THE SIGNIFICANCE OF INCREASED BLOOD PRESSURE IN PEOPLE OVER 40 YEARS OF AGE.

Aim.

The aim of this thesis is to make a clinical investigation of patients with increased blood pressure or hypertension, with the armamentarium of a general practitioner and without the aid of laboratory and radiological investigations.

All the patients were forty years of age or over, and were selected in the ordinary routine of a country general practice during a period of two years. They were all examined on several occasions, and thus were excluded the more temporary types of increased blood pressure.

In addition to a general investigation of the signs and symptoms present, particular attention was paid to the age, sex, occupation, habits such as eating, drinking, and smoking, height, weight, and body build, medical history, family medical history, urine, cardio-vascular system, and the fate of the patients, with a view to establishing a relationship, if any, between these and increased blood pressure.

It was soon observed that the majority of the cases could not come under the classification of Renal Hypertension and a short historical study of the growth of the concept of Primary Arterial or Essential Hypertension was made.

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Concept of Primary Arterial or Essential Hypertension.

Blood pressure is one of the functions of the circulatory system which can be measured quantitatively, and related to it are many physiological and pathological states of the body. By itself, like the pulse rate or temperature, it is merely a sign and in the days before the advent of the sphygmomanometer the pathological states associated with increased blood pressure were classified as Bright's Disease and the kidney was regarded as the primary cause of these states. Gradually, through the aid mainly of post-mortem examinations, the morphologists observed that cerebral vascular lesions, cardiac hypertrophy, and contracted kidneys, were commonly found together. Bright himself noted the connection between the renal and the cardio-vascular systems and "the extent and frequency to which the derangement of one organ is connected with derangements of several others."(1)

Consequently the idea arose that some of the pathological states, which had been classified under Bright's Disease, were not primarily renal.

Through the work of Johnson, and Gull and Sutton, this idea was developed and the suggestion was made that Bright's Disease was a disease of the body as a whole in which the chief pathological process was a diffuse arterio-capillary fibrosis.

With the introduction and application of clinical

methods of estimating blood pressure, it was soon discovered that increased blood pressure was a clinical manifestation of some of the pathological states associated with Bright's Disease. Some puzzling problems arose. Was high blood pressure always associated with Bright's Disease? Was high blood pressure essentially renal in origin? Were there pathological states, manifested clinically by increased blood pressure, which could not correctly be classified under Bright's Disease? These were some of the problems which Mahomed tackled. This physiologist and clinician was able to demonstrate that high blood pressure may be present without renal disease and before any signs of failure of renal function. "Patients with primary kidney disease such as seen in surgical kidneys or scrofulous kidneys.... do not have high blood pressure while patients with acute Bright's Disease..... may lose all signs of high blood pressure during their recovery even at a time when the kidneys are manifestly crippled, the urine being albuminous and the solids deficient in amount."(1) "High blood pressure and the cardio-vascular changes are the primary and most important conditions to recognise while the kidney symptoms are only secondary and are not even essential conditions."(1)

Thus did Mahomed weld the clinical and morphological findings into the modern concept of Primary Arterial or Essential Hypertension, which grew out of, but must be dis-

tinguished from the concept of Bright's Disease which is associated with primary kidney disease.

Primary Arterial or Essential Hypertension means a non-renal hypertension and covers a series of morbid states in which increased blood pressure is a permanent feature, is accompanied sooner or later by a diffuse hyaline fibrosis of the arteriolar system and by definite complications in the cardio-vascular system,which in turn lead to degenerative changes chiefly in the heart, brain, and kidneys. The diffuse hyaline fibrosis of the arterioles must be distinguished from Arterio-sclerosis in which the lesions are to be found in the bigger arteries. The two processes, though distinct, are often present together. It has been estimated that increased blood pressure is present in 50 per cent. of the cases with Arterio-sclerosis.(15) Thus whilst Arterio-sclerosis is not included in the concept of Essential Hypertension, it may be contemporaneous with it.

The present study has revealed that the concept of Essential Hypertension has a solid foundation in fact and that in the great majority of the 106 cases reported,neither the history, clinical investigation, nor observation over two years, nor cause of death in the eight patients who died during that time, have demonstrated a primary kidney lesion, though secondary renal changes were noted.

The Meaning of High Blood Pressure or Hypertension.

What constitutes high or increased blood pressure? There are two pressures which form the quantitative basis of all investigations into blood pressure, viz., the systolic pressure, i.e., the pressure within the arteries at the height of cardiac systole, and the diastolic pressure, i.e., the minimum pressure constantly present in the arteries. The systolic pressure is intermittent, is a measure of ventricular efficiency, and is liable to great and small variations as a result of physiological and psychological processes. The diastolic pressure was found in this study more fixed than the systolic. It is a measure of the peripheral resistance and the permanent strain to which the cardiovascular system is subjected. Normal blood pressure signifies systolic and diastolic pressures that do not fluctuate too violently and which under reasonable conditions of rest and quiet are within certain not too rigid limits. "No fact regarding the blood pressure is better established than its wide range of variation in any individual. It is therefore impossible to speak of a normal value for blood pressure but only of certain upper and lower limits."(3)

The normal limits for adults are - systolic pressure 110 to 140 mm. Hg., diastolic pressure 70 to 90 mm. Hg.. A patient with a sustained blood pressure of 150/90 is a potential hyperpietic. It is generally agreed that a systolic

pressure of 160 mm. Hg. or over in an adult under the specified conditions of mental and bodily rest and quiet is high and that a diastolic pressure of about or above 95 mm. Hg. and certainly above 100 mm. Hg. is high. After an investigation into blood pressures, Dr Hare[15] concluded "that blood pressure readings obtained by ordinary clinical methods of auscultation vary with some frequency up to 15 mm. Hg., and that this variation must be considered together with the other better recognised factors, such as emotion or exertion, causing physiological variations." This is important firstly in estimating whether hypertension exists, and secondly in appraising the results of treatment, for obviously a treatment that claims a reduction of 15 to 20 mm. Hg. must be examined critically.

Hypertension then connotes sustained increased systolic or diastolic pressures or both. In Essential Hypertension both pressures are raised and marked instability is sometimes observed especially in the early stages. Dr Halls Daly says in his book "This notable instability of arterial pressure is more characteristic of Hyperpiesia (Essential Hypertension) than the actual heights of pressure recorded."[3] It is disputed which pressure is the more significant. The important point is that together they give more information than either separately. Together they provide us with the pulse or differential pressure which is of some prognostic

value. "In adult life the normal range of differential pressure is 30 to 55 mm. Hg."(3) In this series of cases the differential pressure ranged from 55 mg. Hg. to 170 mm. Hg., and no uniform relationship was found between the pulse rate and the differential pressure.

In addition to the sustained increased pressures which give a quantitative basis for study, other clinical signs are usually found to clinch the diagnosis of hypertension, especially if the patient is in reasonable health. Blood pressure is a reaction and in certain cachectic states the failing peripheral resistance or heart masks the underlying disease process. The heart, where the blood pressure is increased, sooner or later hypertrophies on the left side, a fact which can be ascertained by percussion aided by palpation and auscultation, - the methods employed here. Radiological examination gives much more accurate information. The second aortic sound as heard over the aortic area is accentuated and if the aorta is dilated or sclerosed it has a ringing quality.

Cardiac enlargement with accentuated second aortic sound was present in over 70 per cent. of this series of cases. The reason for their seeming absence in some cases was due to the stoutness of the patients and in others to emphysema and the encroachment of the cardiac area by the lungs and in others to general weakness.

The pulse in the absence of grave cardiac complications is characteristic and almost pathognomonic in the majority of cases. It is a full pulse, is well sustained and is not easily obliterated. In a few cases the pulse was small and thin.

Thus in the absence of grave complications, the diagnosis of Essential Hypertension rests on a raised systolic and diastolic pressure accompanied by cardiac hypertrophy, accentuated second aortic sound, and a full well-sustained hard pulse, and also on the absence of primary renal disease.

How is the Blood Pressure measured?

"Stephen Hales[40] by fastening a long glass tube inside a horse's artery devised the first manometer with the aid of which he made quantitative estimates of the blood pressure." To-day blood pressure is estimated by means of sphygmomanometers. With the arm at the cardiac level and the patient sitting or reclining in physical and mental rest and not too soon after a meal in a comfortable temperature, a cuff 12 cms. in width is wound round the arm above the elbow and inflated until the pulse disappears at the wrist. At the point of disappearance and reappearance of the pulse, a reading of the gauge (mercurial or aneroid usually) is made. This is called the Palpatory Method and gives only the systolic pressure. A more accurate method is the Auscultatory, which

can be checked by the Palpatory Method as regards the systolic pressure. With the patient as above, a stethoscope is placed over the brachial artery just above the bend of the elbow. The cuff is inflated until all sound ceases and the pulse at the wrist disappears. This latter observation is often necessary because of the silent period that sometimes appears within the sequence of sounds usually heard. The point at which all sound ceases and the pulse disappears, checked by the reappearance of both on deflation marks the systolic pressure. On deflating a distinct click is first heard, then a sequence of blowing murmurs and a crescendo of clear thuds changing, often suddenly, into a muffled sound and then silence. The end of the crescendo of thuds, i.e., the end of the third phase and the onset of the fourth phase, indicates the diastolic pressure. Most authorities agree on this point, though it is disputed.

In this series of cases the diastolic pressure varied from 60 mm. Hg. in a case of aortic regurgitation to 150 mm. Hg. in a case of Menopausal Essential Hypertension. There were 27 cases with a diastolic pressure below 95 mm. Hg. and 79 with a diastolic pressure of 95 mm. Hg. or over. There were 9 cases with a diastolic pressure under 85 mm. Hg. and we may safely say that no case with a diastolic pressure less than 85 mm. Hg. is suffering from Essential Hypertension.(30) In the absence of Aortic Regurgitation such cases are less likely to develop symptoms referable to the circulatory system.(30)

An analysis of the 9 cases, except in the one case of Aortic Regurgitation, revealed very few symptoms referable to the cardio-vascular system. With the one exception they were all reasonably well and active with an average age of 70.

Chart of cases with a Diastolic Pressure below 85 mm. Hg.

Name.	Age.	Pulse & Blood Pressure.		Chief Signs and Symptoms.
Lowes, Mrs.	59	94	200/80	Dyspepsia. Tender gall-bladder.
Barker, Miss.	74	55	200/80	Dyspepsia. Tender gall-bladder.
Glaholme, Mrs.	70	60	170/70	Rheumatism.
McDonald, Mrs.	70	90	180/80	Chronic bronchitis.
Raw, Thomas.	71	60	180/80	Dyspepsia.
Gargett, R.	76	64	170/80	Giddiness.
Laidler, Thomas.	80	70	160/80	Rheumatism.
Jackson, Newby.	80	80	200/80	Alcoholism.
Bonsfield, C.L.	40	100	200/60	Aortic Regurgitation.

