CONTENTS

HIGH BLOOD PRESSURE AND ITS TREATMENT IN GENERAL PRACTICE WITH PARTICULAR REFERENCE TO A SERIES OF 100 CASES TREATED BY THE AUTHOR

By

HAROLD WILSON BOWYER MB. [illegible]h.B.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

CONTENTS

SECTION 1.

INTRODUCTION.

I think it can truthfully be said that the most interesting problems in Medicine are those that are most baffling.

Some years ago Ralph Major[1] wrote these words, "If our knowledge of the etiology of arterial hypertension is shrouded in a certain haze, our knowledge of an effective therapy in this disease is enveloped in a dense fog."

A study of some of the vast literature on this subject does not greatly clarify the obscurity. Rather does it leave one more bewildered by the multiplicity of theories advanced, and by the astonishing number of treatments advocated.

Be that as it may, it at least constitutes a challenge, and according to Sir Humphrey Rolleston,[2] a challenge to the general practitioner.

"The treatment of patients with high blood pressure," said Rolleston, "depended on wise discretion based on personal knowledge of the individual. Several possible etiological factors might be at work," he contended, "the elucidation of which should be more within the power of the regular medical attendant than of anyone else."

Special facilities for useful research are thus available to the general practitioner.

By virtue of my position as a family practitioner in a moderately large mixed industrial practice in the fine-cotton spinning town of Bolton, Lancashire, I have, since 1926, taken the opportunity of studying some of the problems connected with high blood pressure and its treatment.

A critical study of the signs and symptoms exhibited by 100 patients showing arterial hypertension, and a consideration of the results obtained in their treatment, have allowed me to formulate certain definite conclusions.

Close contact with, and knowledge of the environment, daily tasks, worries, and difficulties of these people have somewhat modified the views regarding prevalence, sociology, etiology and prognosis which I had previously gained from standard text books on the subject.

Even the usually accepted statistics and life-tables of life insurance companies are to me rather suspect as far as they relate to systolic blood pressures. I hope to show that in the past too much significance has been attributed to a raised systolic blood pressure per se.

By considering the symptomatology and the physical characteristics of this group of patients I hope to show that there is such an individual as a potential hyperpietic who, in response to certain stimuli, developes the condition of primary hypertension. This characteristics will be described.

The significance of hypertension as a physical sign, and later as a fully developed condition of Hyperpiesia will be considered.

The question of secondary hypertension will also arise. Blood pressure readings obtained over a number of years will, I think, indicate a brighter prognostic outlook than is generally visualised.

The treatment of raised blood pressure will be discussed with reference to the difficulties encountered in General Practice and in particular to the results obtained by drug therapy.

From a comparative point of view and to provide a basis for discussion a general outline of the condition of raised arterial blood pressure will be given in the opening sections. This will embrace the generally accepted views regarding the etiology, pathology, prevalence, signs, symptoms and prognosis of high blood pressure, and will contain a brief historical survey.

In order to avoid undue repitition it is proposed to discuss the question of treatment in Section 4.

Acknowledgments.

Many of the cases in this series have at various times been referred to the Bolton Royal Infirmary for pathological investigation, functional and clinical tests.

I gratefully acknowledge my indebtedness to the Honorary Pathologist, Dr. W. Rolland, for his advice and for his useful reports on these cases.

Several of the cases have been examined by Regional Medical Officers of the Ministry of Health in the course of their duties. Their reports and findings have in some cases been utilised as confirmatory evidence.

SECTION 2.

GENERAL REMARKS.

DEFINITIONS OF GENERAL INTEREST.

Blood Pressure is that pressure which the blood exerts at a given instant upon a given point in the circulatory system. Clinically the term is held to include Diastolic, Mean and Systolic pressures.

Arterial blood pressure therefore is not a fixed and definite entity for any individual, but an amount which is constantly varying between certain minimum and maximum values.

Systolic Pressure. When the ventricles are in the process of contraction, i.e. during ventricular systole, the output from the left ventricle distends the walls of the arteries in the form of a wave. The crest of this wave corresponds with the greatest or maximal pressure - the systolic pressure.

Diastolic Pressure. Between each ventricular contraction, i.e. when the heart is in the resting stage of diastole, the minimal pressure or diastolic pressure occurs. This is the pressure to which the heart, valves and blood vessels are continuously subject, while the systolic pressure on the other hand is intermittent and super-added.

The Pulse or Differential Pressure is the difference

between the systolic and diastolic pressure.

The Mean Pressure is the average pressure at a given point. The terms "systolic pressure" and "diastolic pressure" as used later in the context are intended to indicate the pressures shown on a mercurial sphygmomanometer (Beaumanometer) and taken on the brachial artery or arteries above the elbow by the auditory method with the patient in a recumbent position.

The diastolic pressure has been taken to be the pressure indicated when the pulse sound completely disappears. Technically, however, it should be taken when the clear loud thud of the third phase gives place to a dull sound - described as the beginning of the fourth phase.

The systolic pressure has also been checked by the tactile method, used on the radial and brachial arteries.

Final readings have been taken after several trials.

Hyperpiesia or Persistent Hypertension[3] has been defined as 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.

The name Hyperpiesia was first used by Sir C. Allbutt[3] to delineate a definite clinical syndrome "deserving the name of a disease" as he puts it, and which does not follow the

clinical course of Bright's Disease. In modern terminology the condition is described as Essential Hypertension (a) Benign or (b) Malignant in type.

Hyperpiesis or Hypertension, on the other hand, merely means high arterial blood pressure. Sir C. Allbutt originally applied the term hyperpiesis to a primary state of high arterial pressure characterised by its persistency and occuring apart from chronic renal disease, but he later amplified the term to include any high pressure recorded by the sphygmomanometer. It is not a specific disease but a clinical sign which may be manifest in a large variety of functional and organic conditions. It may therefore be applied to a blood pressure which is raised above the normal level.

Arteriosclerosis or Atherosclerosis. Arteriosclerosis is a pathological name indicating a condition of increased rigidity of the arteries, due to a thickening of their walls. The process commenses in the intima and extends to the media and adventitia.

The arterial tree as a whole, or in parts, may undergo injury or deterioration resulting in arteriosclerotic changes, in the course of more than one series of morbid events. In arteriosclerosis the blood pressure may or may not be increased. Frequently, however, hypertension and arteriosclerosis are found together, but their relationship to each other is a very

moot point which must be considered later.

Normal Blood Pressure.

Before further considering the question of high blood pressure it is necessary to define the limits which are considered as normal.

Various criteria have, during the past twenty years, been advanced to indicate whether a blood pressure reading should be considered as normal, high, or even pathological.

Osler[4] considered that a permanent systolic pressure above 160 mm. of Hg. should be called high and stated that in Hyperpiesia it was usually above 180 mm.

H. Batty Shaw[5] in his clinical series of cases of Hyperpiesia and Hyperpiesis took as his basic figure 150 mm. and only considered as hyperpietics those whose blood pressure was 150 or more.

More recent workers,[6] & [7] however, have realised the even greater importance of the diastolic pressure and they take cognisance of this in their diagnosis and in their criteria of abnormality.

For clinical purposes it may be said, according to Halls Dally,[6] that any diastolic pressure of 105 mm. or over, and any systolic pressure of 160 mm. or over, may be regarded as Hyperpiesis.

Incidentally, the same author considers that a diastolic

pressure persistently above 90 mm. is to be regarded with suspicion, while one of 95 or over is definitely pathological.

According to Reginald Hilton[8] a diastolic pressure of 100 mm. is ominous. It used to be taught that a close approximation to normal systolic pressure could be obtained by adding 100 to a person's age. Halls Dally[9] states that this is erroneous and most inaccurate. As a rough and ready guide 100 should be added to half the age for the normal systolic pressure, and 75 added to a quarter of the age to obtain the diastolic figure.

Life Insurance Companies are guided by published tables based on averages obtained from many thousands of cases of both sexes.

In Symmond's table, for example, for age 60 and over (males) the normal systolic pressure is given as 135·2 mm. and the diastolic as 86·6. For females the figures are 135·5 mm. and 89·8 mm. respectively.

A variation of 15 mm. for systolic pressures is allowed on the published figures, while 10 mm. is considered as a normal diastolic variation.

The average pulse pressure up to 50 years is 44 mm.

From these authoritative expressions of view it would seem reasonable to consider any systolic pressure of over 160 mm. as hyperpietic, or any diastolic pressure over 95 mm.

as pathological.

These figures, therefore, will be used as arbitrary standards of assessment. It is interesting and perhaps not without significance to consider in relation to the foregoing statements the criteria adopted by Medical Boards in their assessment of blood pressure readings in men over 35 years.

I quote from the instructions for the Guidance of Medical Boards[10] issued by the Ministry of Labour and National Service.

"Hypertension. - The blood pressure should be taken in the case of all men over 35, or in any case of unexplained cardiac enlargement, or when arteriosclerosis is suspected. A man with a diastolic reading of between 90 and 100 millimetres (as estimated when the pulse sound disappears) should not be placed higher than grade III. If the diastolic reading is 100 millimetres or over the man will be placed in grade IV."

It will be noted that no mention is made of systolic pressures.

This has since been altered and instructions given to reject any man whose diastolic blood pressure exceeds 100 mm. of Mercury.

Present Views on Etiology.

Until the elaboration of the Sphygmomanometer a simple mechanical theory to account for raised arterial blood pressure

held sway.

It was argued, quite logically, that if the arterioles and capillaries of the arterial bed were contracted or narrowed by occlusive disease, or by the degenerative changes of advancing years, increased muscular work must fall upon the heart.

If the resistance thus produced was to be overcome a rise in arterial pressure must inevitably occur. The increased pressure would be brought about by the extra power exerted by the pump or heart, together with an increase of friction within the blood vessels.

We may accept this argument as far as it goes, but unfortunately it makes no attempt to explain how the increased peripheral resistance is produced. Moreover, the information obtained by later types of sphygmomanometer shows that hypertension may occur without evidence of arterial disease.

I. The Mechanical Theory.

The arguments above outlined may be thus summarised:-

The wear and tear of the cardio-vascular system, especially when associated with the degenerative changes of old age, causes an increased resistance to the blood stream. Similar changes may also supervene at an earlier age in those whose blood vessels are inherently poor.

If these structural changes are sufficiently widespread,

and if vaso-dilatation elsewhere does not sufficiently compensate for them, a rise in blood pressure will result.

Any conviction that such a simple physical view can carry must necessarily be limited to hyperpiesis occurring in the decrescent type of arteriosclerosis as described by Allbutt,[11] or to the hypertension which may follow arteriosclerosis.

II. Renal Origin of Hyperpiesis.

High blood pressure must be considered, at any rate in many circumstances, as a symptom of a known primary disease.

In many renal diseases, for instance, hypertension is a secondary symptom. In diffuse glomerulonephritis, while a moderate rise of pressure may be found in the acute phase, in the chronic condition a very high level of hypertension may be attained.

In over 50 per cent of cases of Polycystic kidney moderate or severe hypertension occurs.[12]

Chronic pyelo-nephritis is stated to be usually associated with raised arterial pressure.[12]

On the other hand, T. Izod Bennett[13] says that thousands of cases labelled as chronic interstitial nephritis are cases of Essential Hypertension in which a generalised sclerosis of smaller blood vessels is producing renal effects and which are in reality only a minor aspect of the complete picture. Prof. W. W. Thompson[14] is of the same opinion. Writing of the

kidney of essential vascular hypertension he states that the term chronic interstitial nephritis included many examples of nephrosclerosis or arteriosclerotic kidney.

I am inclined to agree that in a well marked case of essential hypertension the kidney changes which are found are part and parcel of the disease.

I prefer to disregard the assertion of H. Batty Shaw[15] that hyperpiesia was present in cases whose kidneys were to all clinical intents and purposes normal and were found to be normal on post-mortem examination. His definition of hyperpiesia is not one which would be accepted to-day, including as it did cases with systolic blood pressure of over 150 mgm. without reference to diastolic pressure.

I can well believe, however, that in the early or initial stages of benign hypertension clinical evidence of renal damage is lacking. My case histories support that contention.

McDowell[16] in his Oliver Sharpey Lecture (1941) indicated that it is possible to produce a persistent high blood pressure of renal origin without there being evidence of renal disability from a study of the urine.

Platt[17] in a brief outline of the experimental work of Goldblatt and his co-workers showed that constriction of the renal arteries so as to reduce the blood flow through the kidneys, regularly resulted in elevation of the systemic

blood pressure.

If the constriction was moderate a clinical picture of benign hypertension resulted, but if it was severe renal failure with widespread sclerosis of arterioles was produced, similar to that found in cases of malignant hypertension.

Wilson and Byrom[18] indeed have produced evidence to show that unilateral renal ischaemia may cause hypertension, and have proved that in such a case removal of the ischaemic kidney immediately reduces the blood pressure.

Hypertension, as has been shown, very often accompanies chronic kidney disease. In essential hypertension kidney changes varying in degree according to the severity of the condition are part of the general picture.

These changes can be produced experimentally by constricting the renal arteries and they are accompanied by an increase of arterial blood pressure.

Renal damage, therefore, of such a degree as to result in constriction of the renal arteries or arterioles of one or both kidneys is an important causal factor in hypertension.

If, in view of the evidence produced, we accept this as a fact, we are still without an adequate explanation for the hypertension, particularly in those cases where the damage is unilateral, or is neither widespread nor severe.

That a pressor substance reaching the circulation from

the damaged kidneys may in these cases be the cause of hypertension was considered as long ago as 1898. A protein like pressor substance from the kidney called renin was then described.

Much work has been done in this connection[19] but until recently the relationship between renin and hypertension was maintained more by theory than by fact.

Helmer and Page[19] have shown that renin is an enzyme. It is not a vaso-constrictor but requires the presence of another substance normally present in blood plasma, and termed renin activator. Reacting with the activator renin yields a pressor substance known as angiotonin.

Page[19] has obtained angiotonin in crystalline form.

Renin, therefore, has been shown to be capable of indirectly inducing hypertension. This has been experimentally proved by infusing renin solution into the ear veins of unanaesthetised rabbits. Moreover, the hypertension thus caused was similar to the hypertension which results from clamping the renal arteries.

This work has been carried a stage further, and investigations have shown that healthy blood contains a substance also derived from the kidneys, the function of which is to inhibit the action of angiotonin.

III. Constriction of Arterioles.

That this plays an important part in the causation of hypertension cannot be denied. We have already considered it in relation to the mechanical theory and with reference to renal damage both pathological and experimental.

Although it is by no means decided which may be the cause and which the effect when hypertension and arteriosclerosis co-exist, it is a fact that in many cases of the latter condition hypertension is not found.(20) This may possibly be accounted for by a compensatory vasodilatation of arterioles in unaffected parts.

When we consider the pathology and morbid anatomy of cases of hypertension we will find that primary changes in the blood vessels form part of the picture. Contraction of arterioles, hypertrophy of the muscular walls of the smaller vessels and arterio-capillary fibrosis in the kidneys are some of the changes described.

An increased prepheral resistance is therefore present in many of the cases and is apparently caused by either a permanent vasoconstriction of the arterioles or a temporary arteriolar spasm.

Although our attention has been focussed on this important part of the arterial tree further consideration is necessary in order to decide how these changes are brought about.

IV. Hereditary and Familial Influences.

It has been asserted that hypertension is a constitutional disease with dominant Mandelian characteristics.(21)

We know from experience that some people inherit a predisposition to develop the particular circulatory response which gives rise to hypertension.

A definite family tendency to early arterial degeneration with its attendant sequelae is well known. The death from apoplexy of several members of a family at a similar early age have frequently been recorded.

Investigation of case histories shows this family tendency in more than a third of all histories according to Halls Dally.(22) In my own series of cases, admittedly small, I could only obtain a family history of arterial disease in 15 per cent of cases.

V. Compensatory Influences.

Compensatory influences are apparent in some case of hypertension. More particularly is this seen in those who develop rheumatic disease in later life and in whom mitral stenosis supervenes.(23) Hypertension often develops in these cases but it has a beneficial rather than a deleterious effect.

It should be noted, however, that some observers(24) have attributed this hypertension to the effects of partial

asphyxia on the Vasomotor Centre.

The initial increased cardiac efficiency often seen in hyperpietic cases is probably compensatory and not etiological.

In cases of arteriusclerosis with normal arterial pressure a compensatory vasodilatation may occur in other unaffected parts such as the splanchnic area.

Even in normal health we find evidence of a compensatory mechanism between vasoconstriction and vasodilatation, which keeps the arterial pressure at a normal level.

To the Carotid Sinus at the bifurcation of the Common Carotid Artery, Herring[25] originally ascribed the function of the maintenance of arterial pressure at a constant level. McDowell,[25] however, cites evidence to show that the function of the depressor reflexes from the Carotid Sinus and the aortic depressor nerve is not to prevent a rise of blood pressure but actually to facilitate it.

The blood depots, according to this view, are controlled by the depressor reflexes, a reduction of whose activity can result in more blood being returned to the heart, for example, under mental stress.

VI. Toxic Influences.

In most obscure conditions a toxic theory is involved in explanation. In hypertension this is none the less true and indeed the influence of a toxin on hypertension can be shown

in many cases.

Septic foci from an infected gall bladder, unhealthy appendix or uterus, or from a pyorrhoea, are frequently found in association with hypertension or have been known to precede its discovery. Bacillus Coli infection of the urinary tract is not an infrequent accompaniment.

The detoxicating function of the liver, under constant strain, may prove inadequate to deal with the abnormal products of metabolism caused by organisms present.

This also raises the question of disturbed or inadequate hepatic functions as a factor in hypertension. It gains some support indeed from the frequently successful use of calomel and other cholagogue aperients in the treatment of hypertension.

Apart from the liver, the kidneys of course have to meet the full force of an invasion by germs or toxins. Their liability to damage and the resultant reactions have already been considered.

A direct action by bacterial toxins on the blood vessels themselves or on the nerve endings of the blood vessels is a not unlikely hypothesis when we consider the established findings of the action of pressor bodies such as renin.

A pressor amine formed in the intestines during putrefaction and prolonged digestion was described by Berger[26] in 1914. In this connection it may be noted that the

association of hypertension and intestinal stasis is a very common one. Whether this is the result of a sympathetic inhibition is doubtful, but as a result, poisons of putrefactive origin may be absorbed. At any rate, the treatment of intestinal toxoemia often plays a useful part in the regime advocated for the hypertensive patient.

H. Batty Shaw[27] believes that hyperpiesia and eclampsia are closely allied conditions, both produced by a circulating toxin and both capable of causing all the manifestations of uraemia.

The same author[28] also emphasises the fact that hyperpietic patients frequently succumb to the effects of an infection. Doubtless a lowering of resistance to infection can only be expected in such cases. I do agree that chest complications such as Bronchitis, Pulmonary Oedema, Pneumonia are often terminal features, but the gradual left ventricular failure is surely an adequate explanation.

Those who subscribe to the toxaemic theory believe that hyperpiesia results from the circulation in the body of some toxic agent or agents which are pressor in type and which may be capable of producing the other toxic accompaniments of hyperpiesia.

In so far as it goes the Renin theory does not conflict with this conception. It seems not unlikely that there

remain to be discovered other chemical pressor bodies similar to angiotonin, or enzymes from other damaged organs which can be activated to form angiotonin.

As an exogenous source of poisoning the action of lead in the causation of arteriosclerosis and high blood pressure is well known.

VII. The Metabolic Factor.

The frequent association of hyperpiesis and gout is noteworthy. A disturbance of lipoid metabolism has been suggested by the fairly frequent coexistence of hypertension and diabetes. Several examples of this will be quoted later. In some of these cases I think other considerations arise, such as increased viscosity of the blood, and pituitary and other ductless gland dis-functions.

An increase of cholesterin in the blood stream has been shown in many cases of hypertension.(29) Other observers(30) have reported an increase of the uric acid content of the blood, whilst Oxaluria was a troublesome feature of one of my own cases.

A deviation of the acid balance in the direction of either acidity or alkalinity is considered by some to be an important factor.

A disturbance of body metabolism, whether as a result of toxaemia or otherwise, is probably present in these cases.

This, however, is not surprising when we consider the metabolic imperfections of middle life in general.

VIII. Nervous Influences.

The relationship between nervous influences and hypertension has perhaps received more attention than any other factor. The ever-increasing stress and strain of present day existence takes its toll of the weaklings. Long continued anxiety states, emotional shocks, repeated worries and annoyances, fear and apprehension, figure so prominently in our case records that they cannot be ignored. Insomnia too is a very frequent symptom of hyperpiesia.

It would appear that frequently repeated psychical stimuli at first elicit a physiological response in the individual by which the blood pressure is temporarily increased. Later a transitory hypertension exists, characterised by angiospasm or spasm of arterioles.

Frequent use of the arterial muscle may lead to hypertrophy and as a result the lumen becomes narrowed. There results an increased resistance to the blood stream and a more prolonged rise in blood pressure. Increased cardiac energy is called into play and finally arterial degeneration at the weakest point, be it brain, heart or kidney, is the prelude to an hyperpietic catastrophy.

It would, of course, be ridiculous to suppose that such

stimulation of the higher nerve centres invokes such a reaction in the majority of those exposed to its influence, but many of these people appear to have an over-responsive vasomotor system.

Women of this type, as we know from everyday experience, are particularly susceptible to nervous stresses and influences at the menopause.

It is perhaps significant that the majority of hyperpietics are of a nervous temperament. This is at least abundantly true of my own cases.

Moschowitz[31] describes a characteristic type of hyperpietic in these words: "They have," he writes, "tense, single track minds with narrow mental horizon but within their range pursue their activities under conditions of tension."

John Hay[32] divided his cases of hypertension into groups, the first of which he summarised as "thin, spare and high strung individuals." I find that about fifty per cent of my cases come within this category.

Cases of melancholia, depressive states, anxiety neuroses, as will be shown later, very often show a raised blood pressure during their illness.

That nervous elements can profoundly influence organic functions is of course well known. The effects of stress and worry often do interfere with digestion and metabolism,

which in turn may cause toxic changes.

Further consideration will be given to this aspect of the subject in the sections on symptomatology and treatment.

IX. Endocrine - Sympathetic Influences.

There is substantial evidence that the increased arteriolar tone present in hypertension is due to the discharge of vasomotor impulses from the higher centres through the sympathetic pathway.(33)

This is largely borne out by the results of certain surgical operations devised for the relief of hyperpiesia.(34) Two operations in particular are mentioned.

Briefly, the first operation can be described as a section of the ventral roots from the fifth thoracic to the second lumbar, a rather formidable procedure involving laminectomy and rhizotomy.

The second operation consists of a bilateral subdiaphragmatic extra-peritoneal rescetion of the splanchnic nerves, coeliac ganglia and two upper lumbar sympathetic ganglia.

Symptoms generally were improved and a marked decrease in pressure claimed as a result of the operations. Even a diminution in the transverse cardiac diameter was stated to have resulted in some of the cases.

The chief argument against the theory of increased

sympathetic activity is the relative infrequency of such signs as tachycardia and sweating.

Pickering[35] in a review of the experimental work on the subject of hypertension considers that nervous reflex influences alone are insufficient to explain the facts.

The association between the endocrine glands and the sympathetic nervous system is well known and, to a certain extent, reciprocal. Stimulation of the sympathetic system by such factors as mental stress, fear, anger, pain and cold results not only in a constrictive action on the smaller blood vessels but also in a secretion from some of the ductless glands - particularly the pituitary, adrenals, thyroid and the gonads in varying circumstances.

Actually adenomata of the pituitary (Pituitary Basophilia) of the suprarenal bodies, (Neuroblastoma of the chromaffin cells of the adrenal medulla and of the Thymus are the only definite known causes of hypertension. In the rare tumour of the suprarenal medulla the hypertension is paroxysmal and is due to the intermittent release of adrenalin into the blood stream.

Hyperadrenalism, a condition parallel to hyperthyroidism, is stated by Crile to occur where the cerebral association mechanisms are over-active. Weak stimuli are sufficient to provoke an emotional response in these persons. The adrenal-

sympathetic system is stimulated and the symptoms of effort syndrome produced.

Paul Wood[36] quotes this in his paper on Da Costa's Syndrome, but the cases instanced by him could conveniently be considered under the heading of high blood pressure. In two of the cases the pressures given were 165/100 and 210/115 respectively. The operation of suprarenal denervation did not cause any objective improvement.

Tachycardia and sweating are, of course, prominent features of Da Costa's syndrome, but not as a rule of Essential Hypertension.

In permanent hypertension a search for pressor bodies such as adrenalin has proved disappointing. Notwithstanding the physiological facts that under conditions of emotion and anger excess adrenalin is secreted into the blood stream.

Acromegaly we know is associated with perverted function of the anterior lobe of the pituitary and is accompanied by high blood pressure. In its early stages it is often associated with diabetes or glycosuria. (The fairly frequent association of glycosuria or diabetes and hypertension has already been commented on.)

The chief characteristic of acromegaly is the general enlargement of the bones of the extremities and of the face. It is interesting in this connection to refer to Draper's

assertion[37] that in women, tallness, large skulls and relatively greater length of limb bones, is more prone to be associated with hypertension.

Not without significance also was the discovery of a pressor substance in the posterior lobe of the pituitary.

May it not be that pituitary vagaries are responsible for variations in individuals not clinically acromegalic, but conforming more or less to the type? Patients thus affected may at least form one group of hyperpietics. At any rate, from my own cases I can form a small group of hyperpietics whose physical characteristics can be described as acromegalic in type.

Cushing's rare syndrome of diabetes, hirsuties, hypertension and other phenomena may result from a basophil adenoma of the pituitary or a tumour of the thymus or suprarenal cortex.[38]

The relationship of the thyroid gland to hypertension is even more apparent clinically.

One type of hyperpietic not infrequently encountered is the woman of obese proportions with coarse features, dry, rough skin, and slow cerebration. She is almost myxoedematous in type and often reacts favourably to thyroid extract.

Cases of hyperthyroidism occasionally present a raised blood pressure (mainly systolic) which persists even after

thyroidectomy.

