THE TREATMENT OF HIGH BLOOD PRESSURE IN GENERAL PRACTICE.

THESIS

Submitted for the

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by

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Part 1. SECTION A.

Introduction.

Arterial hypertension may be defined as a state of the circulation in which intravascular blood pressure rises above normal limits. (1) To render this definition intelligible one must naturally define the normal limits. Fortunately ample figures for this purpose are available from the statistics of life insurance medicine.

Hunter's (2) compilation of the blood pressures in nearly a quarter of a million healthy persons from ten years to sixty years inclusive sets an authoritative standard for the average variations with age. From an average systolic and diastolic blood pressure of 120/80 at the age of twenty years, the level increases to 135/89 at the age of sixty years. There is a rise in each half decade of approximately 2 mm. of mercury in the systolic pressure and of 1 mm. in the diastolic and pulse pressures. Within these narrow bounds are the physiologic limits of blood pressure.

The Average Variations of Blood Pressure (after Hunter's Compilation of observations on a quarter million healthy Americans).

Age.	Systolic P.	Diastolic P.	Pulse P.
10.	103	70	33
15	113	75	38
20	120	80	40
25	122	81	41
30	123	82	41
35	124	83	41
40	126	84	42
45	128	85	43
50	130	86	44
55	132	87	45
60	135	89	46

In Fisher's (3) series of 4,165 cases of hypertension without other serious involvements, the extra mortality was 36.1 per cent. when the blood pressure was 10 - 14 mm. of mercury over the standard. The added mortality became 83.8 per cent. when the systolic pressure exceeded the average for age by 15 - 24 mm., and rose to 314.7 per cent. above the expected mortality when the pressure was 50 mm. above the standard.

Rogers and Hunter (4) found in a series of 2,838 hypertensive persons, an extra mortality of 46 per cent. when the pressure was 10 - 25 mm. above the standard, rising to 148 per cent. when the elevation was 36 - 50 mm. When obesity, mitral insufficiency and albuminuria were added to the high pressure the extra mortality became 211, 220 and 290 per cent. respectively.

These are most impressive figures. They serve to fix a normal standard, varying within comparatively narrow limits, showing that the cardiovascular system attaches the greatest importance (if one may so express it) to the maintenance of blood pressure at a physiological normal. In practical clinical work a blood pressure of 150/90 may be taken as the upper limit of normal for any age. Borderline patients are, of course, only to be considered hypertensive after repeated observations. It is a matter of common clinical knowledge, (5), (6), (7), (8), (9), (9a) that blood pressure varies to a remarkable degree under conditions of excitement or apprehension, and a first observation, even in an apparently calm patient, is liable to be very much higher than that subsequently recorded.

Should this cardiovascular regulating mechanism fail to maintain a normal standard of blood pressure, what are the consequences?

Cabot (10) on the basis of 4,000 consecutive necropsies in the Massachussetts General Hospital concludes that the commonest of all types of heart disease with decompensation is hypertensive heart

disease - in fact that it is commoner than all the other types put together, and comes to the rather startling conclusion that 77 per cent. of all heart disease is due to simple hypertrophy and dilatation of the heart (or hypertensive cardio-vascular disease) without valve lesions. He further states that this overwhelming excess of hypertensive cases is entirely in accord with clinical experience.

From the insidious nature of this malady it is evident that most of its victims rarely become hospital cases except when struck down in the final stages with the "defeated heart" of Allbutt, or on the catastrophic advent of an "apoplectic stroke".

Hypertension would appear, then, to be a malady "par excellence" for observation by the general practitioner. By a series of consecutive blood pressure estimations he can gauge fairly accurately the incidence of hypertension in the general population of his district, and apply ameliorative therapy at a much earlier stage than is usually the case.

The present investigation is concerned largely with a working class population (about 50 per cent. miners) in panel practice, and some middle class

private patients. It is intended to show a consecutive series of blood pressure readings extending over twelve months in all patients coming for examination (irrespective of their complaint), and some clinical observations on hypertension with regard to symptomatology, etiology, prevalence, in the particular field, prophylaxis, prognosis and general hygienic management, with details of the results of treatment. In addition it is proposed to make a special investigation into a smaller series of cases with a view to the elucidation of the question of disorder of liver function as a possible cause of hypertension. This part of the investigation has been possible through the kindness of Professor Henderson of the Royal Infirmary, who has lent me every assistance in his wards. Similarly Doctor Douglas A. Adams and Doctor Mavor have very considerately placed their wealth of clinical material in Stobhill Hospital at my disposal for this purpose.

In the investigation of disorders of liver function within recent years, many workers have drawn attention to the fact that much useful

information can be obtained about the existing state of certain abdominal organs by accurately conducted and cautiously interpreted "Function Tests".

Many tests have been devised but the claims of several of them have not been clearly established, and much difference of opinion exists as to their respective values. The best known tests are those dealing with the state of the liver, pancreas and kidney.

Recently, it has been stated by several writers, (11), (12), (13), (14), (15), that the internal hepatic secretion influences the blood pressure. Allbutt, (11) was convinced that in hyperpiesia the cause was a warp in metabolism, possibly in the liver, "an incomplete or skew reduction of noxious waste, turning out some pressor amine, or hampering its exit, or failing to convert it". Major, (13), MacDonald, (12) Major Stoland and Brickstra, (14) have been actively investigating the depressor effect of a certain liver extract (not to be confused with the liver extract used in pernicious anaemia) within recent times. Their work is still largely in the experimental stage,

but if their claims are justified by further research, a great advance of our medical knowledge will have been achieved.

Part 1.

Section B.

The Liver in Relation to Blood Pressure.

The tests for liver efficiency, (20)B, (21)B, may be grouped according to the known functions of the liver. (In this connection it may be recalled that Starling(1) calls the liver the great chemical factory of the body.) The best known tests are the following:-

Carbohydrate Metabolism - Laevulose) Galactose) test.

Protein Metabolism - Amino-acid content of urine.

Fat Metabolism - Lipase test.

Secretory Function - Determination of bile salts and pigments in faeces.

Pigmentary Function - Van den Bergh's Test.

Excretory Function - Dye retention tests.

Urobilin Function - Tests for urobilinogen and urobilin in the urine.

The liver, as is well known, has the power of absorbing and storing large quantities of glucose, laevulose and galactose. Strauss, (2) first suggested that the estimation of laevulose tolerance

might be a test for liver efficiency. He had found that about 90 per cent. of his cases of liver disease showed laevulosuria, whilst only 10 per cent. of normal controls showed this phenomenon. A great deal has been written on the subject of this test, but the majority of observers, (3) agree that in its original form the test is unreliable. Shirokauer, (4) was the first to substitute the estimation of blood sugar in the place of urinary sugar. When a normal person takes 50 grms. of laevulose by mouth, little or no rise occurs in the blood sugar concentration. In disease of the liver, however, the storage capacity for sugar is impaired, and consequently the blood sugar rises considerably on the administration of laevulose. The actual test consists in having the patient fasting over night. The fasting blood sugar is estimated by McLean's method, 50 grms. of Laevulose administered in 500 cc. of water (flavoured with a little lemon juice) and the blood-sugar thereafter estimated every half hour for two hours. (The Laevulose must be pure and free from Glucose). If the maximum blood sugar concentration rises above 140 mgrms. per 100 cc. or if the blood sugar rises more than

30 mgrm. above the resting value, a degree of hepatic inefficiency is indicated. The urinary findings are of doubtful value. (3)

Galactose Tolerance Test.

This test, introduced by Bauer (5) of Vienna, is performed in exactly the same manner as the preceding one, except that the dosage is 40 grm. of Galactose. The latter must be pure and have a rotatory power of at least 76°. Up to the present this test has been mainly used in America (7) and on the Continent (6), but not much in this country. A rise of more than 30 mgrms. above the fasting value is considered to indicate liver damage. (8)

Elmer and Scheps, (8) as a result of comparative tests with these two sugars, come to the conclusion that the Galactose and Laevulose tests do not agree in one-third of all cases of hepatic disease, and that the Galactose test is very much more valuable than the Laevulose test in the diagnosis of hepatic disorders. In a recent communication Shay, Schloss and Bell,(9) as a result of two years' work on the Galactose tolerance test, claim to have demonstrated that in Galactose we have a sugar that is eminently suitable for the testing

of the function of the liver. (They consider the urinary excretion a more accurate index of liver assimilation than the blood sugar curve.) Their conclusion is based on the following points in the metabolism of Galactose.

(a) It is obtainable in pure form.

(b) It is readily absorbed from the digestive tract.

(c) It is converted into glycogen by the liver with some difficulty as compared to other sugars (dextrose and fructose.)

(d) It is practically not utilizable by any other tissues than the liver.

(e) After it reaches the general circulation it is excreted in the urine regardless of either the state of the renal excretory mechanism or the activity of the endocrine glands.

Shay and Schloss, (10) consider Galactose "far superior to Laevulose" for the purpose of this test.

Amino-acid Content of the Urine.

Many attempts have been made to detect impairment of hepatic function by examining for derange-

ment of protein metabolism. Since one of the most important metabolic functions of the liver is the formation of urea from ammonia derived from the deamination of amino-acids, one would expect in liver disease to find an increase in the concentration of amino-acid nitrogen in the blood, and therefore in the urine. There will also be a decrease in the formation of urea, and therefore reduced concentrations of urea in blood and in urine. Such alterations have, in fact, been found, but only to a convincing degree in cases in which disease of the liver is very extensive or severe.

"The normal range of amino-acid nitrogen in the blood is from 4 to 9 mgrm. per 100 cc. Values above 9 mgrm. are not often found except in acute yellow atrophy or eclampsia,- even in these conditions the increase is not always very great". (11)

Low values for blood urea are rarely found, probably because the hepatic lesions concerned are usually accompanied by functional disturbances of the kidneys, which cause urea retention.

The ratio of the urea nitrogen to the total non-protein nitrogen of the blood (or of the urine) is of greater value than either determination taken alone. Thus, the estimation of the urinary

nitrogen coefficient:- $\frac{\text{Urea N.}}{\text{total N.}}$ expressed as a percentage, has been recommended as a test of hepatic function. Normally the value of this coefficient is between 85 and 90, whilst in hepatic inefficiency it is said to fall to 40 or 50[3], indicating a decrease in the ureogenetic function.

On similar grounds the estimation of amino-acids in blood and urine has been recommended, elevation of these being said to indicate deranged function.

