

# FRENCH SUPPLEMENT TO THE LANCET

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## ARTERIAL HYPERTENSION:

ITS CLINICAL MEASUREMENT, PATHOGENESIS,  
PROGNOSTIC VALUE, AND TREATMENT.

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THE earliest research work upon arterial tension and its application to current clinical practice, was carried out at the same time by Potain in France and Bach at Vienna. During the period from 1897 to 1907 practically all the work on the necessary instrumentation, on pressure, and on the methods of use of the knowledge gained, was performed outside France, by Mosso, Sahli, Riva-Rocci, Recklinghausen, Hill and Barnard, Oliver, Janeway, Erlanger, and others. In France, however, Marfan, Teissier, and Bouloumié must be mentioned; also Huchard, who demonstrated in a continuous series of publications the extreme importance of arterial hypertension, making of it the capital symptom of arteriosclerosis. In other countries the various pressures, systolic, diastolic, and mean, together with pulse pressure, were studied; in France up to 1907, before the publications of Amblard (of Vittel), the systolic pressure alone had been the object of measurement and study.

At this time Amblard showed the importance of simultaneous measurements of maximum and minimum pressures, and described the method, based on the work of Marey, and called the "oscillation method," which is now most commonly employed. Shortly afterwards he produced his sphygmometroscope for measurement of the two pressures; by this oscillation method the maximum pressure is obtained automatically and with precision.

Eventually Pachon produced his manometric sphygmometer, a perfected form of Pal's sphygmoscope, the coloured liquid indicator of which is replaced by a needle moved by the modifications of a capsule which retains a constant tension no matter what degree of pressure is applied to it. Since that time new work on arterial tension and its measurement, symptomatology, diagnosis, and prognosis, has constantly been published. Amongst all this research may be especially noted that of Vaquez, Widal, Josué, Amblard, and Gallavardin, in whose recent considerable work on "Arterial Tension in Clinical Practice" are set forth and discussed the most recent advances in this subject. The perfecting of apparatus has facilitated very complete clinical study of tension; the knowledge now at hand of maximum and minimum pressures links this clinical study up with physiological study, and considerably enlarges a hitherto narrow field of clinical research, permitting the hope that in a short while the estimation of the functional power of the heart will become a matter of ease; its difficulty is lessened almost daily by the publishing of fresh information.

### *Causation of Arterial Tension.*

Arterial tension is the result of two antagonistic elements which equilibrate each other: (1) the force of contraction of the left ventricle, which tends to drive the blood from the centre to the periphery, and (2) the resistance offered to the flow by the more or less accentuated closing up of the small peripheral blood-vessels. This resistance to the flow is only susceptible to minimal variations from one moment to another. The force of ventricular propulsion is intermittent in its increase of the blood content of the arterial system. The pressure to which the arterial wall is subjected varies constantly, growing

under the influence of the blood increase to a maximum—the maximum arterial tension, rapidly attained and then rapidly diminishing during the period separating one wave of increase from the next. Maximum arterial tension, then, is the index of ventricular effort; minimum tension is the index of resistance to the blood flow. In a young man the space separating these two pressures—the variable or pulse pressure—is about 60 mm. Hg, and the figures of normal tension range from 120 to 140 mm. Hg maximum, and from 70 to 85 mm. Hg minimum. When either factor—either cardiac contraction or peripheral flow—is modified, modification also results in the figures noted above. If modifications take place in the same direction, the conclusion may be drawn that the degree of tension has varied in the same direction; if the modifications are in opposite directions, no immediate conclusion can be arrived at concerning the modification of the degree of tension. That is to say, it is absolutely necessary, in order to estimate the arterial pressure of a subject, always to note the maximum and minimum pressures.