The systolic pressures ranged from 150 mm. Hg. in a case of Menopausal Hypertension to 290 mm. Hg. in a case of Essential Hypertension complicated with cerebral vascular lesions. The former case was a potential sufferer from Essential Hypertension. Her blood pressure was 150/100 with a slightly accentuated second aortic sound. All she com-

plained of was feeling a little more tired after her day's work than formerly. Her age was 51, the menses had not been seen for a few months and she had put on two stones in weight during the last year, although her diet and daily routine were unaltered.

In the present study a Tycos Aneroid Sphygmomanometer was used although it is generally agreed that a mercurial manometer is more reliable.

How is Blood Pressure increased and sustained?

The cardio-vascular system is a distributing and collecting system which requires pressure to maintain its functions. The pressure is created and maintained through the action of the central pump, i.e., the heart, against a load, i.e., the blood and the peripheral resistance. "The peripheral resistance in the vascular system is directly proportional to the viscosity or internal friction of the blood."(12) Factors acting through the heart or the peripheral resistance affect the blood pressure which thus "varies as the product of the cardiac output and the peripheral resistance."(2)

Increased unit output of the heart, increased volume of blood in the body as a whole, increased blood viscosity and contraction of the vascular bed, have all been invoked as explanations of increased blood pressure.(6)

Examples of increased unit output are to be found in

cases of Aortic Regurgitation and in some cases of Hyperthyroidism. In these, however, only the systolic pressure is raised.

Intake of excessive fluids can raise the blood pressure temporarily and may induce a catastrophe in a case of high blood pressure. Increased volume of blood has not been proved to be present invariably in hypertensive cases. In any case increased volume of blood is an unlikely mechanism because of the range of accommodation of the vascular bed and also because of the facilities of the body for the withdrawal of fluids from the circulation.(6)

Likewise increased viscosity of the blood is an unsatisfactory explanation and hypertension is not always present in the classical example of increased viscosity - Polycythaemia.

Thus we are thrown back on a contraction of the area of the vascular bed as the mechanism whereby blood pressure is increased and sustained. Arteriolar Hyperplastic Sclerosis has been suggested as an explanation of the vascular contraction. Against this view is the fact that Arteriolar Hyperplastic Sclerosis, when present, has a characteristic distribution and is more or less confined to the kidneys, spleen, liver, pancreas, and brain. In comparison with the potential area of the vascular bed a contraction of these areas alone would be insufficient to increase the blood pressure more than temporarily. Further it has been shown that Arteriolar Hyperplastic Sclerosis does not precede but rather follows in the

wake of high blood pressure.(6)

Logic therefore points to a general vasoconstriction as the primary mechanism in the pathogenesis of high blood pressure. This mechanism explains both the systolic and diastolic increase of blood pressure and indicates that Essential Hypertension is functional in its beginnings, of which there is much evidence, and also helps to explain its spasmodic nature often observed in early cases.(6) Vasoconstriction has been actually observed in the retinae of cases of Eclampsia where there is increased blood pressure, and of acute hypertensive cerebral attacks.(6) Also in cases of spasmodic hypertension pallor has been noted in the skin during the spasms of increased pressure. Again cases are reported where increased blood pressure has been restored to normal through the resolution of psychic conflicts which liberates the arterioles from their spasm. This factor of spasm explains the fall in pressure and relief from symptoms experienced after certain therapeutic measures, e.g., the administration of nitrites, or mental and physical rest. One patient, Mrs Pedelty (No. 50) had a blood pressure of 300/130 for several days when first seen with severe occipital headaches. In the course of a few weeks with rest and reassurance the pressure fell to 180/100.

In time changes of structure follow changes of function and gradually come more and more into the clinical picture and no doubt induce a compensatory increase in blood

pressure. These start a series of changes which end with defeated hearts and blood vessels.

Aetiology.

"Knowledge of the earlier stages in the development and in fact the entire natural history of Arterial Hypertension is still lacking," says Dr. Soma Weiss.(1) Whilst several causative factors have been shown to be involved, no well-supported theories of the aetiology of Essential Hypertension have been produced so far.(6)

There is much evidence, experimental and clinical, to show that disordered kidneys are responsible for some cases of hypertension. These latter are classified as cases of Renal Hypertension. Dr. H. McLean(8) says, "Broadly it may be taken for granted that in chronic renal disease the more serious the renal damage the higher the blood pressure." That increased blood pressure and renal disease do not run parallel in many cases was demonstrated by Professor Boyd(8) who "came to the conclusion that blood pressure furnishes no evidence as to the state of the renal functions."

Experimental blocking of both ureters, urinary retention as a result of prostatic hypertrophy or as a result of tabetic vesical paralysis have been found to be followed by increased blood pressure. In some cases when the obstruction has been released the blood pressure has reverted to normal.

Polycystic disease of the kidneys with high blood pressure, Fishberg points out,[6] is a classical example of Renal Hypertension.

The kidney has many functions and consequently many theories have been constructed to explain Renal Hypertension. Disturbed acid-base balance, deficient salt and water excretion, and retained metabolites have all been implicated. None explains satisfactorily all the features of this type of hypertension. Against the disturbed acid-base balance is the fact that acidosis does not develop until late in chronic nephritis. As to deficient salt and water excretion restriction of salt intake in cases of Congestive Heart Failure where hypertension may be present and of Chronic Prenchymatous Nephritis where hypertension is not present, is beneficial but in cases of hypertension where there is no oedema restriction brings no improvement.

The theory of retained metabolites having a pressor action has more support in its favour. In the British Medical Journal[9] an account was given of experiments in connection with the "extraction of pressor principles from blood of hypertonic subjects (arteriosclerosis, acute nephritis, chronic nephritis, essential hypertonia)." The extracts were injected into dogs as indicators. Extracts from a case of Essential Hypertension and Arterio-sclerosis produced no alteration in blood pressure, whilst extracts from a case of Acute Nephritis or Secondary Contracted Kidney regularly

produced a great rise in blood pressure. No quantitative relationship was observed between the degree of hypertension and the induced hypertension in the dogs. In almost all cases in which a pressor effect was found there were positive urinary findings, e.g., casts, blood, and albumin. Another interesting observation was that a similar pressor effect was found with extracts from the blood of true epileptics taken during or just before an attack. In two cases of haemorrhagic nephritis the pressor substance disappeared from the blood when the clinical condition improved.

The above report suggests the presence of pressor substances in the blood of some renal cases with hypertension. On the other hand there are cases of renal disease without hypertension, e.g., Chronic Parenchymatous Nephritis. Occasionally acute tonsillar infections are followed by Acute Interstitial Nephritis with no increased blood pressure.(6) Cases of Adolescent Genu Valgum with late Rickets have a Chronic Interstitial Nephritis and no hypertension. Cases with a latent nephritis, as the one reported in this series, Mrs. Cathleen Keenan (No. 57), have not got increased blood pressure except during the subacute exacerbations. These latter cases probably in time will enter the chronic stage of nephritis with hypertension. There is clinical and experimental evidence of Uraemia without hypertension and convulsive phenomena and of hypertensive convulsive phenomena

without Uraemia, which suggest that the hypertension as well as the convulsive phenomena do not belong to the biochemical syndrome of Uraemia which is directly due to renal factors.[24] Further it has been observed that in the course of Acute Glomerular Nephritis the blood pressure falls to normal and the oedema disappears before the albumin, casts, and blood disappear from the urine.[1,6]. All these facts suggest that the pressor substance responsible for the hypertension does not derive from the kidney but from extra-renal factors. Allergy[6] has been invoked to explain the development of Acute Nephritis, e.g., Scarlatinal, two or three weeks after the original infection and it is a reasonable conjecture that just as the Acute Nephritis so the hypertension may have to do with the processes of Allergy.

Thus the relationship of hypertension to renal disease is not clear and some clinical pictures of renal disease are certainly mixed pictures in the sense that all the blood pressure phenomena are not explicable through the kidneys. Consequently "blood pressure furnishes no evidence as to the state of the renal functions."[8] After destruction of the glomeruli has gone so far a compensatory increase of blood pressure is induced to make up for the glomerular deficiency. This is the condition of compensated renal deficiency which may be decompensated through further glomerular destruction or through heart failure. One patient, Mrs Jackson (No. 31), who had a benign nephro-sclerosis, suffered from periodic

attacks of Congestive Heart Failure without valvular disease. Latterly she developed a polyuria with deficient urinary urea concentration - a condition of compensated renal deficiency. Finally decompensation developed with oliguria, uraemia, and signs of Congestive Heart Failure.

Whilst therefore the exact rôle played by the kidney in the production of hypertension is by no means clear, Renal Hypertension is a reasonable classification. But by far the greater number of cases of hypertension in people over 40 years of age come under the classification of Non-renal or Essential Hypertension. There are few cases in this series which clinically, i.e., by observation of signs and symptoms over two years, by examination of the urine for albumin, specific gravity, sugar and deposits, by observation of the fates of those who died, and by reference to statistical evidence of the cause of death and the age incidence of hypertension, could be classified under Renal Hypertension.

All the cases except one are 40 years of age and over. After the fourth decade of life statistical evidence shows that Chronic Glomerular Nephritis is diminishing whilst the incidence of Essential Hypertension in both sexes is increasing.(6) In addition the weight, body build, absence of anaemia, the presence of albumin in only 20 out of 90 patients, the absence of haematuria in all, point to the probability that infective primary kidney disease was not

the essential aetiological factor in the majority of the cases. Post-mortem discovery of renal lesions is not a sufficient basis on which to build a primary renal aetiology for we know that the kidneys have a wide range of physiological flexibility, and also that a major destruction of kidney tissue is necessary before the blood and urinary chemistry is altered. The clinical pictures on the whole suggest that the renal lesions are incidental or secondary to a more general condition.

As already stated twenty cases had traces of albumin in the urine. No unusual deposits were found and few casts.

Patients with Albumin in Urine.