When the vast amount of reserve liver tissue is called to mind, and the various extraneous factors capable of modifying these figures, any test depending on the above estimations falls to the ground on general principles. (In this connection it is interesting to recall that Mann and Bollman, (12) found that after removing 70 per cent. of the normal liver of a dog, the remaining liver tissue would, in a few weeks, return to approximately its preoperative level.)

Lipase Test[3]. (Estimation of Blood Lipase.)

Into four test-tubes 1 cc. of serum is placed, together with 0.3 cc. of Toluene to prevent decomposition; 3 cc. of water are then added

to each tube, making the total volume 4 cc. To two of the tubes 0.26 cc. of ethyl butyrate is added, and all four tubes are incubated for eighteen to twenty-four hours in an incubator at 37°C. At the end of this time the tubes are removed and a drop of azolitmin solution is added to each. The tubes containing serum alone are alkaline, and are titrated with $\frac{N}{10}$ acid. The other tubes, in which the lipase will have produced butyric acid, are acid in reaction, and are consequently titrated with $\frac{N}{10}$ alkali. The amount of lipolytic action is the sum of the amounts of acid and alkali used. Normally this is between 0.2 and 0.3 cc.; values above these figures, indicating increase of lipase, are said to point to derangement of hepatic function. The urine may be examined for lipase instead of examining the blood. McNee (13) is satisfied with the test.

Determination of Bile Salts and Pigments in the Faeces.

Schmidts' test for Hydrobilirubin. A small amount of faeces is rubbed up in a mortar with a concentrated aqueous solution of mercuric chloride.

The mixture is transferred to a shallow, flat-bottomed dish and is allowed to stand for six to twenty-four hours. The presence of hydrobilirubin will be indicated by a deep red colour being imparted to the particles of faeces containing this pigment. This red colour is due to the formation of a hydrobilirubin-mercury compound. If unaltered bilirubin be present in any portion of the faeces, that portion will be green in colour, due to the oxidation of bilirubin to biliverdin.

Gmelin's test for Bilirubin. Place a few drops of concentrated nitric acid in an evaporating dish, and allow a few drops of the faeces and water to mix with it. The usual colours of Gmelin's test are produced, i.e. green, blue, violet, red and yellow. This test can be performed on a slide, and observed under the microscope.

Hay's test for Bile Salts. This test consists in sprinkling flowers of sulphur on the surface of a watery suspension of faeces in a basin. If bile-salts be present, the particles of sulphur sink to the bottom of the basin.

Bilirubin and biliverdin are formed from haemoglobin from broken down red blood cells.

These bile pigments are converted in the intestine to a number of substances, the most important of which are urobilin and its precursor urobilinogen. These changes are brought about by bacteria. Urobilin and urobilinogen are absorbed into the circulation, and are picked out by the liver cells, and re-excreted into the bile as bilirubin and biliverdin. A very small amount of urobilin is excreted normally in the urine, and special tests are required to demonstrate its presence. Hence the presence of bilirubin or its derivatives in urine or faeces is evidence of derangement of hepatic function.

Van Den Bergh Test. This test is primarily concerned with the differentiation of different types of jaundice, hence it may not be amiss to describe the modern views on this condition and compare them with the older ones.

McNee (13) in his excellent review in the "Quarterly Journal of Medicine" traces the history of medical opinion on this subject. In the past all types of jaundice were regarded as "hepatogenous" in origin, and were explained as resulting from some form of obstruction in the bile capillaries,

which caused the bile pigments to pass back into the blood stream. In view of more recent knowledge, (16) jaundice can be definitely classified as follows. McNee, (14):-

1. Obstructive Jaundice.

(a) The bile pigment is formed normally in the reticulo-endothelial system and is secreted by the liver cells into the bile capillaries. Owing to some obstruction the bile is dammed up and passes into the systemic circulation via the lymphatics or directly into the hepatic vascular capillaries. The jaundice due to stone in the common duct or to carcinoma of the head of the pancreas is thus readily understood. The bilirubin passed back into the blood stream has passed through the polygonal liver cells, and thus gives a (prompt) direct Van Den Bergh. reaction.

(b) In conditions of Hepatitis, the bile capillaries become plugged with an albuminous coagulable substance; this is followed by rupture of the disturbed bile capillaries, and the passage of bile into the blood.

(c) In jaundice due to haemolytic agents the bilirubin content of the bile is considerably

raised, the bile is so viscid as to practically block the ducts, and actual bile thrombi may be detected. Bilirubin may also pass into the general circulation.

(d) In acute necrosis of the liver (acute yellow atrophy, eclampsia) many liver columns are cut off from their bile capillaries, and any bile which they may still form, is passed back into the blood.

2. Toxic and Infective Jaundice.

When the liver cells are poisoned, they lose to a greater or less extent, their power of secreting bilirubin from the blood into the bile capillaries, and consequently bilirubin accumulates in the blood. The jaundice is due to excess of bilirubin in the blood which has not passed through the liver, and hence the indirect (or perhaps delayed direct) Ven Den Bergh reaction is obtained.

An obstructive element may be present in these conditions, and so complicate the Van Den Bergh reaction. A "biphasic" reaction may be obtained, the bilirubin in the blood due to obstruction of the ducts gives a prompt direct reaction, while

the bilirubin dammed up from poisoning of the liver, gives a delayed direct reaction, i.e., a reddish colour first appears which after a varying interval deepens to a violet.

3. Haemolytic Jaundice.

This is due to excessive destruction of red blood corpuscles, and the formation of bilirubin in larger amounts than the hepatic cells can deal with. The excess of bilirubin thus accumulates in the systemic circulation. This type of jaundice is produced by haemolytic agents such as arseniuretted hydrogen, toluidine - diamine.

The lemon-yellow tint of pernicious anaemia is due to a low grade jaundice.

In acholuric jaundice the red corpuscles are abnormally fragile, and so tend to be destroyed in excess, with resulting slight haemolytic jaundice.

Although for theoretical purposes jaundice may be conveniently classified into the above types, clinically there is bound to be a good deal of overlapping. Both forms of bilirubin may then be found in the blood, and the biphasic reaction occur frequently.

The Reticulo-endothelial System.

Piney (15) includes in this system: "many cells of the splenic pulp, of the medullary follicles and "cords" of the lymphatic glands and of other lymphatic structures, of the splenic sinuses, of liver capillaries (stellate cells of Kupffer), of capillaries in the formative bone-marrow, and of the adrenal and pituitary". (Wright (14) includes the interstitial cells of the testis). These cells are capable of picking up certain colloid particles in finely granular form. This capacity for storage distinguishes them from the other connective tissue elements as well as from all forms of myeloid and lymphatic cells.

According to a recent authoritative pronouncement (22) the bile pigments are produced from haemoglobin by the destruction of red blood corpuscles in the phagocytic cells of the reticulo-endothelial system. It is in these Kupffer cells that the bilirubin is produced, and from them it circulates in the hepatic capillaries and passes into the bile capillaries lying in the centre of the liver tubules.

If the liver cells are healthy the bilirubin passes through them and becomes combined in some loose way with bile salts and cholesterin, but when the liver cells are damaged the bilirubin from the hepatic capillaries cannot be fully absorbed by them, and consequently remains in the blood stream.

Van Den Bergh Reaction.

Two types of bilirubin can be distinguished by means of this reaction:-

1. Bilirubin formed as the result of the disintegration of haemoglobin in serous cavities, or during rapid haemolysis in the blood stream.

2. Bilirubin which has passed through the liver cells into the bile.

The reagents used in Van Den Bergh's reaction are as follows:-

A. Sulphanilic acid 1 gm.; concentrated HCl 15 c.cm.; distilled water 1000c.cm.

B. Sodium nitrite 0.5 grm.; distilled water 100 c.cm.

Mix 10 c.c.m. of solution A. with 0.3 c.cm. of solution B. immediately before use. The blood to be tested is allowed to clot (centrifuged) and

the clear serum used for the test.

Two reactions are described:-

(1) (a) Direct immediate reaction: Mix 1 c.cm. of serum and 0.5 c.cm. of reagent. A bluish violet colour develops, and reaches its maximum intensity in 10 to 30 seconds.

(b) Direct delayed reaction: Here a reddish colour, deepening to violet, begins to appear in about fifteen minutes.

(c) Direct biphasic reaction: In this type of response a reddish colour appears immediately, and takes much longer to develop into a violet hue.

(2) The Indirect Reaction. If neither of the first two be obtained the indirect reaction should be proceeded with. To 1.c.cm. of serum add 2 c.cm. of absolute alcohol. Centrifuge, draw off 1 c.cm. of the supernatant fluid, and add to it 0.5 c.cm. of the reagent. A violet-red colour develops, and is maximum instantly. (23)

When these reactions are positive, confirm by adding two drops of concentrated HCl to a portion of the fluid, and the reddish-violet colour will deepen towards a violet-blue if bilirubin is

present. To another portion add strong NaOH solution (2 drops) and a greenish-blue colour indicates the presence of bilirubin.

The bilirubin poured into the blood stream by the reticulo-endothelial system, gives the "indirect" reaction only, i.e. it needs the addition of alcohol to give the colour change. But during its passage through liver cells into the bile capillaries, it is modified in some way, so that now the addition of alcohol is not necessary - the "direct" reaction is obtained.

To be decisive the colour change must come on in 10 to 30 seconds ("prompt" direct reaction). A delayed direct reaction (appearing in 1 to 15 minutes) has the same significance as an indirect reaction.

Biliverdin (14) is only formed in the bile passages as a result of the oxidation of bilirubin, it is not present in the blood stream and does not give the Van Den Bergh reaction.

The indirect reaction may be converted into a quantitative (3) process by matching against a standard of ferric thiocyanate in ether.

Estimation of Icterus Index. (3)

For this determination clear, non-haemolysed serum or plasma should be used. This is compared in a colorimeter against a standard solution of potassium dichromate. The strength of the solution is 1 in 10,000, i.e. 50mgrm. of potassium dichromate to 50 cc. of water. Should the serum be too deeply tinted with bile, it may be diluted with normal saline.

Calculation. $\frac{15}{R}$ x dilution (if any) gives the icterus index.

The normal range is from 4 to 6. The method is useful in following the retrogression or otherwise of jaundice in a patient from week to week.

Dye Tests.