In menopausal hypertension there exists a disturbance of balance between various of the endocrine glands. Diminished ovarian activity and at times possibly disfunction of the ovaries and thyroid point to a rather complex relationship between the hypertension and the endocrine influences. Fortunately, the hypertension in these cases is often of a transitory nature.

X. Allergy as an Etiological Factor.

The recurrent nature of certain of the phenomena met with in hyperpiesia raises the question of a possible allergic condition being present. The periodic acute dyspnoeic attacks of cardiac asthma, which indicate left ventricular failure, bear a great resemblance to ordinary asthma in symptomatology but not in treatment. The dramatically sudden onset of pulmonary oedema, and the sudden collapses occasionally met with, which are not unlike anaphylactic shock, at least attract our attention by their superficial similarity to known allergic reactions. It is possible and even an attractive proposition to suppose that a sensitiveness may develop from some previous acute illness such as cholecystitis, scarlet fever, or food poisoning, and result in a general cellular disorder.

H. Batty Shaw[39] supports the hypothesis that the body

cells in hyperpiesia have become sensitive to extraneous proteins such as bodies of bacteria, and that this has lead to irritation and extensive cell injury. The theory, although unsupported by practical or experimental evidence, is also mentioned by Halls Dally.(40)

My experience of severe allergic conditions, however, is that they are more often accompanied by hypotension. This is particularly the case in anaphylactic shock and is often apparent in asthma.

If an allergic explanation of hypertension is plausible at all it can only be applied to some of the terminal manifestations of it, i.e. when the heart has failed and the pressure has markedly dropped. I do not deny the possible coexistence of hypertension and allergy however. Several of my own cases described in a later section have exhibited eczema, dermatitis, urticaria or migraine coexistent with their hypertension.

XI. Syphilis.

While recent syphilitic infection does not raise the arterial blood pressure, hypertension frequently results from its protean effects.

When we consider the widespread changes which may occur in the vessel walls with a consequent increased peripheral resistance, the rise in pressure is understandable. Apart

from this, secondary changes in other vital structures and organs can be held partly responsible.

XII. Asphyxia.

As has already been noted some observers[24] have attributed some cases of hypertension to the effects of partial asphyxia on the vasomotor centre. This presumably would only explain the hypertension developing in such cases as late mitral stenosis, and certain cerebral tumours.

Summary.

To sum up the position, it may be stated that changes in the smaller blood vessels, particularly in the arterioles, is the one significant fact. Increased peripheral resistance is the obvious result to overcome which a compensatory mechanism must come into play.

If the peripheral resistance is relatively slight, or of a temporary nature, it may be balanced by vasodilatation of the capillaries elsewhere in the body.

If the condition is more severe the blood pressure must be increased in order to overcome the peripheral resistance. This apparently is facilitated by the depressor reflexes in the carotid sinus and by the action of the aortic depressor nerve.

Increased action is necessary from the muscle of the left ventricle which eventually leads to its hypertrophy. Similarly

increased strain devolves upon the media and intima of the large and small arteries causing a vicious circle of changes and a gradually increasing arterial pressure.

If the structural changes in the arterioles are such an important factor in the mechanics of hypertension, what are the controlling or activating influences which can result in such changes?

Let us consider first vasoconstriction of the smaller blood vessels, chiefly the arterioles. This can be brought about by direct and repeated stimulation of the vasomotor centre, or conversely by paralysis of the vasodilator mechanism. Apart from the direct pressure effect of a cerebral tumour, injury, deformity or malformation, direct stimulation of the higher centre is unlikely to occur, except perhaps as a result of asphyxia or partial asphyxia.

Indirectly, however, the vasomotor centre can be stimulated through the sympathetic pathway. Over-stimulation of the sympathetic nervous system by often repeated psychic stimuli, such as worry, stress or strain, and other emotional influences, can elicit an abnormal vaso-constrictor response, resulting in hypertonus and later, hypertrophic changes in the appropriate arterioles.

The role of the endocrine glands must not be forgotten in this connection. By their relationship to each other and

with the sympathetic nervous system they seem to fit in to this rather intricate etiological jig-saw at this juncture.

The physiological response of the suprarenal bodies for instance to reaction of fear whereby adrenalin is secreted into the circulation and the blood pressure is momentarily increased by vaso-constriction of superficial vessels is well known. The effects of over-function or disfunction of the adrenals, thyroid, pituitary and ovaries are similarly manifest by allied action by the sympathetic system.

Pressor substances circulating in the blood stream have the power to cause vaso-constriction by their action on the sympathetic nerve endings. Such substances are found in certain of the endocrine glands, for example in the medulla of the suprarenal and in the posterior part of the pituitary.

They may also be found in other parts of the body as a result of dis-function or disease. A pressor amine for instance has been discovered in the intestine as a result of putrefaction or prolonged digestion.

The formation of angiotonin from renin plus renin-activator, following on renal damage, has also been considered in this respect. In fact, the view that hypertension is caused by renal disturbance, often of an ischaemic nature, probably holds pride of place and has more evidence to support it than other theories.(41)

It is also possible, of course, that where a toxaemic factor is suspect, toxic agents circulating in the blood stream may be pressor in type and have a similar action to angiotonin on the sympathetic nerve endings. Experimental evidence of this is absent. Neither has it been proved that the end products of metabolic imperfections act in this manner.

Although allergy and hypertension may co-exist, I do not consider that the arguments advanced in favour of an allergic cause of hypertension are tenable. The symptoms of cardiac asthma and nocturnal dyspnoea can be explained more easily in terms of left ventricular failure.

Pathology and Morbid Anatomy.

Hyperpiesia has been described as a marked symptomatic occurrence connected with varied pathological conditions.

Many of its symptoms can be explained as either concommitant effects of the underlying cause, or of the resulting complicating arteriosclerosis, renal or cardiac disease.

In considering the pathological aspects of the subject I do not propose to describe in detail the changes which occur in so many varied conditions.

Hypertonia.

As a result of persistent stimulation whether by chemical or psychical causes acting on the sympathetic nerve endings or

on the higher centres, a condition of hypertonus or increased tone occurs in the arteriolar walls. While this at first is possibly physiological, eventually structural changes take place in the walls of the affected vessels.

Essential hypertension produces a characteristic lesion in the arterioles, not only of the kidneys, but in many other organs of the body, e.g. the adrenals, spleen, liver, retina, and brain.(43)

The changes which occur are essentially arteriosclerotic in character. The arteriosclerotic kidney or nephrosclerosis is our old friend the red granular contracted kidney of so called chronic interstitial nephritis.

A hyaline thickening occurs in the subendothelial layer of the intima of the afferent arterioles. The larger interlobular arteries become thickened. This is caused by changes in the internal elastic membrane of the intima, which is eventually converted into collagenous connective tissue. The lumen may thus become obstructed by the thickened intima. Atrophy of the muscular coats occurs and is followed by a replacement fibrosis.

Ischaemia and disuse conspire to the gradual onset of atrophy and obliteration of the glomeruli and tubules. The sclerosis, however, is patchy rather than diffuse and does not obstruct the circulation to any marked extent.

Many normal portions of kidney may intervene between the islands of sclerosis. Thus the kidney function, in spite of changes, is little impaired in benign essential hypertension.

It is assumed that the long continued action of a renal or general poison causes a primary fibrosis, and that the tubular degeneration is due to the pressure effect of the newly formed tissue.[43] On the other hand, the renin theory already discussed suggests that as a result of any damage to the kidney, even to one kidney, causing renal ischaemia, renin is formed. This enzyme, in conjunction with a renin activator in the blood stream, produces the pressor substance known as angiotonin. Angiotonin in turn acts on the sympathetic nerve endings and thus indirectly on the vaso-constrictor centre.

The picture I have drawn of the arteriosclerotic kidney will be recognised as essentially the same as that described in most text books under the heading of chronic interstitial nephritis.

It is, I think, becoming increasingly evident that the latter term is rather a misnomer which in the past has covered a multitude of diagnostic sins. The majority of cases of chronic interstitial nephritis are, according to T. Tzod Bennett[13] and Prof. W. Thompson,[14] really cases of essential hypertension.

Where a true nephritis has existed we are much more likely to get a condition of chronic glomerulo-nephritis with its characteristic pathological features. More often this occurs in a younger person. Hypertension is present in more stable form. Albuminuria, renal casts and blood corpuscles are easily found. The blood urea is usually raised above 40 mgm. per cent. Renal efficiency fails and uraemic manifestations are soon in evidence.

In addition to chronic glomerulo-nephritis and benign essential hypertension, we have already seen that chronic pyelo-nephritis and chronic arterial disease can result in a progressive ischaemia of the kidney.(12) If the resulting hypertension is sufficiently severe in any of these conditions arteriolar necrosis is apt to supervene. Further damage to the kidney is then inevitable and the terminal picture of malignant hypertension with renal failure may thus be a feature of several diseases.

The changes in the small arteries such as have been described in the kidneys may affect the whole cardio-vascular system. Thus prolification of the endothelial cells of the intima, with resultant thickening, takes place. Secondary fatty degeneration or hyaline changes follow with gradual occlusion of the lumen. The entire vessel wall may be involved, lepoid and calcium deposits in the intima causing

atheromatosis, while the muscle coats of the media become fibrosed or even calcified.

Atheromata, when present, may be patchy or diffuse. In the larger vessels such as the aorta they are often distributed hap-hazard round the orifices of the larger branches.

In senile degeneration the changes are more marked in the media. Fatty degeneration and muscle atrophy give place to fibrosis and deposits of lime salts.

The left ventricle of the heart hypertrophies under the constant strain of increased muscular work and may eventually show signs of dilatation.

As has been described the walls of the capillaries, arterioles, and larger arteries, may show changes varying from hypertonus and hypertrophy to fully developed arteriosclerosis. Some of the smaller blood vessels may become completely occluded, but often symptoms arise from a condition of angiospasm or temporary spasm of small arteries. This is rather an important phenomenon. It not only explains many of the transitory motor and sensory paralyses often found in hypertensive cases, but it is stated to be a frequent cause of true cerebral apoplexy.(45)

Although cerebral apoplexy is not infrequently caused by rupture of a blood vessel, particularly where marked arteriosclerosis is present, it is often brought about by angiospasm

of a cerebral artery. Stasis of the circulation in this area occurs, followed by exudation or diapadesis through the vessel walls. The resultant condition is really a "red infarotion."[44]

Attacks of Hypertensive Encephalopathy are also considered to be angiospastic in nature, although in the type of hypertensive cerebral attack which may complicate chronic glomerulonephritis cerebral oedema is probably the main factor.

Infarctions, as a result of cerebral thrombosis, may of course occur. In recovery cases of apoplexy the condition has more often been due to cerebral thrombosis or angiospasm than to cerebral haemorrhage.

In distinction to the condition of essential benign hypertension a small percentage of hyperpietics (stated by Fishberg to be about 7 per cent)[45] may progress to the rapidly fatal condition of malignant hypertension.

The pathological changes met with in this condition are an acute aggravation of those already described.

In the kidneys, for example, a widespread necrosis of the afferent and interlobular arteries is found. This necrosis is widespread and is associated with the escape of blood and fibrin. Endarteritis obliterans is well marked in the small renal arteries and arterioles. Renal damage is therefore extreme. Similarly in the brain the walls of the cerebral arterioles are thickened. Vascular lesions, haemorrhages

and infarcts are frequently found. The latter may be multiple, small miliary infarcts scattered widely through the brain. Gross cedema of the brain and increase in the cerebrospinal fluid is a frequent finding.

No organ is immune from these vascular changes. Thus from closure of the lumena of the smaller arteries and arterioles infarctions and areas of necrosis may develop in liver, spleen, pancreas or gastro-intestinal tract.

Curiously enough, as Professor Thompson points out, and in support quotes evidence from Odel and other workers,(46) the arterioles of the heart show comparatively little change.

A Brief Historical Survey.

The first measurement of Blood Pressure was accomplished in 1733 by a Cambridge physiologist, the Rev. Stephen Hales. His manometer consisted of a long tube, which he inserted into an artery of a mare.

It was almost a hundred years later (1829) when Poiseville improved upon the device by introducing his haemodynamometer - a U-shaped mercury manometer. This was also connected with an artery, but a solution of Potassium Carbonate was used to prevent clotting. Loss of blood too was avoided.

Carl Ludwig made further improvements in this instrument. By adding a float and a pen he made it into a recording instrument, or kymograph - the earliest application of the

graphic method. The Spring Manometer was a further adaptation in order to overcome the lag of the mercury column.

To overcome the obvious drawback of having to open an artery various types of sphygmograph were later invented and by means of which the pulse wave was shown. The better known of these were by Marey (1860) and Dudgeon.

In 1876 came the first clinical sphygmomanometer devised by Von Basch of Vienna. With this instrument pressure was applied through a fluid medium. Potain in 1889 first used an air cushion as a means of compression.

The modern clinical method of circular compression of a limb by air was, however, introduced by Riva Rocci (1896) in Italy, and Hill and Barnard in England a year later. This was the prototype of the modern instrument. Numerous modifications have, of course, taken place. The original bag or armlet, for instance, was much too narrow.

On the clinical side we must go back to Richard Bright of Guy's and of Bright's Disease fame. His momentous work formed the groundwork and inspiration for all future study on Hypertension and allied conditions.

Gill and Sutton in 1872 gave their masterly description of arterio capillary fibrosis.

To Sir Clifford Allbutt, Professor of Physics at Cambridge for 32 years, we owe a tremendous debt, not only

for his brilliant work on cardio-vascular diseases, including his conception of Hyperpiesis and Hyperpiesia, but also for his stimulating influence on our modern thought. He started the general use of the sphygmomanometer in clinical practice.

Lauder Brunton and George Oliver quickly followed in his wake and worthily upheld the prominence of British clinicians in this field. Nor must we forget the work of another famous Cambridge professor who was inspired by Allbutt's work - Sir William Osler.

Of recent years so many of our ablest clinicians and research workers have contributed to the literature of hypertension and its problems, that at the moment it would perhaps be invidious to single out names for special mention. Much of this valuable work is mentioned in these pages with appropriate acknowledgment.

Without in any way belittling the work of other well known authorities, however, I think it can be fairly said that the experimental studies of Helmer and Page on renin and angiotonin[19] in relation to hypertension, and the work of Goldblatt and his followers,[17] mark important milestones of progress in the elucidation of hypertension.

SECTION 3.

THE PRESENT POSITION.

Prevalence of Hyperpiesis.

It is generally assumed that hypertension is becoming increasingly more prevalent throughout the civilised world.

This assumption is based partly on the Registrar General's mortality figures and partly from statistics and analyses published by the larger insurance companies.

Even so, they are indirect as far as they refer to hypertension per se. As it has been computed that one third of the deaths in England and Wales are due to cardio-vascular diseases, the incidence of hyperpiesis so frequently an accompaniment of these diseases, must be considerable.

Statistics published by the Metropolitan Insurance Company of New York[48] in 1900 showed that cardio-vascular-renal diseases were responsible for 35 per cent of total deaths at and over 45 years, whereas in 1935 the figure had risen to 53 per cent. In 1960 it is estimated the numbers will have doubled.

As deaths from intercurrent disease, infections and accidents must include a proportion of hyperpieties it will be seen that estimates based on mortality statistics may err on the conservative side. On the other hand, we know that

it would be fallacious to assume that everyone suffering from a cardio-vascular disease is necessarily a hyperpietic.

In considering the matter from the point of view of the doctor and his patient, rather than from mortality tables, it must be realised that in the early stages of hypertension many people do not seek treatment. The condition may be relatively asymptomatic. In fact, it may even be accompanied by a feeling of increased well being.

Such cases are frequently discovered during Life Insurance or routine examination.

Under war conditions medical examination of recruits to the armed forces and to war industries has become more or less compulsory. The incidence of hypertension in people over 40 has been found by Medical Boards to be fairly high.

Hitherto routine examinations of a comprehensive nature have been required by certain life insurance companies. Their statistics are of undoubted value, but life insurance is not by any means widespread in this country. It caters chiefly for the professional classes and for those of moderate means. Among the working classes, particularly amongst the female population, too much life insurance work is done without medical examination. Blood pressure readings have not been obtained in many thousands of such cases.

In spite of the difficulty of accurately assessing the

prevalence of hypertension it is certainly a fact that from middle age onward hyperpiesis is very prevalent, and under modern conditions of stress and strain is becoming increasingly so.

To revert to statistics the American figures published by the Metropolitan Insurance Company showed that unskilled labourers in urban communities gave the highest mortality rate for cardio-vascular-renal diseases. Males over 50 appear to be rather more prone to the condition than females.

Rolleston[21] states that statistical investigations do not point to greater prevalence among those subject to mental strain. This would seem to contrast with the mortality rates quoted by Lawrence Smith and his co-workers[47] who assert that the mortality rate from cardio-vascular-renal disease is high among professional men, especially surgeons and physicians.

We do find, however, that nervous, hyper-sensitive, hyper-active, quick-tempered people are more likely to be sufferers than their more phlegmatic neighbours. The short-necked, florid-complexioned individual we look upon with suspicion, but we also find a fairly large group of hyperpietics among the sallow, asthenic, toxic-looking type.

The relationship of hypertension to cardio-vascular and renal diseases has been discussed, as has the occurrence of

raised arterial tension in certain mental diseases.

In diabetic cases hyperpiesis is a frequent accompaniment. It is also not infrequently an additional burden to the obese overweight individual.

In considering the prevalence of hypertension we must also remember its association with such varied conditions as thyrotoxicosis, myxoedema, the menopause, eclampsia gravidarum, Cushing's syndrome and coarctation of the aorta.

Clinical Manifestations. Symptoms and Signs.

Children.

A condition of hypertension is occasionally found in children.[48] Such patients are usually pasty-faced and sickly; have dark rings under their eyes; are headachy, sluggish and moody, fretful and excitable. Their tongues are dirty and the bowels irregular.

Adults.

A high pressure of 180 to 200 may cause no symptoms. The individual may even report himself in excellent health.

As high blood pressure is itself a symptom of various pathological processes it would be rather involved were we to attempt to describe its clinical manifestations on such a broad basis.

We must clearly understand that many of our patients showing raised arterial pressure are suffering from one or

other of the maladies which are known to cause high blood pressure.

It is not my purpose here to describe in detail the symptomatology of such conditions as acute and chronic glomerulo-nephritis, polycystic disease of the kidneys or eclampsia except to mention that they are accompanied by a condition of hypertension. Other more rare conditions such as coarctation of the aorta, Cushing's syndrome, and tumours of the suprarenal medulla have previously been mentioned.

It has been estimated by Pickering[49] that hypertension arising from these various primary conditions accounts for probably less than 25 per cent of the cases of high blood pressure occurring in the temperate countries.

The remaining large group has been classified under the heading of Essential Hypertension.

Essential Hypertension.

Essential Hypertension is virtually our old friend the hyperpiesia of Allbutt.

By virtue of a fairly well defined series of clinical manifestations it can now aspire to the status of a disease. Unlike the hypertension which is secondary to or accompanies other pathological conditions already mentioned, the complications and later signs of essential hypertension are consequent to the hypertension.

Both sexes are affected and a family history is common.

Symptoms may manifest themselves during the fourth or fifth decade, but not infrequently the earlier stages are more or less symptomless, and the patient may not consult his medical adviser until some years later.

Simple or Benign Hypertension.

Early Symptoms.

Functional complaints are common, possibly owing to the surcharged vascular system.

Not infrequently, however, the first contact with the patient is made on account of a cerebral vascular accident, angina pectoris, or a coronary thrombosis, or even as a result of failing vision.

Between these extremes we are consulted for a diversity of symptoms many of which should direct our attention to the possibility of essential hypertension.

The systolic pressure may reach up to 300 m.m.Hg. and the diastolic over 100.

Breathlessness, tightness or discomfort in the chest, or severe palpitation, may first alarm the patient. Headaches, fullness in the head, giddiness, and sometimes tinnitus may be distressing symptoms. Emotional disturbances are fairly common. Insomnia is a frequent bugbear. Loss of energy, weakness and exhaustion are complained of. Attacks

of syncope may indicate depressed nervous function. Loss of concentration and impairment of memory may be troublesome. Extreme pallor is sometimes noticeable.

Later manifestations are due to increasing disability of the heart, impaired renal function or to an incapacitating lesion of the central nervous system.

Cardiac Complications.

In established cases hypertrophy of the heart, especially of the left ventricle, is almost invariably present. Increased pulse rate and diminished exercise tolerance are soon evident.

Increased cardiac dulness to the left is manifest to percussion and X-ray, while left-sided ventricular preponderance is frequently evidenced by the electrocardiograph.

Heart failure may be of the congestive type with liver enlargement and dropsy. Auricular fibrillation may occur.

Not infrequently, however, the left ventricle fails before the right, causing pulmonary and pleural congestion without dropsy or systemic engorgement.

Pulmonary congestion is intensified by recumbency or effort. Typical spasms of dyspnoea or cardiac asthma may thus be distressing accompaniments of the failure.

Acute pulmonary oedema may supervene without previous symptoms. Pulsus Alternous and Gallop rhythm, if present, are of ill omen. Dilatation shows itself by increased

cardiac dulness. A blowing systolic murmur is present at the apex and occasionally at the aortic area. A significant fall in systolic pressure usually accompanies the other signs and symptoms of cardiac failure.

Angina pectoris and coronary thrombosis have been mentioned. Arteriosclerosis is usually present in some degree in the kidneys. If it is widespread elsewhere the classical signs of palpable thickening and tortuosity of the arteries, particularly the radial and brachial, will be found, also hypertrophy of the left ventricle and accentuation of the second aortic sound. Dilatation of the aorta and deepening of its shadow on the X-ray screen might complete the picture.

Cerebral Complications.

Apart from cerebral haemorrhage and thrombosis with their well known sequelae, we may get angiospastic symptoms of a more transient nature. Epileptiform fits, coma, paralysis, aphasia, visual disorders. Sensory disturbances may be caused by spasm of the cerebral vessels or from oedema of the brain. Some of these disturbances comprise hypertensive encephalopathy.

Hypertensive Cerebral Attacks or Hypertensive Encephalopathy.

Encephalopathy is featured by very severe headaches, often

frontal and increasing in severity until the onset of convulsions.

A sudden high rise in blood pressure usually precedes the onset.

The extreme suddenness of this cerebral phenomenon is noteworthy.

In addition to convulsions and headache, amaurosis and aphasia and even coma may occur.

Retinitis, papilloedema and retinal haemorrhages are often found on examination of the fundi. No abnormality of renal function need be present.

Two distinct types are described. (1) The above mentioned variety which is commonest in middle-aged subjects, and is considered to be angiospastic in nature, and (2) Encephalopathy accompanied by signs of cerebral oedema, and signs of renal deficiency. This type is commonest below forty. It is seen in acute nephritis and eclampsia and in malignant hypertension.

Some text books classify all the transitory disturbances of cerebral function such as temporary paralysis or paresis, under the heading of hypertensive encephalopathy. Death is usually due to cardiac failure, apoplexy, or intercurrent disease.

Renal Symptoms.

In Essential Hypertension renal symptoms are unobtrusive. Nocturia is probably the earliest and most common of such symptoms. Even this is considered to be of cardiac origin, the increased volume of night urine being due to improved circulation while the patient is at rest. Polyuria is not very prominent. Albuminuria is not as a rule present in the early stages. As the disease progresses albumen may appear, often intermittently, and be accompanied by a few hyoline and granular casts.

Renal function is usually normal. Even if slightly impaired there is no rapid deterioration and uraemia is extremely rare.

Eye Symptoms.

The retina is normal as a rule, but may show the signs of arteriosclerosis. A restriction of the calibre of the arterioles takes place. This shows as an exaggeration of the arterial stripe. A variation in the lumen is also noticeable. This type of retinitis is often unilateral.

Malignant Hypertension.

In its early stages malignant hypertension is almost indistinguishable from the benign form from which it may develop. As a rule, however, it starts at an earlier age, e.g. in the thirties or forties. The diastolic pressure is

usually over 130 mm.Hg.

As the underlying pathological condition is a severe arteriolar necrosis of the kidneys, malignant hypertension may also develop as an end result of kidney disease, but more often is a terminal phase of essential hypertension. Headache becomes more constant, severe, and resistant to treatment. Often it is occipital and not infrequently accompanied by vomiting. A sustained rise of diastolic blood pressure - over 130 mm. - is important. General weakness and loss of weight is striking. Dyspnoea on exertion is often extreme.

Albuminuric Retinitis is almost invariably present, but the appearance of papilloedema is almost pathognomonic of the malignant stage.

The oedema of the disc is usually accompanied by atrophy and pallor. It may vary from hyperaemia to marginal blurring. Haemorrhages and the so-called cotton wool patches of exudale are present as evidence of residual oedema.

The cerebral symptoms show considerable variation as a result, no doubt, of the many small haemorrhages and infarets present in the brain.

Vertigo is common - transient hemiplegias and aphasias - loss of concentration and memory - personality changes - all may be featured.

Cortical lesions may cause epileptifom convulsions. Encephalopathy is not infrequent, but probably has its basis in cerebral oedema, infarcts and haemorrhages rather than in angiospasm. An increased tendency to haemorrhages exists - intracranial, retinal, nasal, subcutaneous, etc.

Renal Symptoms.

In the early stages renal function is normal. Even when the diastolic pressure is high and signs of albumenuric retinitis are present there may be no signs of renal abnormality.

In a few months albumen, casts and blood cells appear in the urine. The power of concentration of the urine falls, specific gravity decreases and the blood urea figure mounts up. The patient finally dies of uraemia unless he has previously succumbed to cardiac failure or cerebral haemorrhage. Few survive for more than two years after the onset of the malignant phase.

According to Fishberg[45] the malignant stage develops in only about 7 per cent of individuals with essential hypertension.

Hypertension in Pregnancy.

I. Pregnancy has an unfavourable effect on Glomerulonephritis already present. The symptoms are aggravated and the hypertension increased.

II. A patient suffering from essential hypertension may, of course, become pregnant with an increase of her hypertension and increased liability to renal damage. Usually, however, the previous level of pressure is afterwards regained.