### *Measurement of Arterial Tension.*

The importance accorded at the present time to arterial tension necessitates precise measurement. Some writers have attempted manometric improvements, others have worked to limit as far as possible the subjective element of measurement, and to substitute the automatism of a tactile instrument. At present two fundamental methods of measurement are employed in France, being undertaken by different means. The first of these methods is that of appreciation of tension by observation of altered blood pressure in a segment of a limb under the influence of a compressor placed on that limb. Of this type is Potain's sphygmomanometer, in which a rubber bulb is used to compress the radial artery, this bulb being connected with a metal manometer which indicates the degree of external pressure on the artery; the indicator, placed on the radial artery below the point of compression, shows the modification of blood pressure caused by that compression; the point of disappearance of pressure indicates the maximum. This instrument, the first to be designed for the purpose, is difficult to handle in spite of its apparent simplicity, and its recording of results is so influenced by personal equation that its employment is rapidly becoming more restricted. Based on the same principle of compression are the method of Riva-Rocci and those derived from it, in which is used a rubber brassard, the moment of disappearance of the pulse at the radial artery being registered. Satisfactory results are thus obtained, mainly because digital palpation is replaced by an oscillating indicator which, as in the methods of Vaquez and Barré, permits very exact measurement of the maximum tension. The Riva-Rocci apparatus also permits study of minimum tension by means of two procedures based on modification of pressure below a compressed point. One of these, called Ehret's method, consists of progressive decompression of a previously distended brassard; the growing amplitude of pulsation is felt by a finger inserted beneath the edge of the brassard. At a certain moment a particular vibration of the pulse is felt; this point past, the pressure rapidly diminishes. The figure read on the manometer at the moment when the amplitude of the brachial pulse begins to diminish is that of minimum pressure. The other method, called that of Korotkow, consists of the same application and inflation of the Riva-Rocci brassard, and of estimation of the sounds made by the passage of the

blood through the brachial artery during decompression. These sounds are noted by means of a simple binaural stethoscope, or of a similar stethoscope in which the end-piece is replaced by a small metal cup at the edge of which is stretched a tense membrane (Laubry's sphygmomanometer). After sufficient education of the ear it becomes possible to recognise the arterial sounds to which the figures of maximum tension correspond, and the cessation of clear arterial sound, to which correspond the figures of minimum tension.

#### *The Oscillometric Method.*

Another method, based on Marey's experiments, is that in which measurement is obtained by the study of modifications in blood pressure induced by various known counter-pressures applied by means of a brassard placed as in the methods previously mentioned. This last is a principle absolutely different from that on which the apparatus described above is founded. In England Hill and Barnard had already utilised the oscillations of a manometric needle to measure minimum tension; I myself first described the technique of measurement of maximum and minimum tensions by this method, which I named the "oscillation method." Its advantage lies in the certainty of the physiological data obtainable, and in the knowledge of maximum and minimum tensions and pulse pressure, and (by means of suitable apparatus) of the action of the circulation during application of various known pressures. My "sphygmometroscope," which I invented for the clinical application of this oscillation method, consists of a brassard 17 cm. broad fitted tightly to the arm and connected with a sensitive manometer of a large size, arranged to record by oscillations of a needle the exact size of impulse, irrespective of the degree of counter-pressure on the elastic capsule. Compression is obtained by means of a rubber bulb, which permits counter-pressure between 50 mm. Hg and 300. After application of the brassard the pressure is raised progressively; the needle of the manometer is displaced like the hands of a watch, soon showing slight oscillations which may be ignored. At about 80 mm. Hg, however, in normal cases a sudden sharper and larger oscillation appears, and the figure then indicated on the dial of the manometer is that of the minimum pressure. If compression is continued a zone of large oscillations is observed, followed by diminution of their amplitude and by their final disappearance. The point of their complete extinction is that of maximum tension. The modifications of amplitude can also be studied during progressive decompression after previous establishment of a high counter-pressure. This method, which is used equally with Pachon's sphygmometric oscillometer, presents one considerable inconvenience; in most cases the observer is compelled to fix the maximum tension arbitrarily. Actually, the oscillations of the needle do not appear abruptly; before they become marked there is a zone, sometimes extending over several centimetres, in which small oscillations of practically equal size take place, due, as I have shown, to the circulatory beat at the edge of the brassard in the zone of the limb just above it. To remove all subjectivity from the reading of the result I have modified my earliest apparatus; and at present my "sphygmometroscope" admits of automatically exact measurement of both maximum and minimum tensions.