Tests based on the excretory functions of the liver depend on the fact that certain dyes are excreted almost entirely by the liver into the bile. Among the dyes which have been used for this purpose are phenoltetrachlorphthalein, bromsulphalein and rose bengal. In these tests a quantity of dye is injected into the circulation, and its rate of disappearance from the blood is noted. Any decrease in this rate of disappearance

compared with what is known to be the normal rate, is regarded as evidence of hepatic inefficiency.

In the phenoltetrachlorphthalein test (introduced by Rosenthal (17)), the dose is 5mgrm. for each kilo of body weight. A solution of the dye containing 5mgm. per 0.1 c.cm. may be obtained in ampoules. The required quantity of this solution is added to 100 cc. of sterile physiologic saline solution, which is then injected into the patient intravenously, the needle being then washed out into the vein with a further small quanity of physiologic saline solution. Fifteen minutes and one hour after the injection specimens of blood are removed from the patient (about 5 cc. in each case), allowed to clot, and the amount of dye in the serum is determined colorimetrically. In normal cases 2 to 6 per cent. of the amount of dye injected remains in the circulation after fifteen minutes, and none arter an hour. The presence of an appreciable amount of dye in the blood at the end of an hour indicates some hepatic disorder.

Shay ~~and~~ and Schloss (18) state that bromsulphalein has practically replaced phenoltetra-

chlorphthalein in usage, largely because of its lesser toxicity. Acting on the suggestion of Greene of the Mayo Clinic they have adopted the dose of 5 mgrm. per kilo of body weight instead of the usual dose of 2 mgrm. They are convinced that the larger dose is a better test load for the liver, and state that they have frequently been able to demonstrate the presence of liver dysfunction with the larger dose when the 2 mgrm. dose had previously indicated this function of the liver as unimpaired.

In the rose bengal test (19), 10 c.cm. of a 1 per cent. solution of the dye in normal saline is injected intravenously and the needle washed through slowly with 5 to 10 c.cm. of saline, held ready in a fresh syringe. The needle is left in the vein, and at exactly two minutes after the injection of the dye a sample of blood (10 c.cm.) is withdrawn from the needle into a clean syringe, and added to a centrifuge tube containing a little potassium oxalate, the tube then being inverted two or three times. The needle is again washed through with saline solution, to prevent clotting within its lumen, and at eight and sixteen minutes

from the time of injection samples of blood are withdrawn as before. The amount of dye in the eight and sixteen minute samples is then compared with that in the two minute sample, which is taken to represent the maximum concentration of the dye in the blood.

In normal individuals the eight minute sample shows from 40 to 60 per cent. of the dye retained in the blood, and the sixteen minute sample shows 23 to 30 per cent. Cases of obstructive jaundice, catarrhal jaundice and arsphenamine jaundice are said to show definite delay in eliminating the dye, the delay being greatest in obstructive jaundice. In cirrhosis of the liver the result seems to be in direct ratio to the amount of liver involvement, marked delay in excretion being shown in advanced cases.

Diffuse replacement of the liver tissue by carcinoma also produces definite retention, and acute hepatic infections, too, show marked impairment as judged by this test. Chronic passive congestion of the liver, nephritic toxaemias of pregnancy and certain types of ascites, other than those associated with cirrhosis, have given negative results.

Urobilin Function. Another test depending on the pigmentary function of the liver is the test for urobilin in the urine. Bilirubin, after passing into the intestine, becomes transformed by reduction into urobilin or its precursor urobilinogen. This is absorbed to some extent, taken to the liver, where it is reconverted to bilirubin, and re-excreted in the bile. In disease of the liver this capacity for dealing with absorbed urobilin and re-excreting it may become impaired, so that instead of being transformed and excreted in the bile, this substance becomes absorbed into the circulation and excreted in the urine. The presence of an excess of urobilin (or urobilinogen) in the urine may therefore be an indication of impaired hepatic function. Positive results to the test are also given in fevers, and in haemolytic jaundice, as well as in lesions confined to the liver. Excess of urobilinogen may be tested for by:-

(a) Ehrlich's Aldehyde Reaction. To 5 c.cm. of urine add two drops of a 3 per cent. solution of paradimethylaminobenzaldehyde in 50 per cent HCl. If the test be positive, the resulting mixture becomes deep red. Occasionally it is necessary to

warm the solution to bring about the reaction, which may take a few minutes to develop. This test, when positive, demonstrates the presence of a pathological amount of urobilinogen.

(b) To a test tube half full of the urine add 1 - 2 c.cm. glacial acetic acid. Shake out with amyl alcohol. Separate the alcoholic extract by centrifuging, and add saturated alcoholic solution of zinc chloride or zinc acetate. A green fluoreseence indicates an excess of urobilin.

In addition, the characteristic spectrum of the solution (a band in the green between b and F) can be demonstrated.

The clinical evaluation of tests of liver function is a matter of extreme difficulty at present. Few pathologists or clinicians have sufficient experience in all these tests to enable them to make a critical comparison of their value. Many of them are of very recent introduction, and important modifications are still being introduced. As in most new work, very conflicting results have been recorded by different observers. But the need for tests which shall add to the knowledge obtainable by ordinary clinical methods concerning the liver is

great. It is therefore advisable that extended use should be made of such tests as appear to have any merit, so that a considerable body of experience may be accumulated, to enable us to arrive at a true estimate of their value. (11).

From a review of the literature one is forced to the conclusion that the most convincing data have been presented by the following tests:-

1. Tests concerned with the pigmentary function of the liver (Van Den Bergh, Icterus Index, Urobilinuria.)

2. Tests based on the ability of the liver to remove foreign bodies from the blood-stream, (Bromsulphalein, phenoltetrachlorphthalein, rose bengal.)

3. Tests of the carbohydrate function of the liver (Laevulose tolerance, Galactose tolerance).

In the present investigation, known cases of hypertension have been submitted to the following tests for evidence of liver inefficiency:-

1. Galactose tolerance test.

2. Van Den Bergh test.

3. Urobilinogen and urobilin in urine.

The accompanying charts record the findings in sixteen cases showing high blood pressure. These include four cases showing other abnormalities, leaving twelve cases of essential hypertension. The age groups of these are as follows:-

Age in years.	40-50.	50-60.	60-70.	70-75.
No. of cases.	1	5	2	4

As indicated in the literature, and as borne out by the controls, a rise in the blood-sugar of more than 30 m.grms. above the fasting level indicates liver damage.

Of the twelve cases of essential hypertension eleven showed definite evidence of liver damage by the galactose tolerance test, and in addition three gave a delayed direct and a positive indirect Van Den Bergh reaction.

None of the sixteen cases gave a positive result for either urobilinogen or urobilin in the urine.

The fact that one case of essential hypertension gave a negative result to the galactose tolerance test does not necessarily indicate absence

of liver damage. Just as with the Wassermann test, a positive result is of much more significance than a negative one.

The incidence of dysfunction of the bilirubin mechanism (25 per cent.) is too high to be mere coincidence. It is noteworthy that the case showing greatest disorganization of carbohydrate liver function (no. 24), presumably a syphilitic liver, gave a negative Van Den Bergh reaction.

The normal curve was constructed on the basis of ten controls. For completeness, the controls originally included one diabetic (sugar free on diet without insulin), and one patient aged 66 years with arcus senilis, marked generalised arterio-sclerosis and severe anginal symptoms. Omitting these two abnormal cases, we are left with the ten controls above cited. The age-groups in these are as follows:-

Age in years.	18-20.	20-30.	30-40.	45-50.	50-60.
No. of cases.	2	2	2	2	2

Of these, one case of mediastinal tumour showed a slight rise above the normal, and presumably

suffered from liver involvement. None of the others showed any evidence of liver dysfunction.

The four cases of high blood pressure showing other abnormalities include:-

1. Uraemia with enlarged prostate.
2. Acute nephritis.
3. Syphilitic Aortitis.
4. Enlarged prostate.

The first three showed definite liver damage by the galactose tolerance test, the fourth gave a normal curve.

The findings as given above and illustrated in the charts would seem to warrant the following conclusions:-

1. Cases of high blood pressure show definite evidence of liver dysfunction.
2. The carbohydrate-metabolizing function of the liver would seem to be dissociated from the bilirubin secreting mechanism.
3. The excretion of urobilinogen (and of urobilin) would seem to connote a more advanced degree of liver damage than is indicated in the text books.
4. Age is not necessarily a factor in liver

dysfunction, as evidenced by the fact that two patients in the series (Nos. 25 and 15) aged respectively 70 and 65 years, gave negative results to all the tests.

(The urine passed before administering galactose was examined for sugar in every case with negative result.)

None of the cases showed any obvious clinical indication of hepatitis.

Number.	Age.	Diagnosis.	Blood-Pressure.	Galac-tose.	Urobilin-ogens and Urobilin.	Van den Bergh.	C = Control
C. 1	35	Gastric Ulcer.	124/72	-	-	-	
2	52	Hyperpiesa.	160/120	+	-	-	
C. 3	18	Normal.	110/68	-	-	-	
4	34	Disseminated Sclerosis.	129/72	-	-	-	
C. 5	45	Mediastinal tumour.	130/84	+	-	-	
C. 6	51	Bronchitis.	134/78	-	-	-	
C. 7	57	Subacute Rheumatism.	136/82	-	-	-	
C. 8	27	Renal Calculus.	138/86	-	-	-	
C. 9	18	Mucous Colitis.	110/66	-	-	-	
C. 10	46	Endocarditis.	140/82	-	-	-	
C. 11	26	Normal	114/78	-	-	-	
12	31	Acute Nephritis.	230/150	+	-	-	
13	60	Uraemia.	210/130	+	-	-	
C. 14	27	Diabetes Mellitus.	114/76	+	-	-	

Number.	Age.	Diagnosis.	Blood-Pressure.	Galactose.	Urobilinogens and Uroholin.	van den Bergh.
15	65	Enlarged Prostate and Hyperpiesis.	210/110	-	-	-
16	70	Hyperpiesia.	210/110	+	-	-
17	50	Hyperpiesia.	190/120	+	-	Indirect +
18	71	Hyperpiesia.	190/112	+	-	-
19	75	Hyperpiesia.	220/125	+	-	-
20	51	Rheumatic Carditis and Hyperpiesis	200/126	+	-	-
21	65	Hyperpiesia.	230/125	+	-	-
22	48	Hyperpiesia.	236/130	+	-	Indirect +
23	66	Hyperpiesia.	190/112	+	-	-
24	51	Syphilitic Aortitis and Angina.	190/110	+	-	-
25	70	Hyperpiesia.	210/130	-	-	-
26	56	Hyperpiesia.	186/112	+	-	-
27	54	Hyperpiesia.	185/112	+	-	Indirect +
C. 28	66	Arteriosclerosis.	120/90	+	-	-

PART II

General Remarks.