Name.	Age.	Symptoms and Signs.	Pulse.	Blood Pressure.
Dean, R.	69	Dyspnoea. Giddiness. Dyspepsia. Polyuria.	100	240/110
Slack, H.	53	Dyspnoea. Angina. Nose Bleeding.	60	160/100
Lodge, J.	78	Bronchitis. Dyspnoea.	90	170/90
Bell, J.	77	Memory bad. Weak and giddy.	64	200/105
Wigham, J.	86	Cardiac oedema. Urinary retention.	80	200/110
Longstaff, R.	69	Dyspnoea. Cardiac oedema.	80	200/130
Brayfield, R.	62	Dyspnoea. Enlarged prostate removed.	90	190/100
Hall, Wm.	73	Dyspnoea. Cardiac oedema.	90	170/110

Name.	Age.	Symptoms and Signs.	Pulse.	Blood. Pressure.
Pedelty, Mrs.	63	Memory bad. Dyspnoea. Weakness. History of puffy eyelids.	70	200/105
Brown, H.	71	Cardiac oedema. Dyspnoea.	80	210/130
Down, Mrs.	74	Bronchitis. Dyspnoea.	80	210/110
Willcock, Mrs.	67	Dyspnoea.	80	190/95
Smith, Mrs.	75	Dyspnoea. Cardiac oedema.	80	260/130
Bell, Mrs.	76	Dyspepsia.	80	200/100
Copping, Miss.	51	Bronchial asthma.	90	150/110
Appleby, Mrs.	72	Nil.	80	180/100
Dobson, Mrs.	61	Oedema of feet and eyelids.	64	220/120
Tarn, Mrs.	51	Angina. Dyspepsia.	80	250/150
Walker, Mrs.	85	Nose-bleeding. Nocturia. Giddiness.	80	170/90
Brown, Mrs. L.	62	Insomnia. Angina. Dyspepsia.	120	250/110

On further analysis it was found that of the five youngest with urinary albumin, two had a history of swellings of the face suggestive of renal oedema. One, Mrs. Pedelty, had a history of Scarlet Fever and Measles with a specific gravity of urine of 1008 and with a deficient urinary urea concentration. The other, Mrs Dobson, had a history of Typhoid, Scarlet Fever and Measles, with specific gravity of

urine of 1010, and a normal urinary urea concentration. No retinal deposits or haemorrhages were observed. These were the only cases suggestive of an inflammatory kidney lesion. They both had rather indifferent health. The majority of the others have been active workers right up to the seventies. Mrs. Appleby, age 72, says she feels fit and lives a life only a little less active than formerly.

The fifth decade marks the beginning of involutionary changes in women, those in men come later. The following chart demonstrates the earlier incidence of hypertension amongst women.

Chart showing the incidence of Hypertension amongst men and women at different ages in this series.

	40-49	50-59	60-69	70-79	80-89
Men	4	8	20	12	5
Women	5	17	15	17	2

There are 22 cases of women in the 5th and 6th decades against 12 men. The men do not reach peak incidence until the 7th decade. The incidence suggests a connection between involution and hypertension. Altogether there are 49 men and 57 women in this series. No doubt some cases of Essential Hypertension

exist symptomless before the age of 40, but there is an absolute increase in the fifth decade and afterwards, and tending to fall again after 70.[6,3]. In this series there is a fall amongst the men then but amongst the women the fall is not evident till 75.

Involutionary changes are partly physiological in which the endocrines, mental and physical make up, all the factors that go to determine constitution, familial tendency and diathesis and consequently metabolism, play a part.

Attempts have been made to explain hypertension through one gland. All the endocrines have been implicated. Imbalance of endocrine and other pressor and depressor substances explains some of the features of Essential Hypertension. Blood and urine examinations of hyperpietics have been made to establish their presence or absence in excess, e.g., cases of hypertension have been reported where the urinary excretion of guanidine has increased as the blood pressure diminished.[3] Adrenalin is the classical example of a pressor substance. It is secreted by the suprarenal glands under emotional stress and causes a vaso-constriction of the arterioles with a rise in blood pressure and blood sugar. A product which is either Adrenalin or something biochemically indistinguishable is liberated from all sympathetic nerve endings when these are stimulated, e.g., by the emotions.[35] It has been shown by experiment that repeated injections of

Adrenalin can produce a condition similar to Essential Hypertension.[6] On the other hand efforts to find Adrenalin in excess in the blood of hyperpietics have failed. The effect of normal quantities of Adrenalin in the blood may be increased considerably by the diminution or absence of the vaso-dilating complementary hormone or hormones. We do know, however, that several cases have been reported of Suprarenal and Chromaffin Tissue tumours with hypertension which has been reduced to normal limits in some cases after removal of the tumours. One case of Basophil Adenoma of the Anterior Pituitary Gland with hypertension is reported here. These are rarities.

Amongst depressor agents with vaso-dilating properties is a substance called Acetylcholine, the direct antithesis to Adrenalin, which is liberated at the vagal nerve endings when these are stimulated. Vagotonine, a pancreatic vaso-dilator, and Adenosine found in all muscles in abundance, in addition to Acetylcholine have been used with some success in the treatment of Essential Hypertension.[20,21]

In cases of Hyperthyroidism the systolic pressure only is raised. Several cases of this series, all women, nervy, emotional, with palpitation and throbbings, suggest glandular imbalance. In one case of Myxoedema, proved by the efficacy of thyroid medication, the blood pressure was very unstable and the patient was emotional and nervy and not always morose and dull.

The different types of case, the stout, the thin, the lethargic, the vigorous, the emotional, suggest a functional disturbance of relationship of the endocrines. The clear cut case traceable to one endocrine or one chemical agent, pressor or depressor, is exceptional.

The ductless glands are in close physiological relationship with the sympathetic nervous system which is very sensitive to mental states of an emotional character. These physiological connections are doubtless concerned in the aetiology of hypertension and will be discussed later.

The endocrines have been called the regulators of metabolism, and with the functional disturbances which often accompany the involutionary changes of middle life, come disordered or "warped" metabolism.(15) It is a fact that many men and women grow stout in the fifth and sixth decades and later in some cases grow thin again. This runs roughly parallel with the incidence of hypertension. In 23 of the female cases of this series there is a definite history of increase in weight round about the climacteric. There are 11 cases in which symptoms associated with increased blood pressure developed with the onset of the menopause. Six showed marked increase in weight.

Chart of cases where there was a close association between the menopause and the development of high blood pressure.

Name.	Age.	Weight.	Pulse & Blood Pressure.		Symptoms.
Walker, Mrs.	43	18 st.	96	160/105	Nerves and flushings.
Stewart, Mrs.	40	14 st.	96	190/110	Dyspepsia. Giddiness. Urticaria.
Hodgkinson, Miss.	48	13 st.	80	180/90	Tiredness
Carter, Mrs.	54	16 st.	90	220/100	Dyspepsia. Dyspnoea. Nerves.
Langstaff, Mrs.	43	15 st.	74	170/90	Dyspnoea.
Watson, Mrs.	54	11½ st.	60	200/100	Dyspnoea. Vague pains. Precordial distress.
Allison, Mrs.	54	10½ st.	80	150/100	Slight tiredness.
Barnes, Mrs.	53	11½ st.	100	160/90	Throbbings. Flushings. Palpitation. Nerves.
Tarn, Mrs.	51	12 st.	80	250/150	Dyspnoea.Dyspepsia.
Gibson, Mrs.	50	14 st.	70	185/100	Dyspepsia and Nerves.
Parkinson, Mrs.	54	11 st.	60	210/90	Dyspepsia and Dyspnoea.

The menopause, obesity, and hypertension are related somehow, though some cases of menopausal obesity do not have increased blood pressure and yet have symptoms in some cases similar to those with increased blood pressure,[14] so that the relationship between the symptoms and the blood pressure is not a direct one.

In the following chart are shown the number of men and women who are overweight, normal, and underweight. The weights were calculated with reference to height and age.

	+6 st.	+5 st.	+4 st.	+3 st.	+ 2 st.	+ 1 st.	+	-	-1st.	-2st.	-3st.
Men	0	1	3	2	6	9	7	6	2	4	1
Women	2	1	4	4	5	9	6	6	4	3	1

To put the matter another way; Of 86 patients whose weights and heights are recorded,

53 per cent. were overweight,

29 per cent. were average weight,

17.5 per cent. were underweight.

The height and weight relationship, the body-build, is an important one.(11) This table shows that a plus weight tends more towards hypertension than a minus one. This agrees with observations made on healthy adults in the Royal Air Force.(11) Wing Commander Treadgold,(11) after an extensive investigation, concluded that between the ages of 18 and 40 increased blood pressure was absent where the body build was good, i.e., where height and weight were proportionate for the age.' He found that cases with raised blood pressures, i.e., cases, according to his standards, with systolic pressures over 140 mm. Hg., constitute 6 per cent. of fit pilots

and that the tendency of these ~~were~~ towards overweight and poorer cardio-vascular efficiency than normal.

This association of obesity with increased blood pressure tallies with therapeutic experience where a diminution in weight, with diminished intake and increased output of energy, is often followed by improved health, release from symptoms and sometimes by diminished blood pressure.

Associated with obesity are conditions such as Cholecystitis, Gall-stones and Diabetes, which often accompany hypertension. It has been said that the prognosis for Essential Hypertension is similar to Diabetes in pre-insulin days.[30] In addition to lower sugar tolerance noted in the literature of Essential Hypertension,[33,34] a very interesting comparison can be made between Diabetes and Essential Hypertension.[30] As a result it has been suggested that Essential Hypertension is a deficiency disease, e.g., due to absence more or less of depressor agents. In both there is increased incidence early in middle life. They run prolonged courses, have a certain familial tendency, are found together, and are more severe and more rapid in their course the younger the patient, e.g., Malignant Hypertension occurs before the fifth decade usually.

There were three cases of Diabetes in this series of cases.

The Three Cases of Diabetes of this Series.

Name.	Age.	Weight.	Pulse.	Blood Pressure.	Urine.	Signs and Symptoms.
McDonald, J.	58	14 st.	120	180/105	No albumin.	Acute cerebral attacks. Paresis Dyspepsia.
Dean, R.	69	12 st.	100	240/110	Albumin slight.	Dyspepsia. Dyspnoea.
Waine, Mrs.	67	12 st.	80	190/100	No albumin.	Dyspepsia, with tender gall-bladder.

All three cases had obvious arterio-sclerosis in the retinal blood vessels, with diabetic symptoms of thirst and polyuria which disappeared with balanced and restricted dietary. The Diabetes may have been due to arteriolar changes in the Islets of Langerhaus but according to the literature this is rare. The youngest and heaviest, McDonald, but with the highest blood pressure, has had acute cerebral attacks with permanent sensory and motor paresis. The patient with the albuminuria enjoys the worst health with clinical symptoms, such as mental and physical weariness and gastro-intestinal disturbances, suggestive of renal deficiency.

Joslin[28] in 1063 diabetics found marked obesity as a precursor in 40 per cent. and that Diabetes does not increase with age except in the obese.