#### *Apparatus Now in Use.*

The apparatus at present in use consists of a leathern brassard 17 cm. wide, lined with two rubber bands, not intercommunicating, but both connected with a compressor and furnished with a delicate manometer of large size. A tap, fitted with a screw, secures it in position, whether inflated or deflated, or half inflated—i.e., the position for record of oscillations—when only the lower band is connected with the manometer. In this way the technique of measurement is rendered rapid and simple. A counter-pressure is first obtained, so that the mano-

metric oscillations have entirely ceased; then decompression is begun, centimetre by centimetre, care being taken to keep the manometer in communication with the lower band only. The highest point at which the slightest oscillation reappears is that of maximum tension, for at that point oscillation can only be due to the blood stream and not to the pressure of blood against the upper edge of the brassard, where there is no counter-pressure. The maximum pressure may thus be read accurately within a millimetre. Minimum pressure is obtained by continuing the decompression; first the oscillations increase, and then are suddenly reduced again; the reading at the latter point gives the minimum pressure.

#### *Consideration of Various Curves.*

In this way may be obtained maximum and minimum pressures, pulse pressure, and lastly the oscillometric index, which has recently been applied to clinical work. In this communication only passing reference to its interest can be made; readers are referred to the bibliography. Certain writers have recently attempted to represent by a curve drawn by hand, after examination of the arterial circulation by the oscillatory method, the modifications of the arterial beat shown by the needle, and thereby to establish an oscillometric index and obtain a curve which may be studied at leisure. This curve is generally obtained by taking for abscissa the successive intensities of counter-pressure, and for ordinates the corresponding amplitudes of oscillation of the needle. Harlé (Bordeaux), insisting on the greater interest of a double tracing for purposes of clinical study of the normal and pathological functions of the heart and blood-vessels, has proposed a curve composed of two branches, of which the upper gives the heights reached by the needle above its starting-point, and the lower curve the depths reached below that point. According to Pachon: (1) In the absence of any complicating factor in the artery concerned, the oscillometric index expresses the strength of the cardiac impulse; (2) in the absence of any complication concerning the cardiac impulse, the oscillometric index expresses the vascular calibre—i.e., the state of constriction or relaxation of the arterial vessels. But the clinical application of these data is a very delicate matter, and I only refer to the methods by which they are obtained in order to indicate the direction now taken by research into methods of diagnosis of functional disorders of the cardio-vascular system.

No curve traced by hand can better a direct tracing, and inquiry is now being directed towards the construction of apparatus for graphic expression of the circulation of blood under various counter-pressures. Strohl has made use of a contrivance designed to record the oscillations of a manometric needle under constant conditions and varying pressures, which has enabled him, together with Barré (Strasbourg), to publish curves reproducing the modifications of the arterial beat under the influence of increasing and diminishing counter-pressures, and has provoked interesting discussion on the precise points of fixation of maximum and minimum pressures. But the drawback of this contrivance lies in its clumsiness for clinical use; it is more of a laboratory apparatus. I have now attached to my own "sphygmometroscope" a recording contrivance; the combined apparatus is neither heavy nor difficult to carry about, and it permits of rapid and accurate registering of variations in the arterial beat, disturbances of rhythm, and maximum and minimum pressure points.

The graphic study rendered possible by means of these apparatus has already roused numerous discussions, notably concerning the points on the curves at which the maximum and minimum pressures are to be recognised. For close consideration of this point, I must refer readers to the works on the subject.