Persistent elevation of the blood pressure without known cause is a disease of high incidence and great seriousness as shown in the introductory section of this paper. Recent studies have shown that it is present in about 1.6 per cent of the population in the United States and Canada, a figure which probably holds for England too. The disease is said to be very rare in China and India and in tropical countries generally. In America it ranks with tuberculosis and cancer as a cause of death. (1)

The malady has been known to Medical Science for centuries. Symptoms resembling those of high blood pressure had been recorded by the Greeks under the term 'plethora'; and Galen had surmised the nutritive function of blood flow in the tissues. The science of haemodynamics goes back to Stephen Hales (2), Rector of Farrington, Minister of Teddington, when he bent his activities from the study of plant physiology to find out the real force of the blood in the arteries, not only in animals of different species but also in animals of the same kind...and in the same animal; Hales wrote in 1733 "the force of the blood in its vessels is continually varying, according to the different kinds and quantities of

food, the various distances of time after taking food, the more or less plethoric state of the blood vessels, also from exercise, rest, different states of vigour or vivacity of the animal and many other circumstances." Following on these discoveries Von Basch in 1833 introduced the clinical sphygmomanometer. Of this instrument Janeway (3) remarks, "In two fundamental respects it differed from all that had preceded it. One was the employment of a bag containing fluid as the compressing mechanism, thus obtaining pressure per unit of surface for the first time; the other was the use of a mercury manometer for the measurement of the pressure within the bag necessary to wholly compress the artery. In this way he made possible a comparison of results with the direct methods of the physiologists." The introduction by von Recklinghausen in 1901 of the cuff 12 inches in width removed a source of inaccuracy due to too narrow an armlet, and the demonstration of the auscultatory method of Korotkoff in 1905 established the clinical study of blood pressure on an accurate and easy basis.

Bright, it has been said, began the story of hypertension and its consequences by writing its

final chapter. As a result of his epoch-making clinical and pathological studies on patients presenting hypertrophy of the heart with albuminous urine, he propounded the following conclusions in 1837 (5),: "The two most ready solutions appear to be either that the altered quality of the blood affords irregular and unwonted stimulus to the organ (heart) immediately, or that it so affects the minute and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system." This mechanical theory on the genetic basis of kidney disease dominated clinical medicine for nearly a century. Gradually, however, it became increasingly evident that many cases of cardiac hypertrophy with hypertension showed no evidence of kidney disease, and finally led to the theory of a common toxic agent acting on both the blood vessels and kidney substance. H.Batty Shaw (6) as a result of his fine clinical study propounds the following theory; "It is a more reasonable proposition to advance that, at the neuro-muscular junction, agencies can operate which are chemical or physical or chemico-physical and that they are bodies which may be carried by the blood. Experimentally

it is established that the toxins of bacterial origin operate at such sites, and that they are almost universally depressor in action - they almost all act, when they do act, by lowering the blood pressure. Are there bodies which could act at this site which are pressor in function? We are aware of a very few, but their action and importance is established. If the blood stream supplying the heart and blood-vessels is charged with pressor agents capable of acting at the neuro-muscular junction, then there is a ready explanation for hyperpiesis, and if the strength of solution in the blood of these bodies varies, there is a ready explanation for the variations in the reading of a hyperpiesic chart."....."With so many hypotheses to choose from there can be no harm in choosing one which seems least objectionable and most in conformity with knowledge, and which opens the way for investigation, clinical, pathological and experimental." Janeway (7) in 1913, came to practically the same conclusion when he stated that the "symptom of hypertension" could arise in three ways.

1. "Through purely quantitative reductions of kidney substance below the factor of safety. It is difficult to conceive of this as other than a vascular hyper-

tonus due to retained poisons of some kind."

2. "In connection with the unknown intoxication which causes disturbances of the central nervous system and which we call uraemia. This intoxication is not one of retention in the strict sense."

3. "In primary irritability of the vaso-constricting mechanism from unknown, probably extrarenal causes, which lead eventually to arteriolar sclerosis. In this type the disease in the kidney is the sequence, not the cause, of the generalized vascular lesion.....In these primary vascular diseases, it is probable that eventual, widespread narrowing of the arterial stream in some cases produces a permanent organic increase in peripheral resistance."

The most recent trend of authoritative opinion is towards the view that the causes of hypertension are extrarenal. The modern conception of hypertension as a general constitutional disease has expanded the field of observation, and increased the possibility of its prevention and control.

Arteriolar resistance to blood flow is fundamental in the pathophysiology of genuine hypertension. (8). The insignificance of the cardiac factor, blood volume and viscosity, as well as sclerosis

of the larger vessels has been repeatedly proved. Attention centres on the arterioles and capillaries. Renal changes are secondary throughout. Clinically the hypertensive diseases exist in two groups; essential hypertension and acute universal capillaropathy (so-called acute glomerulo-nephritis). Essential hypertension is a hereditary constitutional autonomic vaso-motor neurosis with hormonal disturbances. Constitution, the vegetative system and the endocrine glands are inseparably involved in the abnormal reaction of the body. The abnormal reaction is manifested in a multiplicity of phenomena.

Patients or their families tend to have diabetes, gout, adipose constitution, asthma, migraine, spastic colitis and vaso-motor disturbances. The essential hypertonic patient tends towards vagotonia.

In essential hypertension the capillaries are normal as to morphology and tension. The arterial pressure is at its highest in this form of hypertension, and is based on arteriolar spasm. Extensive daily fluctuations are characteristic and attest to the concept of a functional vaso-motor disturbance. Diffuse arteriolosclerosis, which is a concomitant pathologic change, (9), (6), may be interpreted as

an end-stage of arteriolar work-hypertrophy.

Acute universal capillaropathy is considered to be due to pathogenic streptococcal toxins. According to Kylin the toxin produces a universal capillary dilatation with an axon reflex contraction of the arterioles. The vessels of the kidney are merely more sensitive and hence more involved. The primary site of action is the capillary; the arteriolar response is secondary. Edema is based on the increased permeability of the capillaries, increased hydrostatic pressure or diminished osmotic pressure of the blood. Changes in eye grounds constitute advanced, organic vascular injuries. Kylin emphasizes the fact that the rise in capillary and arterial pressure, as well as edema, precede the urinary symptoms. Renal changes are only secondary. Lastly he considers the permanent diseases of hyper tension as end-stages, often being combination forms of essential hypertension and acute diffuse capillaropathy. They are the result of irreversible organic vascular changes.

The pathological changes may be briefly stated as diffuse arteriolar disturbance with hypertrophy of the media and intimal proliferation.

It is rather surprising to find, in a very recent text-book of medicine, (10), the following list of etiological agents; "over-indulgence in food or alcohol or tobacco, auto-intoxication, especially from the teeth, the tonsils, the accessory nasal sinuses, the colon, and the genito-urinary tract, gout and lead poisoning. Other causes of supernormal blood pressure are nephritis - more especially the chronic interstitial form, in the later stages of hyperthyroidism, often in the cyanosis of cardiac failure, and usually in polycythaemia. It will thus be seen that the etiology of supernormal blood pressure is similar to that of arterial hypertrophy." The experience of observers dealing with African and Chinese patients would seem to be in direct contradiction to many of the statements in the above list. ~~Dominion~~ DONNISON (11) working amongst the African Negro tribes of the Kenya Colony, reports 1800 admissions to the Mission Hospital without a single case of hypertension. In these negroes cardiac hypertrophy was rare and arteriosclerosis was late in making its appearance, and then mild in degree. Infections, nevertheless were extremely frequent. Pyorrhoea was universal in adults over thirty years.

Tonsillitis, chronic skin ulcers and constipation prevail in these negroes, and meat is a large element in the diet. The one outstanding factor which seems to differentiate them from their white brethren is the absence of the worry and mental strain of civilized life.

In China high blood pressure is a rare finding. Among 4,000 patients in the medical wards at Changsha, J.H.Foster (~~12~~ 17) found one instance of essential hypertension. This was the case of a highly educated Chinese woman, who had been trained as a nurse in America, and had become completely westernized.

Albutt wrote in 1925, "It now seems to be generally agreed that mere alcohol is, as such, no cause of hyperpiesis.....Furthermore I find no later evidence to weaken my opinion that the effect of tobacco in causing hyperpiesis or senile atheroma, if any, is negligible....."

"That gout, in its uncomplicated arthritic form, is no cause of hyperpiesis is still my opinion....."

"Intestinal poisons are indeed, generally speaking, of the depressor class."

The real explanation of these contradictions is, of course, to be found in the fact that the cause

or causes of persistent arteriolar spasm with its concomitant hyperpiesis has not yet been discovered, and we consequently find diet, auto-intoxication, focal infections, gout and nephritis invoked as etiological agents. In the same text book of medicine (10) we find the following listed as factors in hypotension. "Acute infective diseases, auto-intoxication, especially from the teeth, the tonsils, the accessory nasal sinuses, the colon and the genito-urinary tract.....the excessive use of tobacco."

The Present Position.

In order to obtain a broad view of the incidence of hypertension, as distinct from its origin and course in the individual, it is necessary to make observations on unselected groups of cases. Such observations should be of value from the standpoint of epidemiology and of public health in general. As already stated, the striking incidence of hypertension in the white races, with their intensive civilization, in contrast to its entire absence in the African negro, is of significant import. Beckman's estimate of hypertension as occurring in 1.6 per cent of the population may be compared with Gager's (2) figures in 2,000 consecutive patients with symptoms unrelated by them to hypertension. Aortic insufficiency was excluded from the series and also true nephritis with hypertension. The analysis revealed systolic hypertension in 15.2 per cent of the men and in 16.9 per cent of the women. Diastolic hypertension occurred in 12.6 and 16.1 per cent for the series respectively.

