five policy years, and probably has a demonstrable effect even up to the tenth policy year.

In this chapter many important points which bear directly on medical examination for life assurance have been omitted, since they are fully dealt with in other chapters. Special reference may be made to "Clinical Estimation of Arterial Pressure," "Physiological Variations in Arterial Pressure," "Prognosis," "Pathologically High Arterial Pressures," "Ocular Changes" and "Effect on Mortality of High Arterial Pressures."

"Probably life insurance and general medicine will never regard blood pressures in the same light. Life insurance sees only people who are healthy, or at least think they are. Even the highest systolic pressure of fat elderly people is below 140 mm. on the average, if they are acceptable for life insurance. This also means that practically as many are below 140 mm. as above, and of those above 140 mm. nearly all are below 150 mm. General medicine, on the other hand, sees those who feel that they are sick. If their illness is due to blood pressure, it is usually high, frequently as high as 200 mm. or more. General medicine knows that these high pressures will come down to 170 or 180 mm.

by appropriate treatment, and many of them live for years. But medicine does not realise that a small increase in the number of deaths per year means a great difference to life insurance. At age fifty we only expect fourteen to die in the following year out of 1,000 living, and we call that 100 per cent. mortality. If twenty-eight die, our mortality jumps up to 200 per cent. At age sixty, if the number of deaths among 1,000 living increases from 26·69 to 40·04, the mortality increases to 150 per cent. If a practitioner should see 1,000 patients with high blood pressure at age sixty and bet with himself that 974 would survive the year, and only 960 did survive, he would not feel downcast. In fact, he would probably point to the record with pride and boast of his ability in prognosis. But life insurance would have to tell him that his mortality was 150 per cent. in that group, and a medical director who never made a better guess than that would not keep his position for long. General medicine would look complacently at the living, but life insurance would ruefully regard the dead, for forty claims would have to be paid instead of the twenty-six expected."[260]

Given more opportunity of examination in doubtful cases than is at present afforded, in proportion as diagnosis, and therefore prognosis, is thereby rendered more certain, the protective value of medical examination is likely to be enhanced.

In my judgment, an examiner for life assurance cannot be too good a clinician. Combined with purely medical skill, he should also possess a wide all-round capability of assessing the relationships to assurance of the interwoven and complex individual, environmental, and financial considerations which directly or indirectly affect each proposal.

CHAPTER XV

Epochs in the Clinical Estimation of Blood Pressure

"Men's works have an age, like themselves; and though they outlive their authors, yet have they a stint and period to their duration."—Sir Thomas Browne: *Religio Medici.*

In bygone days, through skilful and experienced palpation of the radial pulse the fathers of medicine became familiar with its characters. These were described so carefully by Aristotle, Herophilus, Erasistratus and other early writers down to the second century of the Christian era, when Galen taught, as to leave little for later observers to add until the advent of objective and graphic methods. After Galen's time, the quest of natural knowledge was brought to a standstill by the subjection of research to authority, from which dominance it was not freed till the revival of learning in the Middle Ages.

The chief epochs in the early history of blood pressure occur about the end of the first quarters of the seventeenth, eighteenth and nineteenth centuries. Still earlier, however, in China, from the time of Pien Ch'iao (255 B.C.), great importance was attached to a detailed study of the characters of the pulse, which in subsequent days became of diagnostic significance in various conditions of disease. Wang Shu-ho (A.D. 280) wrote the Mo Ching (Pulse Classic), in ten volumes, considered to be one of the Chinese standard works on medicine.[261]

1. The Circulation of the Blood.—*The first epoch* coincides with the publication by the immortal William Harvey[1] (1628) of his "Exercitatio." Harvey demonstrated the "motion of blood in a circle," tracing it through the lungs and greater circulation, "down" arteries and "up" veins. The ground had been prepared by his predecessors, Vesalius, Servetus and Fabricius. Long before, Galen had established the existence of the lesser circulation. But Harvey's logical and masterly exposition of the mechanics of the

circulation as a whole, verified by experimental evidence, did for physiology what the "Fabrica" of Vesalius (1543) had done for anatomy.

Worthy successors were Malpighi,[262] Lower[263] and Borelli.[264] Malpighi (1628–1694), the first histologist, was enabled by the microscope to see in the frog's lung a network of tiny channels joining the ends of minute arteries to the beginnings of minute veins—those channels that we now know as capillaries. He thus supplied the missing link in Harvey's discovery by showing that the circulation was even more complete than Harvey had supposed. Richard Lower was one of a band of scientists at Oxford, including Willis, Boyle, Christopher Wren, Mayow and others, who originated researches in physics and physiology of fundamental importance. In earlier days a careful worker in anatomy and physiology, in later days a distinguished physician noted for his experiments on direct blood transfusion, Lower brought to his work a true scientific spirit which dominated his writings. The chief of these was "Tractatus de corde" (1669), the third chapter of which "begins with quantitative estimations of the output of the heart, and of the rapidity of the blood flow, which show Lower to be a worthy successor of Harvey, and predecessor of Stephen Hales." Borelli (1608–1679) developed Harvey's views by proving through physical methods the propulsive force of the heart in systolic contraction of the spiral musculature of the ventricle "after the manner of a wine-press," and by demonstrating the value of arterial elastic recoil during diastole in promoting the onflow of blood.