As already mentioned, "the relationship between cholecystitis, cholelithiasis and obesity is a commonplace."[28] In this series there are recorded sixteen cases with tenderness in the region of the gall-bladder and accompanied with Dyspepsia

of the flatulent type. The majority - 13 out of 16 - are actually overweight or increasing in weight. In two cases gall-stones have been removed, in another gall-stones diagnosed, and in another there have been repeated attacks of Jaundice accompanied by Dyspepsia and Biliary Colic. The others had only gall-bladder tenderness and dyspepsia. With the metabolic disturbances associated with the above conditions there is sometimes a cholesterolaemia, which has been found also in Acute Hypertensive Cerebral Attacks, and in the Lipoid Nephroses where there is no increase in blood pressure.(6)

There were 13 cases with disordered stomachs, chiefly of the flatulent and heartburn type without gall-bladder tenderness. Seven have suffered with rather intractable diarrhoea, whilst three very stout women had periodic bouts of vomiting. The explanation of these is not clear. Some may have been due to liver dysfunction and the result of vigorous eating and living, and some to nervous disturbances.

Whilst, therefore, all cases of obesity do not have hypertension, all these facts taken together form a reasonable case for an aetiological relationship between Essential Hypertension, Obesity, Disorders of the Liver and Gall-bladder, Diabetes, the Endocrines and Involutionary Changes. "Degenerative diseases in heart, arteries, kidneys, and liver of a fatal nature are $2\frac{1}{4}$ times commoner in the obese than in standard weights and $3\frac{3}{4}$ times commoner than in underweights."(28) Certainly Essential Hypertension is more common in overweights

before and after the age of 40.

We now pass on to a consideration of heredity in the aetiology of Essential Hypertension. At present most cases of Essential Hypertension are classed as Constitutional or Familial. The influence of heredity is emphasised by comparison. Dr. Donnison studied 1,000 healthy Kenya natives in 5 year age groups and found their blood pressures identical with Europeans up to the age of 40, after which they fall steadily, whilst Europeans tend to rise.(11) "He found that the natives were singularly free from cardio-vascular disease although pyorrhoea was universal over 30, and infections in general extremely frequent."(21) Sir H. Rolleston records that anthropometric characters of hyperpietics have been tabulated and the influence of heredity illustrated by freedom from hypertension "of pure bred Chinese with their oriental calm of the East."(21) Also it has been observed that "the South African native seems immune from Hyperpiesia whilst he lives in primitive conditions in Africa but when he has been transported to America and adopts a mode of living associated with civilisation, he becomes subject to it."(33) This suggests that inheritance of environment as well as of a bodily constitution plays a part in the development of hypertension, an idea which will be discussed later.

In this study the ages at death of 140 fathers and mothers were collected and causes of death ascertained. The average age at death was 71.

Chart of Causes of Death in 140 Parents.

Heart.	Stroke.	Bright's Disease.	Diabetes.	Old age.	Cancer.	Accident.
22	22	8	1	17	7	8

Bronchitis.	Pneumonia.	Intestinal Obstruction.	Liver.	Appendicitis.
5	6	1	4	1

Rheumatism.	Puerperal.	Urinary Obstruction.	Tuberculosis.	Alcoholic.
2	1	1	2	1

Unknown.

31.

Of the 31 unknowns, 9 died 80 years of age or over,
11 died between 70 and 79.
7 died between 60 and 69.
1 died between 50 and 59.
2 died between 40 and 49.
1 died between 30 and 39.

Probably some at least of the cases of Bright's Disease would be cases of Essential Hypertension. The old age group would contain some hyperpietics and no doubt some of the unknown also. When we reckon that 16 per cent. died of Cardiac Disease and 16 per cent. of Strokes, according to the informa-

tion given, there is some foundation in fact for heredity as an aetiological factor.

The following charts confirm this further. The first chart gives the family history of the 15 cases who were below normal weight. The second chart gives the family history of a number of the overweights.

Family Histories - Chart No. I.

Copping, Miss.	57.	Mother d. 82 - accident. Father d. 77 - heart.
Colling, Miss.	73.	Mother d. 84 - heart dropsy. Father d. 78 - apoplexy.
Layton, Miss.	70.	Mother d. 80 - old age. Father d. Not known.
Dent, Mrs.	56.	Not known.
Walker, Mrs.	85.	Mother d. 40 - apoplexy. (Very stout). Father d. alcoholic. Age unknown.
Barker, Miss.	74.	Mother d. 56.) Bright's disease. Father d. 68)
Appleby, Mrs.	72.	Mother d. 66.) Cause unknown. Father d. 64.) Both stout and tall. Sisters living ages 70, 72, 83. Sister d. 66 - cerebral softening. Sister d. 77 - heart. Brother living age 80.
Bell, Mrs.	76.	Mother d. 73 - heart. Father d. 84 - old age.
Scott, F.	57.	Mother d. 73 - old age. Father living age 80.
Willcock.	70.	Unknown.
Collinson.	71.	Mother d. 65 - bronchial. Father d. 69 - jaundice.

Robinson.	84.	Mother d. 78 - heart. Father d. 94 - heart. Sister d. 73 - stroke. Sister d. 63 - in sleep.
Thurlbeck.	67.	Mother d. 86 - unknown. Father d. 74 - had several strokes. Brothers d. 74 & 65 - heart.
Davison.	71.	Mother d. 74 - bronchial. Father d. 76 - accident.

Family Histories - Chart No. 11.

Spenseley.	62.	Mother d. 47 - inflammation of bowels. Father d. 50 - cancer. Brothers 2 living 68 and 70. Brother d. 63 - sudden death. Sister living 73 - loss of memory. Sister d. 82 - old age and heart. Sister d. 67 - hemiplegia.
Dawson.	57.	Mother d. 45 - apoplexy. Father d. 76 - heart. Brother d. 46 - asthma. Sister living 54 - hyperpiesia. Sister living 60 - stroke. Sisters living 49 & 59 - very stout.
Collinson.	65.	Mother d. 34 - unknown. Father d. 67 - stroke. Several paternal relations died of strokes and heart.
Hodgkinson, Miss.	48.	Mother d. 80 - old age. Father d. 67 - cerebral haemorrhage.
Raw, Thomas.	71.	Mother d. 81 - old age. Father d. 70 - cerebral haemorrhage.
Duthie, Mrs.	65.	Mother d. 80 - old age. Father d. 80 - stroke.
Down, Mrs.	74.	Mother & Father d. over 70. Brother d. 62 - stroke.
Dean, Margaret.	62.	Mother d. 87 - old age. Father d. 70 - bronchial.

Nixon, J.	68.	Mother d. 64 - heart. Father d. 87 - unknown.
McDonald, J.	58.	Mother d. 78.) Unknown. Father d. 80.)
Oliver, Henry.	66.	Mother d. 75 - stroke. Father d. 76 - old age. 4 brothers living over 70. Sister living over 70.
Slack, Harker.	53.	Mother d. 58 - found dead in bed. Father d. 64 - influenza. Sister d. 65 - in sleep. Brother d. 59 - heart dropsy.
Bell, Mrs.	76.	Mother d. 73 - heart. Father d. 84 - old age.

"It is common to obtain a family history of cerebral haemorrhage or death from myocardial disease in those who suffer from Hyperpiesia. Heredity is the one established factor in the aetiology of the disease,"[36] says Dr. Geoffrey Evans.

The appearance of many sufferers from Hyperpiesia (i.e., Essential Hypertension) is fairly characteristic. On the whole they have a sthenic habitus, are big and robust, with florid complexions, short thick necks, and stocky, with good skeletal and muscular systems, broad chests and wide epigastric angles. Following a classification according to appearance suggested by Dr. Ryle[15] into (1) Robust, (2) Average, (3) Poor Physique, and (4) Thin, about 60 per cent. of this series were robust, 19 per cent. average, 17 per cent. poor physique, and 4 per cent. thin.

With regard to tobacco[10] and alcohol, it was found that 54, i.e., just over 50 per cent., neither smoked nor consumed alcohol in any form, and the majority of these had been life-long abstainers. Only two of those who confessed to drinking and smoking were admittedly heavy drinkers. There is no doubt that the majority of the drinkers at least were moderate. This is not so certain with regard to the smokers. There is therefore no obvious and direct connection between Alcohol,[6,34,37] Tobacco and Hypertension, though there may be an indirect one for we know that Alcohol at least is connected with certain metabolic disorders, e.g., Gout, and also that over-eating and drinking often go together. A writer in the British Medical Journal[10] discussing "the vaso-constriction effects of tobacco" concluded that "many consider, where familial or personal disposition, arterial degeneration is hastened by excessive smoking and where already developed tobacco may aggravate it by inducing vaso-constriction."

A survey into the occupations of patients did not reveal anything conclusive. The men followed 30 different occupations. There was one school teacher, one solicitor, three clerks, one salesman, one postmaster, and one postman. The majority of the others followed occupations involving more or less strenuous physical exertion. Mental strain, as estimated by the nature of the employment, was not a prominent feature. The psycho-physical activity of work, however, is an import-

ant factor in determining the strains and stresses of life, but the problem is not a simple one as it involves so many factors, social and economic.

Of the women patients, 46 described their occupations as domestic, which, of course, is too wide a designation from which to draw any reasonable conclusion.

Quantity and quality of food have been considered as possible factors in the production of hypertension. There is no doubt that many of the sthenic type are good eaters and consume bigger quantities of meat than are considered to be physiological. The most direct evidence of the influence of quantity and quality of food in this investigation was the therapeutic effect of restriction of food as a whole and meat in particular. In some cases blood pressures were reduced and symptoms improved. "The effect of large amounts of meat is due probably to the purin and pressor substances rather than because of the actual protein content of the food."(7)

"The national experiment during the war showed clearly that meat in moderation does not alter the arterial tension."(19)

Infection has often been blamed, but the connection is by no means direct. Of 34 patients who were able to give definite histories of illness,

13 had Measles,
9 had Influenza,
8 had Scarlet Fever,

5 had Typhoid,
4 had Pneumonia,
3 had Acute Rheumatism,
2 had Mumps,
2 had Diphtheria,
2 had Smallpox,
2 had Bronchitis,
2 had Bronchial Asthma.

Some had had more than one infection. Only in one patient who had had Influenza three times was any direct connection obvious between infection and his cardiac ailment, though it was by no means clear that the Influenza had been responsible for his hypertension. There were 57 patients who protested that they had never been unwell, but had always enjoyed good health. In comparing those who admitted to having infective diseases and those who said that they had none no distinctive clinical differences could be found. Once Essential Hypertension is established the eradication of focal sepsis does not appear to affect the blood pressure much, though of course on general principles it is wise to get rid of focal sepsis.

As to intestinal toxaemia and the absorption of the derivatives of protein digestion the evidence is conflicting. In a few cases where constipation was diagnosed no Indicanuria was found. There was one case, Mrs. Raw, whose blood pressure after a severe and intractable attack of diarrhoea lasting over a week was reduced to normal limits. This reduction may have been due to the evacuation of pressor

bodies or may have been due to the weakness resulting from the diarrhoea. The therapeutic regulation of the bowels is attended sometimes with reduced pressures and release from symptoms.