#### *The Study of Hypertension.*

The perfecting of apparatus has permitted real and rapid progress to be made in knowledge of hypertension and its consequences. First, definition of

true arterial hypertension has been made possible without danger of confusion with other circulatory states having the appearance of hypertension. Measurement of maximum tension alone, when abnormally high, as in patients with aortic insufficiency, has led to these cases being considered hyperpietic; but the complementary information given by concomitant measurement of minimum tension furnishes an important distinction between pure aortic insufficiency—e.g., in typhoid and rheumatism—and insufficiency due to specific aortitis. In the former class the affected valve no longer opposes during diastole the reflux of blood from the aorta towards the ventricle (this condition is indicated by a diastolic murmur); an abnormal depression of tension in the arterial system at the moment of diastole is the result. There is, then, diastolic hypotension. But as it is necessary to ensure circulation that the blood be impelled from the heart towards the periphery under a certain mean pressure, minimum hypotension must be compensated by an exaggeration of maximum tension; thus in pure aortic insufficiency are found habitual tensions of 170–60, the maximum exceeding the normal maximum of 140 by 30 mm. Hg and the minimum not attaining the normal minimum of 80. In specific aortic insufficiency the pressure readings may appear to be those of pure insufficiency so long as a renal sclerosis has not produced its customary effect of hypertension. At that moment it is not possible to distinguish the nature of the trouble by measurement of tension alone. Only when renal sclerosis has progressed sufficiently does distinction become possible, for at that moment the elevation of minimum tension to the normal level—or above it—indicates the specific nature of the cardiopathy. With the figures 240–100 mm. Hg, for example, specific aortic insufficiency is certainly present, with an already considerable renal lesion. I do not emphasise the fact that the transient hypertension arising by painful crisis in tabes, or that due to excitation of a heart already prodigal of effort, or an excessive tachycardia, can slightly raise the pressure; I prefer to confine my attention to true persistent hypertension.

#### *True Persistent Hypertension.*

Two principal factors may modify the pressure: exaggeration of the force of ventricular contraction (maximum hypertension) and exaggeration of resistance to the flow (minimum hypertension). Except in cases where under some transient influence—e.g., emotion—contractile energy is momentarily exaggerated, the maximum tension is only exaggerated to the precise degree necessitated by that elevation of the minimum tension to which it is subordinate. This minimum hypertension can be referred to two mechanisms: (1) The venous congestion which, supervening in advanced phases of valvular affections, cannot be without effect on the flow of blood in the small peripheral arterioles; (2) much more important, the obstruction of these small arterioles. The true hyperpietic subject is one in whom a peripheral spasm raises the minimum tension and renders necessary a maximum tension sufficient to maintain the outward circulation from the centre to the periphery (Amblard). Normally minimum tension is about 70 mm. Hg, and maximum about 130 mm Hg, the various arterial pressures which arise being contained within the limits of 60 mm. Hg. A minimum hypertension of 40 mm., for example, necessitates a maximum hypertension of 100 mm. But various intervening factors (notably the elasticity of the arterial wall, which varies with the causal malady and with functional abnormality) so modify normal functions that the rise of minimum tension is always accompanied by a proportionately larger rise of maximum tension—e.g., a minimum of 120 mm. Hg demands a maximum of from 200 to 220 mm. Hg. If the necessary space between the maximum and minimum figures is maintained, circulation will be ensured under conditions abnormal but relatively satisfactory. The

cardiac muscle becomes hypertrophied to perform the extra work imposed upon it by the anatomical or functional defect of the arteries until it slowly or rapidly breaks down. Clinically, hypertension sometimes appears, frequently associated with more or less marked troubles relating to renal insufficiency; but sometimes very careful investigation—qualitative and quantitative—of renal function and of the rhythm of urinary elimination gives no sign indicating disturbance of these functions; arterial hypertension is then the only symptom reported. In the first instance it is easy to connect the hypertension with a renal lesion and to act accordingly (hyperpietic nephritis of Widal); in the second instance it is more difficult to confirm this relation.

Any infection can cause lesions of chronic nephritis and the condition of hypertension. But the latter seems particularly closely allied to the evolution of syphilis (which disease should always be examined for in a hyperpietic patient), apart from several very clear cases where supervening events can be unhesitatingly connected with a recent affection (e.g., scarlatina) frequently complicated by nephritis, or with an evident intoxication (e.g., lead poisoning).