2. Direct Estimation of Arterial Pressure—Physiological.—*The second epoch* begins with the year 1733. Up to this time, notwithstanding attempts, mostly erroneous, to calculate it, the blood pressure had not been directly measured. This measurement was accomplished by the Rev. Stephen Hales,[265] who at Cambridge had been well grounded in Newtonian physics, which he proceeded to apply to physiology and biology. His classical experiments, initiated about 1708, are detailed in "Statical Essays, containing Hæmastatics," the first edition of which is now very rare. Into the artery of a mare Hales inserted a long tube which served

as a manometer for quantitative determinations of arterial pressure, cardiac capacity and velocity of blood flow. After somewhat similar fashion he estimated the pressure in large veins. He also arrived at the correct inference that the volume of blood traversing the lungs must always be equal to that flowing through the systemic circulation in the same time. Unable directly to measure capillary pressure, by calculation he deduced that "the force of the blood in these fine capillaries can be but very little; and the longer such capillaries are, the slower will be the motion of blood in them." As Dr. Clark-Kennedy, in his able memoir, has remarked: "Hales was essentially a pure scientist; his concern was with physiology as a branch of science, and not with medicine, of which he knew nothing." Nevertheless, one may add that his direct estimations of blood pressure in animals not only constituted a valuable addition to biology, but also proved of great utility to subsequent observers.

The third epoch, not reached for yet another century, marks the beginning of modern developments. The work of three men—Magendie and Poiseuille in France, and Ludwig in Germany—following that of Harvey and Hales, may justly be regarded as the earliest exposition of the science of hæmodynamics. Magendie, an experimental physiologist and pharmacologist, along with Poiseuille,[266] showed the driving force of the heart to be the chief cause of blood flow in the veins, and observed that arterial pressure increases with expiration.

The Mercurial Manometer.—Poiseuille (1828) invented the hæmodynamometer, thus making a distinct advance on the piezometer of Hales by substituting for the unserviceable long tube a U-shaped mercury manometer, connected with the artery by a lead tip filled with a solution of potassium carbonate to retard clotting, a procedure still in practice. With this instrument Poiseuille [267] brought observations within reasonable space, and avoided the error inherent from loss of blood in the method of Hales. Poiseuille showed that arterial pressure rises with expiration and falls with inspiration, and was able to assess the degree of arterial dilatation at each heart beat.

The Kymograph.—Carl Ludwig,[268] in one of the many manifestations of his genius, took the hæmodynamometer of Poiseuille and made it into a kymograph by the addition of a float furnished with a pen, by which every oscillation of the mercury was recorded on a cylinder rotated by clockwork. The kymographion, as its inventor termed it, which he modestly attributed to "a principle first employed by the celebrated Watt," was introduced in 1847, while Ludwig was still at Marburg, and represents the earliest application of the graphic method. Not only did this notable invention give a tremendous impulse to elucidation of physiological phenomena in general, but it enabled permanent records to be obtained of the mean pressure in any artery. Shortly afterwards, in 1858, Claude Bernard, the greatest physiologist of France, completed his researches on the circulation by demonstrating the sympathetic to be the constrictor nerve, and the chorda tympani the dilator nerve of the blood vessels.

The Elastic or Spring Manometer.—To overcome the lag of the mercury column, and thereby to attain more exact registration of even the most rapid fluctuations of arterial pressure, the elastic or spring manometer came into existence; in this instrument the mercurial mass is replaced by a spring, or other elastic body of small mass, less subject, therefore, to errors arising from inertia. Many labourers worked in this field, notably Chauveau and Marey [269] (1861), Fick [270] (1864 and 1885), and Hürthle [271] (1890), water being used in some instances and air in others for the transmission of vibrations.

3. Indirect Estimation of Arterial Pressure—Clinical.—The next step introduced methods applicable to human investigation, which do not involve opening an artery. The first of these originated from the sphygmograph, invented by Vierordt [272] (1855), and improved by Marey (1860), by which the form of the pulse wave was first clearly defined. This invention, modified by Meyer, Anstie, Mahomed and Sir Benjamin Ward Richardson, and simplified by Dudgeon, retained its vogue for upwards of half a century. By reason, however, of changes in the form of the curve obtained under varied pressures, and because it indicated total

pressure, and not pressure per surface unit, the sphygmograph failed to justify its early diagnostic promise and fell into disuse.

The Weighted Sphygmograph.—Vierordt (1855) made the earliest attempt at indirect estimation by loading a sphygmograph with weights, thus roughly measuring the force requisite for pulse obliteration, and so approximating to one criterion of systolic end-pressure. Varying kinds of apparatus, all possessing the same fatal defect of applying pressure to the artery by a solid pad, were devised by numerous investigators, such as Förster (1867), Landois, Béhier (1868), Waldenburg (1877), Bloch, Verdin, Chéron (1888) and Hoorweg (1889), down to Philadelphien (1897) and Levaschoff (1901).

The fourth epoch marks the construction of the first clinical sphygmomanometer. This applied pressure through a fluid medium, and was free from grave inaccuracies. For this we are indebted to von Basch [273] (1876), of Vienna, as well as for the earliest estimation of systolic pressure in man that has any real value. The instrument consisted of a small funnel capped by a rubber bag containing water (*pelotte*), and connected with a mercury manometer, thus for the first time obtaining pressure per unit of surface.