III. While the two above named conditions may be found at any period of gestation the typical kidney of pregnancy is found during the last three months of pregnancy. A rise of pressure found during this period is usually followed by the symptoms of pre-eclamptic toxoemia. These include albuminuria, severe headache, cramps, vertigo, lethargy, nausea, loss of appetite, tremors, insomnia, loss of memory, melancholia, and raised arterial pressure.

Sometimes the more dreaded eclampsia gravidarium supervenes. Its symptoms hardly come into this purview, but in view of their similarity to those of malignant hypertension they will be briefly indicated. It will be noted that they comprise an intensive hypertensive encephalopathy.

In addition to gross hypertension and albuminuria, the following signs and symptoms may occur:- Headache, vertigo, flashes of light before the eyes, partial or total blindness, drowsiness, mental irritability, insomnia, constipation, oedema of legs, puffiness under the eyes, olegouria and epileptiform fits with or without aura. Sometimes even

acute pulmonary oedema may supervene.

Usually symptoms disappear after parturition, but a permanent hypertension may remain in 50 per cent of the patients.

Menopausal Hypertension.

In addition to the more prevalent symptoms of the menopause, such as hot flushes, nervous irritability, and even even instability, it must be noted that a proportion of sufferers exhibit a temporary rise of arterial pressure.

Probably less than 30 per cent show this hypertension.

If those suffering from essential hypertension and glomerulo-nephritis are eliminated from this category it will be found that normal pressure is usually regained afterwards.

Prognosis.

Various factors must be considered in addition to the height of the blood pressure.

Hypertension associated with chronic renal disease, for instance, carries with it a much graver outlook than an uncomplicated essential hypertension.

A temporary hypertension due to emotional causes often disappears when the mental stress is relieved.

Hypertension associated with obesity is not usually of ill omen, but the increased load on the myocardium requires

careful supervision. Generally speaking, if the hypertension is relieved by proper dieting, the prognosis is good.

The age of the patient is an important factor in hypertension. The younger the patient the greater is the liability to develop malignant hypertension. Therefore the outlook is unfavourable if a high pressure is found at a comparatively early age.

A bad family history, e.g. arterial disease or hypertension in the parents is significant.

The co-existence of hypertension with symptoms and signs evidencing systemic degeneration in arteries, heart, kidneys and digestive tract - with limited effort tolerance gives an unfavourable prognosis.

Where frequent severe headaches of a migrainous type are associated with a raised blood pressure of an unstable type there is a risk of cerebral thrombosis or haemorrhage.(50)

The onset of hypertensive cerebral attacks in a young person indicates a permanently raised pressure. In older patients there is an increased risk of cerebral haemorrhage from the sudden rise in arterial pressure.

In cerebral haemorrhage cases the pressures are not as a rule unduly high. A fall ensues after haemorrhage, but if the pressure is not re-established the indication is that

the haemorrhage is progressive.

The prognosis in coronary thrombosis is not good, but is rather better where the arterial pressure is raised than in cases where it is low.

Hypertension with commencing heart failure, dyspnoea, cyanosis, gallop rhythm and continuously accelerated pulse is ominous.

The onset of cardiac asthma or acute pulmonary oedema is of grave import. The adequacy of renal function is of prognostic importance in all cases of hypertension giving as it does an indication of the amount of damage to the renal arterioles.

In menopausal hypertension favourable results may be expected if there is an elevation of systolic pressure only, if the pressure varies considerably, or if there is comparative freedom from symptoms. Prognosis is more unfavourable if the diastolic pressure is raised, if obesity accompanies the hypertension, or if there are marked arterial changes - or signs of thyroid disfunction (e.g. persistent tachycardia).

The onset of any of the signs of malignant hypertension, papillaedema, etc., as has been pointed out, is of grave omen. There is a greatly increased tendency to intracranial haemorrhage.

Haematuria, in severe cases, gives suspicion of

necrotic changes in the renal arterioles. Petechiae under the pneumatic cuff are also of serious prognostic importance. Cerebral syphilis is often accompanied by raised arterial pressure. The liability to "stroke" is great. If treatment can be afforded before the onset of this complication the prognosis is reasonably good.

Considering actual figures Hilton[8] states that with regard to diastolic pressures "Any figure over 90 mm. must be regarded with suspicion." 95 mm. he regards as definitely pathological, and 100 mm. or over as ominous.

The same author classifies raised systolic pressures as follows:-

145-150 mm. is suspiciously high if below 40 years of age.
150-230 mm. is considered high.
240-275 mm. very high.
280-320 mm. exceptionally high.

Sturrock[51] regards all systolic pressures over 150 and all diastolic readings over 100 mm. as sub-standard lives.

While this is doubtless quite true it is not very helpful from the point of view of prognosis. There appears to be a not inconsiderable gap between the rule of thumb methods of insurance offices and the actualities of general practice in this respect.

Present day opinion rather tends to disregard the significance previously attached to a systolic pressure of

160 mm. Even a diastolic pressure between 90 and 100 is not now deemed sufficient by the Authorities to affect the grading of a recruit.

Certainly the diastolic pressure is a better guide to prognosis than the systolic, being less liable to temporary fluctuations.

If the diastolic rise in pressure is proportionally less than the systolic we can usually assume that the left ventricle is carrying on efficiently.

A diastolic fall is often of good omen, but a rapid fall of systolic pressure while the diastolic pressure remains high, is alarming.

A diastolic pressure of 135 mm. or more presages disaster. In nephritis when the systolic pressure is high, e.g. 180 or more, and the diastolic pressure is disproportionately raised to 120-150 mm. with signs of renal inadequacy, the outlook is grave and death from uraemia or cerebral haemorrhage is likely.

In this connection also a failure to concentrate urine, a high blood urea, a failure to respond to treatment, and the presence of albuminuric retinitis are all unfavourable signs.

Treatment is discussed in Section 4.

SECTION 4.

Observations on a series of 100 cases treated by the Author during the years 1926 to 1942.

Prevalence in the Bolton District of Lancashire.

It has been stated with considerable authority that hypertension is on the increase, both in this country and in America. So much can be gauged from statistics issued by large Insurance Companies[48] and from mortality tables.

If we are only considering the term hypertension without reference to other symptomatology this increase must be considerable.

It must be admitted, however, that such a bald statement of fact is much too vague in its significance. In fact, it is almost as useless to speak of the prevalence of hypertension as it is to give the incidence of any other sign or symptom such as vomiting, headache, or tachycardia.

Stricter and more thorough routine examinations during the last decade have certainly brought many more cases to light.

Repeated and periodic examinations of particular patients have shown many of them to have an hypertensive state where it would not have been diagnosed at one examination.

It is only when we study the other relevant signs and symptoms which accompany the hypertension that our statistics

can be of any assistance.

To be able to classify the cases into the categories of essential hypertension - benign or malignant, or hypertension secondary to another disease, is necessary before one can usefully analyse the prevalence of hypertension.

In my own series of 100 cases exhibiting hypertension over a number of years, I have placed 60 per cent as suffering from Essential Hypertension (Benign 56, Malignant 4). In twenty per cent the hypertension was of the secondary type, while the remaining 20% were doubtfully hyperpietic according to modern standards of assessment. By that, I mean that while they showed a definite systolic rise their diastolic pressures were under 90 mm. of Hg.

Pickering[49] states that maladies which cause high blood pressure account for probably less than 25% of the cases of high blood pressure occurring in temperate countries. The remaining majority constitute Essential Hypertension.

As regards the district in which I practice I cannot claim to give comprehensive statistics to prove that the prevalence of hypertension - essential or secondary - is greater than in other parts of England.

I can and do assert that contrary to the findings of others I have found it to be commoner in females than males in the proportion of three to two. The relative preponderance

of female labour in the Lancashire cotton spinning mills may be a contributing factor.

Among the women suffering from hypertension many have worked in the cotton mills from ten years of age until over sixty. In this connection one is led to theorise as to whether the heat and moisture, combined perhaps with the cotton dust, operating over so many years, has an effect on the vasomotor centre in the medulla, through the sympathetic pathway. The effect of repeated climatic or temperature changes from indoor to outdoor conditions cannot be negligible.

I think it can at least be said that the long working life of many of these female cotton operatives, in an unhealthy atmosphere, for forty or fifty years, has been a predisposing factor.

The life tables of the larger Insurance Companies showing normal blood pressure readings are doubtless based on statistics from a very great number of cases. One wonders, however, whether these figures are fully representative of all classes or whether they are mainly culled from life insurance policy holders or applicants.

In this part of the country at any rate a large proportion of the population, including most of the female cotton operatives, have never undergone a complete medical examination such as is required for life insurance. In fact, a very

pernicious system of life insurance exists among these classes, where policies are issued without medical examination.

From the sociological aspect it is rather important that full cognisance be taken of this comparatively large section of uninsured people of the poorer working classes. The stresses and strains to which they are subject are by no means negligible.

Of the women in this series, 60 in number, 26 were widows. Several were widowed in tragic circumstances, or after the long illness of the husband, and had to return to employment at a late age, or seek Public Assistance. Others were virtually in the same position, having to become bread-winners when their husbands suffered from prolonged incapacity.

Fifteen of the patients were in very poor circumstances and in receipt of Public Assistance.

Of the fifty-six married patients, it could be said of twenty-four of them only that they were happily or contentedly married.

These facts I mention in support of my conclusion that a potent etiological factor is the return to hard and difficult conditions of life, of many women of middle age, consequent upon family and financial tragedies. This combination of grief and worry and lack of family comfort at a critical period of life, is a noticeable feature in many of the cases.

In contrast, twenty cases were from the well educated middle class. They were of the type designated as brain workers.

Twenty patients belonged to the senile or decrescent group. It is perhaps significant that six of them were men who held positions of responsibility associated with worry and anxiety.

My impression as to the increasing prevalence of hypertension among the industrial operatives of this part of the country has been reinforced by my experience as a member of a Medical Recruiting Board during the past two years. Of 400 men aged 35 and over examined by me for blood pressure, 28 (7 per cent) were rejected on account of hypertension.

[illegible]	51	M	178/95	1 "
[illegible]	47	F	190/82	3 "
[illegible]	39	F	180/85	12 "
[illegible]	42	F	184/98	6 "
[illegible]	54	M	190/[illegible]	[illegible] "
[illegible]	51	F	[illegible]/96	[illegible] "
[illegible]	48	F	[illegible]/[illegible]	14 "

Summary of Cases.

No.	Name.	Age.	Sex.	Ave. B.P.	Length of Observation.
1.	Horridge, B.	71	F	210/70	2 years
2.	Bell, M.	78	F	190/75	8 "
3.	Buchan, M.	59	F	160/80	12 "
4.	West, R.	52	F	170/80	7 "
5.	Buchan, J.	60	M	170/85	12 "
6.	Baldwin, J.	74	M	170/80	9 "
7.	Whittaker, S.	48	F	168/85	14 "
8.	Farnworth, M.	78	F	190/85	9 "
9.	Batty, T.	62	M	160/85	7 "
10.	Buckley, R.	65	F	170/80	10 "
11.	Brown, F.	54	M	164/90	9 "
12.	Turner, R.	72	F	190/85	9 "
13.	Horrocks, L.	30	F	178/88	5 "
14.	Lewis, M.	33	F	190/85	3 "
15.	Kealbey, R.	51	M	175/85	1 "
16.	Bradshaw, M.	47	F	190/82	3 "
17.	Denton, F.	39	F	180/85	12 "
18.	Burke, S.	18	F	154/86	5 "
19.	Bentley, S.	81	M	190/85	8 "
20.	Whittle, J.	54	M	190/85	6 "
21.	Brown, E.	48	F	165/85	14 "
22.	Farnworth, G.	49	M	180/85	3 "

No.	Name.	Age.	Sex.	Ave. B.P.	Length of Observation.
23.	Thistlethwaite, E.	65	F	178/90 later 210/95	14 years
24.	Wyatt, S.	72	F	178/90	5 "
25.	Roscow, F.	59	F	170/95	3 "
26.	Cary, P.	60	F	178/95	12 "
27.	Bell, M. A.	76	F	170/98	7 "
28.	Lee, E.	73	F	180/95	3 "
29.	Riley, N.	72	M	168/95 later 260/120	9 "
30.	Birch, A.	74	F	168/90	8 "
31.	Boardman, W.	63	M	168/90	5 "
32.	Longworth, M.	68	F	175/92	12 "
33.	Hoyle, F.	48	F	178/90	3 "
34.	Hurst, M.	55	F	160/90	5 "
35.	Hadfield, C.	34	F	200/95	12 "
36.	Pilling, F.	49	M	180/90	2 "
37.	Morris, J.	75	M	195/95	5 "
38.	Foster, J.	70	M	185/95	8 "
39.	Marsden, E.	80	F	190/95	3 "
40.	Lever, E.	65	F	210/95	7 "
41.	Fletcher, W.	54	M	160/90	8 "
42.	Wood, J. S.	54	M	160/90	14 "
43.	Lowe, E.	65	F	170/90	14 "

No.	Name.	Age.	Sex.	Ave. B.P.	Length of Observation.
44.	Hart, T.	63	M	160/90	10 years
45.	Baldwin, N.	54	F	160/95	3 "
46.	Atkinson, J.	72	M	200/90	4 "
47.	Gregory, M.	69	F	185/90	8 "
48.	Craven, F.	16	M	150/100	5 "
49.	Dutton, E.	70	M	190/100	12 "
50.	Ashworth,	73	F	190/100	2 "
51.	Wyatt, W.	72	M	200/100	5 "
52.	Wagstaffe, W.	59	M	190/100	1 "
53.	Peel, M.	53	F	196/100	14 "
54.	Gratrix, G.	72	M	215/100	2 "
55.	Banks, E.	68	F	210/100	3 "
56.	Stevenson, T.	72	M	204/102	3 "
57.	Barlow, E.	64	F	200/100	2 "
58.	Heyes, J.	55	M	186/100	4 "
59.	Dorning, L.	54	F	190/106	12 "
60.	Flint, M.	57	F	210/100	8 "
61.	McEachran, E.	60	F	210/105	1 "
62.	Farnworth, M.	60	F	178/100	8 "
63.	Downs, A.	55	M	210/110	10 "
64.	Morris, M. E.	54	F	210/110	2 "
65.	McCallister, M.	53	F	190/110	3 "
66.	Yates, W.	65	M	190/110	12 "

No.	Name.	Age.	Sex.	Ave. B.P.	Length of Observation.
67.	Thomas, H.	41	F	170/110	10 years
68.	Rogers, E.	71	F	180/110	4 "
69.	Swyers, E.	33	F	175/110	2 "
70.	Butler, S.	73	F	220/110	5 "
71.	Greenhalgh, E.	75	F	200/110	3 "
72.	Hurst, J.	55	M	200/110	8 "
73.	Orr, T.	60	M	200/110	3 "
74.	Markland, M.	66	F	200/110	5 "
75.	Dowbekin, E.	63	F	210/110	10 "
76.	Ashworth, F.	66	F	220/112	1 "
77.	Morris, E.	60	F	240/110	7 "
78.	Dootson, J.	68	M	165/110	4 "
79.	Moss, S.	49	M	210/110	11 "
80.	Wornwell, W.	67	M	190/110	2 "
81.	Crossland, M.	68	F	240/110	4 "
82.	Prew, J.	68	M	220/110	6 "
83.	Crankshaw, W.	65	M	220/110	9 "
84.	Horridge, J.	53	M	200/115	6 "
85.	Lamb, L.	59	F	210/115	7 "
86.	Parkinson, M.	43	F	220/115	13 "
87.	Craven, J. P.	66	M	220/120	2 "
88.	Hurgatroyd, E.	52	F	200/125	2 "
89.	McMillan, M.	69	F	230/125	1 "

No.	Name.	Age.	Sex.	Ave. B.P.	Length of Observation
90.	Clarke, W.	73	M	220/120	7 years
91.	Dutton, S.	50	F	210/120	3 "
92.	Peel, V.	57	M	205/120	12 "
93.	Walker, M.	58	F	220/120	8 "
94.	McCaffer, S.	69	F	220/130	5 "
95.	Fletcher, D.	63	M	220/130	6 "
96.	Iddon, M.	46	F	220/130	1 "
97.	Parry, E.	78	F	240/135	1½ "
98.	Bradley, E.	60	F	250/140	9 "
99.	Hall, M.	64	F	280/140	1 "
100.	Peirce, W.	56	M	270/160	2 "

The clinical records of one hundred cases exhibiting hypertension over considerable periods have been analysed.

For convenience of reference they have been divided into two main groups. Group 1 (Moderate Group) comprises patients whose systolic pressure is over 160 mm. but whose diastolic pressure is under 100 mm. of Hg. Forty-seven are included in this group. Of those, 30 were females and 17 males. Both systolic and diastolic pressures are shown in table 1. Group 2 (Severe Group) comprises patients with hypertension whose diastolic pressure is 100 mm. and over. This group embraces 53 patients - 31 female and 22 male. Their pressures are shown in the form of a graph in table 2.

Summary.

100 cases	(Group 1 - Moderate (Diastolic pressure under (100 mm.)	47 cases	(Females 30 (Males 17
	(Group 2 - Severe (Diastolic pressure 100 (and over.)	53 cases	(Females 31 (Males 22

41 patients had a systolic pressure of 200 mm. and over.

Sex Distribution.

100 cases - (Females 61 (Males 39 (Table 3)

Age Distribution. (Table 4)

Under 50 years of age	- 17	100 cases.
Age 51 to 60 years	- 33	
Age 61 to 70 years	- 27	
Over 70 years	- 23	

Types of Patient.

An attempt has been made to classify the type of patient according to physical characteristics.

It will be found that the hyperpietic as a rule belongs to one of two main types:-

(1) The thin, sallow, asthenic toxic type, or

(2) The florid, stockily-built, short-necked type.

Both Douthwaite[52] and John Hay[53] agree that these two groups form a large proportion of cases of hypertension.

I found that these two main types were almost equally represented in this series of cases - 51 being of the florid type and 49 sallow.

Although the majority of hyperpietics can conveniently be grouped as above some will also be found who have other striking characteristics.

(3) The obese type of individual forms a small proportion of our cases. Actually six patients in this series, including two with myxoedema fall into this sub-category.

(4) Seven patients in this series were of a type worthy of mention. They were long-boned individuals almost acromegalic in type.

Summary.

Type 1.	Thin, sallow, toxic, etc.	49 cases.
Type 2.	Florid, stockily-built, etc.	51 "
Type 3.	Obese type.	6 "
Type 4.	Acromegalic type.	7 "

Types of Hypertension.

Classification.

Essential Hypertension -	Benign 56 cases Malignant 4 "	60 cases.
Secondary Hypertension		20 "
Unclassified or Doubtful ... ... (Systolic Hypertension) (Diastolic pressure under 90)		20 "

Symptomatology.

(1) Dyspnoea.

Dyspnoea varying from breathlessness on exertion to Orthopnoea was the chief symptom in 28 cases.

It was an early or initial symptom in 12 cases.

Where cardiac enlargement supervened in response to a gradually increasing pressure dyspnoea was usually in evidence. It was, of course, a marked symptom in 4 cases where Aortic Regurgitation was present, and in 1 case of Mitral Stenosis complicated by hypertension.

As a late sign it was found to develop, as could be expected, in those who eventually succumbed to heart failure. Twelve patients in this series died as a result of heart failure of the congestive type, including two in whom Auricular Fibrillation caused death.

A more distressing dyspnoea was found, however, when failure of the left ventricle occurred and was accompanied by signs of acute pulmonary oedema or cardiac asthma. This was evidenced in nine cases.

Co-existent with the heart failure a sudden fall in both systolic and diastolic pressure was a significant finding and was accompanied by an increase in dyspnoea.

In four cases complicated by obesity dyspnoea was troublesome. Douthwaite[52] found that dyspnoea on exertion

was much commoner in the short florid type of patient than in the sallow type. My case records show that in this series the reverse was the case.

Apart from the dyspnoea of terminal heart failure nine patients of the florid type complained of dyspnoea as their chief symptom as against nineteen of the sallow toxic type.

In eight cases dyspnoea was a prominent symptom before any signs of cardiac enlargement or valvular disease could be demonstrated.

Dyspnoea and its relationship to the height of the Blood Pressure.

Thirty-eight per cent in this series (cases 63 to 100) had relatively high blood pressure. They had high systolic pressures combined with diastolic pressures of 110 mm. and over.

Of this number, eleven cases did not complain of dyspnoea during the greater part of their illness. In one case (case 100), although the average pressure for a period of two years was 270/160, there was no complaint of shortness of breath. This man's chief symptom was very intractable headache of the confusional type.

In general, dyspnoea was not a common symptom, except as a terminal manifestation, in those cases who showed effects of cerebrospasm or cerebral haemorrhage.

Where the pressure had been consistently high for a lengthy period and a sudden drop of both systolic and diastolic took place, the acute onset of dyspnoea was very noticeable.

Case 99, for example, showed a blood pressure of 280/140 from June, 1942, until October, 1942. During this period dyspnoea was not greatly in evidence. In October the pressure was found to have dropped to 160/90. Marked dyspnoea was present, and the patient died 10 days later.

A similar sequence of events was strikingly shown in cases 29, 40, 57, 63 and 98. (Table 5)

The importance of this, at any rate from a statistical point of view, lies in the possibility of these cases being wrongly diagnosed.

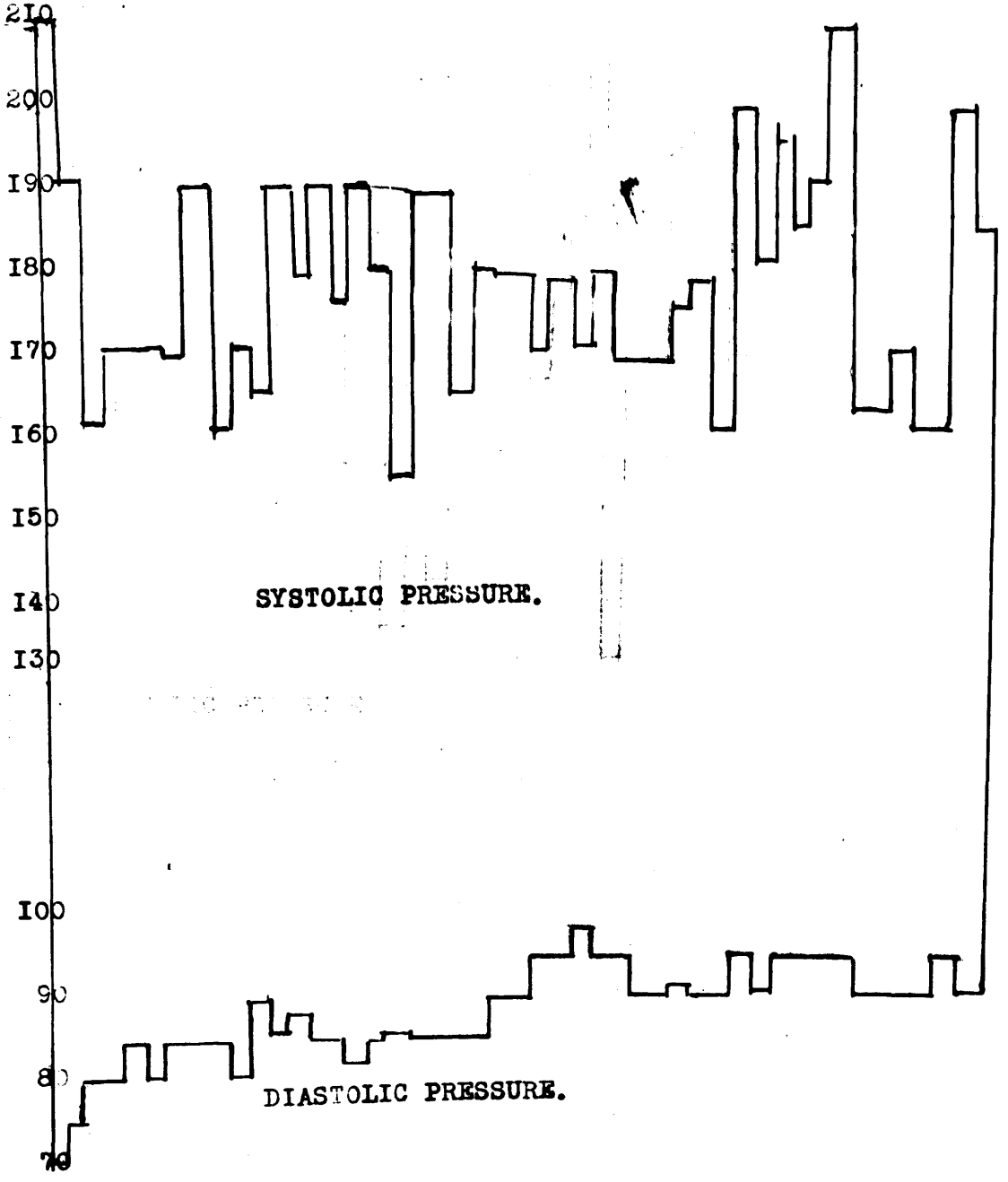
If the patient is first seen by a doctor at this stage of the illness the picture of hypertension is likely to be obscured by the myocardial changes and relatively low blood pressure.

Table 5.

Case NO.	Ave. B.P.	Length of Time	B.P. on onset of myocardial failure	Sequelae
29.	260/120	5 years	180/90	Myocarditis 12 months.
40.	210/95	10 "	150/80	Congestive Heart Failure, death in 3 months.
57.	210/110	1 year	160/80	Myocardial Failure Dilatation, death 6 weeks
63.	210/110	3 years	150/100	Myocarditis & continued reduced B.P., 6 months.
98.	250/140	3 "	170/100	Myocardial Failure - death in 2 weeks.
99.	280/140	3 months	160/90	Myocardial Failure - death in 10 days.

TABLE. I.

Mm. of Hg.



I.2.3..... 48

Cases No. I to 48.

TABLE 2.