#### *Clinical Divisions of Hyperpietic Subjects.*

Clinically, hyperpietic subjects may be divided, from an ætiological standpoint, into several groups, as follows:—

1. Patients, usually young, in whom hypertension follows more or less recent scarlatina or acute infection.
2. Cases of industrial lead poisoning, common in the labouring class, exceptional in other classes.
3. Young hereditary syphilitics, the type of whom is the arterio-sclerotic of about 25 years of age, with hard arterial walls, whose radial arteries "roll" beneath the finger, who have apparently healthy kidneys and an arterial tension of about 170–100 mm. Hg, and in whom arterial hypertension is connected with a sclerosing process resulting from specific hereditary dystrophy.
4. Hereditary syphilitics at a later age, when renal lesions at first insignificant have developed; and also other syphilitics, 20 years after their infection, the type of whom is the hyperpietic patient of 45, who admits infection, or denies it deliberately or in good faith.
5. Senile sclerotics, the type of whom is the hypertensive patient of 65 or 70, in whom the hypertension is of mixed origin, being partly functional, due to peripheral arterial spasm, but chiefly connected with senescence of the arterial walls.
6. Other patients in whom syphilis is not detected, who are perhaps sound in that respect, but have gout or diabetes (although in many cases of hypertension during these two diseases syphilis, hereditary or acquired, must be suspected, and only dismissed from possibility after very careful examination).
7. Lastly, patients with transient forms of nephritis of unknown infective origin, in whom the hypertension remains constant for a while, but diminishes with the causal affection.

I consider all permanent hypertension to be of renal origin, and clinical evidence seems to support the contention; the hypertension, at first isolated, has added to it by degrees various disorders, not previously present, of renal insufficiency. When there is no albuminuria, no abnormality of diuretic rhythm, no disorder of renal function, it does not follow that the kidney is anatomically intact; possibly we owe this apparent anomaly to insufficient means of appreciating renal function. In the statistics of Janeway, and in those of Fischer, dealing with post-mortem findings in numerous cases presenting permanent arterial hypertension, the cases showing indubitable renal lesions were very few.

#### *Causation of Hypertension.*

It is, however, the possible existence of these cases of isolated hypertension which has permitted Gull and Sutton to attribute to hypertension another cause—namely, hindrance to the flow of blood by generalised arterial lesions, the kidney being ultimately affected, but the morbid condition arising in the arteries. The results of Tripier and Brault seems to run counter to this theory; these writers scarcely recognise the existence of generalised arterio-sclerosis, admitting only that of lesions localised in

certain regions. The dependence of arterial hypertension on local and extrarenal arterial trouble, as affirmed by Teissier (Lyons), does not seem to me to be admissible. Lastly, Huchard, who upheld the connexion between hypertension and renal sclerosis, wished to attribute hypertension to a generalised spasm of the peripheral arterioles—a spasm developed under the influence of retention in the organism of vaso-constrictor poisons owing to insufficiency of urinary excretion. If this mechanism is the most probable, the origin of this spasm has not been attributed by all writers on the subject to the same cause. Besides Huchard's theory—which has in its favour results of experience, for in a very general sense the meatless diet leads to a fall of pressure in hyperpietic subjects—other factors have been invoked. The work of Vaquez and Josué, Widai and Boidin, leads readers to think that arterial hypertension can be provoked in subjects presenting no nephritis, and in subjects in whom it has already been found, by suprarenal hyperplasia, and that even in those cases where nephritis is evident hypertension is only brought about by a reaction of the suprarenal capsules. The action on the organism of adrenalin, investigated by Sergent and Léon Bernard, gives a certain probability to this hypothesis. There are, however, two objections to acceptance of this suprarenal origin of hypertension—first, that anatomical lesions are usually found in the cortical region of the gland, since the medullary region is considered to be productive of adrenalin; secondly, that numerous observations have shown the inconstancy of suprarenal hyperplasia in patients suffering from accentuated hypertension, since it was found to exist in other cases where hypertension had never been manifested. Research into the adrenalin content of the blood of hyperpietic subjects has given such contradictory results in this connexion that it is impossible to draw any other conclusion.