In 1887, von Basch brought out an improved model in which the mercury was replaced by an aneroid. The values thus obtained were not absolute, as von Basch admitted, and as Tigerstedt [274] showed in 1893, owing to the variable relation of the artery to bone and soft tissues, causing readings to be invariably too high. Improvements were instituted by many workers, especially by Potain [275] (1889), who replaced the pelotte by an air cushion. To the labours of these two pioneers, von Basch and Potain, we owe the recognition of the importance of clinical sphygmomanometry. Simultaneously with von Basch, Marey [276] (1876) had been engaged upon a series of researches from which our clinical methods of determining diastolic pressure have been developed, so that the next link in the chain is represented by Marey's plethysmograph, which enabled a variable counter-pressure to be applied to the whole surface of a limb or digit. Two years later (1878) Marey reached

the true conclusion that the point at which oscillations of the recording lever cease denotes the maximal or systolic pressure, while the point at which the amplitude of oscillation is greatest indicates the minimal or diastolic pressure.[277] These exceedingly valuable observations stand out as a landmark, since they have provided subsequent observers with an accurate basis for the clinical determination of arterial pressure. Marey's procedure was elaborated by Mosso [278] (1895) and Hürthle [279] (1896), who, with other observers, proved the correctness of Marey's criterion of diastolic pressure, which stands out clearly as *the fifth epoch*. "Marey, however, was ahead of his day, and notwithstanding the fundamental importance of these two criteria of maximal and minimal pressures, during the next twenty-five years his observations attracted little attention, although his methods and conclusions are almost identical with the best and more modern work of von Recklinghausen and Erlanger" (Janeway). Coincidently with von Basch and Marey, the Cambridge school of physiologists—all pupils of Sir Michael Foster (1836–1907)—were creating an epoch in teaching well-nigh equalling that of Ludwig. In this connection the names of Balfour, Gaskell, Langley, Sherrington, Roy, Adami and H. N. Martin speak for themselves.

Next came the hæmodynamometer of Oliver,[280] and Hill and Barnard's [281] pocket sphygmometer, both measuring pressure through direct application to the radial artery, and thus sharing the fallacies of the instruments of von Basch and Potain. Gartner's [282] tonometer (1899) afforded another index of systolic pressure by the point at which colour returned to the skin of the blanched finger after removal of compression.

The above-mentioned instruments are purely of historic interest, for no sphygmomanometer introduced prior to 1896 complied with all the essential requirements for general use by physicians, namely accuracy, freedom from subjective errors, ease of application and portability." [30]

In 1894 von Basch made the first systematic study of arterial pressure in children,[283] while the first clinical observations of importance were recorded by Cook in America (1903).

The sixth epoch is noteworthy in that it introduces the modern clinical method of circular compression of a limb by air. Such was the procedure adopted by Riva-Rocci [284] (1896) in Italy, for estimating systolic pressure by a mercurial manometer, and by Hill and Barnard (1897) in England, for estimating diastolic pressure by an aneroid. In reality both these instruments constituted an ingenious extension of the method of von Basch, whose small pelotte was expanded into a rubber bag (originally 5 cm. in width) which, encircling the limb and connected by a tube with the manometer, was covered by an inelastic silk bandage (Riva-Rocci), or by a leather cuff (Hill and Barnard). Von Recklinghausen [285] (1901) and Erlanger [286] (1904) showed that the chief remaining error lay in the narrowness of the 5 cm. bag. With this, all readings were too high, particularly in the case of high arterial pressures, for since the bag becomes more circular in cross section as it distends, the compressing surface proportionately decreases.

Von Recklinghausen [287] invented a machine registering in centimetres of water. It was little used outside Germany since it was bulky and expensive, and possessed no advantages over the other instruments. The useful piece of work, however, which he did was to prove that for adults, in obliteration of the brachial artery by any method, the cuff must be at least 12 cm. wide, for, when narrower cuffs are used, much pressure is wasted in squeezing the tissues, and in proportion as the pressure is raised, so the compressing surface decreases, since the bag, as it distends, tends to become more circular in cross section. Further, it has been shown that, by the use of a 12-cm. armlet, accurate values are obtained independently of the amount of muscle and fat surrounding the brachial artery.

The Sphygmomanometer of Erlanger [288] is based on the principles of Marey and Riva-Rocci, and constitutes the earliest attempt to furnish graphic records of both maximal and minimal pressures. It is accurate, but too complicated and bulky for clinical use. It differs from the Riva-Rocci type of instrument with broad armlet solely in the introduction of a T-tube between the mercury manometer and armlet connecting with a rubber pressure bag in a glass bulb.

Pressure oscillations in the armlet are transmitted to the pressure bag, and its oscillations in turn are communicated to air contained within the glass bulb, and are registered by the writing lever of a Marey tambour on the smoked paper of a revolving drum. By a valve the pressure can be released gradually, when small oscillations of the lever appear, due to impact of the compressed artery on the upper margin of the cuff. Sudden increase in the size of these wavelets soon occurs, and fixes the maximal pressure which is read on the manometer scale. Below this point oscillations wax and then wane. The point of maximal oscillation marks the minimal pressure. Systolic readings average 5 mm. higher than with the Riva-Rocci instrument with broad armlet, and diastolic readings correspond within 5 to 15 mm.

Hirschfelder [289] increased the usefulness and complexity of this apparatus by the addition of two tambours for simultaneous registration of the carotid and venous pulses.