The close relationship between the ductless glands and the sympathetic nervous system has already been noted. These in turn are under the control of the vasomotor centre in the Medulla Oblongata. We have seen that Essential Hypertension runs in families and there is a theory that the hypertension is the result of an over-responsive vasomotor centre[15] which may be hereditary, or acquired, consequent upon a high pressure existence involving mental stress.[33] There is evidence to show that an over-responsive or disordered vasomotor system does exist in some people. "Dawson[15] found in 8 per cent. of 650 school children examined that the systolic blood pressure was definitely above the normal limits for their age, i.e., it exceeded 130 mm. Hg. In two classes which were working for the Oxford Senior Examination the incidence of hypertension was two and a half times as great as in the school as a whole. These observations are of great interest, for they show:- (1) that hypertension may set in at an early age; (and we have already seen that hypertension tends to run in families), (2) that predisposed subjects may respond excessively to emotional or other stimuli and it is possible that the vaso-constriction thus produced

may persist, after the exciting stimuli have been removed."[2] Also several observers[3,6] have noticed that potential hyperpietics have rather violently oscillating blood pressures, which point to disorder of the vasomotor system, for physiologists[2] tell us that under normal conditions the vasomotor centre "aims" at keeping the blood pressure within relatively narrow limits. "This notable instability of arterial pressure is more characteristic of Hyperpiesia than the actual heights of pressure recorded."[3] Women at the menopause often suffer from vasomotor and blood pressure instability, some of whom develop Essential Hypertension. Several cases in this series were related in time at least with the menopause.

One of the chief effects of stimulation of the vasomotor centre is vaso-constriction and whilst there is no uniformity of opinion in the literature[2] there are sound theoretical reasons supported by post-mortem evidence[15] for asserting that "vaso-constriction is the essential factor in the pathogenesis of Hyperpiesia." The vaso-constriction, if maintained, leads to a muscular hypertrophy of the arterioles,[6] which according to some writers is the primary pathological lesion, and which has some histological evidence.[15] The intimal hypertrophy is considered to be secondary to the induced high blood pressure or to some circulating toxin.

The reactions that follow stimulation of the vasomotor

centre normally include vasoconstriction, a rise of blood pressure and blood sugar, and secretion of Adrenaline into the blood stream, which have been grouped together and called Cannon's Emergency[33] reaction, because this is a physiological reaction induced by the emotions which seek immediate expression through some form of muscular activity and is "directed towards efficiency in physical struggle." The "physical struggle" brings the reaction to an end; it counterbalances the reaction and probably activates "the vaso-dilating complementary hormone or hormones and nerves" but concentration on this known reaction, viz., Cannon's Emergency Reaction, helps to clarify the subject under discussion. The contention is that through disordered vasomotor action, hereditary, or acquired through unfavourable environment or both, unbalanced reactions are produced which originate that series of morbid changes known as Essential Hypertension.

The vasomotor centre is influenced by many factors. Pressor and depressor impulses pass from all over the body to the centre, and by suitably selecting the type of stimulation rise and fall of blood pressure can be obtained.[2] It is possible that hypertension is produced through excess of pressor stimuli as a result of disease. Some writers have suggested a connection between fibroid disease of the uterus and increased blood pressure.[3] One case of uterine fibroid, Mrs. Hunter, No. 20, is reported here. Removal of

the fibroid by operation made no alteration in the Hyperpiesia. Other writers have noted a connection between increased blood pressure and conditions like fibrositis where the pressure has been reduced by such means as massage.[3,6]

Cerebral oxygen lack stimulates the centre "though in the human subject acute anoxaemia produces little rise of pressure and most of that is emotional in origin.... nor does anoxaemia, which develops more gradually, produce a marked rise of pressure in man."[2] Anoxaemia may result from vascular changes in the centre itself or from a cerebral tumour. Cases have been reported where the arteriolar changes in the vasomotor centre were considered to be primary[15,21] and resulted in a compensatory hypertension.

Circulating poisons or toxins may have a selective action on the vasomotor centre. The toxaemias that were present in many of the patients of the present series were of the metabolic rather than the infective type. Cases have been reported where the fall in arterial pressure has coincided with a gradual rise in the output of guanidine.[3,15]

Excess of CO_2 in the blood or a rise of H ion concentration stimulates the vasomotor centre and tends to raise the blood pressure. Acidosis is not commonly found in cases of Essential Hypertension and as already pointed out in cases

of chronic nephritis with hypertension it does not develop till late on in the disease.

The higher centres are potent stimulators of the vasomotor centre. "A rise of blood pressure commonly occurs in emotional stress and in anticipation of muscular exercise."(2) There is considerable evidence to show that the stimuli which come from the higher centres originate in or are associated with social and economic conditions, i.e., the nature of the civilisation, the soil in which people live. They are probably of a psychological rather than a physical nature and produce mental states of an emotional character. These states, it is contended, tend to produce over-responsive vasomotor systems or to further disorder of an already over-responsive system.(33,15). The evidence is partly theoretical but "the geographical and racial distribution of the disease (i.e., Essential Hypertension) affords some definite facts." A world survey, though not complete, shows that Hyperpiesia is associated with particular communities rather than with particular races. "The most outstanding fact known regarding the aetiology of Hyperpiesia is its dependence upon civilised conditions for its incidence."(33) This suggests that the stimuli responsible for over-activation of the vasomotor centre exert their influence through the mind rather than the physical body. It is a trite fact that mental strains and stresses increase and physical strains and stress diminish in the growth of civilisation. Man

has become less dependent upon his physical organism for the means of his subsistence and he has evolved curious powers of discrimination, and of choice. Instinctive guidance is no longer adequate to the occasion as under primitive conditions, and no adequate substitute, such as "reasoned conscious control," (39) has been adopted. The increase in the mental strains and stresses is shown by the increase of mental disorders and suicides in civilised communities.

Relevant to this factor of mental strain and stress is the fact, as the literature and the present series of cases show, that there is an absolute increase in the incidence of Essential Hypertension in both sexes after the age of 40, - a time of life when physical exertions are beginning to diminish and emotional and mental strains and stresses to increase, e.g., in the matter of rearing families.

Also it is noteworthy how many, in fact the majority, of the symptoms which accompany Hyperpiesia in this series can be explained through vasomotor disturbances.

Drs. Riseman and Weiss conclude a study of the symptomatology of Arterial Hypertension by saying that "the majority of symptoms are referable to disturbances of the central nervous system." "It is of interest," they continue, "that all the complaints referable to the heart and kidneys, with the exception of haematuria, may also result from vasomotor disturbances of central origin."

Cannon's emergency reaction, which is a physiological one, has been observed in animals, and in the civilised and the uncivilised, after emotional stimulation.(33) Like all other reflexes often repeated stimulation leads to a lowering of the threshold for the reaction and a heightening of the response, as facilitation occurs.

How does a physiological reaction lead to a pathological result? What distinctive feature or features distinguish the life of the civilised from the uncivilised? Some of the features are these. Firstly, the uncivilised are dominated more by nature. Their environment is comparatively stable, which conditions their reactions and allows, through long repetition of similar stimuli, of the growth of true and appropriate reactions, which become almost instinctive. On the other hand the civilised tend to dominate and to alter nature. Environment becomes more fluid and less and less stable, and consequently instinctive reactions become less and less true or appropriate to the circumstances. This, in the absence of something to replace instinct, such as, for example, "reasoned conscious control,"(39) as a guide to the constantly increasing new experiences, leads to maladjustment and malcoördinations between the individual and environment which are fruitful of emotional states which require expression. Secondly, these emotional states are not temporary but are constantly recurring, a fact which conditions a lowering of the threshold of stimulation of the vasomotor system, through

which the emotions discharge themselves, and a heightening of reaction, such as Cannon's Emergency Reaction. Thirdly, in the civilised communities of to-day with the diminution in the physical struggle for existence, which provided the essential muscular activity for the discharge of emotional reactions, and at the same time with the increase of mental states, especially of an emotional character, there is over-stimulation of the vasomotor centre without the necessary and appropriate muscular activity.

Experiments have been made which show that the rise in blood pressure and blood sugar induced by an injection of Adrenaline, which produces a similar reaction to emotional stimulation, returns to normal quicker after muscular activity than after rest.(33) Further, the therapeutic effect of release from mental and physical strain, with fall in blood pressures, often observed in the course of this study, illustrates the effect of environment, and emotional reactions thereto, on blood pressure.

Certain forms of mental disease are accompanied by hypertension. Three cases of Psychoneurosis were noted in this series.

Chart of the Three Cases of Psychoneurosis.

Name.	Age.	Pulse.	Blood Pressure.
McDonald, Eliz.	55.	80	200/100
Stafford, Hilda.	46.	106	170/95
Layton, Margaret.	70.	60	200/100

All three cases have been in mental institutions. Two have been discharged but still have periods of mental instability/ and hypertension. In a Psychoneurosis the emotions of past events which are forgotten are associated with present stimuli and as a consequence are not discharged through the appropriate activity. "Barton Hall investigated the blood pressures in various forms of psychoneurosis and demonstrated that in neurasthenia and psychasthenia blood pressure is abnormally low, whilst in cases of prolonged mental strain and anxiety neurosis it may be abnormally high, falling with treatment of the neurosis."(33) Dr. Donnison in review of 117 cases of Hyperpiesia says "a perusal of the histories reveals the existence of symptoms suggestive of past neurosis in 65 per cent."(33)

In this series a perusal of the histories reveals the existence of symptoms suggestive of past neurosis in about 30 per cent. This comparatively low figure is partly due to the fact that many of the cases are elderly and their increased blood pressures have been established for many years.

In conclusion "the relationship between psychoneurosis and arterial hypertension is probably closer than is recognised at present. Psychic trauma and conflict, abnormal sensitivity of the psyche as well as the constant strain of life, play most important rôles in the development of psychoneurosis. These factors probably are often responsible for

the development of hypertension and therefore the elimination of the psychic conditions plays a primary rôle in the prevention and treatment of hypertension."(14)

On the other hand Dr. Treadgold concluded after a study of "Blood Pressure in the healthy young male adult" in the Royal Air Force that "the psychoneuroses seem to play little part in the causation of raised blood pressure in healthy young adults." The youthfulness of the latter, however, and the environmental conditions, which in times of peace are comparatively stable, secure, and routine, would tend to diminish the emotional strains and stresses and also permit of suitable "physical struggle."

We may now summarise our discussion of the aetiology of hypertension or hyperpiesis.

(1) There is a definite type of hypertension associated with some forms of renal disease - Renal Hypertension.

(2) After 40 years of age Hyperpiesia or Essential Hypertension is more common than Renal Hypertension. The theories of the aetiology that command most general support centre around (a) the toxaemic theory, and (b) the mental strain and stress theory in combination with an over-responsive vasomotor system. Heredity is a prominent feature of both theories.