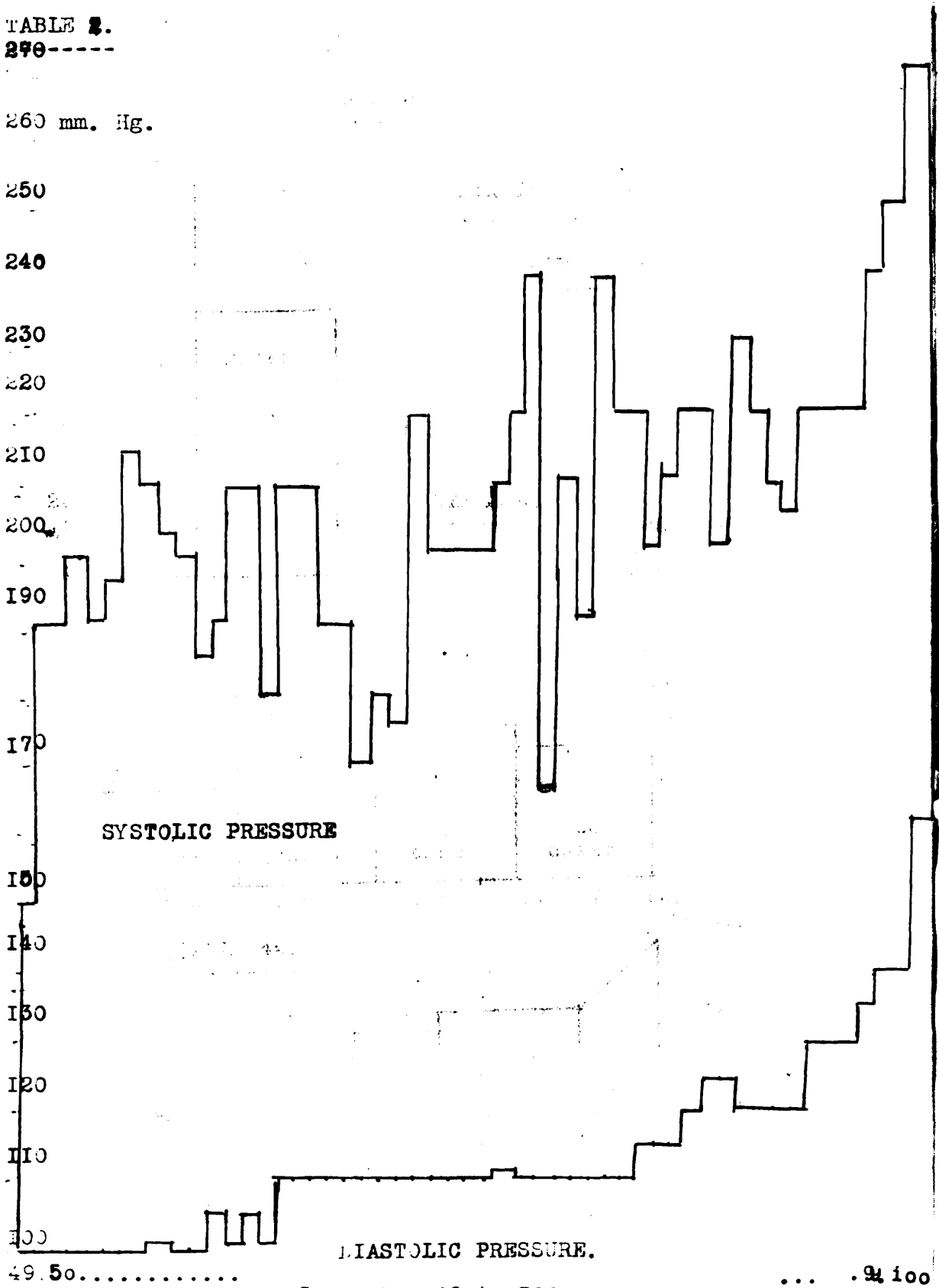
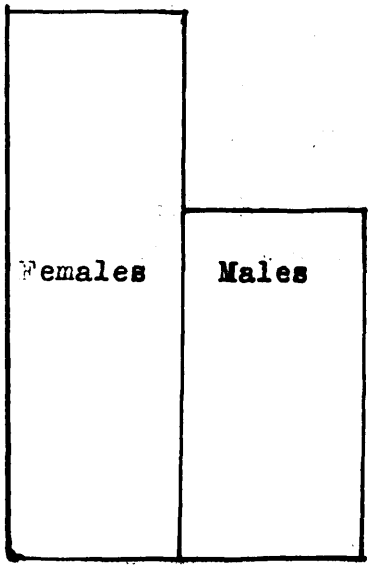

260 mm. Hg.
250
240
230
220
210
200
190
170
SYSTOLIC PRESSURE
150
140
130
120
110
100
DIASTOLIC PRESSURE.
49 50
91 100
Cases No. 49 to 100.

TABLE 3.



Sex Incidence.

females 6I cases.
males 39 ..

TABLE 4.

Age Incidence.

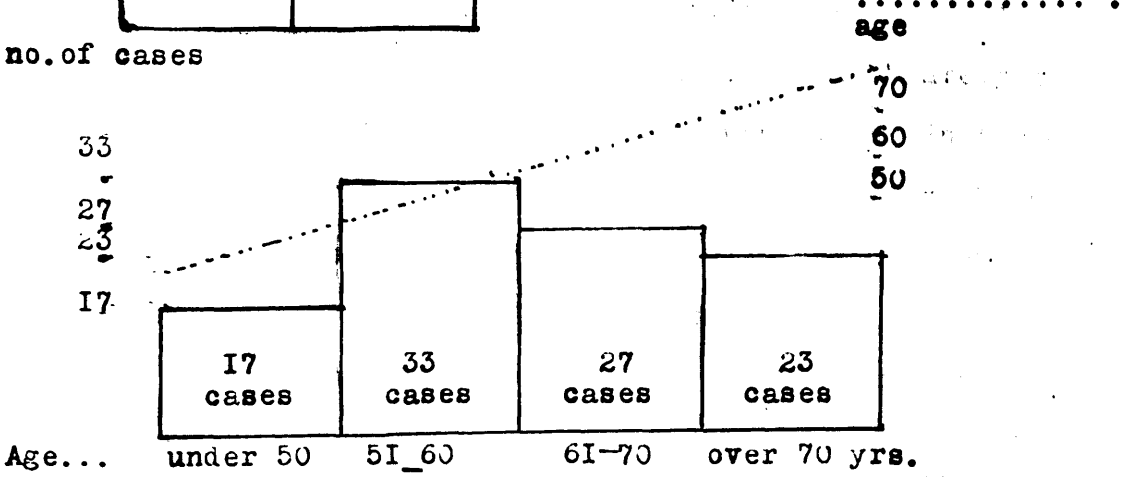
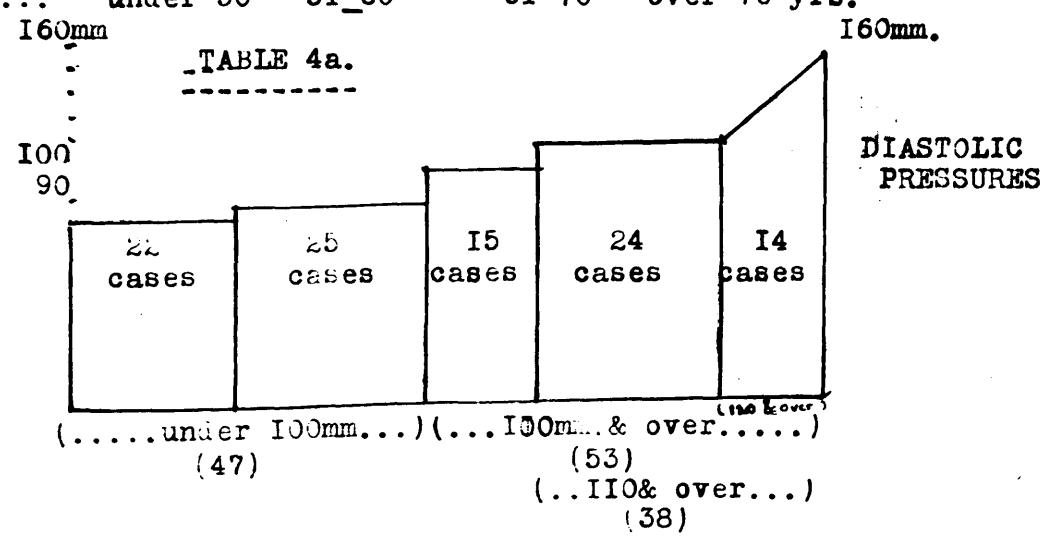


TABLE 4a.



(2) Tightness of the Chest and substernal region rather than a true dyspnoea was a distressing feature in several of the cases. (Cases 23, 29, 76 and 98.)

(3) Headache. Headache was an extremely common symptom. It varied considerably in type and intensity.

Table 6.

Severe Headache	- 25
Moderate	- 24
Mild or Infrequent	- 18
Absent	- 33

Thus in 67 per cent headache was mentioned as a symptom. In 25 cases it was of a severe type and in 24 moderately severe.

The most extreme examples were encountered in the malignant group. In two of these cases in particular, both of whom had papilloedema (cases 88 and 91), the headache was described as agonising. During a severe attack of what might be described as hypertensive encephalopathy the pain was obviously unbearable and was referred mainly to the frontal, parietal and retro-orbital areas.

Severe frontal headache was very marked in two cases who showed a positive Wasseman reaction (cases 72 and 86). In one, cerebral haemorrhage later developed and in the other General Paralysis of the Insane ultimately supervened.

In both patients the headache was blinding in character, affected the vertex and occiput, and was accompanied by a confused, muddled feeling.

Where the renal function was disturbed headache was almost invariably complained of, but where cardiac symptoms predominated headache was not a common symptom.

In this series of cases 33 made no complaint of headache.

Among those with medium or moderately raised pressures headache was more frequently encountered than in the very high pressure group. The headache, however, although more persistent, did not attain the same severity.

In the pre-eclamptic cases the headache was similar in type to that complained of in the malignant group.

Severe headache accompanied by vertigo was found in 8 of the cases. No deafness or middle ear trouble was found in these cases.

In all the patients of the menopausal group headache was a troublesome symptom.

Headache which was accentuated by stooping or by reverting to the horizontal position was exemplified in 4 of the series (cases 27, 67, 100 and 86).

A confused, muddled or muzzy, heavy feeling in the head, rather than a definite head pain, was a fairly frequent complaint.

Headache of a migrainous type was found in 4 of the cases.

Three patients who had frequent attacks of epistaxis always had preceding frontal headaches (cases 23, 21 and 49).

Suspicion of hypertension in two of the patients was aroused by their complaint of waking in the morning with severe, dull, persistent frontal headache, which persisted until about 11-0 a.m. No sign of sinusitis was evidenced, but hypertension was found to be present.

(4) Mental Confusion.

(a) Mild - a rather confused, dulled feeling, described as "muzzy" or "woolly" was a fairly frequent complaint. It was noted in the case histories of sixteen cases.

(b) Moderate - a fairly well marked degree of confusion where the patient was at times flustered, and could not concentrate or even speak coherently, was shown in five further cases.

(c) Confusional States severe enough to require institutional treatment developed in five of the series. In three it took the form of Manic Depressive Insanity with delusions and morbid depression. One other case (No.16) suffering from menopausal symptoms with hypertension committed suicide during a bout of extreme depression. Suicide was attempted in case 61.

Kidney function in these cases was sufficiently good to negative the possibility of uraemia.

Uraemia, however, was the cause of death in 5 cases in this series. Mental symptoms of relatively short duration

such as delusional ramblings, temporary violence, convulsions and coma were in these cases probably attributable to the uraemia.

Both syphilitic cases exhibited severe melancholia.

Mental confusion was found mostly in the sallow type of individual, but bore no apparent relationship to the height of the pressure.

In the mental cases, however, reduction in pressure which occurred occasionally or which was achieved by treatment, caused amelioration of the mental symptoms.

5. Vertigo or Giddiness was a rather common symptom. In the mind of the layman it is probably more often associated with hypertension than any other single symptom. Its onset in a middle-aged person apparently free from gastric upset or middle-ear disease is certainly strongly suspicious of hypertension.

Thirty cases in the present series complained of this symptom. It was variously described as "a top-heavy feeling," "a light headedness" or a giddiness brought on by sudden change of position.

Occasionally it was accompanied by vomiting (case 30) and in five of the cases by headache (cases 2, 32, 83, 85 and 98).

Vertigo without tinnitus or deafness is rather an interesting symptom and, as I have shown, a fairly frequent one.

C. P. Symonds,(52A) the Neurologist, states that it is reasonable in such cases where there is no evidence of any disease of cerebellum or brain stem, to presume a vascular lesion either in the labyrinth or in the brain stem.

He also points out that in Menière's Disease vertigo may precede the other symptoms of the head by many years.

Menière's syndome itself, however, according to this same authority, may result from arteriosclerotic degeneration in cases of arterial hypertension.

6. Tinnitus was present in seven cases, (cases 9, 23, 30, 42, 49, 66 and 68). Arteriosclerotic changes were noticeable in four of these cases. Solerosed and thickened, indrawn tympanic membranes suggested a definite labyrinthian origin in the other cases. (Meniere's Syndrome)

7. Loss of Weight was both an early and an important symptom in ten of these cases. It commenced with an aggravation of symptoms and appeared to give a bad prognosis. (Cases 9, 23, 28, 29, 31, 32, 40, 55, 57 and 85.)

8. Loss of Energy accompanied by a feeling of weakness was a very frequent and striking complaint. Often it was the only symptom for a time, or at least it was the first one complained of. (Cases 10, 40, 83 and 94.) It was specially mentioned by 25 per cent of the cases.

9. A type of paraesthesia - exemplified by a feeling of

tingling and numbness in the hands and feet was an occasional finding in six of the cases (3, 4, 7, 27, 54 and 68). This was not accompanied by Neuritis.

10. Neuritis, however, was present in fourteen patients in the series. In the form of Anterior Crural Neuritis it was at times a very severe and troublesome symptom in three of the cases (49, 70 and 83).

11. Rheumatic Manifestations (Fibrositis, Lumbago, etc.). At least ten of the patients suffered from chronic rheumatic disorders in addition to their hypertension. (Cases 9, 10, 27, 31, 51, 68, 75, 83, 87, 90 and 98.)

12. Intermittent Claudication was troublesome in only one case (No.4).

13. Flushing of the Skin (apart from the Menopause) was a prominent feature in twelve cases, particularly in the florid type of individual.

14. Cerebrospostic Symptoms. Monoplegia, diplegia, hemiplegia or paresis of one or more limbs was present at some stage in eighteen cases. In eight cases, however, it was a temporary feature lasting from a few hours to several days. The temporary nature of the condition suggested an angiospostic origin.

Temporary aphasia of short duration was sometimes present. Case 63, a bank clerk, aged 55, during a busy

period at the counter, suddenly lost his speech for a period of thirty minutes. Afterwards he complained of weakness of the right arm and leg. I examined him that evening. He had extreme difficulty in starting to speak and also in his articulation. His arterial blood pressure was 210/110. Twenty-four hours later, however, he expressed himself as being fit and well. No weakness was then apparent in arm or leg. Blood pressure was 190/100.

In three cases these temporary cerebrospastic symptoms were eventually followed at a later date by a true cerebral haemorrhage with resultant hemiplegia (cases 4, 5 and 65).

In case 5 a temporary monoplegia followed by rapid recovery was succeeded three days later by a true and lasting hemiplegia.

15. Cerebral Haemorrhage and Hemiplegia. Twelve of the series developed hemiplegia following cerebral harmorrhage or thrombosis (2 cases). (Cases 4, 12, 19, 28, 37, 38, 50, 52, 65, 71, 88 and 90.)

16. Syncope. Sudden collapses of a syncopal or vaso-vagal type occurred in two cases (12 and 57). In case 57 they were very frequent.

17. Pre-eclamptic symptoms were present in three cases (13, 14 and 35). In two eclampsia developed in spite of treatment, but all three regained normality. Albuminuria

disappeared, urea concentration became normal and arterial tension dropped to normal level.

Severe headache, frontal and occipital, albuminuria, oedema of legs, raised arterial pressure, insomnia, dimness of vision (in one case temporary blindness) were the chief symptoms of the pre-eclamptic stage.

18. Arteriosclerosis. Definite signs of arteriosclerosis were present in fourteen cases. These included palpable thickening of the radial and brachial arteries; accentuation of the second heart sound; enlargement of the heart to the left and eye changes - arcus seniles, thickening of the arteries of the fundi.

19. Hypertensive Cerebral Attacks or Encephalopathy. Various cerebrospastic signs have already been mentioned (para.14). Hypertensive encephalopathy of one type is believed by McAlpine[34] to be a similar phenomenon.

It is a well known complication both of nephritis and essential hypertension. Cerebral Oedema is thought to be the etiological factor where the attacks supervene in nephritis and in young hypertensive adults.

In the above group severe frontal or occipital headache, sometimes accompanied by cerebral vomiting, papilloedema, and a raised pressure of the cerebrospinal fluid, are usually found. Attacks of epilepsy, generalised or Jacksonian, may

occur. Loss of vision may be present. Albuminuria is usual. The blood pressure is found to be much increased above its former level during one of these attacks. Signs and symptoms gradually disappear in the course of a week or two.

One of the cases in this series exhibited this type of encephalopathy. She was a patient of 52, however. Sudden attacks of most acute and blinding headache in the temporal and occipital region supervened periodically. They were usually accompanied by vertigo and nausea and were followed by a temporary blindness lasting from twelve to twenty-four hours. Papilloedema was present, along with constriction of the retinal arteries. Albuminuria was present. During one attack the blood pressure rose from 190/110 to 230/125. (Case 88.)

The other type of encephalopathy which occurs usually in the over forty group was typically shown in three cases.

In a woman of 72, on three separate occasions, severe head pains were followed by epileptiform convulsions and rather prolonged coma. Slight albuminuria was present. There was no papilloedema. Blood pressure was temporarily increased from 200/100 to 280/120 on one occasion. All symptoms subsided in 6 or 7 days, but a cerebral haemorrhage with hemiplegia resulted eighteen months later. (Case 50.)

Case 63 is that of a bank clerk, aged 55, who had a complete Aphasia at the counter one morning. He had almost completely recovered when I saw him on the same evening but his arterial blood pressure was 210/110. On three occasions subsequently he had complete loss of consciousness followed by coma of short duration. I examined him during one such attack and ascertained that he had gone through all the stages of a major epileptic attack. His blood pressure was falling when I examined him. It was 220/115 mm.Hg., but by the following morning had come down to 170/100.

Case No.57, a married woman, aged 64, had very frequent epileptic attacks - several times a week - always preceded by blinding headache and followed by prolonged coma of several hours' duration. Her average arterial pressure between the attacks was 200/110. I was not able to demonstrate a rise in pressure at the time of attack.

Case No.92 presented classical symptoms of hypertensive encephalopathy, including unbearable headache followed by epileptiform convulsions. The symptoms were ultimately discovered to be due to haemorrhage into a malignant cerebral tumour. (Glioblostoma)

20. Eye Changes - Ocular Manifestations.

Temporary Blindness occurred in four cases (7, 13, 50 and 88).

Arteriosclerotic Changes in the arterioles of the fundi, shown by a thickening of the walls and variation in the lumena, were frequently found. (Cases 4, 24, 27, 56, 66, 84, 87, 88 and 98.)

Albuminuric Retinitis was present in four cases (50, 61, 73 and 97).

Diabetic Retinitis was seen in one case (25).

Papilloedema was found in four cases (76, 88, 92 and 99). In each of these cases death supervened within a period of a few months of this finding.

Diplopia was complained of in one case only (69).

21. Nervous Symptoms. Nervous symptoms usually associated with the term "functional" were exhibited by a considerable proportion of the cases in this series. Actually 31 per cent were of a noticeably nervous temperament. They could be characterised as "highly strung" or "nervy". Twelve of them in particular were more or less continually seeking treatment on account of trivial symptoms. They could be described as hypochondriacal or even neurasthenic in that physical signs were usually lacking in substantiation of their many complaints.

They differed from the asthenic type of Neurasthenic whom we usually associate with visceroptosis and hypotension, by virtue of their raised arterial pressure.

They were rather unstable emotionally, apprehensive and rarely free from worry and anxiety. Their worries, however, were plausible and reasoned, and were often engendered by fear of a definite serious illness such as a stroke, a heart attack, or a cancer.

In one case the frequency and severity of her occipital headaches gave rise to a genuine anxiety lest insanity should supervene.

These phobiae in three of the cases did actually attain the severity of an acute mental illness resulting in one case of suicide and in attempted suicide in another. Mental treatment in an institution was necessary in two other cases in this series (cases 53 and 91).

In considering the nervous make-up of these patients it is perhaps noteworthy that the type of nervous symptoms mentioned occurred predominantly in those with a moderate degree of hypertension. I think, therefore, that they may be considered as early symptoms due in all probability to the hypertonic condition of the cerebral blood vessels.

In the later stages, if the blood pressure increases, or in those in whom a very high pressure is discovered, these functional or neurasthenic symptoms are not marked. This, I think, is explained by the more definite physical findings, and the severity of more or less clear cut symptoms referable

to cardiac, renal or cerebral systems.

The patient is often at this stage seriously ill and takes little cognisance of the milder and more varied ailments which previously monopolised his thoughts.

Although described as "nervy" and highly strung, paradoxically enough many of these people are shrewd and full of well reasoned arguments. They are not mentally unbalanced like their neurasthenic counterparts with normal or low blood pressure, but they are often abnormally touchy, easily upset and easy prey to external stimuli.

In one case in particular (case 23), a widow of 65 years, living alone, I found that under the constant strain of frequent air raid alerts, bombings, gun-fire, and blackout conditions, her blood pressure increased from an average figure of 178/90 mm. to 210/100 during a period of three months.

22. Chronic Constipation and Hypertension. Chronic constipation was a very frequent finding. Actually, although a history of it was not often volunteered, twenty-five cases were found to have long-standing constipation. It was probably present in others of the series, but as many of them had embarked upon a self-prescribed purgation routine, it was difficult to assess the real frequency of this symptom. As a sequel Haemorrhoids were not infrequently found to be

present.

23. Other gastro-intestinal symptoms were, of course, encountered, such as Dyspepsia and Gastric Catarrh, but they did not appear to be related in any way to the hypertension.

24. Chronic Infection of the Gall Bladder. Three of the series were found to have pathological Gall Bladders as shown by Graham's Test. (Cases 5, 7 and 27.)

25. Chronic or Subacute Appendicitis necessitated operation in two of the cases. The hypertension still remained.

Haematemesis occurred in one case (case 90). X-ray examination failed to reveal evidence of an ulcer.

26. A thickly furred tongue was persistently present in two early cases - a boy of sixteen (case 48), and a girl of 17 (case 18). The only findings in both cases, after full investigation, were hypertension and Bacillus Coli urinary infection. In the boy's case the blood pressure registered 150/100 mm. and the girl's 154/86.

27. Haemorrhage (excluding Cerebral Haemorrhage). Repeated attacks of Epistaxis occurred in three cases; Haemoptosis in two cases; Haematemesis once; Haematuria in one case, Melaena in one case, and Menorrhagia in two of the series.

28. Bacillus Coli Urinary Infection. Bacillus Coli was

found in the urine in nine cases (3, 7, 10, 18, 34, 48, 57, 67 and 85). In each case treatment was successful in clearing up the infection, but the hypertension was not substantially reduced.

29. Albuminuria. Albuminuria was found to be present in thirteen of the cases. In five of them it was present when the patient was first seen (cases 50, 61, 69, 73 and 76), but in the remainder it developed at a later stage (cases 28, 30, 54, 62 and 97).

Of the five cases presenting Albuminuria when first seen by me, four on investigation proved to be suffering from chronic nephritis. Renal casts and blood corpuscles were present in the urine. Urea concentration was low and the blood urea was 35, 35, 40 and 45 mgm. per cent respectively. Two others of the series were subsequently found to be suffering from chronic nephritis (cases 30 & 97). Of these six proved cases of nephritis with hypertension, five eventually died of Uraemia.

Seven cases during the period of observation showed Albuminuria varying from a trace to a moderate amount. Three of them (cases 57, 88 and 99) who had previously been free from urinary signs and symptoms developed gradually increasing amounts of albumen, togethers with casts and red blood corpuscles at a late stage in their illness. As

this was coincident with a marked deterioration in their condition and all three showed papilloedema and increased diastolic pressures, the diagnosis of Malignant Hypertension was made.

The remaining four cases showed intermittent and slight Albuminuria unaccompanied by Polyuria and without renal casts. Renal efficiency was normal (cases 28, 50, 59 and 62).

30. Glycosuria was discovered in seven of the patients. Six of them on investigation proved to be suffering from Diabetes Mellitus (cases 3, 25, 53, 74, 85 and 89). Insulin therapy was necessary in four cases, while in the other control was established by an 8 line Lawrence Diet (case 94). In one case Glycosuria was only temporally present. Control of the Glycosuria did not relieve the hypertension in any of these cases.

31. Cardiac Abnormalities.

Aortic Regurgitation was present in four cases (87, 89, 96 and 1). In only one of these patients was there a low diastolic pressure (case 1). Case 87 had well marked arteriosclerosis and an average arterial blood pressure of 220/120. Case 89 was further complicated by Diabetes Mellitus. Her blood pressure was 230/125. Case 96 showed a pressure of 220/130. All four showed considerable cardiac enlargement.

Mitral Stenosis was found in one case (case 69).

Coronary Thrombosis was the cause of death in three cases whose blood pressures estimated 2 years, 14 months and 9 months respectively before death were 180/80, 175/85, 180/90. (Cases 15, 22 and 36.) Case No.42 has a well marked Coronary Thrombosis with a pressure of 160/90.

Hypertrophy of the left Ventricle was a frequent finding at some stage of the illness. This sign was noted in thirty per cent of cases.

Cardiac Dilatation and failure was a terminal manifestation in twelve cases. A marked fall in blood pressure was found to be coincident with this complication.

Auricular Fibrillation was a troublesome symptom during the last twelve months of her life in case 39. It was frequently associated with Cardiac Asthma in this patient.

An Accentuated Second Aortic Sound was well marked in six cases (cases 51, 56, 66, 77, 88 and 91).

Gallop Rhythm and Pulsus Alternous were in evidence in the terminal stages of cases 71 and 75.