The Sphygmomanometer of Gibson [290] (Fig. 42) also takes advantage of the principle of circular compression in estimating the maximum pressure, while it gives the minimum pressure by the oscillations of the mercury. It has a mercurial manometer, the lumen of which is exactly that of the ordinary physiological kymograph. The air contained in the armlet can be augmented by a large syringe, and the pressure may be raised quickly or slowly. By means of a valve the pressure may also be lowered quickly or slowly. A float rests upon the mercury, surrounded by alcohol, and an upright rod of aluminium leads to a horizontal arm which writes on the revolving cylinder. In order to find the absolute zero, a fixed arm traces the abscissa upon the cylinder, which is driven by a horizontally placed clock, as in Erlanger's instrument. The pulsations of the artery below the point of compression are recorded by means of a transmission sphygmograph. This consists of a tambour placed in contact with the brachial or radial artery, as may be most convenient, by a pelotte resting upon the vessel. This tambour is brought into communication by rubber tubing with another tambour, the movements of which are recorded on the cylinder simultaneously with the move-

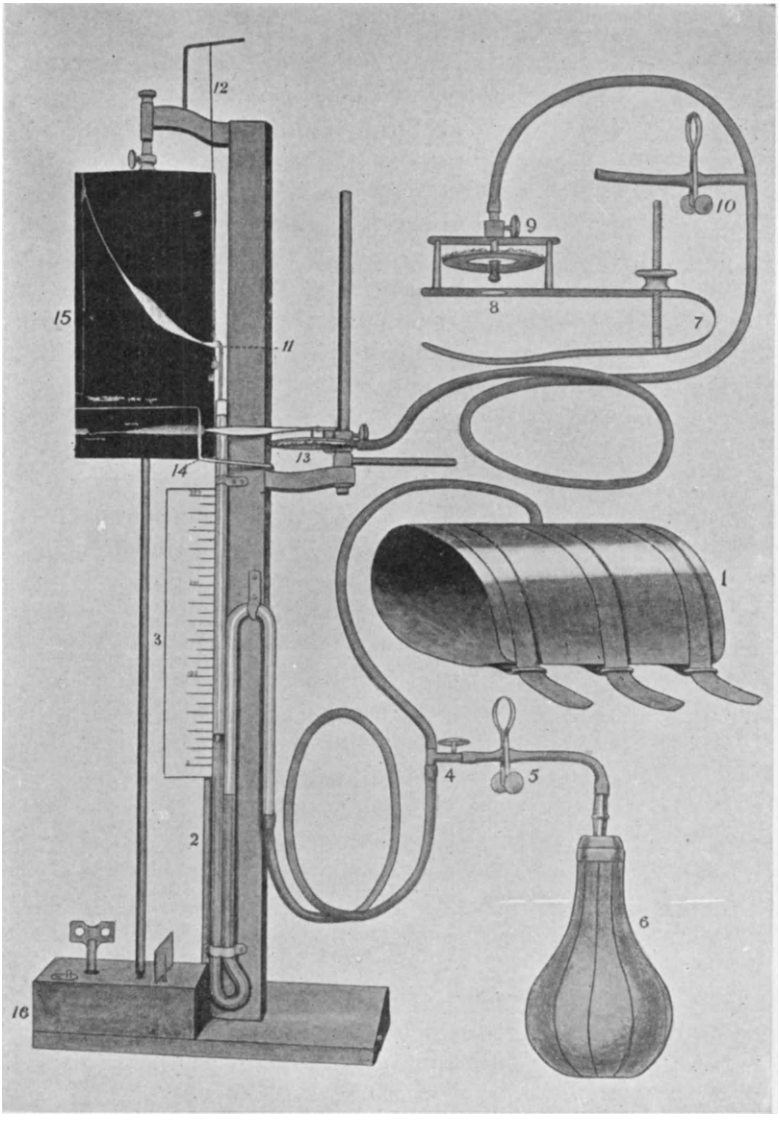


FIG. 42.—Gibson's recording sphygmomanometer. 1. Armlet. 2. Manometer. 3. Scale. 4. Valve for regulating pressure. 5. Clip, acting as a valve to 6, inflation bulb. 7. Support for transmission sphygmograph. 8. Pelotte. 9. Screw for adjusting sphygmograph to artery. 10. Clip for arranging pressure. 11. Recording lever of manometer. 12. Weighted thread for adjusting lever. 13. Tambour recording movements of transmission sphygmograph. 14. Arm marking abscissa. 15. Revolving drum. 16. Clockwork.

ments of the kymograph. The best tracing is obtained when the tambour in contact with the artery is larger than that connected with the recording lever, by means of which the movements are amplified.

"In using the instrument the pressure within the cuff may be raised gradually or quickly, the latter being the more usual course. If it is slowly raised, the tracing of the kymograph shows at first a line of ascent with small oscillations, but as it rises the pulsations become more and more marked, and the excursion of the index more extensive, until a maximum point of amplitude is attained, when they begin to diminish and gradually disappear. Simultaneously the transmission sphygmograph records a gradual diminution in the amplitude of the pulsations, which finally cease. When all the movements of the kymograph, as well as of the sphygmograph, have come to an end, the pressure is allowed to fall by the escape of air from the valve, and the events which follow are the converse of those just described." [291]

Such a tracing is shown in Fig. 43.

Since the manometer has a double column of mercury, the height of the tracing above the abscissa must be doubled in reading the record; *e.g.*, if the reading is 60 mm., the pressure must be recorded as 120 mm. Hg.

The mid-point of the kymographic curve at the point where the pulsation returns in the vessel below the seat of compression is taken as the index of systolic pressure, whilst the mid-point of the kymographic curve where the greatest amplitude of oscillation occurs is chosen as the index of diastolic pressure.

"The advantage of this instrument over that of Erlanger lies in the fact that the height of the mercurial column is automatically recorded, and that the personal equation is entirely eliminated. Its disadvantage in relation to the former is due to the inertia of the mercurial column, which renders it less sensitive to delicate pressure changes." [291]

Gibson's sphygmomanometer was modified for clinical use by Singer,[292] who reduced the size of the instrument, and substituted ink pens writing on white paper.