Infections probably are not primary causes in the

majority of cases, nor Smoking nor Alcohol nor occupation.

The toxaemias concerned result from disordered metabolism and tend to develop at times of physiological stress, e.g., the involutionary changes such as the menopause.

The two theories are not necessarily in opposition and in time may be synthesised.

The multiplicity of phenomena associated with Hyperpiesia suggest disordered function and relationships between inborn psycho-physical tendencies and environment which lead to wrong or exaggerated reactions or as someone has said to "physiology gone mad."

Pathology.

It is generally agreed that High Blood Pressure is a reaction to an increased peripheral resistance brought about by a contraction of the arterioles and possibly the capillaries. This condition causes an hypertrophy of the arteriolar media which in time is followed by degeneration and fibrosis. As a result of long-continued increased blood pressure in conjunction with chemical or toxic disorders, characteristic changes appear in the intima of the arterioles such as hypertrophy and fibrosis accompanied by a hypertrophy of the left ventricle. The changes in the arterioles are not universal but appear chiefly in the kidneys, spleen, liver, pancreas and brain. The changes are most characteristic and constant in the kidneys.

There is no "obligate connection" between Arterio-sclerosis of the larger vessels and High Blood Pressure. Arterio-sclerotic and arteriolo-sclerotic lesions are to be distinguished because of their distinct pathological significance.(6)

As already stated, the characteristic Arteriolo-sclerosis occurs most frequently in the kidneys. In the smaller arterioles a subendothelial hyaline degeneration takes place with secondary fatty changes and narrowing of the lumen.

In the larger arterioles a hyperplasia takes place in the internal elastic membrane which undergoes regressive changes, marked by fatty and hyaline substances and reactive proliferation of connective tissue cells resulting in collagenous connective tissue in the intima.

The arteriolo-sclerosis leads to glomerular and tubular atrophy. The kidney contracts and becomes granular. In chronic glomerular nephritis the primary lesions are in the glomeruli which leads to increased blood pressure, which in turn gives rise to an Arteriolo-sclerosis with a distribution similar to that of Essential Hypertension.

The cardiac left ventricular hypertrophy is not due to a hyperplasia but to increased thickness of the muscles, cells and all its elements. In time the hypertrophy may be succeeded by dilatation with interstitial fibrosis and degenerative changes in the muscle cells.

Symptomatology.

The relationship between raised blood pressure and symptoms has not been investigated very extensively. Many sufferers in the beginning probably have no symptoms. In this series there were nine cases who had no symptoms. They all looked and felt well and able to do their work. Their diastolic pressures were 100 or under except two, one of which was 105 and the other 130. The latter/was much overweight. Their systolic pressures ranged from 150 to 220.

There were seventeen cases with one symptom. Amongst these there were a few who could not do as they used to. There was more disability in this group. The pressures showed similar variations to those of the latter class - the diastolic pressures varied from 120 to 90, and the systolic pressures from 220 to 160. Their symptoms centred largely around the cardio-vascular and vasomotor systems.

The remainder had more than one symptom each. This is the largest group, and disability was definitely greater than in the other groups. The pressures were on the whole a little higher especially the diastolic pressures - the diastolic pressures varied from 80 to 150 and the systolics from 170 to 290. The symptoms centred largely around the cerebral, cardiac, peripheral vascular and gastro-intestinal systems.

These three groups correspond roughly to the development of definite organic changes though there are cases with

many symptoms which are functional and in which organic changes are few, e.g., the menopausal cases. The quantitative difference of the blood pressures of the groups is not striking, and no exact relationship was observed between the number, discomfort, and severity of the symptoms and the blood pressures. The symptoms are inconstant and diverse. There are cases, with many symptoms, who drag on a weary existence for years whilst others die suddenly and with little or no warning. One patient, William Young, until a few weeks of his death was going about seemingly well, then symptoms of nocturnal dyspnoea quickly developed with slight substernal oppression and he died suddenly one night.

Chart of symptoms of patients in this series with frequency expressed as percentages.

Dyspnoea	34%	Paresis (Sensory & Motor)	7%
Giddiness	29%	Hot Flushes	4%
Dyspepsia	28%	Bad Memory, Insomnia, Defective hearing	4%
Tender Gall-bladder, Tiredness	14%		
Precordial distress, Nocturia	14%	Tinnitus, Palpitation, Arthritis, Encephalopathy, Mental, Constipation	3%
Aches and pains	13%		
Headaches, Nervousness, Nose bleeding, Diarrhoea and vomiting, Oedema	10%	Paraplegias	2%
		Polycythaemia	1%
		Urinary retention	1%

How long it takes for symptoms to develop is not known. Some writers reckon that it takes roughly ten years. (6)

From the chart it will be seen that the symptoms centre around the cerebral, cardiac, peripheral vascular, gastric and renal systems.

The cerebral symptoms, which include the majority, consist of Giddiness, Nocturia, Headaches, Nervousness, Paresis, (Motor and Sensory), Hot Flushes, Failing Memory, Insomnia, Tinnitus, Palpitation, Mental Instability and Convulsive phenomena.

Around the heart centre such symptoms as Dyspnoea, Dyspepsia, Precordial Distress, Nocturia, Oedema and Palpitation.

Around the gastro-intestinal system centre Dyspepsia, Diarrhoea and Vomiting.

The peripheral lesions give rise to Nose-bleedings, Cramps, Bowel Bleeding, Aches and Pains, and Weakness.

The kidneys may give rise to Nocturia, Oedema, and Haematuria - though none of the patients in this series had Haematuria.

Some of these symptoms can be explained through more than one system and it is striking the large number that can be attributed to the nervous system.

There are vague symptoms of weariness and distress amongst hyperpietics which are difficult to classify. A full-blown symptom is a gross manifestation and between it

and no symptoms are many gradations.

The symptoms are inconstant and diverse and are found in conditions without increased blood pressure, e.g., the menopause with normal blood pressure, obesity, and psychoneurosis.(14)

Dyspnoea on exertion was found to be the most common symptom and due in many cases to crippled hearts. In one patient, Harker Slack (No. 46) Dyspnoea with vague pains around the left nipple was the most prominent symptom. It was considered to be cardiac in origin. Electrocardiograms, however, revealed no cardiac lesion. Nor was there any other physical condition to explain his condition which probably was of a nervous nature, a neurosis.

Dyspepsia occupies a high place and probably had to do in many cases with the vigour and lusty appetites of the patients.

Precordial distress is a mixed symptom and includes the anginas, and also vague discomforts around the sternum and nipple. No doubt coronary disease - from fleeting angiospasm to coronary thrombosis - is responsible for some of these. This symptom may be vasomotor in origin.

Nocturia in three cases was diabetic, and in the majority of the others probably cardiac and in some of nervous origin.

Aches and pains were referred to different parts of the body, chiefly the lower limbs and back.

Headaches were present only in 10 per cent. It was not constant all day: often it was complained of first thing in the morning; sometimes occipital, sometimes frontal.

Nose-bleeding was a disturbing symptom and invariably brought the patient to the doctor, but was never serious.

Oedema of the feet was never marked except as an end result. Two cases reported swelling of the face suggestive of oedema of the face.

Bouts of Diarrhoea and Vomiting, either singly or together, were annoying, but did not happen frequently. The Vomiting sometimes followed on headaches and was suggestive of Migraine. The Diarrhoea was more intractable.

Nine cases had some form of motor and sensory paresis. One case had attacks of temporary paresis probably due to angiospasm before the onset of more permanent symptoms. "Focal angiospasm is a well-authenticated process in the complications of High Blood Pressure."(27)

Hot Flushes, Insomnia, Tinnitus and Palpitation were found more amongst the women and were propably of vasomotor origin.

Failing Memory was noted in the more elderly.

Arthritis centred around the knees - mostly in one at a time and accompanied by gross creaking and swelling.

The Mental Symptoms were of the anxiety neurotic type.

Polycythaemia was present in the one case of Anterior

Pituitary Basophil Adenoma.

There was only one case of urinary retention, which was not complete. The patient was aged 85.

One patient, when questioned regarding her symptoms, said that she could stand cold better than heat, whilst one complained that she could not stand a stuffy room.

Giddiness consisted chiefly of light-headedness, though in one case at least, with Menière's Syndrome, it was a true vertigo. This symptom often brings the patient to the doctor. It is often noticed when changing position, e.g., getting up in the morning.

In conclusion we can say that the symptoms are not specific as they are varied and are found where hypertension does not exist.

Convulsive phenomena were observed in four cases - two had generalised convulsions followed by unconsciousness and then complete recovery. No blood pressure estimations were made during the attacks, but the feel of the pulses was suggestive of increased blood pressure. In none of the four did the clinical picture point to changes in the blood chemistry associated with Uraemia.

Significance and Prognosis.

"The clinical significance of High Blood Pressure is obviously that it is the potential antecedent of cardio-vascular and renal complications."(21) The hypertensive

degenerative processes are diffuse and progressive. Arteriolosclerosis leads to increased peripheral resistance, which leads to an impoverished capillary circulation, which leads to degeneration of the finer type of cells and to replacement fibrosis. The brunt of the pressure is borne by the brain, heart and kidneys. A patient with hypertension is necessarily restricted physically and mentally. It has been observed that disaster visits those, who disregard its significance, sooner than those who do not.(22) The extent of the restriction is determined by the height and the range of the pressures, the symptoms, and by hereditary and constitutional factors. At optimum pressures there may be no symptoms and consequently production of symptoms is significant. Prognosis is based partly on the height of the pressures, plus a proper assessment of the symptoms and the underlying pathology. The following chart shows the effect on mortality of high arterial pressures. The chart was published by the North Western Life Insurance Company of America.(3)

Accepted or Rejected.	Average Systolic Blood Pressure.	Approximate Extra Mortality.
Accepted	141 mm.	10%
do.	146 "	35%
do.	153 "	60%
Rejected	160 "	110%
do.	170 "	165%

Whilst a study of the symptoms is important some cases die suddenly and almost symptomless. The presence or absence of Arterio-sclerosis, the condition of the Cardio-vascular system, as shown by its response to effort, by the size of the heart, the nature of the pulse, the presence of irregularities such as Pulsus Alternans and Gallop Rhythm and the existence of oedema are important. The kidney functions must also be investigated. The reaction to treatment such as rest and dietetic and medicinal is of prognostic value. The personal and family medical history and the condition of the ocular fundi and the age should all be considered. The prognosis is worse in patients before 40 than after 40, and is also worse for men than women. The presence of Neuro-retinitis alone, or especially in conjunction with Haematuria, is ominous, and indicates Malignant Hypertension.