#### *Possible Renal Origin of Hypertension.*

Is the kidney itself productive of a vaso-constrictor substance, accumulation of which results directly in arterial hypertension? A non-dialysable and hyperpietic substance has been extracted from the cortical layer of the kidney—i.e., renin—which is found in the renal veins, but the results of this research have not been confirmed in practice. The study of cholesterin content in the blood of cases of Bright's disease—which shows a large increase of this substance—has led Chauffard to consider that there may be a connexion between hypertension and the accumulation of cholesterin in the blood, the latter being considered as a defensive element. The presence in the blood of products of the oxypurins—hypoxanthin, xanthin, and uric acid, which are powerfully hyperpietic—may also, according to this writer, originate modifications of pressure. That hypertension may frequently be associated with chloruræmia or with azotæmia is easily understood once we have admitted its essentially renal origin, but it seems to me that it cannot be derived from these two affections; it is frequently met with separately, representing, sometimes at least, a type apart—hyperpietic nephritis—accompanying the two other groups, azotæmic and chloruræmic nephritis.

#### *The Latent Phase of Hypertension.*

Recognition of hypertension can only be made possible by systematic research; in an apparently healthy subject the first phase of its evolution is not betrayed by any functional symptom noticeable to the patient himself; this is the "presclerotic" phase of Huchard, the "latent" phase of Gallavardin. There may exist no functional symptom at all, not even dyspnoea—which nearly always dominates the clinical picture at some stage of the condition—or albuminuria, or disorders of urinary elimination. Radioscopic examination, however, often shows slight hypertrophy of the left ventricle, for it is the heart's effort in face of the demand made upon it which permits this phase of latency or compensation. As to the duration of this phase, the possibility of

checking it, and the fatal results of the disease, practical difficulties prevent clear answers being given. Most frequently hypertension, when unaccompanied by any appreciable disorder, and only noted by accident, has doubtless existed for some time; in some cases in the course of acute nephritis, the increase of pressure will be rapid; but in chronic nephritis the slowness of development, the possible arrest of progress in the condition, and the absence of knowledge concerning its onset, render it impossible to make even an approximate estimate of the latent phase. As to a return to normal of a pressure constantly raised for some time, although it is never reported after the condition is clearly established and allied with other signs of renal insufficiency, it is difficult to deny its possibility in cases where hyperpietic conditions have been under observation from their onset.

#### *The Phase of Sclerosis.*

To this first phase succeeds another, which is often the earliest to be clinically observed—a phase in which other symptoms of a toxic nature are ranged alongside hypertension; this is Huchard's period of sclerosis. To name only affections of the whole circulation, there arise certain aphasias and transitory pareses, epistaxis, retinal hæmorrhage, tinnitus aurium, and dyspnoea, the latter due partly to mechanical hindrance of cardiac function, and partly to intoxication of the subject. The evolution is variable, and the terminal events which arise from toxic troubles only, or from their association with mechanical disturbances, may be determined exclusively by circulatory troubles, and are then manifested by vascular ruptures due to excess of pressure in vessels previously weakened by the causal malady or by cardiac insufficiency.