To ensure uniform compression, Gallavardin [51] (1922) devised an armlet with two independent pressure bags,

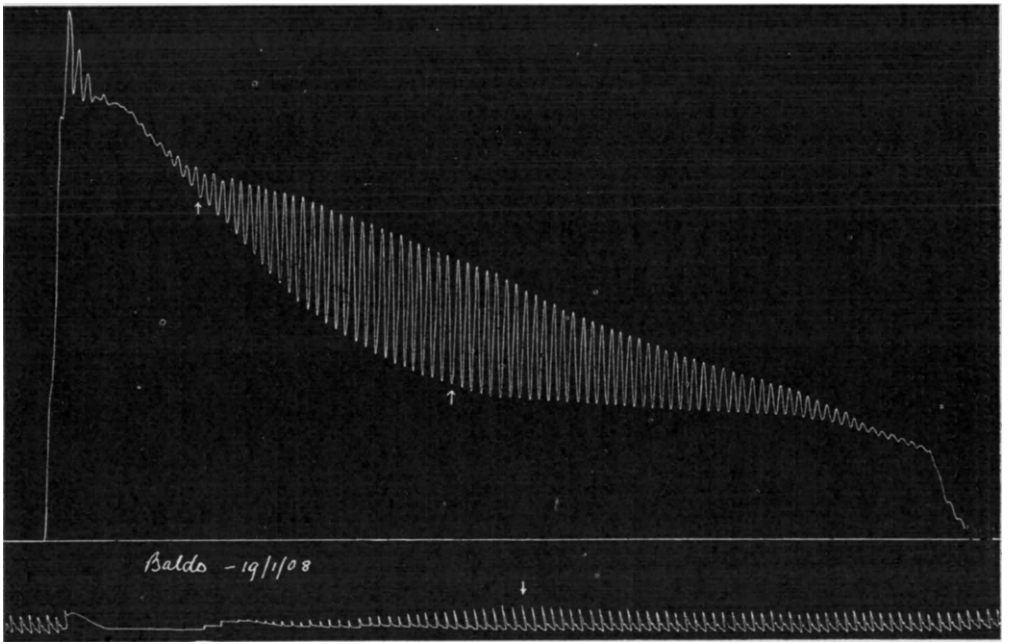


FIG. 43.—Tracing taken with Gibson's sphygmomanometer by rapid inflation and gradual escape on a quickly revolving drum. The point of the curve at which the first definite pulsation returns in the artery below the seat of compression is the index of systolic pressure, S. Preceding S is a zone of small waves due to impact of the pulse waves against the slight funnel-shaped depression of the brachial artery at the upper edge of the armlet, no separation of the walls of the segment of the artery under compression taking place till the point S is reached. The mid-point where greatest amplitude of oscillation occurs is taken as the index of the diastolic pressure, D. (Figs. 42 and 43 are from Gibson's original paper in *Proc. Roy. Soc. of Edin.*, 1908.)

which overlap by one-third of their width, and have a combined width of 12 cm.

The Riva-Rocci instrument was thus the prototype of the modern mercurial manometer. Numerous modifications were made by Oliver,[33] French, Mummery and Martin [293] in this country; Gallavardin [16] and Vaquez [52] in France; Cook, Stanton, Janeway (Fig. 44), Brown, Faught [295] and Nicholson in the United States; Sahli in Switzerland; Bruhns-Fahræus in Norway; and many others. The most

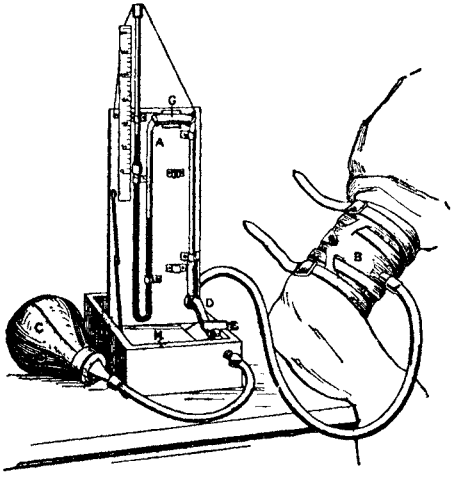


FIG. 44.—Janeway's sphygmomanometer.

complicated machine, called the sphygmomanometroscope, embodying five different instruments, was invented by van Westenrijk (1908) of Petrograd, and the simplest by Professor C. J. Martin (1905).[293]

The Martin Sphygmomanometer (Fig. 45).—This comprises a manometer of the U-tube variety, 12 inches in length, made of thick opal-backed glass tube, the internal diameter of which is $\frac{1}{10}$ inch. Each end is trapped so that the mercury cannot escape. The tube, with attached millimetre scale, is secured to a piece of wood which stands upright in a socket fitted to the inside of the box.

Note.—One millimetre of the scale really indicates 2 mm. of pressure, as the rise of the mercury in one limb is accompanied

by a corresponding fall in the other. In graduating the scale, allowance is made for this, so that the pressure can be read directly.