A study of the comparative incidence of the chief types of disease in various heart clinics (20) reveals that hypertension, which is so often inex-

tricably mixed with arteriosclerotic disease, makes up from 30 to over 50 per cent of all the patients. At necropsy, of all heart disease not due to valvular lesions or syphilis, 70 per cent were hypertensive. A conservative calculation, based on the mortality statistics of the United States registration area in 1924, estimated that 143,000 deaths in persons of fifty years of age and over were associated with hypertension. This observer comments on the fact that hypertensive heart disease is frequently incorrectly diagnosed as myocarditis or mitral insufficiency, that true nephritis in contrast with hypertensive renal disease is rare, and that in apoplexy, hypertension is present in the majority of cases.

Gilman (4) investigating the influence of occupation on blood pressure concludes that elevation is more frequent in labourers than in brain workers. He believes that the variations in pressure occurring during heavy manual labour lead to permanent structural changes.

Amberg (5) studying persistent elevation of blood pressure in the young, has described six different types.

1. Hypertension limited to the upper part of the body associated with coarctation of the aorta.

The retinal arterioles were contracted. Renal function was normal.

2. Hypertension with organic cerebral lesion.

3. Essential hypertension without evidence of renal damage.

4, Malignant hypertension with rapid fatal evolution and extensive arteriolar disease.

5. Hypertension associated with a variety of lesions in the kidney.

6. Hypertension following congestive heart failure.

The incidence of hypertension rises rather rapidly after middle age. Most of the text books describe essential hypertension as a disease of middle life, usually occurring in individuals between forty and fifty years, with equal distribution between the sexes. Such an age distribution is only to be expected when one considers the inevitable loss of elasticity in the tissues and organs of the body with advancing years and the consequent alteration in physiological reactions.

The symptomatology of hypertension naturally varies greatly according to its mode of origin, rapidity of development, and the varying body

areas and organs involved. As already mentioned established hypertension is often found in plethoric and well nourished individuals. Douthwaite (6) gives the following list of common symptoms, based on a clinical survey:- Palpitation, shortness of breath on exertion and headache, palpitation, tachycardia and inability to lie on the left side, giddiness or dizziness, tinnitus aurium, anginal pain, mental symptoms (nervousness etc.,) haemorrhages (nasal and retinal), pain in the limbs or a clutching sensation in the throat.

Acute hypertension, on the other hand, is purely a disorder of function. It usually arises in patients with a normal blood pressure, and is found to have an exciting basis in eclampsia, crises of pain or severe emotional disturbance, acute lead poisoning, suprarenal tumour, or in chronic glomerulonephritis as an exacerbation of existing high blood pressure. The sudden dramatic development of the symptoms and signs of vasoconstriction and consequent ischaemia stands in sharp contrast to the slow and often insidious onset of the benign chronic disease. Crises of paroxysmal hypertension have been found associated with suprarenal tumour tissue. Villaret (7) discussing the causes

of this interesting condition gives the following list of known etiological agents;'tumours of the adrenal medulla, in which the systolic blood pressure may rise suddenly and without apparent reason from 120 to 250 mm of mercury, causing great distress, numbness of the limbs and pallor; injections of adrenal substance, stimulation of the adrenals by metabolic poisons in the blood and by nervous impulses, for example a tumour involving the vagus nerve or a lesion of the Gasserian ganglion, acting by an increased output of adrenaline. Peripheral pain, such as biliary and renal colic, has a similar mechanism and effect. Emotion, cerebral compression, embolism and thrombosis, poisons such as ergotamine, and acute and chronic nephritis are other recognised factors. Among the obscure associations of these hypertensive crises are lead poisoning, eclampsia, angina pectoris, and pulmonary oedema and tabes dorsalis.'

Far different from these acute manifestations of purely exogenous origin is the insidious evolution of chronic benign hypertension. Starting often with symptoms which are non-specific for hypertension: irritability, psychic instability,

easy physical and mental fatigue, imperfect digestion, palpitation, flushes, cold blue hands and other vasomotor phenomena, frequent urination, possibly sexual impotence - the progressive course of hypertensive vascular disease leads slowly to three chief syndromes (2), the cerebral, the cardiac and the renal. These are important as being the chief basis of symptoms and the major causes of mortality.

Dizziness, fullness or tightness in the head, "douleur en casque", the occipital headache occurring in the early morning hours and wearing off after rising, described by Janeway, migraine or hemicrania, sensations of throbbing and noises, are the symptoms which suggest cerebral vascular lesions. Shortness of breath, sensations of premature beats, substernal heaviness and pain point to coronary sclerosis and myocardial degeneration. Frequency of urination, especially nocturia and an excessive amount of urine with low and fixed specific gravity, indicate involvement of the kidneys. The occurrence of fullness or distress in the abdomen, of cramps in the legs (intermittent claudication), of impairment of vision, are evidences of vascular disease in these several domains of the body. The symptoms and signs of

diabetes mellitus arise from pancreatic arteriolar disease. The joint and muscle pains (often very severe in nature) which are called "high pressure rheumatism" have been ascribed to regional angiospasm. In the menopausal group, obesity, decreased sugar tolerances and various forms of arthritis are commonly found associated.

While the hypertensive is inevitably a cardiopath in the making, his progress towards this end is interrupted in rather more than half the cases by vascular disease in regions other than the heart. The brain is the second important danger area. The kidney and pancreas come next in order of importance.

The cerebral type of hypertension is evidenced by the "hypertensive headache", throbbing in the head, sleeplessness, irritability, dizziness, mental weakness, psychoses and sometimes dementia. Vascular spasm induces objective signs of disturbed arteriolar function, leading to clinical syndromes simulating organic structural lesions of vessels and brain substance, thrombosis, haemorrhage and softening of circumscribed cerebral areas.

The appearance of paroxysmal dyspnoea or cardiac asthma is now regarded as being due to anoxaemia of

the respiratory centre, as a result of left ventricular insufficiency, and is a phenomenon of serious prognostic import. (Relatively it does not occur as often in hypertension as in central vascular syphilis). The average duration of life after this symptom appears is between one and two years.

In his stimulating book on the study of constitutional types Draper (8) states that hypertension nephritis people are differentiated from other structural types by their relatively narrow faces and broad eye zones. On the other hand those predisposed to tuberculosis have the same narrow head, but eyes closely set together. He claims that, amongst women, the hypertension types are larger, heavier and more masculine - more of a "pituitary type" than are people with a tendency to nephritis.

Apart from these rather speculative data, the sequelae and complications of high pressure give rise to well-marked clinical signs. No system or organ of the body escapes in some measure the consequences of persistent hypertension. The important practical point is to investigate the 'status praesens' of the patient with special reference to (1) the vascular changes present in the various areas of the body,

(2) their extent and (3) the rate at which they are progressing.

Retinal changes in hypertension.

Within comparatively recent years the retinal picture in hypertensive disease has come to assume more and more importance, not only from the standpoint of diagnosis, but more particularly from that of prognosis. Fishberg and Oppenheimer (9), recently investigating 274 cases with 39 post-mortems, conclude that the concept of albuminuric retinitis includes three distinct pathogenetic entities which can most often be differentiated with the ophthalmoscope:

(a) Arteriosclerotic retinopathy.
(b) Malignant hypertensive neuroretinitis.
(c) Choked disc due to increased intra-cranial pressure from oedema of the brain.

Keith, Wagener and Kernohan (28), on the basis of extensive investigations, believe that they can often differentiate the retinal lesions in the severe cases of essential hypertension which they term malignant hypertension. They found the following:- In malignant hypertension the oedema of the retina is less extensive and less dense, and there is little tendency to the formation of peripapillary snow-bank exudates. The hyperaemia

of the disc is in marked contrast to the anaemia of the disc and the retina that is seen in the retinitis of nephritis. Sclerosis of the retinal vessels (arterioles) is always present in malignant hypertension and is usually absent in chronic nephritis.

It is of great diagnostic and prognostic importance to differentiate the ophthalmoscopic pictures that may appear in patients suffering with diseases characterized by hypertension.

In retinal arteriosclerosis and arteriosclerotic retinopathy the changes in the fundus are the result of retinal arteriosclerosis. The retinal arteries show the characteristics of more or less marked arteriosclerosis, namely irregularity of lumen, so-called arteriovenous compression, irregular light reflex, copper-coloured appearance and white lines accompanying the blood columns (perivasculitis). Generally some constriction of the arteries, particularly of the smaller branches, can be made out, but this is, as a rule, not nearly so marked as in malignant hypertensive neuroretinitis. Increased tortuosity and a brilliant light reflex are often also considered as signs of arterioscler-

osis. The disc shows no abnormalities in most instances; in others there is slight haziness of outline. The absence of papilloedema is the most important criterion for differentiation from malignant hypertensive neuroretinitis. Haemorrhages are often present. White spots (areas of degeneration or of proliferation) may be present in small numbers or profusely. When the individual opacities are large they often appear shiny, and their outlines are sharply delimited from the adjacent retina ("hard opacities"), thus differentiating them from the "cotton wool" patches found in malignant hypertensive neuroretinitis. Evidences of choroidal sclerosis with pigment changes are often present. The large majority of the cases in which the picture occurs are those of essential hypertension.

The significance of arteriosclerotic retinopathy, severe though the changes in the retina may be, is purely that of sclerosis. It does not necessarily indicate, as does malignant hypertensive neuroretinitis, that renal insufficiency either is present or threatens almost inevitably.

Malignant hypertensive neuroretinitis.

This variety of retinal change is of much more ominous significance than the arteriosclerotic lesions just described. It is characterized by the presence, generally though not invariably, from the onset, of papilloedema in addition to the retinal lesions. As a rule the appearance of papilloedema is the first change in the fundus (apart from the usually antecedent constriction of the arteries), though most cases are first observed when retinal lesions are also present. The disc is usually reddened; in the retina haemorrhages are generally present. White spots are usually present also. Contrary to those found in the arteriosclerotic retina these opacities are most often of the soft, indistinctly bordered type described as cotton-wool. Hard, sharply-delimited opacities may also be present. The arterial blood columns are narrowed generally to a striking degree. There can be no doubt that the narrowing of the arterial blood columns is the result of functional vasoconstriction of the musculature of the retinal arteries, for it may appear early in the course of acute glomerulonephritis or the hypertensive toxaemia of pregnancy

before there is any opportunity for narrowing of the lumen by a proliferative process. The veins are generally distended, particularly when there is marked papilloedema.

Malignant hypertensive neuroretinitis occurs in glomerulonephritis and essential hypertension. It is frequently present in the hypertensive toxaemia of pregnancy, with or without eclampsia gravidarum. It may also appear in lead poisoning with hypertension. This may occur in the absence of renal involvement.