In this series the majority of the cases showed complications in the cardio-vascular system as shown by irregular pulse and enlarged hearts, but apart from arterio-sclerosis nothing more, except in two cases, was noted in the ocular fundi. Little of positive prognostic value was obtained from ocular examinations.

In conclusion we may fairly say that we know of no prognosis in cases of Essential Hypertension apart from its complications.

End-Results.

"Persistently elevated arterial pressure with associated morbid changes of the vascular tree is probably responsible directly or indirectly for more disability and death than any other single pathological condition including Cancer and Tuberculosis."(1)

It has been estimated that it takes ten years for symptoms to develop on the whole in cases of Essential Hypertension and that on an average the duration of life after the appearance of symptoms is ten years.(6)

Blackford, Bower and Baker observed 202 patients with systolic pressures over 175 from 5 to 11½ years. At the end of that time they found 50 per cent. dead - 70 per cent. males to 39 per cent. females. The causes of death were tabulated in order of frequency as follows:- (1) Heart, (2) Cerebral Vascular accidents, particularly haemorrhage, (3) Intercurrent infections, e.g., Broncho-pneumonia, (4) Kidney.(3,6).

Christian found the causes of death in 131 patients with High Blood Pressure in hospital as follows:-(6)

32%	Cardiac	25%	Cerebral.
25%	Intercurrent	4.5%	Uraemic.

In this series eight patients have died since study began two years ago.

Chart of Patients who have died, with causes of death.

Collinson	(M)	-	Cerebral Haemorrhage.
Jones	(F)	-	Congestive Heart Failure.
Young	(M)	-	Coronary Occlusion.
Hall	(M)	-	Congestive Heart Failure and Uraemia.
Tallentire	(F)	-	Cerebral Haemorrhage.
Jackson	(F)	-	Uraemia and Congestive Heart Failure.
Brown	(M)	-	Congestive Heart Failure.
Lodge	(M)	-	Coronary Thrombosis.

There are five men and three women.

Six died with obvious heart affections. Four had congestive heart failure, and two coronary artery disease. The remaining two died with cerebral haemorrhage. None had valvular disease.

M. A. Cassidy, M.D., in the "British Medical Journal," January 13, 1934, in an article entitled "Aetiological Classification of Cardiac Disease in consulting cardiac practice" gave these figures of incidence:-

(1) Valvular Disease - 5.6%
(2) Hypertensive " - 31.3%
(3) Decrescent " - 31.6%

In all the patients who have died, arterio-sclerosis was well marked in the radial and retinal arteries.

Amongst the congestive cardiac cases three towards the end developed uraemic symptoms, probably as a result of pre-existing nephrosclerosis with possible compensated renal deficiency.

Four of those living suffer now from attacks of Congestive Heart Failure. All have irregular pulses and one, Mrs Smith, shows retinal macular starring suggestive of renal deficiency. It has been observed that patients with marked cardiac enlargement and symptoms of heart failure do not develop cerebral vascular lesions. Certainly in this series the six cardiac cases who died and the four living had no motor or sensory paresis. They all had Arterio-sclerosis. The two cerebral cases who died had several attacks of angiospasm, thrombosis, and haemorrhage, at different times, spread over several years with consequent temporary and permanent sensory and motor paresis, and in one case latterly complete hemiplegia developed.

No quantitative relationship was found between the height of blood pressures and cerebral haemorrhage. H. O. Gunewardene, M.B., in the "British Medical Journal," "concluded that cerebral haemorrhage does not occur with a diastolic pressure below 115 (whatever may be the systolic) and that prognosis may be based on this observation."(22) In this series there were no massive cerebral haemorrhages. Seven cases showed signs of cerebral vascular lesions with

temporary or permanent sensory and motor paresis, and of these only one had a diastolic above 115, viz. 290/130. Of course blood pressures may rise suddenly, e.g., in dreams, and cause a haemorrhage.

In conclusion we may say that the fate of the majority of people with Essential Hypertension is death with the essential lesion in the heart, brain or kidneys.

Treatment.

The treatment of High Blood Pressure is that of the cause, but as the latter is so obscure in the majority of cases treatment is unsatisfactory. It is largely palliative and directed towards the complications. The determination of the cause or causes even if theoretical is important. Most cases can be grouped as renal or non-renal which determines treatment to a certain extent. This done, a general survey should be made of the patient's life, both mental and physical. Sometimes it is necessary to put the patient to bed to make this survey and also to test the patient's reaction to rest and quiet. Too vigorous efforts towards reduction of blood pressure should not be made, for sometimes the increased pressure is compensatory and necessary, e.g., for efficient coronary circulation or kidney excretion. Also too vigorous and too fussy a treatment has a bad effect on the patient's mentality.

Work should not be forbidden on general grounds but only for some specific reason. If pleasurable and within physical and mental capacity work is useful. A cheerful and congenial environment should be encouraged and psychic conflicts should be eliminated.

Congenial and non-competitive exercise is beneficial. Baths should be taken regularly at body temperature. A good plan is for the patient to recline in the bath at body temperature for 10 or 15 minutes daily at a convenient time. There are special baths obtainable at spas such as immersion baths, massage douches, in addition to electrical procedures such as diathermy which all help to enlarge the peripheral vascular bed temporarily at least. Some authorities claim that the results, if the baths and electrical procedures are persevered with, are cumulative.(31) In any case even if a permanent lowering is not achieved a temporary lowering helps to ease the strain and is beneficial. Spa treatment provides not only special procedures but for many people a congenial and pleasant atmosphere. Life is regulated.

Where weight is excessive, its reduction should be attempted. This involves, where possible, exercise and also attention to diet and the emunctories. In renal cases the diet will be regulated by the state of the blood chemistry and urine and may mean rigid restriction of proteins. In non-renal cases on the whole no complicated dietaries are

necessary. A patient's optimum weight should be estimated and diet arranged accordingly. This invariably means cutting down the diet as a whole and maintaining a reasonable balance of proteids, fats and carbohydrates and vitamins. Where there is obvious arterio-sclerosis fats should be diminished and liver, brain, egg and kidney excluded.

Salt-free diets are unnecessary unless oedema is present, though some restriction of salt may be wise.

We have little knowledge of the pressor bases but they are believed to arise in the bowel. This reinforces the need for regulation of the bowels. Sometimes it may be necessary to reduce protein intake and to adopt some form of bowel lavage, e.g., Plombière's.

Venesection and Lumbar Puncture are useful in Congestive Heart Failure and Hypertensive Encephalopathy.

Drug treatment is necessary but empirical and symptomatic.

The nitrites are commonly used in times of crisis where temporary relief is essential, e.g., in attacks of angina or arterial spasm anywhere. For quick action Amyl Nitrite, Nitroglycerine, and Sod. Nitrite are used. For more prolonged action Erythrol Tetranitrate, and Bismuth Subnitrate are used. The effect is more or less temporary, but nevertheless beneficial.

Mercury, Salines, Iodine, Iodides, Acetylcholine,

Sulphocyanates, Thiocyanates, Ergotamine Tartrate, in nervous cases particularly Bromides, Luminal, and Chloral, all have their uses though in some the division between therapeutic and toxic dose is small. Water-melon seeds 50 mgms. in capsules have been advocated.(19) It is claimed that their action is lasting, with no toxic effects and are easy of administration. Recently Vagotonine has been isolated from the pancreas.(21) In uncomplicated hypertension tonus of the sympathetic is increased and tonus of the vagus and parasympathetic is diminished. Also sometimes there is a low sugar tolerance. Vagotonine increases reflex excitability of the vagus and parasympathetic, has a hypoglycaemic effect, and diminishes blood pressure. It is claimed that its action is more gradual and more lasting than that of Acetylcholine.

In this series of cases no special drugs were used. Bromides, Luminal, and Chloral, Mercury and Salines and Nitrites were used where indicated in conjunction with wise advice and regulation of life, mental and physical. Suitable exercise and fresh air and congenial environment and regular and proper bathing and attention to all the things that go to make life were advised. Any obvious foci of infection were eradicated on general principles though no dramatic fall of blood pressures was observed.

Complications as they arise demand special procedures.

Many cases were benefited and some pressures were reduced but the results were so varied and uncertain that they could not be tabulated.

SUMMARY AND CONCLUSIONS.

(1) The great majority of patients with increased blood pressure can be grouped under (a) Renal Hypertension, (b) Primary or Essential Hypertension.

(2) The concept of Primary Arterial or Essential Vascular Hypertension has a sound basis in fact.

(3) The great majority of the cases of this series were cases of Essential Hypertension.

(4) Essential Hypertension means a permanently raised Diastolic and Systolic Blood Pressure not due to primary kidney disease, and is much more common than Renal Hypertension in people over 40 years of age.

(5) The incidence of Essential Hypertension increases amongst women in the fifth and sixth decades and amongst men in the sixth and seventh decades.

(6) Vaso-constriction is the primary fact in the pathogenesis of increased blood pressure, and is followed by hypertrophy of the media and intima of the arterioles, ~~and is~~ found characteristically in the kidneys, and by an hypertrophy of the left ventricle. Signs of degeneration and fibrosis appear sooner or later.

(7) The aetiology of Renal Hypertension (as the name indicates) is related to kidney disease.

The aetiology of Essential Hypertension is more obscure. Such factors as heredity and environment, body-build, habits of eating and drinking, involutionary changes such as the menopause, disordered metabolism, psychic trauma and conflict, all play a part.

(8) Tobacco and Alcohol are not direct causes.

(9) The part played by infection is not clear. Its influence is probably secondary and not primary.

(10) Essential Hypertension has no specific symptoms. The symptoms, if any, which accompany this condition are probably due to the cause or to the complications. The majority of the symptoms are referable to the central nervous system.

(11) Once established cure is rare. There is no specific treatment. Treatment consists in the wise regulation of life in general, Diets need not be complicated but should be suited to needs. Procedures, e.g., baths, psychotherapy, exercises, alteration of environment, which release angiospasm and increase the area of peripheral circulation, should be adopted as indicated by events. Complications must be treated on their

merits. Drugs may be helpful, but should be used with reference to specific circumstances.

(12) The significance of Essential Hypertension is, that it is the precursor of cardio-vascular degeneration.

(13) Its prognosis, worse for men than for women, is that of its complications.

(14) The chief causes of death are cardiac, cerebral, intercurrent and renal disease and in that order of frequency.

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Brief Summary of Cases.