32. Acute Pulmonary Oedema. Acute Pulmonary Oedema occurred in 4 cases (cases 39, 54, 70 and 75). In each of these cases it was the direct cause of death, but in two of the cases (54 and 75) there had been previous attacks with recovery.

33. Cardiac Asthma. Although Acute Pulmonary Oedema was the terminal event in the other two cases they had each suffered severe attacks of Cardiac Asthma during the previous twelve months, indicating failure of the left Ventricle.

34. Pulmonary Congestion. Pulmonary Congestion in the form of a basal consolidation proved troublesome in the terminal phases of cases 39, 40 and 99.

35. Hypothyroidism. Two of the patients presented signs of Myxoedema. They were both obese and had the classical coarse thickened features, dry and roughened skin, and loss of expression. Case 53 developed Dementia and was admitted to a Mental Nursing Home. Recovery took place within nine months. A cerebral haemorrhage with hemiplegia subsequently developed but she made a good recovery. Case 94, in addition to Myxoedema, had marked Glycosuria which was controlled by 8 line Lawrence Diet, and marked Arteriosclerosis.

36. Hyperthyroidism. Mild degrees of Hyperthyroidism evidenced by fullness of the thyroid gland, tachycardia, palpitation, moist skin, and abnormal nervousness were noticed in cases 26, 32 and 57.

Increased thyroid activity appeared to account for some of the symptoms in the Menopausal Group.

Large thyroid adenoma and substernal thyroid were both present in case 11.

37. Allergic Manifestations.

Recurrent attacks of Urticaria and Angio-neurotic Oedema were seen in two cases (cases 29 and 83).

Dermatitis was at one stage a troublesome feature of the latter case.

Asthma of the bronchial type occurred in seven of the series (cases 3, 6, 23, 29, 51, 63 and 70). It occurred more frequently in the moderate pressure group. In three of the cases (cases 23, 29 and 63) an appreciable rise in arterial pressure at a later stage of the illness coincided with a cessation of the asthmatic attacks. I have not seen many severe paroxysms of true asthma in patients with really high blood pressure. The only one in this series was case 51. He had a very marked arteriosclerosis and his dyspnoea at times was most distressing. It was asthmatic in type, being mainly expiratory. Three of the cases were resistant to adrenalin.

Cardiac Asthma. Cardiac Asthma, on the other hand, may be a severe and distressing symptom of ventricular failure in the hypertensive patient. It does not appear to be allergic.

Migraine was considered to be a factor in two cases (23 and 100)

38. Chronic Bronchitis. Chronic Bronchitis was

co-existent with high blood pressure in seven of the cases in this series (cases 6, 46, 51, 58, 75, 83 and 84). It appeared to have an aggravating effect on the hypertension. After severe spasms of coughing the systolic pressure was found to be increased in three of the cases (cases 75, 83 and 84). For instance, in case 84 the arterial blood pressure had been estimated at 200/115 two weeks before an exacerbation of Chronic Bronchitis. He was examined at the time of an attack. The pressure was then 220/115. Two days later, after some amelioration of his symptoms, the pressure was 215/115 mm. of Hg. Moreover, a sharp attack of Bronchitis was generally found to aggravate the hypertensive symptoms in each of these cases.

39. Obesity. Extreme obesity was a feature in two cases (74 and 75). In case 74 insulin therapy was required. Well marked obesity was present in five others (cases 8, 33, 53, 62 and 94). Cases 74 and 94 showed persistent glycosuria.

40. Insomnia. Insomnia was a fairly common and troublesome complaint in this series. No fewer than sixteen complained of this disability (cases 3, 7, 9, 16, 23, 26, 30, 32, 57, 59, 60, 67, 68, 83, 90 and 100).

41. Other Complications.

The unusual combination of arterial hypertension co-existent with Pernicious Anaemia, Diabetes Mellitus and

Bacillus Coli urinary infection was found in case 85.

In addition to hypertension, case No.11 had attacks of Angina Pectoris attributed at first to Coronary Thrombosis. Further investigation, however, revealed a large substernal thyroid. Surgical removal of this tumour and the normally placed enlarged thyroid gland, relieved the angina for the time being. The arterial pressure remained at its former level. Two years later, anginal attacks again returned, although not of their former severity.

A malignant brain tumour (Glioblastoma) in case 92 provided all the signs and symptoms of a malignant hypertension.

The foregoing tabulation of signs and symptoms are those met with in 100 consecutive cases in which arterial hypertension was persistently present.

It is not intended to imply that all the symptoms mentioned above are part and parcel of the hypertensive state.

Many of them fall logically into the picture of essential hypertension. Others, by their appearance in so many cases of the series, cannot be regarded altogether as extraneous. Even where other definite but co-existent diseases and their symptoms are mentioned they are worthy of consideration from the point of view of the hypertension.

Mortality Table - Table 7.

During the period under review 45 deaths occurred in this series.

Immediate Cause of Death.			Average age at Death.
53 per cent	Heart Failure	17 deaths	63 years
	Coronary Thrombosis	3 "	50 "
	Acute Pulmonary Oedema	4 "	72 "
26.6 per cent	Cerebral Haemorrhage	12 "	68 "
11 per cent	Uraemia	5 "	62 "
	General Paralysis of Insane	1 death	
	Diabetic Coma	1 "	
	Suicide	1 "	
	Cerebral Tumour	1 "	
		45 deaths	

Further Notes regarding the cause of death, the type of individual, and the severity of the hypertension.

The three main immediate causes of death were:-

(1) Conditions affecting the heart, representing 53 per cent of deaths.

(2) Cerebral Haemorrhage, representing 26.6 per cent of deaths.

(3) Uraemia, representing 11 per cent of deaths.

The average age at death was similar in the heart failure and Uraemic groups (age 62 to 63), but was 68 years in the apoplexy group.

Of the twelve deaths from apoplexy, ten were florid short-necked individuals. Seven of the twelve had only moderately severe hypertension.

Cardiac failure accounted for seventeen deaths. Both types (i.e. florid and sallow) were about equally represented. Nine were florid complexioned and eight sallow. A severe degree of hypertension had existed in ten of the seventeen patients.

Those who succumbed to Acute Pulmonary Oedema were of the florid type. They had severe hypertension.

Uraemia was the cause of death in five cases, four of whom were sallow in type and had relatively high arterial pressures.

Table 8 - Summary of Signs and Symptoms found in 100 cases of Hypertension.

	Signs and Symptoms.	No. of Cases
1.	Dyspnoea	28
2.	Substernal Discomfort and Tightness	4
3.	Headache - Severe 25) Moderate 24) Mild 18)	67
4.	Mental Confusion - Mild 16) Moderate 5) Severe 5)	26
5.	Vertigo + Vomiting 1) + Headache 5)	30
6.	Tinnitus	7
7.	Loss of Weight	10
8.	Loss of Energy	25
9.	Paraesthesia - Tingling and Numbness of hands and feet	6

	Signs and Symptoms.		No. of Cases.
10.	Neuritis		14
11.	Rheumatic Manifestations (Fibrositis, Myalgia, Lumbago, etc.)		10
12.	Intermittent Claudication		1
13.	Flushing of the Skin (excluding Menopause)		12
14.	Cerebrospostic Symptoms. Temporary Hemiplegia, Paresis, etc.		18
15.	Cerebral Haemorrhage and Hemiplegia		12
16.	Syncope		2
17.	Eclampsia		3
18.	Arteriosclerosis		14
19.	Hypertensive Encephalopathy		5
20.	Eye Changes - Temporary Blindness	4)	24
	Arteriosclerotic Changes	10)	
	Albuminuric Retinitis	4)	
	Diabetic Retinitis	1)	
	Papilloedema	4)	
	Diplopia	1)	
21.	Nervous Symptoms - Neurosis		31
22.	Chronic Constipation		24
23.	Gastro-intestinal Symptoms - Dyspepsia, Gastric Catarrh		12
24.	Chronic Cholecystitis		3
25.	Appendicitis		2
26.	Thickly furred tongue		2
27.	Haemorrhage (excluding Cerebral Haemorrhage) - Epistaxis	3)	10
	Haematemesis	1)	
	Haematuria	1)	
	Haemoptosis	2)	
	Melaena	1)	
	Menorrhagia	2)	
28.	Bacillus Coli C. Urinary Infection		9
29.	Albuminuria - Chronic Glomerulo Nephritis	6)	13
	Malignant Hypertension	4)	
	Essential Hypertension	3)	
30.	Glycosuria - Diabetes Mellitus	6)	7
	Glycosuria	1)	
31.	Cardiac Abnormalities:-		
	Aortic Regurgitation	4)	
	Mitral Stenosis	1)	
	Coronary Thrombosis	4)	
	Hypertrophy of the left Ventricle	35)	

	Signs and Symptoms.		No. of Cases.
	Cardiac Dilatation	12	66
	Auricular Fibrillation	1	
	Accentuated 2nd Aortic Sound	6	
	Pulsus Alternous	2	
	Coarctation of Aorta	1	
32.	Acute Pulmonary Oedema		4
33.	Cardiac Asthma		2
34.	Pulmonary Congestion		2
35.	Hypothyroidism - Myxoedema		2
36.	Hyperthyroidism (excluding Menopause)		4
37.	Allergy - Urticaria	2	14
	Angio-neurotic Oedema	2	
	Dermatitis	1	
	Bronchial Asthma	7	
	Migraine	2	
38.	Chronic Bronchitis		7
39.	Obesity		7
40.	Insomnia		16
41.	Other Complications and Associated Conditions:-		
	Pernicious Anaemia with Diabetes		1
	Glioblastoma of Brain		1
	Coarctation of Aorta		1

Further discussion of the Symptomatology in this series with illustrative case histories.

Hypertension in its earliest manifestations cannot, of course, be considered as a disease or a syndrome, but merely as a sign denoting reaction to certain stimuli.

It is rather significant to find on analysing my early cases that a common factor was operative.

Case 48.

A boy aged 16, always had a thickly-coated tongue; he complained of soon being tired, and of frequent aching in his legs. Occasionally he had a sensation of faintness.

Tachycardia was present. He was tall and very thin, clear skinned and growing rather quickly. He had a narrow undeveloped chest. No cardiac enlargement could be detected nor were any murmurs present. The arterial blood pressure was 150/90 mm. of Hg. The urine was of low specific gravity. A catheter specimen showed faint trace of albumen, a few pus cells and red blood corpuscles and a large number of coliform bacilli. No tube casts were present. Blood urea was 23 m.gm. per 100 cc. Urea concentration was satisfactory. Intake of red meat, soups and other purin containing foods was restricted. Alkaline diuretics and later Mandelic Acid (Mandelex) were prescribed. Three months later the urine was reported normal. Blood pressure was then 150/100 mm. Twelve months later, although the urine was clear, the tongue was still coated. Tachycardia was present and the blood pressure 140/90. In January, 1941, almost 4 years after his initial investigation, his pressure was 160/90.

In March, 1941, I was a member of an Army Recruiting Board and had a further opportunity of examining this case along with my colleagues. On further examining his arterial blood pressure I found that it was then 175/100 on both brachial arteries, but on the femoral arteries it was only 140/90. A systolic bruit was heard over most of the cardiac area. The pressure readings were verified by the Chairman

and the other members of the Board. We, of course, rejected the recruit on the diagnosis of Coarctation of the Aorta.

Case 18.

Six months ago a girl of 18 consulted me on account of severe headaches which had been consistently troublesome for three months. She also complained of faint feelings and aching of the legs and back. Her urine contained a trace of albumen and a large number of coliform bacilli. Her blood pressure was 154/85. Blood urea was normal. The urinary condition cleared up in a few weeks, but the blood pressure 5 months later was still 150/85 mm.

In both these cases the Bacillus Coli urinary infection was of a rather insidious nature, in that the patient did not complain of any urinary discomfort.

Going through my case histories I found definite evidence of Bacillus Coli C. urinary infection in Case 7 when she was aged 36; in case 67 when aged 34, and in case 69 at the age of 33. Three of the older patients (cases 3, 34 and 82) had attacks of Bacillus Coli urinary infection when aged 59, 55 and 59.

In addition a suggestive history was obtained in six further cases (9, 26, 64, 70, 76 and 98).

Seventeen patients had a history of chronic constipation, three of gall bladder infection, and two of chronic or subacute

appendicitis. One patient had a faecal fistula.

It would seem, therefore, that an early search for infection by Bacillus Coli and its elimination is of paramount importance. Unfortunately, treatment of this condition did not in the majority of cases relieve the hypertension.

Since writing the above case histories I was very interested to find a certain amount of confirmation and explanation in a recent paper in which Weiss and Parker (1939)(55 suggest that 15 to 20 per cent of cases of malignant hypertension arise originally from pyelonephritis.

"In acute pyelonephritis or pyelitis the inflammation is primarily interstitial and recovery is the rule. In the chronic or healed varieties," these authors state, "vascular changes are often prominent and there may be concentric proliferation and even necrosis of arterioles. Cases are frequently associated with hypertension and the vascular changes are diffuse and severe, and often identical with those of malignant hypertension."

So protean is the symptomatology in the group showing moderate hypertension, particularly regarding nervous symptoms that the question of susceptibility immediately obtrudes itself.

If the seed in the shape of a Bacillus Coli infection, a toxin, a pressor substance, or other form of primary stimulus,

is scattered widespread, it would seem that a fertile soil is requisite for its germination. In other words, a type of individual exists who reacts to certain stimuli in such a way that hypertension results from the arterial responses. This may be an inherited characteristic or susceptibility, even one, as suggested by Rolleston,[21] with dominant Mendelian characteristics.

It is, of course, forcibly brought to mind by studying the family histories of many of these cases, particularly where there is evidence of repeated apoplectic catastrophies in one family, but it seems to be even more subtly present when one considers the characteristics of a series of hyperpietics.

A family history of arterial disease or cardio-vascular weakness was obtained in 15 cases.

What type of individual then is the potential hyperpietic?

Enter suspect number one, and I use the term advisedly because even to the layman he is suspect from the start. He is often self-diagnosed even before the hypertension is in evidence.

We can all visualise him, that plethoric, florid, rubicund, often jovial but quick-tempered, short-necked, stockily built character, dynamic and bristling with activity

yet withal dogged and stubborn.

In his early fifties his pressure gradually creeps upward, and before arteriosclerotic changes are noticeable, or his danger is even appreciated, Angina Pectoris may call a sudden halt to his activities or Coronary Thrombosis with dramatic suddenness end the story.

If he weathers the storm, or if he escapes, he still must run the considerable risk of a cerebral haemorrhage with its attendant sequelae.

Again he may be fortunate. Arteriosclerosis becomes more than a suspicion, perhaps even a protection, because as the blood pressure becomes higher the cerebral arteries become more able to withstand it.

His character, however, gradually changes paripassu with his changing arteries. Gone is that dynamic activity. His actions, his step, even his concentration and cerebration are perceptibly slowed down. Slowly and gradually dyspnoea develops, but before it is fully evident to him he complains of exhaustion. Aching thighs and leg weariness are disturbing features of his failing health. Insomnia mars his nights and confusion of thought and idea hinder his already diminished daytime activities.

His florid complexion waxes and wanes under the influence of frequent flushes. An Urticaria, Erythema or Seborrhoeic

Dermatitis may cause him further distress.

Attacks of Dyspnoea leave no doubt as to its cardiac origin even if the enlarging left ventricle were not so easily demonstrable, or that second aortic sound so audible on auscultation.

About this time perhaps attacks of dizziness may complicate the picture. Even before this stage resistance to infection is so lowered that bronchitis and rheumatism are often present or some other intercurrent illness or infection may accelerate the end.

Not all my suspects are of this well defined build and appearance. A minority of them, while conforming to the other characteristics of the florid group, are tall and big-boned, almost of acromegalic type. The long narrow jaw and large skull give one the impression of pituitarism. Usually of florid type their symptomatology is that of the group already described, but their hypertension is at a more consistently high level.

In this series seven cases belonged to this sub-group.

To complete the picture one must mention the opposite type of individual who is perhaps a hyperpietic in the truest sense of the word, and whom we encounter equally as often.

In contra-distinction, however, we do not suspect him unless we know him as a nephritic. In my experience females

predominate in this group.

Excluding cases of proved nephritic origin, however, we often find our most difficult cases in the group of sallow, thin or frail, often toxic looking, high strung individuals. Apprehensive and narrow-minded, as they frequently are, their hypertension often remains unsuspected until acute dyspnoea, cardiac asthma or pulmonary oedema focuses attention, all too late, on the obvious.

In the earlier stages the care-worn facies is accentuated by the already sallow complexion and is not belied by the neurasthenic history so frequently given.

Later, however, they tend to assume a more placid and resigned mien. Possibly as a result of relative cerebral congestion, more clear thinking and reasoned arguments are now advanced. Their sole thoughts are centred on their immediate distress which is now very real.

Although some anxiety is still written on their features it is not from fear of the unknown but from a knowledge of their own inevitable decline.

Renal function may become progressively impaired with all its attendant features and Uraemia as its terminal stage.

More often cardiac failure by slow and uncomfortable stages with increasing dyspnoea, cardiac asthma, or even pulmonary oedema completes the history.

Death may come from apoplexy or intercurrent disease such as pneumonia.

I have thus briefly from my knowledge of these patients attempted to draw a composite picture of the individual who seems to be susceptible to hypertension.

Both main types as described above appear to carry with them a definite mental make-up or characteristic personality.

The florid type of hyperpietic is more often than not dynamic, over-active, sensitive, and quick-tempered, but still pleasant and cheerful withal. On the whole he is uncomplaining.

The thin sallow victim of hypertension has often a typical facies with care-worn, well creased forehead and heavy sunken eyes. More commonly of the female sex, she is a chronic grumbler, irritable, narrow-minded and introspective. Whether she is of the extreme type depicted above, or merely very high strung and touchy, she presents a definite and recognisable nervous symptom complex.

A theorist might call into question the stability of the vaso-motor system of these patients and label them vaso-labile.

Having considered the type of individual who is most often afflicted by hypertension and its concomittant symptoms, let me for a moment try to draw a distinction between

secondary hypertension and essential hypertension.

I have considered hypertension in rather general terms in the foregoing discussion. All my patients in this series have at least had one clinical sign in common. They have had raised arterial blood pressure.

A consideration of the group symptomatology and of the individual case histories shows us, I think, that hypertension has a definite place both as a symptom and as a disease.

It is perhaps only when we come to consider the frank, really high blood pressures illustrated in my second group of cases that we realise that we are dealing with a definite entity which can be characterised as a disease per se.

Many in this group have not gone through the gamut of intermediary symptoms. Stripped of most of the trappings of vague symptomatology encountered in our milder cases we find a genuine illness, characterised by a definite group of signs and symptoms which are directly attributable to the hypertension.

These signs and symptoms have already been described in an earlier section and are further illustrated in the case histories given.

The diastolic pressure is maintained at a persistently high level of 100 mm. of Hg. or more, while the systolic

pressure may soar to over 300 mm.

We are familiar with the almost classical headache, either frontal or occipital, which increases as the day goes on or is aggravated by stooping or sometimes by lying down; the dyspnoea sometimes in evidence before definite cardiac signs are manifest; the loss of weight, inertia and weakness; the pallor; the ocular signs of retinitis, arteriosclerotic changes, haemorrhages and even in extreme cases papilloedema; later evidence of renal defects, and the final manifestations of cerebral damage or cardiac failure.

If we substitute the name of Essential Hypertension for Hyperpiesia in our description of this condition, we must at least pay tribute to the discernment of the late Sir C. Allbutt who showed us how wrong we were in attributing so many of these cases to Chronic Bright's Disease.

In Secondary Hypertension, on the other hand, the rise in pressure can usually be attributed to a definite primary disease or to physical changes occurring in the body as a result of a definite clinical condition.

I have already considered the association of hypertension with renal disease. Six cases in this series were suffering from Chronic Glomerulo Nephritis.

One case only (Case 48) was found to have Coarctation of the Aorta.

Other conditions met with in this series in association with hypertension were Pyelonephritis, Pregnancy Kidney, Aortic Regurgitation, Menopausal symptoms, Obesity, Glycosuria and Cerebral Tumour.

Twenty cases could be grouped in the category of secondary hypertension. Pickering states that this type of hypertension accounts for probably less than 25 per cent of the cases of high blood pressure occurring in temperate countries. The remaining majority, according to this authority, constitute essential hypertension.

In this series I consider that sixty of my patients came within this category. Fifty-six of them suffered from benign essential hypertension and four from malignant hypertension.

Clinically I found that I could divide them into two main types:-

(a) A cerebral or cephalgic type is encountered in which there is no marked dyspnoea until the terminal stages, but unusually severe headache is persistently present.

(b) A cardiac type exists in which the dyspnoea is the most prominent symptom while headache is absent or insignificant.

Vertigo is extremely common in both classes.

Malignant hypertension, although it may follow benign

essential hypertension, is not confined to that category. It may supervene as a terminal phase where there has been considerable kidney damage from other causes than essential hypertension.

Although not included in this series I recently encountered such a case.

A woman of 54 complained of urinary symptoms. Initial investigation revealed many coliform bacilli in the urine. Cystoscopy failed to reveal anything other than cystitis. In spite of treatment by alkalis, sulphonamides and Mandelic Acid, the patient's condition did not improve. Her blood pressure was 205/135 mm. Hg. I arranged for further investigation and it was discovered that she had a very large renal calculus filling the pelvis of the right kidney. The right kidney was not functioning. The left kidney only functioned to the extent of 5 per cent normal. The blood urea was over 60 mgm. per cent. Operation was contra-indicated. Meanwhile the blood pressure rose to 250/150 mm. Hg. Severe headaches were complained of. Well marked papilloedema and retinitis were discovered. Haematuria occurred several times. Death took place from Uraemia three months after she had originally consulted me.

A further analysis of the series of 100 cases reveals a group of cases representing approximately 20 percent, whose

category is difficult to assess. Actually twenty-two of these patients were considered by me to have arterial hypertension. They all showed systolic pressures of over 160 mm. Hg., some of them considerably over that figure, but their diastolic pressures were below 90 mm. Hg. One patient had aortic regurgitation; three died from coronary thrombosis, and two eclamptics were included in this group.

The remaining 16 per cent, including five patients who had menopausal symptoms, suffered mainly from nervous manifestations. They were not seriously ill nor did they subsequently develop any of the signs and symptoms of essential hypertension.

A consideration of their histories has led me to the conclusion that a systolic arterial pressure of 180 or 190 mm. Hg., unless it is accompanied by a diastolic pressure of over 90 mm. Hg., is of no more significance than a rapid pulse rate.

My experience as a member of a Ministry of Labour and National Service Recruiting Board has convinced me that a systolic pressure of 170 mm. Hg. is not incompatible with a Grade I standard of fitness. Indeed, the diastolic pressure alone is the criterion for assessing hypertension, as laid down by the above Ministry.

The definitions of hypertension based on systolic

arterial pressures given by Osler,[4] H. Batty Shaw,[5] Halls Dally,[6] and Reginald Hilton,[8] for example, can no longer be admitted; nor should the systolic pressure tables of Insurance Companies be used as a standard of fitness per se.

Further Illustrative Case Histories.

Simple Benign Hypertension.

Lever, E. (Case 40). Spinster of slight, spare build, sallow, of rather toxic appearance, aged 65. Her mother died of Cancer when aged 52 and her father of Pneumonia at 49. Although she was timid, nervous and apprehensive, she had a bright and cheerful disposition; she worked in a cotton spinning mill for 50 years until her illness forced her to give up.

Prior to 1934 she had no serious illness.

In December, 1934, then aged 59, she first complained of Dyspnoea. This was accompanied by a slight, purposeless, nervous cough. Blood pressure was 200/100 mm. Hg. In spite of advice she persisted, except for short periods, in keeping to her work until November, 1935.

In addition to increasing Dyspnoea she complained of loss of weight. No cardiac enlargement was present and Exercise Tolerance was fairly good. Some arterial degeneration was evidenced by Arcus Senilis of both eyes. No cardiac valvular

lesion was present. Blood pressure was 215/100 mm. Hg.

Since that time until her death in December, 1940, she was under constant supervision. Regarding her incapacity for work the Regional Medical Officer reported in July, 1936, "Feeble heart sounds, shortness of breath, B.P. 210/100. She has a cough and harsh breath sounds. General physique is feeble." And in December, 1936, "Shows cardio-vascular changes with a high blood pressure, and complains of dyspnoea on effort. Renal function satisfactory." X-ray of the chest and sputum tests were negative for tuberculosis. With rest, diet, graduated exercises and drug therapy the dyspnoea improved. Except for periodic attacks of Bronchitis she remained fairly comfortable. The blood pressure in March, 1938, was 168/75. In March, 1940, she developed Pleurisy of the right base which gradually resolved. After 6 weeks in bed the pressure was 175/75, and in July, 1940, when she was getting about again 180/80. Moderate dyspnoea on effort was present, and the heart was slightly enlarged to the left. In December, 1940, dyspnoea became more acute, the heart became further enlarged, and consolidation appeared at the right base. At first she was afebrile, but after 5 days the condition was indistinguishable from Lobar Pneumonia. She died on December 31st, 1940, aged 65.

Drug Therapy. (Case 40)

Strychnine in for form of Liq. Strychnin. Hydrochlor. M.V. combined with Tinct. Strophanth. and Tinct. Card. Comp., or Tinct. Nucis Vom. MX with Sodii Bicarb. and Infus. Gentiae, almost invariably gave symptomatic relief of the dyspnoea until the later stages.

Most benefit, however, was obtained from the extended use of a prescription originally advocated by the late Sir William Gower for cases of migraine.

Sodii Bromide	gr.X
Tr. Gelsemini	MX
Liq. Strychnine	MV
Liq. Trinitri	M½
Ac. Hydrochlor. dil	MV
Inf. Gent. Co. ad	℥j Ft. Mist.

One tablespoonful in water after food, night and morning.

Comment.

The original picture presented by this patient was that of Essential Hypertension (cardiac type). Both systolic and Diastolic pressures remained high for at least 2 years. Gradual ventricular failure was accompanied by a lowering of arterial pressure. Headache was absent.

Essential Hypertension - Myxoedematous Type with Glycosuria.