In the efforts to clear up the pathogenesis of "albuminuric retinitis" attempts have been made to correlate the retinal lesions with five different manifestations of Bright's disease.

(1) Renal insufficiency.

(2) Retinal arteriosclerosis.

(3) Increased intra-cranial pressure.

(4) Hypercholesterolaemia.

(5) Arterial hypertension.

(1) Is untenable, for retinal lesions of even extreme severity may occur in the presence of intact renal function. That (2) cannot be incrim-

inated in the pathogenesis of the retinal process in acute glomerulonephritis or the toxaemia of pregnancy is obvious. Endarteritic lesions of the retinal and choroidal arteries (characterized by endothelial proliferation) are often present in malignant hypertensive neuroretinitis, but they are too inconstant to be regarded as the primary cause of the lesions.

(3) Increase in cerebro-spinal tension is present in only some patients with hypertensive neuroretinitis.

(4) This hypothesis is not of general validity, for in the large majority of instances of essential hypertension or chronic glomerulonephritis of many years standing, with retinal lesions, hypercholesterolaemia is absent.

(5) So-called "albuminuric retinitis" is never seen in cases in which there is not some evidence of hypertension, past or present.

Volhard advanced the theory that the retinal lesions are manifestations of ischaemia of the retina produced by a spasm of the retinal arteries which is a part of the widespread vasoconstriction that is present in arterial hypertension. Recently Haselhorst and Mylius (29) not only observed, but photographed

cramplike and rapidly changing contractions of the retinal arteries in a patient with eclampsia gravidarum. After two days the constrictions became more constant and involved longer stretches of the arteries. At this time the first white degenerative lesions in the retina appeared.

Fishberg and Oppenheimer (9) in their thirty-nine necropsies (essential hypertension with malignant hypertensive neuroretinitis), found, in all cases, necrosis of the arterioles in the kidney. In all instances the anatomic picture of the kidneys was that described by Fahr as "malignant sclerosis", namely, necrosis, endarteritis and thrombosis of the renal arterioles, necroses and proliferative changes in the glomeruli, in addition to the arteriosclerosis and glomerular hyalinization which are also found in the usual (benign) case of essential hypertension. On the other hand, necrosis of the renal arterioles was not present in any of the eleven cases of essential hypertension without malignant hypertensive neuroretinitis in which the patients came to necropsy.

It would appear from these cases that the presence of malignant hypertensive neuroretinitis is

diagnostic of the occurrence of necrosis of the renal arterioles. This necrosis, with its attendant changes in the renal parenchyma results in severe and generally rapidly progressive renal insufficiency.

Allbutt (10) quotes Weiner and Wolfner to the effect that in cases of high blood pressure the pupil on first exposure to light contracts at once, and then dilates again; although still under the light. They consider this reaction, when present, to be "pathognomonic of arteriosclerosis with high blood pressure" or at any rate to be of undoubted clinical value", and that it is almost constant.

Estimation of blood pressure.

The importance of allowing for the normal and abnormal variations in blood pressure readings has already been pointed out. Beaumont and Dodds (11) give a very clear account of the auscultatory method introduced by Korotkow in 1905, ("a simple and accurate method"). The patient should be lying on a couch or sitting on a chair, with the arm resting comfortably on a table at the level of the heart. The armlet is applied as high up the upper arm as possible, with the centre of the rubber bag lying over the brachial artery. Oliver (12) recommends "that the auditory method with systolic tactile check be used in all cases. Whichever method be followed, the reading should be made by the falling pressure after overstepping the obliteration of the pulse; and not by the rising pressure." The rationale of this is, of course, to overcome any spasm of the blood vessel under consideration.

It is now generally recognised that the diastolic pressure is reached when the loud, clear sound of the so-called"third phase" changes its character and becomes muffled. That this point, and not the total disappearance of the sound, corresponds

accurately to the diastolic pressure has been repeatedly demonstrated by experiments on animals. At the critical moment of the determination the patient's attention may be distracted by an inquiry concerning the bowels. The manometer should lie outside his line of vision, and to avoid a secondary reflex rise the reading should be taken expeditiously. Before a patient is labelled hyperpietic his blood-pressure should be estimated on at least two separate occasions, preferably oftener.

~~of Mean Dynamic pressure~~. A diastolic level of 120 mm. is often not borne more than two years, and one of 150 mm. ordinarily conveys the prospect of death within a few months. (13).

Janeway (21), writing in 1904, stated: "To-day the intelligent physician can no more afford to dispense with some form of sphygmomanometer in the study of certain types of disease, than he could discard the thermometer, and trust his trained sense of touch to detect the variations of body temperature in a case of typhoid fever.

In the case of children, whose arms are very small, the thigh is to be preferred for blood pressure estimations. This obviously necessitates the recumbent posture to keep the level of the heart.

Cardiovascular Signs.

The heart of hypertension ranks with the cor bovis of aortic insufficiency and of adhesive pericarditis in exhibiting extreme degrees of eccentric cardiac hypertrophy (20). In view of the recognized effect of physical strain in the production of hypertrophy of the heart, freedom from the necessity of heavy labour is one of the first requirements of the patient who must safeguard his diminished cardiac reserve.

Besides the increase in size, the two other cardinal signs of the heart working against increased resistance are the heaving apex thrust and the ringing accentuated aortic second sound. These may, however, be masked by other physical factors such as obesity and emphysema. On exertion there is a marked increase in pulse and blood pressure.

The apical systolic murmur of relative mitral insufficiency is the most common finding in the failing heart of hypertension. It arises from stretching of the mitral ring caused by enlargement of the left ventricle, with consequent incompetence of the mitral valve. It is the consequence of heart failure and not its cause. Systolic aortic murmurs

are less common, and less significant. They cannot be accepted as evidence of aortic stenosis unless accompanied by a palpable thrill.

As a late manifestation of heart failure from hypertension, auricular fibrillation like coronary thrombosis and myocardial infarction, may greatly lower the blood pressure and mask a virtual hypertension.

The electrocardiogram shows no distinctive features until the advent of myocardial changes as a result of late vascular complications. (14).

Renal Function.

Renal function often remains unchanged during the course of benign hypertension, and is often little affected in malignant hypertension. More than two-thirds of the kidney substance must be destroyed before insufficiency ensues, (2) while it is clinically demonstrable that relatively few patients with hypertension die of uraemia. The evidence accumulated by Addis and his co-workers co-relates haematuria and glomerular inflammation, increased excretion of epithelial cells with tubular degeneration and a continuing slight excess of casts, protein and cells in the urine with probable vascular changes in the kidney. In many cases of hypertension the only evidence of renal damage (apart from the more technical delicate biochemical determinations) is a slight amount of albumen in the urine.

Prognosis as ordinarily accepted.

According to Price (15) the prognosis depends very largely on renal involvement. In hyperpiesia the condition may last for many years and only be terminated by haemorrhage into the brain. When,

however, the kidneys are affected, as shown by persistent albuminuria with casts in the urine and a blood pressure well over 200 mm Hg, the condition is grave; and when well-marked albuminuric retinitis is present death usually occurs within six months, though very rare cases have been recorded where a certain amount of ocular change has persisted for years.

It has already been pointed out in a previous section of this paper how unfavourable a view is taken of hyperpiesis by insurance companies, and it must be remembered that they have unrivalled opportunities for estimating the comparative mortality from different diseases.

Clinical Pictures.

Kernahan, Anderson and Keith (14), as a result of an intensive study of fifty-three cases in the Mayo Clinic, have divided high blood pressure cases into three clinical groups. (1) Benign hypertension, (2) Early malignant hypertension, (3) Malignant Hypertension. Biopsy was performed in each case in order to study the condition of the arterioles in living subjects. Muscle tissue was chosen

because it composes about 35 to 40 per cent of the body and thus an assumption that there is a widespread lesion of the arterioles could be readily proved or disproved. After the injection of a local anaesthetic, a small portion of the pectoralis major muscle was removed in each case, and examined microscopically.

The most pronounced and most constant change was in the media (of the arterioles) which was quite definitely hypertrophied, and there was an increase in the nuclear elements of this tissue. The entire change was due to proliferation of muscle. In the intima the change was widely varied, but the most common modification was proliferation of the lining endothelial cells. Complete occlusion of a vessel was rare, but partial occlusion was the rule. Thus the fundamental changes occurring in these vessels were hypertrophy of the media and proliferation of the intima. This is not a degenerative change, nor a change which can be attributed to senile regression.

The arterioles in the kidney were more extensively and seriously involved than anywhere else (in the cases coming to necropsy). Some of these were almost completely occluded.

The serious prognosis in the malignant group was evidenced by the fact that only two of the twenty-three patients observed have been reported as well, two are failing and fourteen have died. Of the fourteen, eleven have died from two to six months after dismissal.

The results of this study indicate that associated with hypertension a diffuse disturbance of the arterial side of the vascular system exists, both clinically and histologically, in varying degrees of severity. In benign hypertension lesions in the peripheral arterioles may be absent or moderate in degree. (Allbutt considered dynamic narrowing rather than organic changes in the arterioles, as the primary lesion). In the second group of cases those of severe benign or early malignant hypertension, the clinical observations indicated more serious involvement. In spite of this, many of these patients had remarkably good retinal, cardiac and renal function. In the cases of malignant hypertension there was a definite type of retinitis (already described). Cardiac enlargement and disturbance of renal function were more often present than in either of the other two groups, and the blood

pressure was usually very high. There was almost invariably a marked thickening of the wall and narrowing of the lumen of the arteriole in muscles. The clinical course was rapidly progressive.

These observers conclude that the primary cause of diffuse hypertensive vascular disease is still mysterious. Syphilis is so rarely present that it does not seem to be a factor. Hypertension, it seems, may be due to an inherent disturbance of the sympathetic nervous system or it may be due to the action of a hypothetic pressor substance on the sympathetic chain, or sympathetic endings in the wall of the arteriole, or directly on the smooth muscle of the vessel wall.

They suggest that the presence or absence of the above described lesions in the arterioles of muscle may determine the diagnosis and prognosis in the individual case of hypertension.

Malignant Nephrosclerosis (Fahr).

Allbutt, recognising the primary importance of hypertension in the causation of cardiac or cerebral failure, stressed the distinction betwwen Bright's disease and another malady (Hyperpiesia) which " abuts upon cardiac defeat or apoplexy, and does not, at any stage, even of a fatal career, present uraemic symptoms." He apparently did not recognize or include cases of primary hypertension with renal insufficiency.