#### *Myocardial Insufficiency and Pulmonary Œdema.*

Insufficiency of the myocardium appears rapidly or slowly; when rapid there is acute œdema of the lung. In a hyperpietic patient, often apparently healthy but generally presenting aortic insufficiency together with hypertension (type of pressures, 250–100 mm. Hg), a rapid series of sequelæ is disclosed by extreme dyspnoea, pain at the apex of the lung, and characteristic salmon-coloured sputum; at the same time peculiar râles indicate rapid serous invasion of the lungs. This is the crisis of acute œdema, which sometimes results in death in several hours or even in a few seconds, before any help can be given to the patient. But if the patient can be brought through the crisis by therapeutic means, amelioration may be obtained, and the pulmonary sequelæ may die down, sometimes disappearing completely. But later, either with no apparent cause, or following a gap in treatment, or some emotion, the disorder will reappear, more or less rapidly—ending in death, or receding into the background again, but always threatening reappearance, and constituting a chronic subacute form of pulmonary œdema. The progress of the arterial pressure explains these various sequelæ. At the moment of appearance of the earliest sequel the pressure becomes rapidly modified. At first there is parallel rise of maximum and minimum pressures. The maximum is raised from one to two points, the minimum from two to three; thus the cardiac effort becomes much greater. Then the state of pressure is suddenly modified—the minimum falling three or four points, but the maximum even more. At this moment death may supervene, if the maximal depression persists. But with appropriate treatment (first bleeding, then cardiac tonics) the modification may be reversed; the minimum will rise one or two degrees, the maximum two or three; and the patient presents for a time pressures of maximum 180 and minimum 100 mm. Hg. Amelioration becoming more pronounced, the maximum tension may rise again, but never to the figure recorded before the crisis. Then it will once more fall rapidly, perhaps to rise no more, perhaps again to rise a few degrees; by a series of weakening changes it may come about that



the pressure is not compatible even with very defective function of the vascular system, and the patient dies of progressive asphyxiation.

In other cases the heart fails slowly, with a terminal phase of progressive weakening of maximum pressure, whilst the minimum pressure remains at a sufficiently high level on account of the spasmodic toxic element which previously necessitated its elevation, and of the disorder induced in the peripheral flow by venous congestion associated with dilatation of the cavities on the right side of the heart. This failure is marked by an important auscultatory sign—i.e., presystolic gallop—and by modifications of the pressure curves during routine exercises, which were used, notably during the war, to record the coefficient of myocardial resistance to effort in soldiers undergoing examination. When the heart is beginning to tire, and can scarcely continue to perform the work entailed by persistent hypertension, the maximum and minimum pressures rise, but the elevation of maximum is not what it should be, because of ventricular deficiency; the necessary effort is made through a rapid and considerable increase of pulse-rate. Signs of circulatory trouble (œdema of the limbs, hepatic and pulmonary visceral stases, ascites) habitual in valvular patients correspond, in the last phase of arteriosclerosis, to these disorders of pressure.

#### *Importance of Recording Pressures.*

The possibility of recording hypertension when the condition apparently exists alone and only displays a slight degree of renal insufficiency makes of the pressure reading a clinical sign of capital importance, since it permits treatment of nephritis from its onset, before it has been manifested by any trouble otherwise appreciable. At this stage modifications of diet and certain drugs have more chance of slowing down the process of sclerosis; arterial hypertension is in this case an important danger-signal—its mere appearance proving an apparently completely healthy subject to have already received serious damage. As regards prognosis, hypertension seems to assume a degree of gravity in accordance with the age of the subject. In the aged, where the sclerotic and atheromatous process is long established and where renal insufficiency often exists, hypertension does not seem so important as in younger subjects. By the latter are meant patients of 45, in whom the symptom is always serious, and sometimes very serious. Although the gravity of very high figures cannot be denied in a general sense, the degree of maximum hypertension does not exactly indicate the patient's danger. The relation between maximum and minimum is more important in this respect, and the pulse pressure, which shows the functional value of the left ventricle. As the minimum pressure is raised, the maximum pressure is also raised—for reasons which cannot be detailed in this short study—but to a greater extent.