To bring the two columns of mercury to their respective zero points, run the mercury into one of the oval end

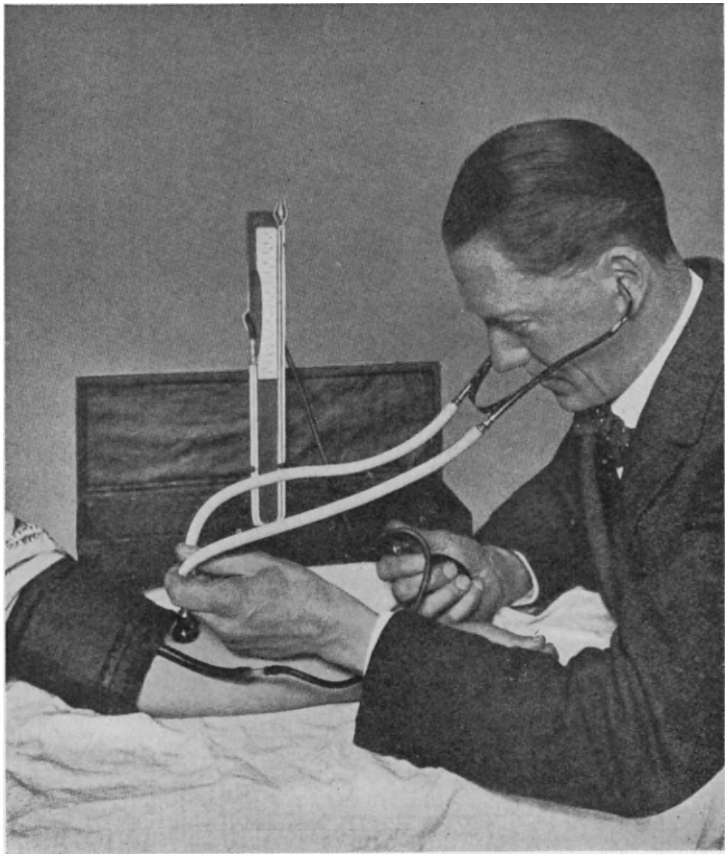


FIG. 45.—The Martin Sphygmomanometer.

chambers, then place the finger over either of the open ends of the non-spillable tube, hold it upright and allow the mercury to run slowly down.

The armlet consists of a thin rubber bag, $\frac{1}{30}$ inch in thickness, $13\frac{1}{2}$ inches long, and $4\frac{1}{2}$ inches broad, the

interior of which communicates with a piece of small-bore rubber tubing. The rubber bag is covered with thin, soft material. A rubber inflating bulb with release screw is connected to the pressure bag and manometer respectively by rubber tubing through the medium of a metal T-piece.

Fold the armlet evenly and comfortably around the patient's arm, and tuck in under the preceding turn the last 2 inches of the tail end. Loosen the brass screw at the neck of the inflating bulb, and see that the mercury level stands at zero on the scale. Attach one end of the tubing to the manometer, and the other to the glass tube connection of the armlet. Close the brass release valve by screwing it up.

The pressure in the armlet is now slowly raised by squeezing the inflating bulb whilst listening with a stethoscope or auditory tambour placed over the brachial artery below the armlet, as detailed on p. 26.

The Nicholson Pocket Sphygmomanometer.[295] (Figs. 46, 47).—In this instrument the right limb of the U-manometer is replaced by a cylindrical glass reservoir, partly filled with mercury, into the top of which is sealed a wavy capillary tube. At its upper end the reservoir is connected with (*a*) a needle valve air release, (*b*) a nozzle for the tube joining it with a 14-cm. soft cuff, and (*c*) a stopcock in union with the pump. Parallel to the lower section of the left limb of the U-manometer, which ends in another stopcock, is a metal scale hinged for folding up. Alongside the glass reservoir is a metal tube which carries the upper section of the left limb of the U-tube. These various parts are held securely by clips fixed to the lid of a metal case which contains and protects all the metal and glass. The lid, when raised, automatically locks in the upright position, thus forming the back of the instrument (Fig. 46).

To operate, fix the ground end of the glass tube into the lower section of the U-manometer by opening the stop-cock. Next unfold the scale, and adjust so that the zero is at the level of the mercury. The needle valve should be closed, and the stopcock to the pump open. The cuff being applied to the arm, now inflate to above obliteration

point. Then open the needle release slightly, using the auscultatory method in estimation.

For sphygmomanometers of the mercurial type to be reliable it is also necessary for the bag to be connected by

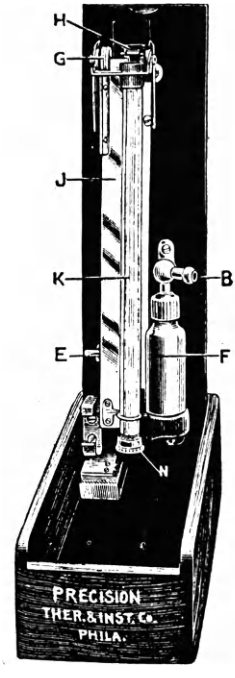


FIG. 46.

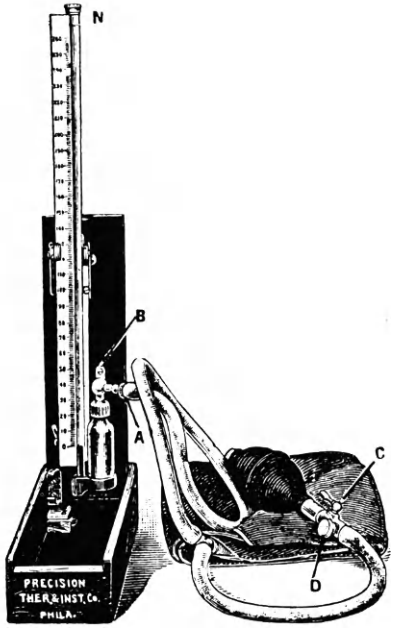


FIG. 47.