The strict clinical differentiation between primary hypertensive conditions existing with and without renal insufficiency, and the recognition of pathologic criteria of two types of vascular renal disease corresponding to these two clinical forms were first established by Volhard and Fahr. Whereas only arteriosclerosis was supposed to be associated with the simple or benign hypertension, a combination of atherosclerosis with inflammatory renal changes was held to be responsible for a malignant form of hypertension, the "Kombinations-Form." A toxic factor was postulated by Volhard and Fahr, which, superimposed on the atherosclerosis of the small vessels, produced the combination form.

Since 1918, Volhard has changed his views, and no longer believes in the inflammatory nature of the glomerular and vascular changes. He now considers these lesions as the result of a prolonged ischaemia caused by a permanent vascular spasm.

Klemperer and Otani (30) made a detailed macroscopic and microscopic study of eighteen cases (classified as malignant nephrosclerosis) which came to autopsy. As renal insufficiency had developed in all the cases before death, it is suggested that malignant nephrosclerosis represents the terminal phase of "malignant hypertension (characterized by progressive severe hypertension and neuroretinitis"). The microscopic features that permit a differentiation of these cases from other vascular, or from inflammatory renal diseases are, (1) arteriolonecrosis and extreme cellular intimal thickening of the larger interlobular and so-called arcuate arteries, and (2) degenerative, proliferative and slight exudative focal glomerular lesions. Focal glomerular alterations characterized by fusion and necrosis of capillary loops, endothelial swelling and epithelial proliferation and desquamation, even with occasional crescent formation and slight accumulation of

leucocytes within the capillaries were a constant feature in the microscopic observations. The great majority of the malpighian corpuscles showed, in every case, a conspicuous anaemia of the capillary tufts. These observers, from comparison with the microscopic appearances in aseptic renal infarcts, concluded that these glomerular changes were due to ischaemia. They maintain that the epithelial proliferation is a compensatory reaction to the original epithelial damage brought on by inadequate blood supply and that the accumulation of leucocytes within the loops is due to the chemotaxis of the necrobiotic cells, a process similar to that seen in the vicinity of aseptic infarcts. They found fatty degeneration always present in the arteriolar wall, and in opposition to Fahr, inflammatory changes always absent in the necrotizing arterioles. They therefore believe that the process is primarily degenerative and is an expression of a severe grade of arteriolosclerosis. The acceleration of the atherosclerotic process in the small cortical arteries of the kidney is the essential pathogenic principle and leads subsequently to ischaemic damage of the arterioles and glomeruli. The extensive

haemorrhages in numerous places in the kidneys of these cases were due to rhexis of small necrotic arterioles, as well as to diapedesis from capillaries that apparently were overfilled because of retrograde passage of blood into the areas where the arterioles had become thrombosed. In all cases extensive, macroscopic haemorrhages were dotted over the surface of the kidneys.

The average age in this series of cases was thirty-nine years. In agreement with Fahr and other authors, these investigators found that the majority of cases occurred in persons below fifty years; and this is contrasted with the situation in cases of benign sclerosis in which the majority of the patients were above fifty years. Cases occurred equally in both sexes, and in several cases the hypertension had been discovered accidentally in examination for life insurance, with the patient apparently in perfect health. Especially striking was the rapid course of the illness, which went on without remission and could not be influenced by therapy once the uraemic symptoms had appeared. The heart was markedly enlarged in every case.

The constriction of the lumen of arterioles

was caused in every case by cellular proliferation of the intima, the breadth of which exceeded by far that of the media.

The authors consider that the rapidly developing obliteration of the renal (vascular) bed is responsible for the sudden onset of fatal renal insufficiency.

The etiology of the vascular condition is unknown. It is conceivable, and in accord with Volhard's conception, that a constitutional or acquired angiospastic factor plays a determining role in producing the accelerated atherosclerotic form.

Malignant Hypertension.

Keith Wagener and Kernohan applied the term malignant hypertension to a clinical condition with the following characteristics; persistent hypertension with a progressively downward course, moderate or no renal failure, absence of anaemia, cardiac hypertrophy, and distinctive changes in the retina. Anatomically they emphasized the widespread involvement of the arterioles of the whole body.

Murphy and Grill (17) group under this heading; "malignant hypertension", "malignant nephrosclerosis of Fahr", "genuine contracted kidney", and "chronic interstitial nephritis". They found headache an outstanding symptom in all cases; it was the first symptom complained of in most cases, and often made life almost unbearable. Marked weakness and loss of weight were present in all cases, and a constant feature was choked disc or retinal oedema accompanied by haemorrhages. The arteries were narrowed, and in some cases straight, in others tortuous. The retinal changes were indicative of various degrees of neuroretinitis. In common with the findings of Evans (18) and Shaw (19) in diffuse

hyperplastic sclerosis, medial hypertrophy of the arterioles of skeletal muscles was usually found. Evans (18) stated that medial hypertrophy was not however, the essential element, nor a constant feature. The main feature, in his opinion, is an intimal thickening. The medial hypertrophy he considered, might be attributed to the hypertension.

Clinically it appears that malignant hypertension is a late phase of benign hypertension, differing from the benign form in the greater severity of its symptoms, the more rapid functional breakdown of the essential organs of the body, and the more persistent and more excessive hypertension. Some persons who have had benign hypertension for years may, for unknown reasons, develop the malignant form. In this form there are no remissions; progress downwards is so rapid that it appears justifiable to call this the most malignant of all renal disease.

Murphy and Grill (17) studied sixteen cases, thirteen of whom died, with autopsy on twelve. The changes in the smallest arteries and arterioles found by them may be summarised as follows:-

(1) The lumina were almost universally narrowed and were sometimes obliterated by thickening of the vessel walls.

(2) Hyperplastic elastic thickening of the intima was found, with proliferation of the connective tissue of the intima.

(3) Fatty and hyaline degeneration involving the intima, and frequently the media, was prominent.

(4) Medial thickening, consisting of hypertrophy of the muscular tissue, frequently occurred alone or in association with degenerative lesions of the intima.

(5) Degenerative and proliferative lesions in the intima were usually accompanied by medial atrophy.

(6) Occasionally there was periarterial lymphocytic infiltration of the interstitial connective tissue surrounding the smallest arteries and arterioles of the kidney.

(7) Necrotic lesions were present in the walls of the arterioles and in the loops of the corresponding glomeruli in six cases.

(8) Combinations of these lesions were at times, found involving the same vessel; in other cases the various lesions could be demonstrated

side by side in the same histologic specimen.

(9) The vessels of the skeletal muscles frequently showed thickening of the media, with normal intimal tissue.

(10) The vessels of the lung were normal in all but one case.

Arteriosclerosis.

Janeway (21) as the result of his painstaking clinical investigations, was of opinion that arteriosclerosis of the larger superficial vessels, is without marked influence on blood pressure; that high blood pressure argues involvement of the small arteries, especially in the splanchnic circulation, and that such cases should be classed as a separate category.

Gager (2) quotes Lange and Pal as to the antagonism between high pressure and arteriosclerosis. Lange concluded that the vascular sclerosis in the latter is due, not to the organically narrowed vessels but to a primary weakness of the media. Conversely the vascular hyper-irritability in the patient with hypertension is apparently based upon excessive sympathetic stimulation of the musculature of the small vessels.

In spite of the early confusion between the two conditions, it is now a well-recognised clinical fact that even very severe grades of arteriosclerosis may exist with a normal blood pressure.

Modern Lines of Treatment.

Sir Humphrey Rolleston (22) states that high blood pressure is a manifestation of some underlying disorder - metabolic, renal, dietetic, toxaemic, infective or psychological (this is spreading the net rather widely), and though it may reasonably be regarded as responsible for subsequent arteriosclerosis, renal fibrosis, and cardiac failure, many if not most of the symptoms found in persons with abnormally raised blood pressure, and in common usage regarded as due to it, can be better explained as either concomitant effects of the underlying course or of the resulting or complicating arteriosclerosis, renal or cardiac disease. It is far more important to treat the underlying conditions or causes just mentioned than to concentrate on reducing the height of the blood pressure, which to some extent is a compensatory mechanism, for to bring it down rapidly and considerably may only make the patient, if indeed, he was one before,

worse. Prophylactic measures in the initial stages are, of course, the most effective means of treatment.

Preventive or Prophylactic Measures. (Rolleston 22)

A periodic overhaul, once or perhaps twice a year, is an insurance against chronic disease, and is particularly desirable in those with a family history of hypertension. The discovery that the blood pressure is rising steadily or is considerably above normal indicates a review of the conditions of life in order to obviate any causes considered as predisposing to the malady, or even intensifying it. A hard working man of affairs must relax the stress and strain of life, and in fine, order his life at a lower level of tension. An injunction to cease from worry is best backed up by a prescription of bromide or some nerve sedative. "The moral is moderation in all things, and freedom from strain of every kind, bodily and mental. A search must be instituted for any chronic focus of infection in the teeth, tonsils, accessory nasal sinuses, gall-bladder, ~~and~~ appendix, AND prostate, and indigestion and intestinal disorder should receive attention." Janeway (21) recommends, "the regulation of the patient's entire

life as the starting point of treatment." It should include the avoidance of all those forms of exertion and excitement which cause a considerable increase of arterial pressure, especially of a sudden nature.

Management of fully established hypertension.

The above-mentioned prophylactic measure of slowing down the rate of life - living one's life at a lower level, so to speak,—also holds good in these cases. Among the many, very many, therapeutic agents listed in the treatment of this disease we find - diet, drugs, diathermy, rest in bed, baths and venesection.

Diet. The general practice is to reduce the protein and to allow some white meat, chicken and fish, but to proscribe red meat, a distinction (as Rolleston 22 says) making little or no difference to the patient's condition. Rather more recently a salt-free diet has been enthusiastically recommended, but it has not apparently come up to the expectations of its originator. Tobacco is generally regarded as detrimental in hypertension, though without any really established basis in fact. (The therapeutic effect is an immediate, though

transient, rise of blood pressure, followed by a fall to below normal). As Allbutt (23) truly remarks, "If we expect by however skimpy and meticulous a dietary to control the blood pressures, we shall be disappointed." The same author remarks that saline laxatives in moderation no doubt have their occasional value, but their regular morning use in respect of the reduction of blood pressures, brings no advantage.