Case 94. McCaffer, age at commencement of treatment 63. This patient is of heavy build, rather stout, with puffy and

coarse features. Her previous history shows frequent attacks of muscular rheumatism, chronic constipation and haemorrhoids. She has been under my care for 6 years.

Initial Symptoms:- No energy, dyspnoea on exertion, general discomfort in lower abdomen associated with extreme constipation, rather vague rheumatic pains. Her arterial blood pressure then was 210/100 mm. of Hg. Later she complained of thirst and muscular weakness. Some glycosuria was present, but was controlled by an 8 line ration Lawrence diet. In spite of freedom from glycosuria, dieting, Potassium Iodide gr. XXX thrice daily, and adequate purgation, the blood pressure in May, 1940, had increased to 220/130. Attacks of dizziness were becoming troublesome at this time. Urinary investigation did not show any signs of renal deficiency - no albumen or casts were found on repeated examination. The heart was slightly enlarged to the left, but no valvular lesion was present.

The administration of Thyroid Extract improved the symptoms. A reduction in weight and an improvement in the skin and facies resulted, but no change in the level of blood pressure was achieved. Theominal Tablets (Bayer) (Theobromin et Phenobarbitone) gave the patient considerable comfort and after 14 days caused a reduction of the hypertension to 190/110.

In January, 1941, after an interval without drug treatment, the pressure was 220/110. The blood pressure reading in June, 1943, was 230/130 mm. Hg.

During the previous 6 months her only treatment had been by rest, diet and purgation.

From 1941 to 1943 there had been little variation except during 3 periods when Theobromin and Phenobarbitone was used and a general lowering of the pressure, both systolic and diastolic, resulted. (200/110, 215/115, 215/120)

Her present condition is one of chronic invalidism. She is unable to walk beyond the confines of her home. Dyspnoea on effort is fairly severe, but while resting she complains little. She has not complained of headache. Apart from arteriosclerotic changes in the fundi no other abnormality was noted. The blood pressure in July, 1943, was 210/110 mm. Hg. Slight Albuminuria, but no casts, were found in the urine - further investigation was declined.

Comment.

This case also is one of Essential Hypertension (cardiac type). Although complicated by some obesity, glycosuria and a mild degree of myxoedemia, the fact that treatment of these complications did not alleviate the hypertension rather tends to negative the suggestion of secondary hypertension.

This patient has been under constant observation for a

period of 6½ years.

Apart from slight variations in her blood pressure above the initial level, the pressure is 210/110 mm. Hg. compared to 210/100 six years ago. Her present physical condition has not deteriorated more than one would expect after this period of years.

Essential Hypertension - Acromegalic Type.

Case 83. Crankshaw, male, single, age 65, Cabinet-maker. Tall, big-boned, florid, with large skull and heavy jaw - nervous and apprehensive. Previous illnesses - Seborrhoeic Dermatitis, Urticaria, Angio-neurotic Oedema, Inf. Influenze (wice) with severe post-influenzal debility, Anterior-crural Neuritis.

Early symptoms of present illness.

March, 1938. At first he complained mainly of exhaustion, and weakness of the thigh muscles, stiffness and neuralgic pains. Considerable loss of weight had occurred.

In February, 1939, he felt very weak, particularly in the legs, and had attacks of extreme dizziness. Blood pressure was 200/105 mm. Hg.

In April, 1939, always felt tired; had lost another 5 lbs. in weight; complained of pains in knees and ankles; very constipated.

June, 1939. Still very exhausted, light-headed at

times and dizzy. Later he had severe headaches, felt confused and could not concentrate.

In March, 1940, pains in both thighs were still troublesome. Dyspnoea on exertion was now felt. Blood pressure was then 220/110 mm. Hg. The heart was found to be enlarged slightly to the left. No murmurs were present. No evidence of renal efficiency was found.

In October, 1940, after playing a game of chess he found himself unable to rise. Paresis of the left leg and arm and of the right side of the face was present. Recovery ensued in 48 hours but a true hemiplegia occurred a few days later. Three months later complete recovery from the hemiplegia had taken place.

In January, 1941, his blood pressure was 230/115 mm. Hg. Gradually increasing weakness, attacks of dizziness and lapses of memory made it necessary to send him to hospital for nursing treatment. He died there 9 months later from a cerebral haemorrhage.

Drug Treatment.

Strychnine was most helpful in countering the exhaustion, but small doses of Phenobarbiton gave most symptomatic relief.

Comment.

This patient was observed over a period of 4 years. His most troublesome symptoms were physical exhaustion and loss

weight.

Essential Hypertension with Bad Family History and High Diastolic Pressure later becoming Malignant Hypertension.

Case 98. Bradley, widow, age 60. Slight build, sallow, nervous, but very active. Her father and sister died of apoplexy. Her husband had been an invalid for 8 years before his death.

Previous Illnesses.

These included Rheumatism. Haemoptysis occurred 3 years before the present illness. No signs of Tuberculosis were discovered.

Recent History.

In April, 1938, she complained of severe vertigo - felt top heavy. Her blood pressure then was 230/110 mm. Hg. Urine S.G. 1015. Slight trace of albumen. No casts; blood urea 30. Improvement of subjective symptoms resulted from Gower's Mixture. Iodism precluded the use of Potassium Iodide.

In May, 1939, the pressure was 240/115 mm. Hg. A course of Dentensyl Tablets failed to relieve her condition.

In September, 1939, she complained of increasing dyspnoea on exertion. Gower's Mixture again gave considerable relief.

By November, 1939, the dyspnoea had become more severe.

Some cardiac enlargement to the left was found and an apical systolic bruit was present. Complaint was frequently made of a tight feeling of the chest, and a confused sensation in the head.

In May, 1940, she still complained of dyspnoea on exertion, but it was not constant. Her head felt muzzy. Haemorrhage was present on left conjunctiva and ophthalmoscopic examination showed marked arteriosclerotic changes but no papilloedema. The blood pressure was then 250/140 mm. Hg. Two courses of Elixir Sulphocyanate failed to give any relief. Caffein Iodide in the form of the Elixir (Nat. Formulary 1939) did not cause Iodism and was helpful, but Theominal (Bayer) was easily the most successful drug in alleviating symptoms.

In January, 1941, loss of concentration and vertigo were more marked. An examination of the fundi revealed retinal haemorrhages and a well marked papilloedema. Blood pressure was then 270/140. This evidently marked the onset of Malignant Hypertension, but strange to say severe headache was not complained of. Albumen was now more abundant in the urine and casts and red blood cells appeared. Slight haematuria was present for 10 days in February. Further urinary investigation was not carried out as the patient refused hospital treatment. The patient's cardiac symptoms

gave more trouble, especially with nocturnal attacks of dyspnoea. Her pressure on January, 1942, was 280/150.

In June, 1942, eighteen months after the apparent onset of the Malignant phase, she finally succumbed during an attack of cardiac asthma. This was actually over 2 years after the diastolic pressure had reached a level of 140 mm.Hg.

Hypertension co-existent with Pernicious Anaemia, Diabetes Mellitus and Bacillus Coli Urinary Infection.

Case 85. Lamb - widow, age 59. Florid, very apprehensive and nervous.

In May, 1937, she had an acute febrile illness with pain in her left loin. The urine contained pus and coliform bacilli in large numbers. She was rather anaemic and slightly dyspnoeic. Investigation of the blood revealed a typical Pernicious Anaemia; Haemoglobin was 62 per cent; Erythrocytes numbered 3,200,000; Mean diameter 7·8 m. Anisocytosis, Poikilocytosis and nucleated reds were present. In addition, sugar was found in the urine and a blood sugar reading of 368 obtained. Insulin was given in doses of 8 and 10 units daily, "Hepol" Liver Extract 4 cc. twice weekly, and Mandelex by mouth to control the Bacillus Coli Infection. In spite of treatment the anaemia worsened.

On 27th August, 1937, Haemoglobin was 42 per cent; Erythrocytes 2,020,000; Colour Index 1·05.

In October, 1937, she had an attack of Hyperglycaemia when the urine was found to be loaded with sugar and acetone. The blood sugar was 442.

By March, 1938, the blood picture was normal. Haemoglobin 104, Erythrocytes 5,080,000; Colour Index 1·04; Mean diameter 7 m. The blood sugar then was 177 and was stabilised on 12 units per day of Insulin. Liver Extract was continued as injections of 2 cc. every fortnight.

In May, 1938, the patient complained of throbbing headaches, particularly first thing in the morning. Attacks of vertigo were frequent and feelings of faintness on standing. Insomnia was troublesome. Frequent flushings of the face occurred. She was very apprehensive and nervous. Blood pressure was found to be 210/115. Theominal ·T tablet 3 times daily gave immediate symptomatic relief and was prescribed at intervals during the following five years always with acceptance.

In July, 1940, the arterial blood pressure was 180/110 mm. Hg. Although frequently tested it was never found to be below this figure.

In June, 1943, the reading was 200/115. With diet, purgation, rest and occasional resource to Theominal, she kept comparatively symptom free. Maintenance doses of Liver Extract and Insulin, controlled by laboratory tests,

kept her free from anaemia and diabetes. There has not been a recurrence of the Bacillus Coli Urinary Infection. No ocular signs have been discovered. Heart is normal.

Comment.

In the rather complicated picture presented here the possible relationship of the hypertension to the other conditions present or to the treatment given gives much room for thought.

Pernicious Anaemia per se is most often accompanied by hypotension, while Hepatic extracts have actually been advocated in the treatment of hypertension. Hypertension is a not infrequent associate of diabetes or glycosuria, but not a symptom of it. (Cases 3, 25, 53, 74, 85 and 89.)

The frequent finding of Bacillus Coli urinary infection in hypertension cases has already received comment. Except where a pyelo-nephritis has been present little support has been accorded to the possible hypertensive proclivities of the Coliform Bacilli.

It would seem most probable that this is a case of Essential Hypertension in association with other diseases, rather than a secondary hypertension arising from the associated conditions.

Essential Hypertension and Arteriosclerosis.

Case 29. Riley, male, age 72. Florid, short-necked,

plethoric type.

Previous History.

His medical record card gives a history of hypertension since 1921, but in 1934, although I took his blood pressure on several occasions, it never exceeded 168/80 mm. Hg.

An attack of Herpes Zoster occurred in 1934, and in 1937 Urticaria and Angio-neurotic Oedema were troublesome.

Since 1937, he has had severe recurrent attacks of dyspnoea with tightness and pain in the chest. During these attacks he was fatigued by the slightest movement and prolonged rest in bed was necessary.

In July, 1937, the heart was slightly enlarged, but no other sign of cardiac disease was present. No signs of renal deficiency were present.

During the intervals between attacks dyspnoea was not troublesome, but vertigo was frequent in occurrence. Blood pressure was then 200/110. Arteriosclerosis was well marked, the radial arteries being very tortuous and even the brachial arteries being palpable. The second aortic sound was accentuated. Steaming of the cornea was present and on ophthalmoscopic examination the arteriosclerotic condition of the retinal veins was visible.

In July, 1939, dyspnoea was more acute and the heart was further enlarged to the left. He complained of extreme

weakness of the legs and at times almost staggered like a drunken man. He felt top heavy. The blood pressure was then 260/120 mm. Hg.

During the last 4 years, since July 1939, he has been very feeble and his activities have been restricted to moving about the sitting room for a few hours daily.

In January, 1941, the blood pressure was still 260/120 but on July, 1943, was 190/90 mm. Hg. Dyspnoea supervenes if he attempts to move.

Drug Treatment.

Tinct. Nucis. Vomic. with Gentian and Soda gave relief for his earlier symptoms. Potassium Iodide in 15 grain doses was helpful, while Gower's Mixture gave great symptomatic relief from time to time.

Comment.

Here again we have a patient who is able to carry on in a restricted way for a period of 7 years with a systolic pressure of over 200 and diastolic over 110 mm. In fact, for almost 4 years the average pressure has been 260/120 mm.Hg.

Strychnine has been the sheet anchor in the drug therapy of several of these cases. Restriction of activities has had to be severe. Once this is obtained it is surprising how symptom free these patients can be kept in face of such a high arterial pressure.

Malignant Hypertension.

Case 100. Peirce, male, married, age 56; fairly tall, big build, rather florid. Occupation - newspaper reporter.

Two years ago he complained of very severe occipital headache which was getting worse. His blood pressure was found to be 260/140 mm. Hg.

Dr. Ferguson of Manchester Royal Infirmary reported kidney efficiency normal. Skull X-ray negative. Blood pressure 270/150 mm. Hg. Hyperpiesia was diagnosed and migraine was suggested as a possible factor.

A mixture containing Potassium Iodide and Potassium Bromide was prescribed together with Phenobarbitone gr. ½, night and morning - later increased to gr. ¾.

The arterial pressure, however, remained consistently above 250/140 mm. Hg.

Eighteen months later he again consulted me as his headaches were getting unbearable and were worse in recumbency. Phenobarbital was making him too drowsy in the daytime. He had been taking a mixture containing Sodium and Magnesium Sulphate with some apparent relief. His blood pressure was 270/160 mm. Hg. The heart was slightly enlarged to the left, but no dyspnoea was complained of. Theominal was substituted for the Phenobarbital and was much more acceptable.

On examining the fundi some marginal blurring of the discs was apparent. Some small haemorrhages were present round the left disc.

This slight degree of papilledema points to a diagnosis of Malignant Hypertension, but the interesting feature of the case is the extraordinary high pressure, particularly diastolic, which has been present for over 2 years. During this period the man has continued at his employment as a newspaper reporter (against medical advice).

I afterwards learned that this patient was killed in a motor accident twelve months after the date of my last consultation with him.

Chronic Nephritis with Hypertension - later developing Malignant Hypertension.

Case 99. Hall, female, age 63.

In August, 1940, she complained of severe occipital headache. Her blood pressure was 180/100 mm. Hg. Albumen was present in moderate amount, renal casts and blood corpuscles were found. No ocular signs were found then. Treatment was advised for the Nephritis and further investigations arranged. Unfortunately she left the town for a period and did not return to me until June, 1942. She then complained of vomiting, thirst, polyuria and constipation. Loss of weight was most marked. The urine contained a

large amount of albumen; no sugar was present. The blood urea was 44 mgms. per 100 cc. Papilledema was well marked in the right disc. Blood pressure was 280/140 mm. Hg. Malignant Hypertension was obviously present.

In addition to general treatment - advice regarding diet, rest, laxatives, hot baths - this mixture was prescribed:

Potas. Iodid. ℥ij
Sodii Bicarb. ℥ii
Spt. Aether Nitros. ℥iv
Inf. Gent. Co. ad ℥viii

1 tablespoonful 3 times a day.

Although the patient had been able to attend my rooms and visit the Infirmary for investigation, she was soon unable to leave the house.

Nocturnal attacks of dyspnoea became increasingly severe. On three occasions convulsions occurred.

The patient's condition rapidly deteriorated. On June 17th, 1943, her blood pressure had dropped to 160/90 mm. Hg. Twelve days later she died in a Uraemic Coma.

Prognosis.

From my personal experience I consider that hypertension, and more particularly essential hypertension, is a very restricting and incapacitating malady of slow and insidious growth. In spite of this, I maintain that the immediate or early prognosis is much more favourable than is generally accepted.

The words "Blood Pressure" once so ominous to the patient should no longer have the same dread significance. We realise now that many useful years may lie before him.

In a moderately severe case of hypertension a fairly normal pressure may be obtained by treatment and the patient maintained in a satisfactory degree of comfort for some years. Eventually the battle will be a losing one as the hypertension gradually reasserts self and the cardiac muscles give in to the constant strain or the cerebral arteries weaken.

To me it is remarkable, however, to find how long a patient can carry on with a very high blood pressure. Hilton[8] for instance states that a diastolic pressure of 100 mms. of Hg. is ominous, and other observers, including Halls Dally,[9] affirm that if the diastolic pressure is over 130 mm. disaster is imminent. That this is by no means necessarily so is shown in the following table:-

Table 9.

Case	Name	Blood Pressure	Length of Time
83	Crankshaw	220/110	7 years
84	Horridge	240/115	5 "
85	Lamb	210/115	8 "
29	Riley	260/120	4 "
95	Fletcher	220/130	6 "
94	McCaffer	220/130	7 "
98	Bradley	270/140	7 "
100	Peirce	270/160	3 "

Each of these patients has been under constant supervision during the periods indicated and the pressures taken repeatedly.

Four of these patients, be it noted, have had persistent diastolic blood pressures of 130 or over for periods of 3 years, 6 years and 7 years (two). Two of them have had a diastolic pressure of 140 and 170 mms. of Hg. respectively for 7 years and 3 years.

Prognosis is not necessarily immediately ominous or catastrophic on the basis of a high diastolic reading, although it undoubtedly indicates the possibility of impending deterioration in many cases.

Of more serious import is an increasing dyspnoea accompanied by a drop in systolic pressure, while the diastolic pressure is maintained at a high level.

The onset of cardiac asthma or acute pulmonary oedema is a case of hypertension is ominous.

Not infrequently the terminal phase of essential hypertension is ushered in by a basal pulmonary congestion. Although the causal mechanism is usually left ventricular failure, secondary infection is apt to occur thus causing a condition hardly distinguishable from lobar pneumonia.

The development of hypertension at an early age is, of course, to be received unfavourably. The prospect of

invalidism from cardio-vascular crises in later years is not inviting.

Loss of weight in an advanced hyperpietic is of bad prognostic significance.

Where angiospastic symptoms, e.g. temporary monoplegia, diplegia, etc., are present, I anticipate the future onset of a cerebral harmorrhage or thrombosis as almost inevitable.

The combination of high blood pressure and Diabetes Mellitus is usually serious. Control of the blood sugar by insulin and diet does not ameliorate the progressive nature of the hypertension.

I have always found that chronic or severe bronchitis aggravates existing hypertension.

Contrary to text book opinion I consider that hypertension associated with obesity carries a much worse prognosis than sumple uncomplicated essential hypertension.

Papilloedema, as we have seen, is of most serious significance, indicating as it does an alteration to the malignant phase. Survival after the onset of this sign rarely exceeds 2 years.

If dyspnoea is not a symptom in essential hypertension, for instance in the cephalgic type which I have described, I regard the prognosis as more favourable.

Marked arteriosclerosis accompanied by high arterial

tension is not usually as serious as one would assume to be the case. It is compatible with a useful existence up to the 7th decade.

On the whole I consider that the immediate prognosis in high blood pressure is more favourable than is generally accepted.

Treatment.

Prophylaxis.

The rather obvious corollary to the foregoing is the necessity for the periodic medical examination of each adult.

The progressive dental surgeon reminds his patients, often by letter, that twelve months have elapsed since his previous dental overhaul, and fixes a date for an appointment which the patient can cancel if he so desires. This is accepted by a more enlightened section of the public as a matter of course and many avail themselves of the opportunity.

Surely a periodic overhaul of the more vital structures of the body would even more commend itself to sensible people.

Much propaganda on the part of the medical profession and the Ministry of Health would be necessary to attain such an ideal.

A comprehensive routine examination which would, of course, include the cardio-vascular system and estimation of the blood pressure, should be advised say every three years

for the ordinary apparently healthy individual. The plethoric or short-necked, florid individual, however, should be suspect, and treated as a potential hyperpietic when he is over forty. He should be examined every year, and without imposing on him many irksome and worrying restrictions, he should receive definite instructions as to his future mode of life. He must not, of course, be frightened into an apprehensive frame of mind, nor even must a routine suggesting semi-invalidism be imposed, but I do submit that in a frank and tactful manner this type of person can be very helpfully influenced. A general health talk, with advice regarding diet, and council against overwork and worry, with emphasis on the necessity for liberal holidays and relaxation, and adequate rest, will carry much weight. This is within the province of the conscientious family practitioner.

Actually, the instructions given must vary according to the actual condition of the patient and at times may not fall far short of the treatment advised for the hyperpietic himself.

We must also be on the lookout for the nervous, apprehensive, anxious, often sallow and careworn, individual, of the type already described, and knowing her by experience we must not too lightly be led to a diagnosis of neurasthenia. If kidney disease can be eliminated this type in particular

is more amenable to treatment in the early stages.

The necessity for periodic examination is still requisite, but the approach must of necessity be different on account of the already existing apprehension. Knowledge of and insight into the difficulties of this patient may necessitate incursions into the realms of psychotherapy. Hidden complexes and the origins of mental conflicts may have to be sifted. Reassurance and the engendering of a philosophic and balanced outlook and a quiet mind must be the keynotes in treatment.

Apart from the treatment of these types of potential hyperpietics we fairly frequently encounter the patient whose symptoms strongly suggest that he has a raised blood pressure. I sometimes find them already diagnosed and the word "Hyperpiesis" written on their National Health Record Cards, and yet on examination I find that their arterial blood pressures are within normal limits.

I can recall five who came within this category, each one of which, after periods of from seven to twelve years, developed definite hypertension (cases 23, 29, 51, 62 & 63). In the light of this experience I would treat such cases as potential hyperpietics.

All those who give a family history of early cardiovascular disease, e.g. death from apoplexy occurring in several members of one family, should receive advice on

prophylactic lines.

Bacillus Coli infection, especially of the urinary tract, should be looked upon not as an unimportant and easily-irradicated infection, but as a prelude to further trouble if not energetically combated. It must be searched for more often, and not only must the acute attack receive adequate attention, but the patient must be kept under observation for several years at least.

Chronic constipation must be promoted from the scrap-heap of minor maladies. Propaganda has indeed found it a fertile field, but the publicity agent has been the patent medicine vendor, and his subject matter has been culled from the researches and publications of the medical profession. This second-hand and not disinterested dissemination of knowledge should be forestalled by direct communication between the profession and the people, i.e. by public health propaganda. The man in the street must be educated in the elements of physiology. The subject must become compulsory in the senior classes at school. The rudiments of dietetics come under the same heading and are surely, at least, as important as geometry or modern languages.

With the help of the wireless, and by suitable Health talks to factory workers, and even by well-prepared films, the people of this country can thus be prophylactically

treated against many crippling and life-destroying diseases including more especially Cardio-vascular Diseases and Hypertension.

In industry, every employee over forty should be examined by the Industrial Medical Officer or by his own doctor. His blood pressure should be estimated, as is required of every army recruit of that age. (Age for blood pressure readings has subsequently been altered to 35 and over.)

Diabetics and those with persistent Glycosuria should have their blood pressure estimated more often.

Prolonged convalescence after any illness is as important in the potential hyperpietic as it is in the fully developed condition.

Life Insurance without a medical examination should not be allowed.

Many of these desiderata are coming within measurable distance of attainment. The future health planning for the nation envisages that degree of positive health which can only be reached by such methods as I have outlined. Moreover, it is likely that all economic barriers will be removed between the patient and his ability to obtain adequate diagnosis and specialist treatment. The Beveridge Plan, if adopted, should remove much of the financial worry

which militates against the successful treatment of hypertension.

Treatment.

The actual treatment of the hyperpietic depends, perhaps more than in any other condition, on the data or information which must be obtained from the patient, so that in a sense it commences in the consulting room. It embraces a complete investigation and a thorough overhaul of the patient even after the diagnosis is made.

Apart from the more obvious physical and laboratory investigations of the various systems, including estimations of renal function and ocular examination, an attempt must be made to discover any aetiological factors.

We must deal with the individual and not with his blood pressure specifically, and we must never focus his attention on or refer his symptoms to the height of his blood pressure. We must learn of his difficulties, his responsibilities, his worries and anxieties, his repressions and hidden conflicts, his activities, his daily routine, relaxations, holidays, family economies and financial stability; his habits and mode of life. Only by so doing can we properly censor his activities and correct his faulty habits.

We must also endeavour to ascertain his optimum pressure, i.e. that pressure below which it is inadvisable to attempt

further reduction.

General Hygiene.

Optimistic encouragement must be the keynote of treatment. It will almost certainly be necessary to enlist the help of a wife or near relative because it will probably be necessary to remodel his life as unostentatiously as possible. This will not be successfully accomplished if too many irksome restrictions and "don'ts" are directly imposed. A balanced, philosophical outlook and a quiet mind must be our aim. A reduction of stress and strain must be obtained by altering the daily routine. Working hours are shortened and adequate rest periods interposed including one complete rest day each week. Adequate sleep must be ensured.

Moderation in all things is essential and is only to be obtained by a steady routine, free from hurry and excitement.

Occupations and habits must be modified consistent with financial stability and happiness. All extra responsibilities and public duties must be drastically cut or forbidden. Instructions as to meals, times of meals and rest periods after meals must be given. He must have plenty relaxation, a definite hobby should be encouraged, and holidays must be real and liberal. Every endeavour must be made to obtain for him adequate rest, physical and mental.

Rest.

Complete rest in bed for a period will of itself considerably reduce the pressure, but without other treatment the pressure will return to its former level on resumption of activities.

In a moderate or severe case, however, I consider that treatment should be commenced by keeping the patient in bed for fourteen days on a low diet consisting chiefly of potato soup flavoured with fresh vegetables to taste. Orange juice, water or tea may be given every 3 hours. In order to restore nitrogen balance, however, the patient's usual diet is given on one day per week.

Diet.

In my early days in general practice I must confess that from various standard text books I drew up a very imposing diet sheet which I issued to my hyperpietic patients. In essence it consisted of specimen diets of non-stimulating foods and beverages with avoidance of excess starch and reduction of animal protein. It allowed white meat and fish, but no red meat, and, of course, forbade extractives, gravies, soups, stews, etc., and it advised against the use of salt both in cooking and on the table. I soon, however, realised that even if scientifically sound it was not practicable. Economically, it was a difficult proposition

for the family man, and to the palate it must have been anathema.

Experience, and knowledge acquired from the writings of our leading clinicians, soon convinced me that more benefit accrues to the patient from lessening the total food intake to the minimum needs of the patient than from mere protein restriction. I do not believe that meat in moderation has any effect on arterial tension, nor that red meat is any more detrimental than white.