#### *Treatment of Hypertension.*

In spite of the knowledge recently acquired concerning the pathogenesis of hypertension, the secretion of hyperpietic renal substances, and the excess in the blood of adrenalin, cholesterin, or uric acid, therapeutic progress has not been remarkable. Whatever may be the starting point, the essential cause, of excess of pressure, it appears that a slow progressive thickening of the kidney necessitates a continuous and progressive exaggeration of arterial tension in order to ensure satisfactory urinary depuration. The excess of minimum pressure, due to peripheral spasm, only exists to the degree in which it is useful, and the excess of maximum pressure only to the degree rendered indispensable by the elevation of minimum pressure. It would be illogical to attempt directly to lower the maximum pressure, for such a lowering would only express the insufficiency of myocardial effort to perform an indispensable supplementary task. What must be aimed at is the lowering of maximum pressure by removal of its cause—i.e., the elevation of the minimum pressure. The latter is due to toxic disorder;

as Huchard has constantly pointed out, the treatment of arterial hypertension resolves itself into treatment of organic intoxication by decreasing the supply of toxin with an appropriate dietary régime, and by facilitating the removal of endogenous toxins by stimulation of normal functions of intestinal, renal, and cutaneous elimination.

With the exception of certain cases in which sudden pressure arises during habitual hypertension and necessitates urgent methods (bleeding, cupping by scarification, use of amyl nitrite, sodium nitrite, erythrol tetranitrate, &c.), the treatment of hypertension is lengthy. Together with antitoxic measures, treatment of the arterial walls is attempted; potassium iodide has been used with success, although the mechanism of its action remains unexplained. At the same time direct attempts to reduce pressure may be cautiously initiated; the Royat baths are indicated as inducing a lowering of pressure. Bubbly and thermo-electric baths, particularly the latter, have also a violent effect upon circulation; their administration should be carefully supervised, and since the effect produced is rapid but transitory, they can only be considered as an adjuvant form of treatment. Electric treatment with high-frequency currents is also an adjuvant, having no clearly perceptible effect on hypertension, but being of use in the restoration of sleep and diminution of nervous troubles associated with hypertension in irritable patients.

It must be borne in mind that the condition begins, has its continuation, and ends through intoxication (Huchard); a diet as little toxic as possible is therefore indicated. Milk, cream, butter, fresh cheese, well-ripened fruit, and vegetable soups form the basis of treatment. It cannot be too often emphasised that only by continuous adherence to the alimentary régime can a marked and lasting lowering of pressure be obtained. Only small quantities of salt must be permitted, less on account of the disputable hyperpietic action of sodium than on account of knowledge of the nephritic origin of hypertension and the fear of more or less latent chloruria.

Although the absorption of meat seems in some hyperpietic patients to have no effect on the pressure figures, it is nevertheless certain that in the majority of cases the dropping of meat diet is accompanied by a diminution in those figures, together with disappearance of toxic troubles accompanying pressure, notably dyspnoea and headache. Although a continued milk, or milk and vegetable, diet may often be unacceptable to the patient, and although in some cases it may even be impossible to overcome individual intolerance to it, there is at present no other means of maintaining a lowered pressure. Intestinal stasis is combated with frequent purgatives, and diuresis is ensured by the use of theobromine; the former difficulty of the insolubility of this very active medicament has been overcome by utilisation of a new composition sufficiently soluble even for subcutaneous injection. To various diuretic preparations may be added tisanes of couch-grass, cherry stalks, and maize. A few doses are sufficient.

In conjunction with these methods of securing diuresis it is necessary to prescribe a diuretic mineral-water cure at Vittel or Evian; the waters at Vittel are more active, being cholagogue and laxative as well as strongly diuretic. Diuretic cures cannot be satisfactorily carried out except in these spas. There is no doubt that the ingestion of large quantities of water augments œdema or raises the arterial pressure. It can be affirmed, in accordance with the views of specialists in these treatments, that, far from augmenting the quantity of water retained in the system, the diuretic cures—however paradoxical the statement may seem—are essentially dehydrant cures; the dechloridising action of the Vittel waters also contributes to the explanation.

In conclusion, the foregoing has shown that if the diuretic cures almost always provoke hypotension on rational lines, this is only one of their many advantages; the disappearance of other signs of intoxication, when the latter are present, affords a proof of the fact.