FIGS. 46 AND 47.—The Nicholson pocket sphygmomanometer. Fig. 46 shows the instrument semi-folded, Fig. 47 in open position. A. Terminal of tubing. B. Socket for same. C. Stopcock. D. Needle valve. E. Clamp level to set scale at proper mercurial level. F. Reservoir of mercury. G. Hinge to allow folding of graduated scale. H. Automatic valve to allow folding of manometer tube and to retain mercury in transit. J. Scale folded. K. Tube folded. N. Screw-cap for removal of mercury.

stiff tubing with a mercury manometer whose bore is fairly wide (2 mm. diameter), and graduated accurately. Such may be either straight or U-shaped, the latter being the more portable. The amount of mercury contained therein usually varies between 35 and 45 gm., but such difference in weight causes no variation in blood pressure readings.

In addition to mercury, water and compressed air have also been utilised as media. Water has distinct advantages in that (1) it has less inertia than mercury, (2) its specific gravity is almost the same as that of blood and (3) centimetres of water can readily be converted into grammes. The facts that militate against its general adoption are (*a*) custom ; (*b*) difficulty in expressing millimetres of mercury in terms of centimetres of water.

The compressed-air manometer, suggested by Oliver [33] possessed considerable merits, but had the drawback of being influenced by changes in temperature and in barometric pressure. It was in use until recent times.

Another good dial instrument was the Faught,[294] which possessed certain minor differences, but gave similar readings.

Of aneroid instruments there are many patterns, all, with the exception of the sphygmo-oscillometer of Pachon, which possesses various refinements, working on the same general principle. An early model was evolved in France by Amblard, who divided the single wide pressure-bag of the armlet into two smaller ones, absolutely separate when necessary, and thus suggested an idea which Gallavardin later improved. One that attained much favour was introduced by Sir Lauder Brunton, who, as a pupil of Ludwig, took a lively interest in the subject of blood pressure.

In France the oscillometric method has long been utilised since Pachon [49] (1909) devised the first model of the sphygmo-oscillometer (p. 45).

Turning for a moment to the clinical side, one may draw attention to the influence of the Guy's school, beginning with the researches of Richard Bright [296] (1836), who drew attention to the " full and hard " pulse of the disease which has been named after him, and by his valuable work provided an inspiration for later investigations. It has been aptly said that Bright began the story of hyperpiesis and its consequences by writing its final chapter. The findings of Wilks (1859) were largely amplified by Gull and Sutton [297] (1872), who described under the title of arterio-capillary fibrosis a pathological state, which long afterwards has reappeared under the guise of diffuse hyperplastic sclerosis. Between

1874 and 1881, Mahomed [298] also published various papers, and, in the words of Dr. Ryle, "may be regarded as one of the chief pioneers of blood pressure observations in disease. . . . With the aid of his fingers, his own modification of Marey's sphygmograph and the roughest methods of measuring pressures . . . he amassed records and marshalled arguments which are a monument to his zeal and brilliant intellect. More clearly than anyone previously, he defined clinically that form of Bright's disease which tends to terminate with apoplexy or heart failure. . . . He accounted reasonably for many of the symptoms of high blood pressure, and described in detail the ways in which the heart may fail." Further, Mahomed described a pre-albuminuric stage in scarlatinal nephritis which, with von Basch's conception of "latent arterio-sclerosis" and Huchard's pre-sclerosis, led up to the recognition of primary high blood pressure or hyperpiesia.

"The logical basis of chronic interstitial nephritis was expanded in 1868 by George Johnson's [296] account of widespread muscular hypertrophy of the arterioles, which, by exerting a "stop-cock action," protected the tissues from the access of blood rendered toxic by renal disease, and Janeway (1913) established the association of a raised blood pressure with renal disease." [113]

General use of the sphygmomanometer in this country was largely due to Clifford Allbutt, Lauder Brunton and George Oliver. The latter (1841–1915) will chiefly be remembered for his insistence on the value of blood pressure estimations in diagnosis, and for instruments that he devised for this purpose.

To Sir Clifford Allbutt, for thirty-two years Regius professor of physic in the University of Cambridge, and a distinguished successor of William Harvey at Caius College, we owe the setting forth and development of much of our modern thought. At various times and places, in his scholarly and inimitable manner, he postulated his view that arteriosclerosis is not the cause of raised arterial pressure,[62, 63] and reviewed the steps by which he had reached his conception of hyperpiesia.[300] On February 27th, 1895, in an address before the Hunterian Society on "Senile

Plethora, or High Arterial Pressure in Elderly Persons," Allbutt indicated that persistently raised arterial pressures were not limited to the elderly, and advanced his conception of a primary state of high arterial pressure characterised by persistency and occurring apart from chronic renal disease. This state he at first called "hyperpiesis," but on further developing his theme he finally amplified the term "hyperpiesis" to include any high pressure recorded by the sphygmomanometer, reserving for the clinical syndrome the name "hyperpiesia," by which it has since been recognised. Clifford Allbutt's original views on this subject, at first contested, were later supported by many writers and now receive general acceptance. Osler,[301] his "brother Regius," at Oxford, was also interested in blood pressure, and in one of his clinical lectures picturesquely stated that "a man's life may be said to be a gift of his blood pressure, just as Egypt is a gift of the Nile."

In the United States the first report on measurements of human blood pressure was not made until 1903, by Cabot, to the Association of American Physicians. This constituted a careful clinical study of the effects of strychnine in fifty-eight cases: the Riva-Rocci sphygmomanometer and the original Oliver hæmodynamometer being the instruments employed. A year later another paper on arterial pressure estimation was read by Cabot, and two by Stengel, one of the latter being in collaboration with Stanton, who later played an important part in disseminating knowledge of this subject which has rapidly become widespread. In 1905, Korotkow,[8] of Petrograd, introduced the auditory method of estimation, which since then has been adopted as routine measure.