An easy assimilable well-balanced diet of simple well-cooked food with caloric value of 2000-3000 should be aimed at. Eggs, liver, sweetbreads and fats should be restricted in order to lower the cholesteral in the blood. Meat extracts and meat soups are forbidden, as is strong tea and coffee. Beef, lamb or mutton is allowed in moderation. Poultry or fish are preferred as often as possible. Fresh fruit, salads, lettuce, celery, vegetables, honey and milk are available. Rich feeding is forbidden. A salt-free diet is unnecessary unless oedema is present.

Any tendency to obesity must be countered by restricting fats and starch:- Butter, cream, bread, cakes and puddings. It may be necessary to restrict diet to 1000 calories at first. A loss of weight of about 1 lb. per week is necessary in excessive obesity, with a view to reduction in weight to

the patient's former level when younger.

Diluted alcohol is allowed in moderation. Beer is not encouraged. Excessive smoking is detrimental. In moderation tobacco is soothing.

In moderate and severe cases a fast day each week is advised during which lemonade, orangeade, weak tea and biscuits are allowed - or milk and milk pudding.

Eliminative treatment is conveniently combined by the use of a purgative on the previous evening. Calomel followed by a morning saline, for instance, helps to prevent the accumulation of toxic products.

Exercise.

Natural exercises graduated according to the tolerance of the body play an important part in the treatment of hypertension. In moderation their beneficial action appears to be due to the washing away of impurities from muscular areas, and the reduction of tension sometimes achieved may be caused by the flushing and dilatation of superficial arterioles and capillaries.

Walking. Quiet walking, graduated and gradually increased to include the lower hills, is suitable for all but the most severe cases and those associated with angina of effort. Gentle games that do not require sudden effort have a useful place, especially if obesity is present.

Golf is ideal exercise for milder cases providing that certain very definite restrictions are imposed. Plenty of time must be allowed for the game. It must be leisurely, in relatively calm weather, on a not too hilly course, and restricted to a definite number of holes. Thus competitive golf is barred. A caddy is advisable in order to avoid the constant stooping entailed in teeing up.

Gentle cycling on the level is beneficial.

Easy horse riding has its advocates, but I have no experience of its benefits in hypertension.

Tennis and Squash I consider to be too strenuous.

Rowing, except for the young and very mild case, is I think inadvisable.

Gardening is very difficult to graduate. Over-enthusiasm is apt to overweigh discretion. Sensibly employed, however, it can be utilised especially where facilities for games are lacking.

Bowls can usually be allowed.

Skating and Swimming are only for the younger and more vigorous patients.

Regulated Hill Climbing may be attained by milder cases with good tolerance.

Breathing Exercises help the circulation and venous return.

In severer cases instead of active exercise one must have resource to passive exercise and massage.

Massage.

General massage and passive exercises three times a week are necessary to preserve muscular tone, aid elimination and prevent obesity. Active exercises may later be prescribed.

Warmth and massage to the splanchnic area is helpful.

Spa Treatment.

As this is somewhat without the province of the general practitioner I cannot speak authoritatively regarding its efficacy. Three patients in the present series (cases 15, 22 and 57) have undoubtedly benefited by periodic courses at Harrogate. Economic or financial reasons very often put it out of court.

The dietetic restrictions, exercise and definite regimen prescribed and given under supervision seem to be more cheerfully tolerated than at home.

The aim of spa treatment is to produce vaso-dilatation by immersion in baths of mineral water at or above body temperature. This peripheral dilatation induces a fall in pressure which is said to be not merely transitory but is cumulative over a series of baths. Early hyperpiesis may thus be arrested or ameliorated.(56)

The regimen, of course, can be carried out at home with

the help of the appropriate waters obtained in bottle, and the various baths can usually be obtained locally, but the patient soon tires of seeking a spa cure under the difficulties encountered at home.

Mineral waters most frequently prescribed at home include Apenta Hunyadi, Vichy and Contrexville.

The spas considered most suitable for cases of hypertension are Harrogate, Llandrindod, Carlsbad and Marienbad.

Hydrotherapy.

This, of course, is part of spa treatment, but its prescription may often be called for when spa treatment is unobtainable.

The Aix Douche or colonic lavage is often usefully prescribed, combined with massage. Needle baths of alternating temperatures are sometimes helpful, while the warm pack, as has been indicated, is decidedly useful at times.

Diaphoretic methods on the whole, however, are disappointing, including the not unpopular vogue for Turkish and Hot Air baths. Hot baths, as Rolleston[57] pointed out, cause a rise in systolic pressure and a lowering of the diastolic, thus increasing the work of the heart.

Diathermy has a useful place in the treatment of resistant cases. Long wave diathermy is advised.[58] A

current of ½-1 ampere is passed between electrodes placed on both wrists for fifteen minutes, three times a week. Where there is angina of effort Jones and Cumberbatch[59] advocate ultra short wave diathermy through the chest. I have had no personal experience of this method of treatment nor of the use of High Frequency currents in Hypertension.

Climatic Considerations and Holidays.

Where it is possible a mild climate in Winter is advisable; similarly extreme hear in Summer is to be avoided. Living at too high an altitude is not helpful, e.g. not above 3,000 feet above sea level. Sea level is preferable. Bournemouth, Torquay, Sidmouth, Paignton, Madeira, Canary Islands, Egypt and Algiers are considered suitable.

For the mild and moderate case an annual cruise is ideal. In any case, a prolonged holiday, as often as can be arranged, to a pleasant environment, is advisable.

Much of the foregoing may be criticised on the grounds that it is full of banal generalities and common-sense dicta hardly worth committing to print. I feel, however, that unless these points are stressed one is inclined to put too much of a burden on to the therapeutic side.

I further believe that treatment conscientiously carried out on these lines, and without having recourse to drug therapy

at all, would enormously decrease the incidence of hypertension and would cure early and moderate cases besides ameliorating the established and severe case of hyperpiesia.

In actual practice, however, such a council of perfection is most difficult to attain for several reasons.

Working-class people, in whose ranks are so many of our hyperpietics, cannot re-order their lives at a moment's notice. Their financial and economic position does not allow for them adequate holidays, special diets, spa treatments, rest periods, changes of occupation - or even relaxation in its true sense. Their worries are mainly financial or family ones which are aggravated by enforced idleness. Pensions and sick benefit are inadequate and Public Assistance still carries its stigma from "Poor Law" days. Moreover, their education is insufficient to allow them to grasp the significance or the wisdom of the advice we try to impart. Many are elderly, single or widowed people, with no near or living relative; people who live alone and who dread hospitalisation because of the fear of losing their homes.

These are but a few of the difficulties which the general practitioner will encounter when he attempts to put his precepts into practice.

At rock bottom he will find that problem of hypertension,

both etiologically and prophylactically, is essentially a social one. Bound up with it are innumerable difficulties which can only be remedied by future Ministers of State. One must envisage a post-war Utopia in which the worries and anxieties of the poorer classes are mitigated by such measures as adequate employment, pensions; more convalescent and holiday homes; semi-communal flats, with trained nurses attached, for lone, elderly people, etc.

Without entering further into politics, however, I think I have shown that in general practice, at least in the industrial areas, only partial success can be achieved by the ideological general measures advocated. As far as is practical they must be put into operation, but much more will be demanded even if only in the nature of a placebo to reinforce the advice given.

Especially is this true in the County of Lancashire where, unfortunately, the fetish of the medicine bottle still persists and where a blinding faith in its contents far outweighs any "incidental" advice.

Rehabilitation.

Since writing the foregoing it has been most interesting not only to find confirmation of the difficulties expressed but to find a practical way out of many of them.

The Tomlinson Report[60] on the Rehabilitation and

Resettlement of Disabled Persons published by H.M. Stationery Office as a White Paper offers scope for a tremendous advance in the treatment of hypertensive and allied conditions.

While it is recognised that a proportion of cases will be compelled to lead an invalid life, the remainder, it is pointed out, will be able to resume their former activities to a greater or less extent, or to undertake work in an occupation requiring little physical effort.

Special centres providing continuous medical supervision, somewhat on the lines of a Sanatorium rather than a training centre, are envisaged for this group. Thus full responsibility would be maintained for their convalescence.

Basil Parsons-Smith[61] in the British Medical Journal amplifies this theme in a note on the Rehabilitation of Heart Patients. This is applicable in all its details to Hypertension.

By means of graduated exercises and other remedial exercises and physiotherapy, the patient graduates through the ambulatory phase, and in the absence of complications, arrives at the stage where he has accommodated himself to appropriate forms of exercise and normal routine of domestic habits. He may now feel capable of employment, although this may have to be an alternative occupation appropriate to his diminished circulatory reserve.

Drug Therapy.

The question of effective elimination has already been partially discussed under the heading of Spa Treatment. It must, however, be fully considered in every case and with special reference to bowel action.

Constipation being such a frequent concommitant of hypertension must be adequately dealt with.

Purgatives must not be used as a routine. I do not favour daily salines even, except where the gall bladder is suspect. Then I do advise a small dose of Magnesium Sulphate in hot water half an hour before breakfast.

I prefer a regular, gentle laxative taken each night, such as Liquid Paraffin, or an Emulsion of Liquid Paraffin, Agar and Phenolphthalin.

For those whom it suits Cascara alone (Cascara Evacuant) or in combination with Liquid Paraffin, or with Eunonym and Tridin, is useful. Vegetable Laxatives are preferred by some patients. Whichever laxative is used, the forming of a Habit Time and the gradual re-education of the bowel is important.

Liberal fluid intake, particularly in the early morning, e.g. a glass of hot water or even 1 pint of cold water on rising, will often help.

Periodically each week-end or every other week-end a

more drastic regime is employed. Calomel gr. $\frac{1}{8}$ every half hour for 8 doses, or gr. $\frac{1}{4}$ to $\frac{1}{2}$ at night is prescribed, followed by a morning saline. Pil. Hydrarg. gr. $\frac{1}{2}$ to v may be substituted if the calomel does not agree.

In the treatment of hypertensive encephalopathy Magnesium Sulphate in 50% solution (one tablespoonful of Magnesium Sulphate to one tablespoonful of water - three times a day) may be prescribed alone or along with an intramuscular or intravenous injection of 10-20 cc. of 10% solution of Magnesium Sulphate.

Table 10.

Drugs commonly used in Hypertension.

I. Alkaline Mixtures.

II. Sedatives.

Ammonium, Potassium or Sodium Bromide.
Mist. Bromide et Valerian.
" Bromide & Phenazone (Phenazone Co.).
" Bromide et Aspirin.
" Bromide & Ac. Hydrobrom. dil.
" Bromide & Sod. Phenobarbital.
" Bromide & Chloral Hydrate.
" Bromide & Gelsemin.
" Bromide & Pot. Iodide.
Gower's Mixture.
Calcibronat.
Luminal or Phenobarbital.
Tabs. Theominal.
" Pro-Theonal.
" Veganin.
" Veramon.
" Codeine Compound.
Passiflora (Elix. Passiflorine).

Hypnotics.

Sedobrol.
Tabs. Allonal.
" Dial.
" Medinal.
Bromide & Chloral.

III. Iodine and Iodides.

Potassium or Sodium Iodide.
Tinct Iodi.
Lugol's Solution of Iodine.
Collosal Iodine (Crookes).
Tiodine (Cognet).
Alphidine (Oppenheimer).
Ioxantin Pulv. (Oppenheimer).
(a non-toxic iodine & Sod. Salicyl.)
Iodalbin Caps. (Parke Davis & Co.).
(Iodoprotein Compound).
Elixir Caffeine Iodide.
Iodo Calcium Diuretin (Knoll).
Pot. Iod. c Amenophyllin (Battle).

IV. Vaso Regulators or Dilators.

(a) Nitrites and Nitrates.

Amyl Nitrite.
Nitro-glycerin gr. $\frac{1}{100}$ - $\frac{1}{200}$
Sod. Nitrite gr.$\frac{1}{2}$ - gr.1.
Erythrol Tetra Nitrate gr.$\frac{1}{2}$ - gr.1.
Bismuth Subnitrate.

Tabel Natric (Drug Products Co. Inc.).
Pot. Nitrate gr.2.
Sod. Nitrate gr.1.
Nitro-glycerin gr. $\frac{1}{250}$
Crataegus Oxyacantha gr.1.

Pulv. Lithium Hippurate Co. (Oppenheimer).
Lithium Hippurate gr.2.
Sod. Nitrate gr.1.
Nitro-glycerin gr. $\frac{1}{200}$

Tabs. Hypotensive (Parke Davis & Co.).
Lithium Hippurate gr.2.
Sod. Nitrite gr.1.
Glyceryl Trinitri gr. $\frac{1}{200}$

Hypotensive Ampoules and Drops (Fraixe).
Sodium Nitrite 0·01 gm.
Ext. Visci Alb. 0·05 gm.

Tabs. Lithium Hippurate Co.
Sod. Brom. gr.iii.
Lithium Hippurate gr.ii.
Sod. Nitrite gr.i.

(b) Xanthin Derivatives.

Theominal (Theobromine and Luminal) (Bayer).
Calcium Diuretin (Knoll).
Iodo-Calcium Diuretin (Knoll).
Rhodan-Calcium Diuretin (Knoll).
Raminal (Napp).
(Theobrom. Calc. Salicyl. gr.1½.)
(Chlorophyll. gr.½.)
(Ferri Phos. gr.½.)
Euphyllin (Theophyllin & Ethyl Diamine).
Aminophyllin do. do.
Aminophyllin c Luminal gr.¼ - gr.½.
or Pot.Iod. gr.1.
Perphyllon.
Glucophyllin (Abbott).
Glucophyllin c Nembutal.

(c) Choline Derivatives.

Doryl (Merck).
(Carbamincylcholine Chloride).
Pacyl (Joachim Wiemik & Co.).
Acetylcholine (British Drug Houses).
(Roche).

(d) Potassium Thiocyanate.

Elixir Sodium Sulphocyanate (P. D. & Co.).
Elixir Sod. Sulphocyanide (Legat).
Rhodan Calcium Diuretin.

(e) Drosera.

Collosal Silicia with Drosera Rotundifolia (Crookes)

(f) Veratrone (Park/Davis & Co.).
(Veratrum Viridi).

(g) Animal & Vegetable Extracts.

Padutin (Bayer) (Pancreatic Ext.).
Detensyl
(Mistletoe, Liver, Pancreas, Lung).
Phyllosan.
Hypotensyl
(Vix. Ext., Hepat. Ext., Pancreatic Ext.).
Hypotensive Drops (Fraisse).
Guipsine (Le Prince) (Vixun Alb.).
Viscysate.
Liver Extract.
Renal Extract.

V. Antispasmodics.

Belladonna & Atropine.
Bellergal (Sandoz) (Luminal, Belladonna).
Morphine.
Nembutal.

VI. Diuretics.

Caffeine.
Theobromine.
Diuretin & Xanthin Derivatives.
Salphyrgan. (Salyrgan)
Elix. Caffeine Iodide.
Digitalis.
Scillae.

VII. Hormones.

Thyroid Ext.
Corpus Luteun Ext.
Stilbestrol.
Proklemen (Ciba).
Matronax (Knoll).

VIII. Lithium Hippurate.

IX. Stimulants.

Strychnine.
Strophanthis.
Digitalis.

The somewhat formidable list of drugs here tabulated (Table 10) is not by any means a complete one. It does, however, I think, include most of the types of drug used in the treatment of hypertension. Moreover, each one of them has been tried by the author in his search for a reliable therapeutic weapon with which to combat this troublesome condition.

That so many remedies are offered, is of itself a confession of failure on the part of the prescribing physician.

Theory and practice are sadly at variance in this field and it needs but a casual glance at many of the preparations described to appreciate some of the difficulties.

A curious type of polypharmacy is evidenced. We find various differently acting drugs, each one of which has been sponsored or has had a vogue in the treatment of high blood pressure, slumped together into one homogenious mass. An iodide may be tacked on to a Xanthin derivative, or instead you may have a sedative or antispasmodic radicle included. An assortment containing Bromide, Iodide, Nitrate, and Alkali must surely have one saving grace, but if the result is satisfactory how much wiser are we?

I suppose really the pharmacist ought to be congratulated on his success in so elegantly combining the results of past endeavour. "Many a mickle makes a muckle." If this be blunderbus prescribing I must plead guilty to it.

Each hyperpietic must be an individual problem and must be considered individually. There is no specific drug for him as there is for the Pernicious Anaemic or the Diabetic. If his symptoms are not relieved his hypertension will not be improved. Hence symptomatic treatment more often than not must be given and, if necessary, combined with any hypotensive treatment that may be further required.

Alkalis are merely useful from a symptomatic point of view. They form the basis of many of our placebos, given perhaps to reinforce more useful and necessary advice. They form part of our armenterium in the treatment of Bacillus Coli Infection, and they certainly have their place in modifying and helping the actions of more potent drugs.

A prescription I have found most useful is one which was recommended to me by Dr. D. Tindal of Glasgow.

Sod. Bicarb. ℥ii
Sod. Sulph. exsicc. ℥i
Sod. Iodide ℥ss
Sod. Nitrate ℥ss
Sod. Phosph. ad ℥iv

Sig in a tumblerful of hot water thrice daily.

Here again, of course, for its efficacy we have the

conflicting claims of the Alkalis, the Aperients, the Iodide and the Nitrate. In fact, I sometimes wonder why Sod. Bromide was omitted. Mistura Gentian Alkaline & Tr. Nucis Vom. has frequently been prescribed in this series of cases with considerable benefit, especially in the mildly dyspnoeic cases.

Sedatives.

By sedatives alone much help can be given. In fact, I think that by the judicious use of this type of drug we can do more for the hyperpietic than by any other means at our command. Indirectly, by allaying his fears and soothing his irritable vasomotor system, we can, at the same time, reduce his blood pressure to a more satisfactory level.

Any medicament which is capable of reducing the blood pressure by direct action must be of only temporary utility if at the same time other exciting factors such as worries, anxieties and other nervous manifestations are ignored.

Bromides, therefore, may be considered to have a definite place in the treatment of hypertension.

In combination with Valerian their greatest field is in the Menopausal type of hypertension.

Along with Phenazone we have a useful weapon against the hyperpietic headache.

Ammon Bromide
Sodii Salicyl
Phenazone a.a.gr.x
Spt. Ammon. Arom. m.x.
Aq Camph ad ℥ß.

Potassium Bromide in combination with drachm doses of dilute Hydrobromic Acid, I have found most useful in relieving the Tinnitus which is often present.

As a sedative mixture of real worth in hypertension I give pride of place to the following combination:-

Sodii Bromide gr.x.
Tr. Gelsemii m.x.
Liq. Strychnin m.v.
Liq. Trinitri m.
Ac. Hydrochlor dil m.v.
Inf. Gent ad ℥ß. ft. mist.

Originally it was recommended by the late Sir Wm. Gower for Migraine. I have consistently used it in hypertension cases during the last sixteen years and almost invariably I find that the patient expresses himself as having benefited from its administration. I have been unable to prove that any direct hypotensive effect results, but I cannot ignore the definite improvement in symptomatology which resulted in nineteen cases of the present series (Cases 4, 7, 12, 23, 30, 31, 40, 47, 50, 59, 68, 70, 83, 85, 88, 91, 94, 98 & 99).

Luminal or Phenobarbital I value very highly in the treatment of high blood pressure. Twenty-two cases

definitely improved under Luminal (Cases 3, 7, 9, 10, 22, 23, 26, 27, 31, 32, 41, 47, 57, 59, 60, 63, 67, 80, 83, 91, 98 & 100). In seven the systolic pressure was moderately reduced after a period of two weeks on Luminal, not I imagine by any direct effect, but as a result of general regime plus sedative effect of the phenobarbital. Usually it was exhibited in half grain doses, night and morning, but in three of the cases undue drowsiness made it necessary to give one quarter of a grain three times a day. Case No.100 received one grain night and morning but this had to be discontinued because of its soporific effect. To avoid any cumulative effect alternative treatment was substituted at intervals of 2 to 3 weeks.

In view of the beneficial effects of Phenobarbital, either alone or sometimes combined with Bromide, I was not surprised to find that the drug named Theominal gave me even greater assistance.

Small doses of Luminal failed to relieve the severe and frequent headache. They often caused a certain amount of confusion, and larger doses certainly caused drowsiness; moreover no direct hypotensive effect was evident.

Theominal, a combination of Luminal gr.$\frac{1}{2}$ and Theobromine gr.v., seemed to combine the almost ideal sedative with a Xanthin derivative of known diuretic power. The diuretic

effect of Theobromine is caused partly by dilating the renal vessels and partly by invigorating the circulation. (Prominal, which is not as soporific as Luminal, is also combined with Theobromine in a preparation also made by the firm of Bayer.)

Theoretically, Theominal should be a useful adjunct in the treatment of hypertension. In practice, I have found it the most helpful of all the drugs I have used in this condition. Twenty cases received definite benefit from the administration of this drug (Cases 8, 10, 11, 22, 46, 47, 51, 57, 59, 60, 67, 68, 78, 83, 85, 93, 94, 98 & 100). Eight of these were particularly severe cases who had failed to respond to other preparations (Cases 29, 78, 83, 85, 93, 94, 98 & 100). Symptomatic relief was rapid even when the blood pressure readings were practically unaltered. Each patient expressed himself or herself as having experienced a definite improvement. Headache, confusion, vertigo, insomnia and nervous symptoms such as anxiety and apprehension were relieved. The diastolic pressure was virtually unaffected, but a definite fall in the systolic pressure was observed in five cases.

At the end of a fortnight's treatment the readings in the case of Lamb (85) were 180/110 - the average reading for the previous three months being 210/115.

McCaffer (94) whose pressure had been consistently over

210/110 became 190/110 after 7 days on Theominal. This level was maintained for a period of 2 months.

Bradley (98) experienced a reduction from 250/140 to 220/130 after 2 weeks' treatment.

Cases 100 and 27 were reduced 30 mm. and 20 mm. respectively.

The striking feature, however, in Theominal therapy was that having once employed it and later discontinued or altered the treatment, I was very frequently again asked for it by the patient.

Iodine and Iodides.

No drug has been more universally prescribed in the treatment of hypertension than Potassium Iodide. Perhaps it would also be correct to say that no drug has been more abused.

Common usage and empiricism die hard. Although based on no scientific grounds we still find many of our more recent preparations for hypertension pre-fixed by the syllable "Iodi."

Non-toxic iodides or iodo protein compounds have tended to replace the original Potassium or Sodium Salts.

Seventeen patients of the present series were treated for periods of four weeks at a time with Potassium Iodide (Cases 4, 8, 12, 19, 29, 37, 65, 66, 72, 76, 84, 86, 87, 91,

92, 94 & 98).

It will be seen from the Table (Table 11) that a reduction in pressure was obtained in only one case, although in two other cases the reduction in pressure could not be directly attributed to the Iodide.

Improvement in symptomatology was effected in the two syphilitic cases and, to a lesser degree, in three cases with predominant signs of arteriosclerosis.

I do not consider that Iodides have any useful place in the treatment of hypertension except in syphilitic cases. Symptomatic relief may occasionally be obtained where there is marked arteriosclerosis.

Table 11.

Table showing effect of Iodides on seventeen cases of Hypertension

Case No.	General Effect	Hypertensive Effect
4	No improvement in symptomatology	Nil
65	No improvement	Nil
29	(Arteriosclerotic) Improvement in general condition; Dyspnoea relieved.	Reduction of 15 mm. & 20 mm. on two occasions. (Systolic only.)
66	(Arteriosclerotic) Improvement in symptoms such as Tinnitus and Fibrositis.	Nil
8	No effect	Nil
12	No effect - badly tolerated. Iodalbin substituted.	Nil
37	No effect	Nil
19	No effect	Nil
94	No effect after prolonged treatment gr.XX td. Marked contrast to results with Theominal.	Nil

Case No.	General Effect	Hypertensive Effect
84	Relief of Fibrositis only	Nil
87	(Arteriosclerotic) Slight improvement	Nil
98	Caused Iodism. Elixir Caffeine Iodide gave slight relief.	Nil
86	Moderate improvement (Wasserman positive)	Slight - difficult to estimate
72	Marked improvement (Wasserman positive)	Slight - as intravenous arsenical preparation used
91	No improvement	Nil
92	No improvement	Nil
76	No effect	Nil

Nitrites and Nitrates.

By the very evanescent nature of their action they appear only to be of use where an immediate hypotensive and antispasmodic or vaso-dilator effect is required.

No one can gainsay the relief obtained in Angina Pectoris by amyl nitrites or the preventive action obtained in these cases by chewing tablets of Trinitrin preparatory to any increased exertion. This latter effect was abundantly proved in Case No.11.

The more prolonged effect of Sodium Nitrite has been sought in various combinations such as Tab. Hypotensive (Park/Davis) and Pulv. Lithium Hippurate Co. (Oppenheimer). These contain Lithium Hippurate gr.2 along with Sodium Nitrite gr.1 and Nitroglycerine gr. $\frac{1}{200}$.

I used one or other of these two preparations in twelve

cases for three separate fortnightly periods, but was unable to convince myself that any improvement resulted either in the relief of symptoms or the reduction of the hypertension. Case No.88 reported relief of her very severe headache, however, and she used Tabs. Hypotensive in preference to any other preparation for this purpose alone.

Xanthin Derivatives.

Theominal has already been considered. Of the others enumerated in Table 10, I consider that only Euphyllin or Aminophyllin are of any definite utility in certain cases of hypertension.

This preparation consists of a compound of Theophyllin and Ethyline Diamine. It is a diuretic and also appears to have the effect of dilating the coronary arteries.

Apart from its beneficial effect in Angina Pectoris, of which I had evidence in Cases 22 and 11, I have found it of inestimable value in relieving the distressing dyspnoea of the advanced hyperpietic whose left ventricle is failing under the increasing strain. Cases 54, 84 and 29 obtained this benefit from its employment. In the former case it was used intravenously on one occasion with dramatic effect. In the other cases it was prescribed in tablet form thrice daily.

Choline Derivatives.

Pacyl. For a period of twelve months (1933) I used this drug for all my cases of hypertension then under treatment (9 cases). Allowing for temporary fluctuations and a "placebo" or psychological effect in a few cases soon after commencing treatment, I was very disappointed in the results.