	Name.	Age.	Pulse.	Blood Pressure.	Diagnosis or Reason for Consultations.
1.	Walker, Mrs.	43	96	160/105	Menopause.
2.	Tallentire, Mrs.	71	68 Hard arteries.	280/110	Cerebral vascular lesions.
3.	Robson, Mrs. J.	56	64	180/110	Cholecystitis.
4.	Brown, Mrs. L.	62	120 Full and hard.	250/110	Myocardial degeneration.
5.	Barker, Miss.	74	55	200/80	Influenza.
6.	Stewart, Mrs.	40	96	190/110	Menopause.
7.	Fraser, Mrs.	69	80 Arteries palpable.	210/110	High blood pressure. Simple.
8.	Blackburn, Mrs.	75	100 Palpable arteries. Extra systoles.	170/90	Acute vascular cerebral attacks.
9.	Jones, Mary Ann.	64	70	190/100	Coronary disease.
10.	Hodgkinson, Miss.	48	80 Full and hard.	180/90	Menopause.
11.	Carter, Mrs. G.	54	90 Extra systoles. Small pulse.	220/100	Myocardial degeneration.

	Name.	Age.	Pulse.	Blood Pressure.	Diagnosis or Reason for Consultations.
12.	Langstaff, Mrs.	43	74 Full.	170/90	Menopause.
13.	Waine, Mrs.	60	66	220/90	Cystitis.
14.	Watson, Mrs.	54	60	200/100	Menopause.
15.	Allison, Mrs.	54	80	150/100	Acute tonsillitis.
16.	Barnes, Mrs.	53	100 Extra systoles.	160/90	Menopause.
17.	Jones, Mrs.	54	80	170/110	Arthritis of knees.
18.	Lowes, Mrs.	59	94	200/80	Malignant uterus.
19.	Walker, Mrs.	85	80 Extra systoles.	170/90	Acute cerebral attacks.
20.	Hunter, Mrs.	52	80	180/105	Uterine fibroid.
21.	Waine, Mrs.	67	80	190/100	Diabetes.
22.	Dent, Mrs.	56	100	210/120	Simple high blood pressure.
23.	Wrathall, Mrs.	74	80 Hard arteries. Irregular pulse.	180/105	Insurance Examination.
24.	Collinson, Mrs.	72	80	170/90	Giddiness.
25.	Appleby, Mrs.	72	80	180/100	Accident. Essential hypertension.

	Name.	Age.	Pulse.	Blood Pressure.	Diagnosis or Reason for Consultations.
26.	Brewer, Mrs.	54	80	200/110	Essential hypertension with cardiac hypertrophy.
27.	Dobson, Mrs.	61	64	220/120	Cardio-renal sclerosis.
28.	Jones, Mrs.	76	120 Irregular pulse.	190/100	Congestive heart failure.
29.	Peart, Mrs.	89	80 Tortuous arteries.	180/100	Cerebral arterio-sclerosis.
30.	Walker, Mrs.	64	80	170/100	Simple high blood pressure.
31.	Jackson, Mrs.	75	90	180/90	High blood pressure with congestive heart failure.
32.	Bell, Mrs.	79	72	150/100	High blood pressure with cerebral arterio-sclerosis.
33.	Tarn, Mrs.	51	80	250/150	Menopause.
34.	Gibson, Mrs.	50	70 Full and hard. Regular.	185/100	Menopause.
35.	Garbutt, Miss.	64	84 Palpable arteries. Irregular pulse.	190/90	Cardio-vascular degeneration.
36.	Elleray, Mrs.	56	60 Extra systoles.	250/120	High blood pressure.
37.	Duthie, Mrs.	65	70 Regular.	160/90	Cerebral vascular lesions. "Parkinsonism."

	Name.	Age.	Pulse.	Blood Pressure.	Diagnosis or Reason for Consultations.
38.	Down, Mrs.	74	80 Small pulse.	210/110	Chronic bronchitis.
39.	Dean, Mrs.	62	80	170/110	Emphysema.
40.	Peacock, Mrs.	66	70	200/100	Simple high blood pressure.
41.	Parkinson, Mrs.	54	60	210/90	Prolapse uteri.
42.	McDonald, Eliz.	55	80	200/100	Psycho-neurosis.
43.	Kyle, Miss.	68	70	170/100	Giddiness.
44.	Raw, Mrs.	65	90	180/100	Substernal oppression.
45.	Willcock, Mrs.	67	80	190/95	Cardiac oedema.
46.	Smith, Mrs.	75	80 Thin irregular pulse.	260/130	Cardio-renal disease.
47.	Glaholme, Mrs.	70	60 Palpable arteries.	170/70	Raised systolic blood pressure, and arterio-sclerosis.
48.	Bell, Mrs.	76	80 Irregular.	200/100	Cardio-renal disease.
49.	Copping, Miss.	51	90 Regular and thin.	150/110	Bronchial asthma.
50.	Pedelty, Mrs.	63	70 Full and sustained.	200/105	Cardio-renal disease.
51.	Layton, Margt.	70	60 Hard arteries.	200/100	Anxiety neurosis. Cerebral arterio-sclerosis.

	Name.	Age.	Pulse.	Blood Pressure.	Diagnosis or Reason for Consultations.
52.	Colling, Miss.	73	80 Hard and tortuous arteries.	185/90	Menière's Syndrome.
53.	Stafford, Hilda.	46	106	170/95	Psycho-neurosis.
54.	Hillary, Mrs.	73	120 Hard and tortuous arteries.	220/100	Cardio-vascular degeneration.
55.	McDonald, Mrs.	70	90	180/80	Emphysema and asthma.
56.	Ward, Mrs.	59	80	200/120	Menière's syndrome.
57.	Keenan, Mrs. Cath.	42		180/80	Subacute nephritis.
58.	Daglish, Jessie.	26		175/110	Cushing's syndrome. Basophil adenoma of anterior pituitary.

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	Name.	Age.	Pulse.	Blood Pressure.	Diagnosis or Reason for Consultations.
1.	Waine, Watson.	66	60 Extra systoles.	160/100	Myocardial Degeneration.
2.	Tallentire, Wm.	76	62 Palpable arteries. Extra systoles.	205/100	Cerebral arterio-sclerosis.
3.	Robinson, Thomas.	84	60 Hard and tortuous arteries.	200/100	Congestive heart failure.
4.	Cummings, W. G.	66	90	190/110	Influenza.
5.	Collinson, G. W.	65	76	180/100	Congestive heart failure.
6.	Clarkson, Abrams.	67	76 Hard and tortuous arteries.	205/100	Arterio-sclerosis.
7.	Brown, Henry.	71	80 Auricular fibrillation.	210/130	Congestive heart failure.
8.	Brown, L.	60	64 Regular. Palpable.	180/110	Nose-bleeding.
9.	Evans, A.	51	80	160/100	Acute vascular cerebral attack.
10.	Raw, Thomas.	71	60 Hard and tortuous arteries.	180/80	High blood pressure and arterio-sclerosis.

	Name.	Age.	Pulse.	Blood Pressure.	Diagnosis or Reason for Consultations.
11.	Lynn, J.	86	90 Hard and tortuous arteries.	220/110	High blood pressure. Arterio-sclerosis.
12.	Hall, William.	73	90 Auricular fibrillation. Hard and tortuous arteries.	170/110	Congestive heart failure.
13.	Young, William.	66	90 Hard and tortuous arteries.	170/90	Coronary Disease.
14.	Gargett, R.	76	64	170/80	Giddiness.
15.	Bainbridge, J.	62	80	170/90	Alveolar abscess.
16.	Miller, J. W.	54	70	170/100	Simple high blood pressure. Health Insurance Exam.
17.	Bell, William.	47	70	200/120	R. cerebral thrombosis.
18.	Hodgson, W.	51	80	220/130	Health Insurance Exam.
19.	Turton, S.	47	80	170/105	Varicose veins.
20.	Coates, William.	56	100	160/110	Synovitis of knees and arthritis.
21.	Collinson, J. W.	71	80 Hard and tortuous arteries.	210/90	Cerebral thrombosis and softening.
22.	Wigham, Joseph.	63	80 Hard arteries.	200/100	Biliary colic.

	Name.	Age.	Pulse.	Blood Pressure.	Diagnosis or Reason for Consultations.
23.	Laidler, Thomas.	80	70 Palpable arteries.	160/80	Cerebral arterio-sclerosis.
24.	Bell, J.	77	64 Hard and tortuous arteries.	200/105	Cerebral arterio-sclerosis.
25.	Dawson, C. R.	57	70 Irregular pulse. Hard arteries.	160/100	Cerebral motor and sensory vascular lesions.
26.	Willcock, H.	70	80 Palpable arteries.	180/100	Dyspepsia with flatulence.
27.	Wigham, J.	86	80 Hard, tortuous and nodular arteries.	200/110	Congestive heart failure.
28.	Thurlbeck, J.	67	80 Palpable radials.	170/100	Flatulent dyspepsia.
29.	Atkinson, A.	47	80 Sclerosed arteries.	180/100	Basal meningitis and asthma.
30.	Longstaff, R.	69	80 Auricular fibrillation. Sclerosed arteries.	200/130	Congestive heart failure.
31.	Watson, Col.	70	70	160/100	High blood pressure.
32.	Hull, J.	77	60 Palpable arteries.	200/100	Bronchitis.
33.	Bailey, J.	62	70	200/100	Nose-bleeding.

	Name.	Age.	Pulse.	Blood Pressure.	Diagnosis or Reason for Consultations.
34.	Brayfield, R.	64	90 Extra systoles.	190/100	Emphysema.
35.	Spenceley, T.	62	70	200/110	Biliary colic and jaundice.
36.	Gifford, F.	64	60 Hard and tortuous arteries.	290/130	Cerebral thrombosis.
37.	Nixon, J.	68	80 Tortuous arteries. Irregular pulse.	190/110	Cerebral arteries sclerosed.
38.	Morton, J.	63	80 Palpable arteries.	180/110	Cerebral arteries sclerosed.
39.	McDonald, Jas.	58	120	180/105	Diabetes and cerebral thrombosis.
40.	Jackson, Newby.	80	80	200/80	Alcoholism.
41.	Oliver, Henry.	66	66 Hard arteries.	170/105	Coronary disease.
42.	Dean, R.	69	100 Palpable tortuous arteries.	240/110	Diabetes and arterio-sclerosis.
43.	Hind, Frank.	65	90 Hard nodular arteries.	170/110	Diarrhoea.
44.	Jennings, G. T.	62	80 Sclerosed arteries.	210/120	Cerebral arterio-sclerosis.

	Name.	Age.	Pulse.	Blood Pressure.	Diagnosis or Reason for Consultations.
45.	Davison, William.	71	66 Hard and tortuous arteries.	190/110	Cardio-renal sclerosis.
46.	Slack, Harker.	53	60 Irregular pulse.	160/100	Coronary sclerosis.
47.	Scott, Fred.	57	80	180/100	Arthritis of knees.
48.	Bonsfield, C. L.	40	100	200/60	Rheumatic. Valvular heart disease.
49.	Lodge, James.	78	90 Hard nodular arteries.	170/90	Coronary thrombosis.