For many years in clinical medicine, through lack of any satisfactory objective mode of recording arterial pressures, a numerical formula has had to suffice. More recent advance has been along the lines of graphic registration. Erlanger (1904) and Gibson (1907) were the first to provide graphic records of maximal and minimal pressures.

Recognition has now been generally accorded to the greater stability of the diastolic pressure, and thus to its greater diagnostic value as compared with the systolic, to the importance of the differential pressure as an index of

cardiac load, and to the meaning of abnormal variations, whether high or low, from standard limits.

In this condensed historical summary, though much has necessarily been omitted, I have traced the outlines of this subject from its early days in the endeavour to show how tactile and subjective impressions of pulse values, assisted by increasing knowledge of circulatory events, have gradually given place, by the aid of instrumental methods, to accurate and objective determinations of arterial pressure.

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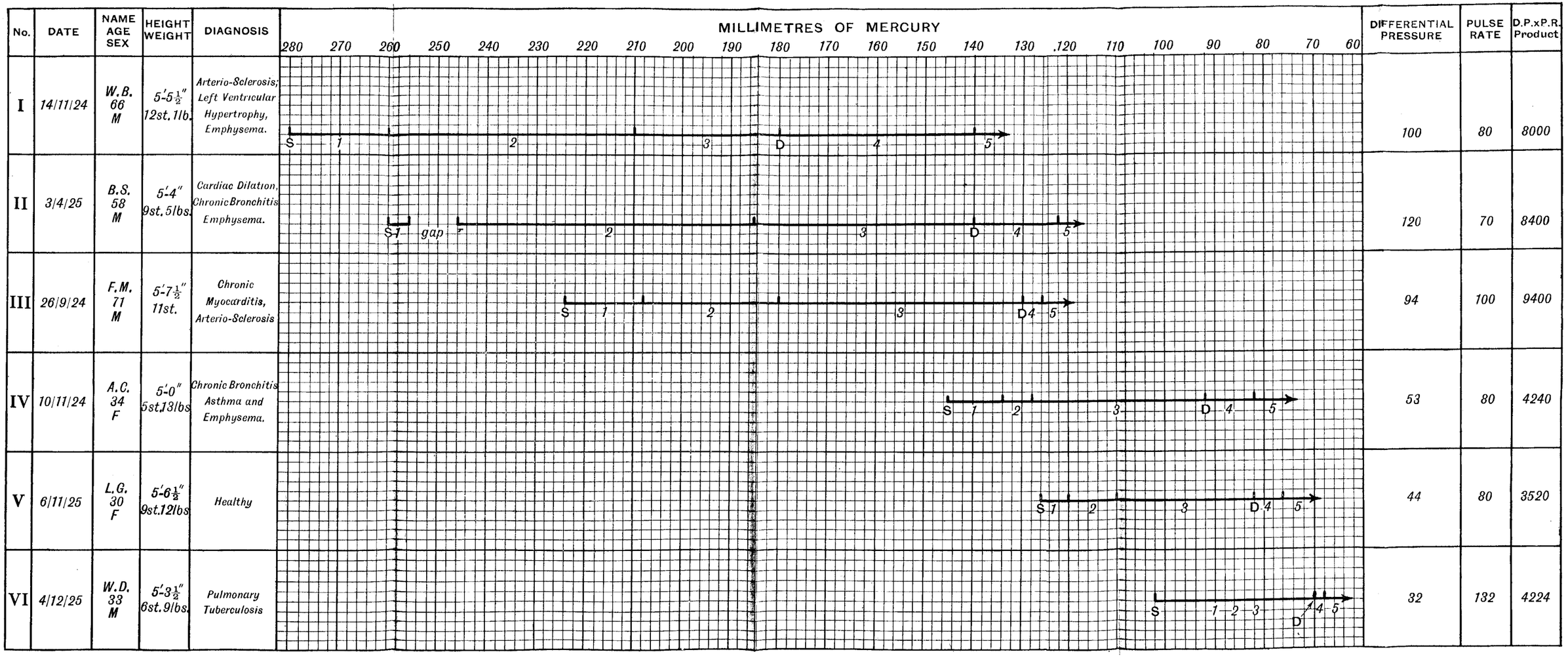

No.	DATE	NAME AGE SEX	HEIGHT WEIGHT	DIAGNOSIS	DIFFERENTIAL PRESSURE	PULSE RATE	D.P.xP.R. Product
I	14/11/24	W.B. 66 M	5′-5½″ 12st. 1lb.	Arterio-Sclerosis; Left Ventricular Hypertrophy, Emphysema.	100	80	8000
II	3/4/25	B.S. 58 M	5′-4″ 9st. 5lbs.	Cardiac Dilation, Chronic Bronchitis Emphysema.	120	70	8400
III	26/9/24	F.M. 71 M	5′-7½″ 11st.	Chronic Myocarditis, Arterio-Sclerosis	94	100	9400
IV	10/11/24	A.C. 34 F	5′-0″ 5st. 13lbs	Chronic Bronchitis Asthma and Emphysema.	53	80	4240
V	6/11/25	L.G. 30 F	5′-6½″ 9st. 12lbs	Healthy	44	80	3520
VI	4/12/25	W.D. 33 M	5′-3½″ 6st. 9lbs.	Pulmonary Tuberculosis	32	132	4224

FIG. 6. -The Author's Complete Arterial Pressure Picture.

A series of specimen cases are here recorded from actual observations to illustrate the author's graphic method of representation on squared paper of various grades of arterial pressure :—
I. = Excessively high. II. = Very high. III. = High. IV. = Suspiciously high, if before age 40. V. = Normal. VI. = Low.

[To face p. 28.