Acetylcholine (British Drug Houses) which contains Acetylcholine Bromide gm.0·1 per c.c. has a powerful hypotensive action. It is given intramuscularly commencing with an initial dose of ·05 gm. raised subsequently to 0·1 gm. daily. It causes a steady fall in pressure in three to four hours, but its effect has passed in seven or eight hours. Its only field of usefulness, therefore, would seem to be where the pressure has reached a dangerous level and must be reduced at all costs. I used it in a particularly resistant case - that of Murgatroyd (Case 88). A definite improvement both in symptoms and reduction of hypertension resulted after 7 days' treatment, but a further and more disastrous experience with another patient caused me to refrain from further experiment with this drug. Being anxious to relieve the acute symptoms of very severe headache, vertigo and dyspnoea, in this other patient, a widow of 68, whose blood pressure was 210/100, and feeling pleased at my apparently successful results in the afore-mentioned case, I again used

Acetylcholine. Following the second intramuscular injection I was recalled to the patient's home to find, to my dismay, that she had suddenly died soon after my departure. Until then I had been unaware of the dangerous potentialities of this drug.

Sulphocyanates.

I used Elixir Sodium Sulphocyanate (Parke Davis) which contains Sodium Sulphocyanate gr. 2½ in each drachm.

It was prescribed for periods of three weeks as recommended. For the first week 3ʃ three times a day. During the second week 3ⁱ twice daily, and for the third week once a day.

Owing to its known toxicity and cumulative effect I did not care to exceed the above dosage.

The following cases received two courses of treatment each - (Cases 23, 83, 85, 91 & 98). In none of the five cases was the average pressure reduced, although a fall in the systolic pressure of 15 mm. was observed after the first week in Cases 83 and 85. This was not maintained.

Bradley (Case 98) complained of light-headedness and nausea, and on each occasion refused to continue with it after the first week. In this case a different preparation with an entirely different taste (Elix. Sod. Sulphocyanide - Legat) was used on the second occasion.

Lamb (Case 85) obtained a sedative effect at first and felt relieved, but during the second and third weeks and after subsequently obtained no benefit.

In the case of Dutton (Case 91) it was found to increase her mental confusion.

Crankshaw (Case 83) exhibited a morbiliform rash after the second week. He had a sensitive skin and had previously had attacks of Urticaria and Seborrhoeic Dermatitis.

Thistlethwaite (Case 23) complained of nausea. Apart from this no symptomatic relief was obtained.

It is, of course, impossible to be hypercritical on the results of a very limited trial, but in practice one does not care to persevere in the face of the obstacles presented by a few carefully chosen and typical cases if it is known that benefit can be obtained by other means. At any rate, my impressions are that this drug in the recommended dosage is toxic and that it gives neither symptomatic nor physical relief in hypertension.

Drosera.

Drosera is an antispasmodic of utility in the treatment of whooping cough, but its efficacy in hypertension has yet to be proved. It is said to contain an enzyme which converts albumen into soluble peptone. I have used a preparation marketed by Crookes Collosol and containing Collosol Silica

with Drosera Rotundifolia.

In only three cases (4, 98 & 85) was this preparation used, but no apparent result was achieved.

Veratrone.

Veratrone (ParkeDavis & Co.) is a non-alcoholic solution of the active principle of Veratrum Viride. Each c.c. contains 2·5 mgm. of the alkaloids of Veratrum. Its arterial and spinal depressant action is powerful, in addition to which it has a strong diuretic and diaphoretic effect.

As a hypodermic injection of 0·5 c.c. I found it of unmistakable value in two cases of Eclampsia (13 & 35). It appeared to aid the elimination of the toxin. It certainly lowered the pulse rate. During the acute crisis of an Eclamptic fit it was not practical to use the sphygmomanometer in these cases. Nevertheless, I feel sure that there was a considerable drop in blood pressure even if only of short duration.

I consider that only in such an acute emergency, where the pressure must be quickly reduced, is this drug a useful hypotensive weapon.

Animal and Vegetable Extracts.

Many preparations of this type have been advocated. Padutin (Bayer) contains a Pancreatic Extract. It certainly has an antispasmodic and vasodilator action which I have found

useful in providing temporary relief in Raynaud's Disease.

In the case of West (No.4) numbness and tingling of the hands and feet, "dead" fingers, and intermittent claudication in the legs, were distressing and early features. Padutin, in this case, gave considerable symptomatic relief, but did not prevent the blood pressure from slowly increasing.

Mistletoe is perhaps one of the oldest remedies used in the treatment of hypertension. Its sponsors have endowed it with a rapid and steady hypertensive action. As a diuretic they say it is equal to Squill. Several well-known preparations contain the extract of this plant, including Detensyl (Ext. Mistletoe, Ext. Liver, Ext. Pancreas, Ext. Lung); Hypotensyl (Anglo French Drug Company), Hypotensive Drops (Fraise), Gimpsine (Le Prince) and Viscysate.

My experience with them has been disappointing. Detensyl received an extensive trial in five cases (4, 28, 49, 95 & 98). Case 49 afterwards received Viscysate 15 drops three times a day and case 98 Hypotensyl. The blood pressure appeared to be entirely unaffected in all these cases, nor was any symptomatic relief manifest.

Liver Extract.

Several writers have stated that hepatic extracts provide one of the most efficient remedies against hypertension.[29]

In the case of Lamb (Case 65) who was suffering from the

rather unusual combination of Pernicious Anaemia, Diabetes and Hypertension, the intra-muscular action of Liver Extract, whilst remedying the Anaemia, had no effect whatever in reducing the blood pressure or even in preventing it from increasing.

Kidney Extract.

Page and his co-workers[62] have produced an extract from the kidney which they claim is effective in lowering the blood pressure. Further confirmation of this is necessary, however.

Antispasmodics.

Morphine has a definite place in the treatment of some of the complications of the hypertensive states.

It may be required in severe Angina Pectoris.

In cardiac failure, where Cardiac Asthma or severe nocturnal dyspnoea is present, it is the only drug which I have found capable of giving dramatic relief (Cases 39, 40 & 75).

Atropine was a necessity when Acute Pulmonary Oedema occurred (Cases 54, 70 & 75).

Diuretics.

Very many of the drugs already mentioned have a strong diuretic action.

Of the mercurial diuretics Salyrgan 1-2 c.c. intra-

venously proved helpful when an attack of Acute Pulmonary Oedema was subsiding (Case 75).

Digitalis is, of course, particularly useful in left-sided cardiac failure, cardiac asthma and uncontrolled auricular fibrillation.

Hormones.

Thyroid Extract was particularly helpful in the cases of Peel and McCaffer (53 & 94). In both these cases obesity and some Myxoedema was in evidence.

Stilboestrol in doses of 0·5 mgm. twice daily gave considerable relief in two of the Menopausal group (7 & 17), but in this series I have insufficient data to pronounce on its efficacy.

The two preparations I have found to give most relief in Menopausal cases where the blood pressure is raised are Proklæmen (Ciba) and Matronax (Knoll).

Proklæmen (Ciba) contains:

Ovarian Hormone	gm.	0.02
Peristaltin	gm.	0.015
Kryofine	gm.	0.2
Caffeine Sodium Salicytate	gm.	0.05

Matronax (Knoll A.G.) contains:

Ovarian Substance	gr. ½
Thyroid	gr. 1/10
Bromural	gr. 2¼
Calcium Diuretin	gr. 2¼

(2 tablets to be swallowed 3 times a day)

Six cases (7, 16, 17, 22, 33 & 67) received treatment with

these preparations during the course of their symptoms. Only one (Case 16) failed to respond satisfactorily.

Improvement in symptomatology was most marked, although the hypotensive effect was variable. Taking into account the normal fluctuations and the natural tendency for these cases to gradually regain normality, it was impossible to ascribe a slight fall in pressure to the direct action of the drug.

Stimulants.

The mild stimulating action of Strychnine and Strophanthus and its usefulness in counteracting the milder dyspnoea of the early case has already been commented on.

Other Drugs.

Pilocarpine has, of course, been advocated for its eliminative and diaphoretic properties. A hypodermic injection of gr.1/10 daily for 12 days, followed by a hot wet pack, and then by massage, is prescribed, but this is essentially hospital routine which I have not utilised in private practice.

Venesection has not altogether vanished from our armentarium. It still has a place in the treatment of certain types of hypertension. It is purely palliative in its results and to recommend it as routine treatment to be performed at six monthly intervals for all cases of hypertension is foolish. It does, however, diminish the intensity of very severe headache and often mitigates the annoying Tinnitus,

Vertigo and mental confusion of some of these people.

About twenty ounces of blood should be withdrawn. A sharp laxative should be prescribed, and fluid intake reduced to a minimum for the following twenty-four hours, or alternately, a 24 to 48 hour fast ordered.

I have employed Venesection in seven cases in this series (Cases 8, 29, 35, 54, 88, 91 & 92). Although the effect was transient it relieved the very acute headache of a case of hypertensive encephalopathy (Case 88). Some relief was obtained in a case of Eclampsia (Case 35). In Acute Pulmonary Oedema it appeared to be a useful adjuvant in Case 54. Symptomatic relief of mental confusion was apparent in Cases 91 and 92.

Lumbar Puncture. This may occasionally be advisable where there is much cerebral congestion or compression (Case 92).

Renal Antipressor Therapy.

Page and his colleagues[19A] have successfully extracted the antipressor substance and used it in the treatment of renal hypertension. Thirty-seven patients were treated, including twenty-four who suffered from Malignant Hypertension. The results obtained were dramatic. Changes noted included reversal of changes of fundi, decrease in diastolic pressure; increased cardiac output and an increase in renal flow.

Further confirmation of such successful antipressor

therapy is awaited with great interest.

SECTION 5.

Conclusions.

A critical survey has been made of the medical histories of 100 patients who showed arterial hypertension. The members of this group were all encountered in private practice and were under the care and observation of the author for considerable periods, in some cases for as long as seventeen years.

Although each one of the series had a persistent systolic arterial blood pressure of 160 mm. of Hg. or over, the significance of the hypertension was found to vary considerably.

A consideration of the clinical factors operative lead to the conclusion that hypertension must be considered under three main classifications.

I. Physiological or Systolic Hypertension is a definite clinical entity. It may be defined as a raised systolic arterial blood pressure in association with a diastolic pressure below 90 mm. of Hg. and unassociated with organic disease (such as aortic regurgitation for instance). This type of hypertension is frequently found in patients of nervous temperament. It is functional, and tends to fluctuate, but may persist for many years at a supernormal level. Twenty patients in this series of 100 cases fall

into this category. Of these, six have been under observation for at least twelve years without showing any sign of ventricular hypertrophy or other complication of high blood pressure. I believe that this type of hypertension is of little or no clinical significance and that contrary to the published statistics and tables of Life Assurance Companies the prognosis in these cases is good.

II. Secondary Hypertension.

Secondary Hypertension is an increased arterial blood pressure which is incidental to a definite morbid condition of the body. It is a symptom which develops as a result of the pathological changes induced by a primary disease.

The primary disease may be renal, such as Acute or Chronic Glomerulonephritis or Chronic Pyelonephritis. Examples of such cases have been given.

One case of coarctation of the aorta with raised arterial pressure of the upper extremities is cited.

Syphilis affecting the arterial tree or the central nervous system may be accompanied by a secondary hypertension. Two such cases are reported.

Aortic Regurgitation and late Mitral Stenosis are often found in association with morbid hypertension.

Other examples of secondary hypertension are found in such conditions as pregnancy kidney and eclampsia; in certain

brain tumours (a case of Glioblastoma is illustrated); not infrequently in Diabetes Mellitus.

Classical examples of this type of hypertension are found in the rarer conditions of Basophil Adenoma of the pituitary, tumour of the thymus or suprarenal cortex, or tumour of the suprarenal medulla.

Twenty per cent of this series have been classified as cases of secondary hypertension.

In so many and varied conditions as those mentioned no single etiological factor can be held responsible for the hypertension.

Peripheral arterial resistance is increased in some cases by the mechanical or pathological structural alterations in the blood vessels and vital organs, and in others more indirectly by the possible chemical action of pressor bodies.

This applies, of course, to secondary hypertension in the rare case of tumour of the suprarenal medulla.

Regarding the renal group of diseases the renin theory elaborated by Helmer and Page appears to me to be most worthy of acceptance.

III. Primary or Essential Hypertension.

This constitutes the main or most important classification of hypertension. Sixty per cent of my series come within this category.

No longer are we dealing with a symptom but with a definite morbid condition whose later manifestations are directly due to the hypertension.

One cannot call it a well defined disease because its earliest manifestations are so often lacking.

The signs and symptoms shown by the patients in this series have been studied in Section V.

It is noteworthy that in those with moderately raised arterial pressure the nervous symptoms predominate. In fact, in these cases, although hypertension is present, it is difficult to correlate the hypertension with the other many and varied symptoms present and give the condition the status of a disease.

Where the hypertension is not obviously secondary in type, however, this stage probably represents the initial phases of essential hypertension.

When we consider the frank, really high blood pressures exhibited by the patients in my second group (Table 2) we get a more definite tabulation of signs and symptoms, which by their repetition in various members of the group leads to the assumption that a definite disease is present.

Essential hypertension usually follows a benign course. More rarely a malignant form supervenes. Four cases in the series belong to the latter category. Both types are described.

Benign essential hypertension, although I have not seen it so described, can be further sub-divided into two main types. I find that there is a cerebral or cephalgic type in which unusually severe headache predominates and in which dyspnoea is not severe, and a cardiac type in which dyspnoea is the most prominent symptom while headache is practically absent.

In essential hypertension various causal factors are implicated. They are factors, however, which we would not expect to have any direct structural effect upon the peripheral circulation in a normal individual.

These factors include the various stresses and strains of modern life, environmental difficulties, marital and family conflicts, economic instability, bad and uncongenial working conditions, and other long continued psychological stimuli.

Nor can we exclude the effects of certain germs and toxins in such conditions as bacillus coli infection and intestinal stasis. A number of case histories have been given which tend to show that chronic or recurrent bacillus coli infection of the urinary tract may play an important role in the etiology of hypertension.

The work of Weiss and Parker[55] throws further light on this aspect and shows that vascular changes, concentric proliferation and necrosis of the arterioles of the kidney are

often present in these cases where hypertension is associated with or follows chronic or healed pyelonephritis.

I believe that in cases of chronic bacillus coli urinary infection the kidney damage is such that renin may be released into the circulation there to become activated into the pressor substance angiotonin.

The part played by dysfunction of the endocrine glands is rather more doubtful. That such changes do take place in the thyroid, pituitary or ovaries in some cases of hypertension is shown in case histories, but inter-related as the endocrines are with the sympathetic nervous system, it is difficult to differentiate cause and effect.

A small proportion of my cases - 7 per cent - although not suffering from Acromegaly had such physical characteristics that they could be termed acromegalic in type. In these cases I am inclined to postulate a perverted functioning of the anterior lobe of the pituitary.

In primary or essential hypertension the causal factor does not appear to act directly on the periphery, but indirectly on the vasomotor centre in the medulla via the sympathetic nervous system.

Although it is contended by Pickering that the vascular narrowing which eventually takes place is not brought about by nervous means a study of the sociological and environmental

conditions and hardships of many of these patients leads me to believe that prolonged psychical stimuli of a strong emotional character are definite etiological factors in a proportion of these cases.

I believe that angiospastic symptoms may be so produced and that a hypertorna of the arteriolar walls is a prelude to a permanent thickening of the walls.

My main thesis, however, is that the several factors I have enumerated do not evoke a hypertensive response in everyone who is subject to their influence. Indeed relatively few develop hypertension.

These various stimuli constitute the seed, a seed that is sown broadcast but only germinates on suitable soil.

Such a theory pre-supposes a susceptible individual whom I shall term the potential hyperpietic.

In an analysis of a series of cases of hypertension two great facts are apparent.

In essential hypertension in particular a family history is common. In a third of all cases of hypertension such a history may be obtained according to Halls Dally.[22] On similar grounds Sir H. Rolleston[21] suggests that hypertension is a constitutional disease with dominant Mandelian characteristics.

My second point is abundantly exemplified in my case histories. I find that my cases of hypertension feature two

main types of individual in almost equal proportion. Their characteristics have been described by me.

Briefly, there is the short-necked, florid, stockily-built person, dynamic, over-active, sensitive and quick-tempered. He often gives a family history of cardio-vascular disease, and he is potential hyperpietic number one.

Not quite so easily distinguished is the second type characterised by a sallow, often careworn facies, a thin spare frame, and a high strung irritable nervous temperament.

John Hay[32] in 1931 first drew attention to the characteristic build of his hyperpietic patients.

Constitutional predisposition and the exciting factors of mental strain and anxiety clearly play a large part.

Prevalence.

Statistics have been quoted to show that cardio-vascular diseases are definitely on the increase. By inference the prevalence of hypertension must be a gradually increasing figure.

It is my contention that this is so, and reasons are given for this conclusion.

I find that females are rather more prone to hypertension than males. Although this has been my experience, other observers report the reverse to be the case.

The extreme importance of sociological and environmental

problems in their relation to hypertension has been considered.

I have indicated my views regarding the prognosis in hypertension. The immediate or short term prognosis in very high blood pressure, for example, is not by any means as ominous as various authorities portray. In fact, it is remarkable to me how long many of these patients survive the onslaught of a very formidable systolic and diastolic pressure. This is well illustrated in Table 9.

While I have taken as my standard of assessment the definition of hypertension as given by such authorities as Halls Dally, H. Batty Shaw, and Osler, I have come to the conclusion that insofar as it applies to systolic arterial pressure it is erroneous.

Twenty per cent of the patients in this series suffered from hypertension by virtue of a systolic arterial blood pressure of over 160 mm. of mercury. Their diastolic pressures were below 90 mm., and in only three instances did the diastolic pressure eventually exceed that figure.

The significance of a raised systolic pressure per se has been greatly exaggerated, and whilst one would be wise to keep such a patient under observation his pressure should not be considered as any more abnormal than a rapid pulse rate.

Treatment.

I have considered at some length the prophylactic and

general hygienic treatment of the hyperpietic because I believe that if the measures I have advocated could be carried out a great measure of success in the prevention of hypertension would be achieved.

A considerable number of mild and moderate cases could be cured and severe cases considerably ameliorated by such a regimen.

I realise, however, that at the present time it is impossible to put such precepts into practice when dealing with a poor or working-class community, but I entertain great hopes that these difficulties will be removed by our future health plans for the nation and by the implementation of the Beveridge Scheme.

Until such time as it is realised by the profession in general and by the Ministry of Health in particular that the well being of the people is profoundly influenced by social circumstances, domestic and occupational surroundings, and, above all, by lack of knowledge of physiological facts and their relation to economic and social circumstances, we will have to struggle on in the semi-darkness.

A working man cannot re-order his life at a moment's notice, nor can a confidential talk, or a psycho-therapeutic interview dispel the fears and anxieties which are so often of an economic or social character.

The treatment must start at an earlier stage. This can only be achieved by:-

(1) Periodic examination of all adults and particularly of those who might be considered as potential hyperpietics.

(2) General hygienic treatment for all potential hyperpietics on the lines suggested for hyperpietics.

(3) Compulsory routine examination of all industrial workers of 40 years of age, such examination to include sphygmomanometric readings.

(4) An increased understanding in the medical profession and particularly in the medical teaching schools regarding the value of social hygiene and its fuller application.

(5) Education of the individual in elementary physiology by the State, and of the patient and family by the family doctor and social worker.

(6) Improvement of economic security in middle and old age by State measures such as insurances, adequate pensions, and

(7) Compulsory life insurance with medical examination.

I was delighted to hear my arguments reinforced and succinctly stated by the Representative Body of the British Medical Association in September, 1943, in their first basic principle which says, "The health of the people depends primarily upon the social and environmental conditions under which they live and work, upon security against fear and want, upon nutritional standards, upon educational facilities, and upon the facilities for exercise and leisure. The improvement and extension of measures to satisfy those needs should precede

or accompany any future organisation of medical services."

Drug Therapy.

This constitutes an individual problem against a condition which is so varied in etiology and symptomatology that no drug is specific. One hopes, however, that the promise of successful anti-pressor therapy will be realised.

By indirect action, however, we can relieve the hypertension by alleviating many of the distressing symptoms.

Drugs of the sedative group are of proved utility in this field. Of these I consider Theominal and Phenobarbital to be most useful.

Indications for the use of these drugs and of various Bromide combinations, including a formula by Sir William Gower, have been described.

Iodides, although supported by common usage for many years, are, in my opinion, useless in hypertension except where manifestations of syphilis exist.

Nitrites are of limited utility. In certain emergencies they are most helpful.

Euphyllin (a compound of Theophyllin and Ethyline Diamine) I have found to be a powerful adjuvant, particularly in the severe dyspnoea of the later stages of essential hypertension. Various other preparations and formulae have been used in this series of cases and the results recorded.

Bibliography.

1. Hay, John - "The Treatment of Patients with High Blood Pressure."
Disorders of Blood Pressure, London 1942, 22.

2. Rolleston, Sir H. - B.M.A. Annual Meeting, Sect of Medicine Proceedings, 1933.

3. Allbutt, Sir C. - Arteriosclerosis, London 1925, 12.

4. Osler & MacRae - Principles and Practice of Medicine. London 1924, 834, (9th Edit.).

5. Shaw, H. Batty - "Hyperpiesia and Hyperpiesis", London 1922, 4.

6. Dally, Halls - High Blood Pressure, 3rd Edition, London 1934, 104.

7. MacLaren, I. Patterson - "Modern Methods of Medical Insurance Examination." 2nd Edition, London 1929, 270.

8. Hilton, R. - Practitioner, London, June 1936, 729.

9. Dally, Halls - "High Blood Pressure", London 1934, 93.

10. Ministry of Labour and National Service - Instructions for Guidance of Medical Boards, MRB 1-9.

11. Allbutt, Sir C. - Index of Treatment, Bristol 1925, 9th Edition, 87.

12. Pickering, G. W. - Disorders of Blood Pressure, London 1942, 5.

13. Bennett, T. Izod - Practitioner, London, October 1936, 577.

14. Thomson, W. W. D. - Disorders of Blood Pressure, London 1942, 43.

15. Shaw, H. Batty - "Hyperpiesia and Hyperpiesis", London 1922, 177.

16. McDowell, R. J. S. - British Medical Journal, July 1941, ii 77.

17. Platt, R. - Practitioner, 1939, 143, 384.

18. Wilson, C., & Byrom, F. B. - Lancet, 1939, I, 136.

19. Helmer and Page - Leading Article, British Medical Journal, Renin and Hypertension, May 1940, I, 857.

19A. Parsons, Smith, P. T. - Practitioner, November 1943, 274.

20. Osler and MacRae - Principles and Practice of Medicine, London 1924, 836.

21. Rolleston, Sir H. - B.M.A. Annual Meeting, Sect. of Medicine, 1933.

22. Dally, Halls - "High Blood Pressure", London 1934, 126.

23. Gibson, A. G. - Medical Annual, Bristol 1930, 74.

24. Gibson, A. G. - Medical Annual, Bristol 1930, 74.

25. McDowell, R. J. S. - British Medical Journal, 1941, II 41.

26. Shaw, H. Batty - Hyperpiesia and Hyperpiesis, London 1922, 128.

27. Shaw, H. Batty - Hyperpiesia and Hyperpiesis, London 1922, 130.

28. Shaw, H. Batty - Ibid - 169.

29. Wheeler's Handbook of Medicine, Edinburgh, 6th Edition, 286.

30. Rolleston, Sir H. - Modern Technique in Treatment, London 1927, 258.

31. Gibson, A. G. - Medical Annual, Bristol 1933, 223.

32. Hay, John - British Medical Journal 1931, II 43.

33. McDowell, R. J. S. - British Medical Journal 1941, II 77.

34. Smith, Shirley - Practitioner, Oct. 1938, 425.

35. Pickering, G. W. - British Medical Journal 1939, I 1.

36. Wood, Paul - British Medical Journal 1941, I, 810.

37. Gibson, A. G. - Medical Annual, Bristol 1933, 223.

38. Pickering, G. W. - Disorders of Blood Pressure, London 1942, 5.

39. Shaw, H. Batty - "Hyperpiesia and Hyperpiesis", London 1922, 174.

40. Dally, Halls - "High Blood Pressure", 3rd Edition, London 1934, 136.

41. Pickering, G. W. - Disorders of Blood Pressure, London 1942, 10.

42. Thomson, W. W. D. - Ibid - 44.

43. Campbell, Harry - Pathology Aids, London 1920, 159.

44. MacDonald, Critchley - Medical Annual, Bristol 1939, 53.

45. Fishberg, A. M. - "Hypertension and Nephritis", London 1939, 33.

46. Thomson, W. W. D. - Disorders of Blood Pressure, London 1942, 51.

47. Smith, Lawrence W. - "Cardio Vascular Renal Diseases", New York 1940; Review Practitioner, August 1940.

48. Allbutt, Sir C. - Index of Treatment, Bristol 1925, 8.

49. Pickering, A. G. - Disorders of Blood Pressure, London 1942, 6.

50. McAlpine, Douglas - Ibid 39.

51. Sturrach, A. C. - Disorders of Blood Pressure, London 1942, 17.

52. Douthwaite, A. H. - British Medical Journal, 1929, II 844.

52A. Symonds, C. P. - Practitioner, August 1938, 189-191.

53. Hay, John - British Medical Journal, 1931, II 43.

54. McAlpine, Douglas - Disorders of Blood Pressure, London 1942, 36.

55. Weiss, S., and Parker, F. Jun., - Medicine, 1939, 18, 221.
Ref. Platt, R. - Practitioner, Nov. 1941, 691.

56. Edgecumbe, W. - British Medical Journal 1932, 1, 58.

57. Rolleston, Sir. H. - Modern Technique in Treatment, III, London 1927, 256.

58. Ince and Hay, J. - Lancet 1926, II, 799.

59. Jones, H. W., and Cumberbatch, E. P. - Diathermy, London 1937, 250, 342.

60. Tomlinson Report - Rehabilitation and Resettlement of Disabled Persons, H.M. Stationery Office 1943.

61. Smith, B. Parsons - British Medical Journal 1943, II, 299.

62. Platt, Robert - Practitioner, November 1941, 692.