Drugs. Rolleston (22) maintains that drug treatment is the least important, and it is significant that so many have been advocated. Bromides are useful in relieving mental strain. The vasodilators (nitrites) have only a transient influence, but have their uses in anginal crises, and sometimes relieve headache. Calomel in $\frac{1}{2}$ to 1 gr. doses weekly seems to benefit some cases. Iodides are given a prominent place in drug treatment, but mainly from a fictitious traditional reputation in this disorder. It has been suggested that they act indirectly by stimulating the thyroid, but the disappointing action of thyroid extract by mouth is not convincing.

Diathermy. Most authors speak well of this method. Allbutt (23) believed it was the most valuable immediate aid we possess for hyperpiesia. It keeps the pressure down much longer than high frequency currents, so that eventually monthly treatments are sufficient to maintain a reduction permanently at a level appropriate for the individual patient. The treatment is given for about ten minutes daily on a couch.

Rest in bed, undoubtedly lowers the blood pressure in nearly every case - sometimes to quite a considerable extent. Unfortunately, however, the pressure quickly returns to its former level when the patient resumes his daily occupation. Shaw recommends that a patient with proclivity to high blood pressure, if engaged in business, should take an occasional week-end in bed. On the other hand, too much bed is unwholesome (22).

Baths. Warm baths have been recommended as likely to relieve vascular spasm. Their value, however, has not escaped criticism, for they raise the systolic pressure slightly while lowering the diastolic pressure to a greater degree, and thus

increase the work of the heart. (22).

Venesection is a temporary remedy - its effect is very transient. The one crisis for which it is indispensable is pulmonary oedema (23); then it must be prompt and combined with an injection of atropine. Douthwaite (6) says that thyroid extract is useful in obese, menopausal hyperpietics ($\frac{1}{2}$ gr. t.i.d., and up to ten times that amount). He found that Veratrone (veratrum viride) has a definite effect in lowering blood pressure. Given by mouth ($\frac{1}{2}$ - 1 ccm.) it will, in many cases, lower the blood pressure from 20 - 30 mm. Hg., and keep it about this level for two hours. The drug must be used with great caution in view of the possibility of idiosyncrasy (sudden large drop in blood pressure). Intramuscular injections of $\frac{1}{2}$ ccm. reduces blood-pressure by 70 - 50 mm Hg.

Acetylcholine stimulates the para-sympathetic system and antagonizes adrenalin. Its effect in lowering arterial tension is by dilatation of the arterioles, the capillaries being unaffected. Douthwaite used Acécoline, which must be dissolved immediately before use, and the solution injected intramuscularly. (It is supplied in sealed ampoules

with similar ampoules of doubly-distilled water). Oral administration is useless, while intravenous injection is extremely dangerous (because of sudden large drop in arterial pressure). For this reason the piston of the syringe should be withdrawn before injection, to ensure that the needle is not in a vein. The initial dose should be 0.05 grm, raised subsequently to 0.1 grms daily. It may be used for fifteen consecutive days each month for three months. M. Villaret (24) speaks very highly of this therapeutic agent, and, as a result of a prolonged clinical trial extending over five years, recommends it in the painful complications of hypertension involving angiospasm, notably in senile arteritis and intermittent claudication. He does not find that it is of lasting value in the actual reduction of hypertension, but maintains that it is of undoubted value in lead colic and the gastric crises of Tabes. (Incidentally it is recommended as the remedy'par excellance' in Raynaud's disease).

Liver Extract.

The use of a special liver extract by certain Canadian and American investigators has already been commented upon. Beckman quotes Major's most recent pronouncement on the remedy; "We still feel, as previously stated, that definite conclusions can be drawn only after a large number of patients have been treated over a long period of time. The evidence at the present time, that the liver extract is effective in certain cases of arterial hypertension, is very suggestive, but is not conclusive.

Sulphocyanates. (Thiocyanates, "Rhodan").

The sulphocyanates were first used by some German investigators, and were found to have a distinct hypotensive action. On the theory that cholesterol was the chief of physico - chemical factors involved in the development and persistence of contraction in the smooth muscle cell of the arteriole, they were introduced as agents for dissolving the cholesterol and bringing about relaxation of the vascular musculature. Owing to the large doses used at first (1 grm t.i.d.) ~~several~~ toxic symptoms arose in several cases, but with a dose of 0.1 grm t.i.d., these toxic symptoms (eruptions of the skin

and catarrh of the mucous membranes) do not appear. Gager (2) who has made extensive use of Potassium Sulphocyanate, speaks very highly of the remedy. "While the results of sulphocyanates have been almost uniformly satisfactory when the patients were co-operative and the physical obstacles to be overcome were not too great, it is not a panacea for hypertension. It does not supplant any of the fundamental hygienic and dietary measures for the control of high blood pressure, and its ultimate action on the arteriolar smooth muscle cell may not be different from the effect of weight reduction or other means of influencing cellular metabolism." Insufficient renal excretion is a contra-indication to its use.

Bismuth Subnitrate.

This drug has been much used by foreign experimenters of recent years. Stieglitz (31) attributes its value to a continuous slow absorption of nitrite ions, and a chloride diuresis, produced at the same time that arteriolar relaxation is taking place.

Bensaude and Cottet (25), as a result of extended clinical trial, conclude that in dyspeptic hypertensives treated by Bismuth Subnitrate there is

produced a fall in arterial tension which leads them to believe that the hypertensive state is often conditioned, at least in part, by the gastro-intestinal condition.

In this connection it is of interest to recall that Oliver (12) lays stress on the sedation of the gastro-cardiac reflex in supernormal pressure of the paroxysmal type, as in Angina Pectoris. To soothe the nerve-endings in the stomach he recommends Bismuth among other sedatives.

Cerebral Haemorrhage.

The treatment of this serious complication of hypertension seems to resolve itself in many cases into adopting a policy of masterly inactivity! According to Beckman (1), during the period of bleeding the application of an ice-bag to the head and the placing of the feet in a warm mustard bath are time-honoured remedies. It is the usual practice to raise the head and lower the feet. The attempt to reduce blood pressure and decrease the haemorrhage by venesection is now discountenanced. After recovery from the acute attack, potassium iodide in large doses (beginning with gr. $\overline{v}$ t.i.d. and increasing to the limit of toleration) is employed

in the attempt to promote resolution of the clot; but this medication must not be begun until several weeks after the haemorrhage.

Stimulants of whatever sort are contraindicated in the acute stage.

Janeway (11) says that large haemorrhages produce marked hypertension, which is more extreme the greater the rise in intracranial tension. A high and rising blood pressure indicates progressive failure of circulation in the medulla, and an increasing haemorrhage. On account of the 'circulus vitiosus' established under such conditions, this rise in pressure is productive of more rapid bleeding; while an artificial reduction of tension by drugs, derivation or bleeding, will be likely to kill the patient by the cessation of cerebral circulation, which results the moment the general arterial pressure falls below the intra-cranial. In consequence some surgeons advocate immediate operation with relief of intra-cranial tension and evacuation of the clot, if possible. With a stationary or falling pressure it should not be thought of.

Albutt (10), from his long experience, suggested that hyperpiesia is an hereditary malady, and so

also is the disposition to its particular consequence of cerebral haemorrhage, as contrasted with the cardiac mode of death.

Gunewardene (27) in a clinical survey of one hundred and fifty cases of apoplexy observed by him, points out (a) that sudden motor and sensory changes may occur lasting minutes, hours or days; (b) that these are not uncommon and the diagnosis and prognosis are not satisfactory; (c) that although these strokes, both motor and sensory, are frequent, they do not occur when the diastolic pressure is under 115 mm. Hg. He also draws attention to the fact that patients with hyperpiesia without symptoms of cardiac failure are those specially prone to cerebral haemorrhage.

Hypertension a Symptom.

It will be noted that practically all the above-mentioned therapeutic agents act by lowering the blood pressure, or at least are employed with that object in view. Martinet (26) emphasizes that high blood pressure is not a disease, but a symptom, which is common to disorders widely diverse, and of complex pathologic physiology. Accordingly there is

no specific for high blood pressure. "There is no single treatment for lowering blood pressure, and the therapeutic plan to be followed should be adopted to the existing clinical and pathophysiologic modality of high blood pressure."

For practical purposes it may be regarded as a fact that any reduction of the systolic pressure which is accompanied by a rise of the diastolic pressure is an index of failure of the myocardium, and is of ominous significance. Any progressive reduction of the systolic pressure which is accompanied by an appreciable reduction of the diastolic pressure is an index of a true primary low blood pressure, and is generally of favourable significance. Clinical observation shows, however, that in a given case of high pressure, there is an irreducible lower limit of high pressure which cannot be overstepped without prejudice to the myocardium.

Author's Observations.

The following figures are based on blood pressure estimations of 403 consecutive patients in an industrial area. The total number investigated represents 225 males and 178 females of all ages.

Of the 225 males, 39 were found to have abnormally high blood pressures. Of the 178 females 49 were found to be suffering from hypertension.

Any doubtful or border-line cases were observed repeatedly to avoid error due to excitement, etc.

Most of the readings in younger patients were taken from the lower limb, with the patient lying horizontally.

The following are the relevant findings:-

1. Total percentage high systolic B.P. = 21.8
2. Percentage high systolic B.P. in women = 27.5
3. Percentage high systolic B.P. in men = 17.3
4. Total percentage high diastolic pressure = 19.2
5. Percentage high diastolic B.P. in women = 21.7
6. Percentage high diastolic B.P. in men = 17.3

These figures bear out the common finding that both systolic and diastolic pressures tend to show a higher incidence in the female sex.

The percentage incidence in different decades is shown in the following table:-

High B.P.-Percentage Incidence in Different Decades.

(See Graph.)

	Percentages.						
Males.	0.	7.7.	9.3.	13.0.	26.2.	40.7.	50.0.
Females.	8.3.	7.7.	30.8.	47.6.	61.9.	63.6.	71.2.
Age.	10-20.	20-30.	30-40.	40-50.	50-60.	60.70.	70-80

The findings here indicate a much higher incidence of hypertension in females than in males. The sharpest rise in males occurs in the age period 60-70 years, whilst in the females there is a sharp rise in the 30-40 decade, and a somewhat similar one in the 50-60 decade. Marked rise of hypertension evidently manifested itself in females a decade or two earlier than in males.

The three to one preponderance in females in this series is not the usually accepted finding, though Gager (1) mentions a two to one preponderance

Name

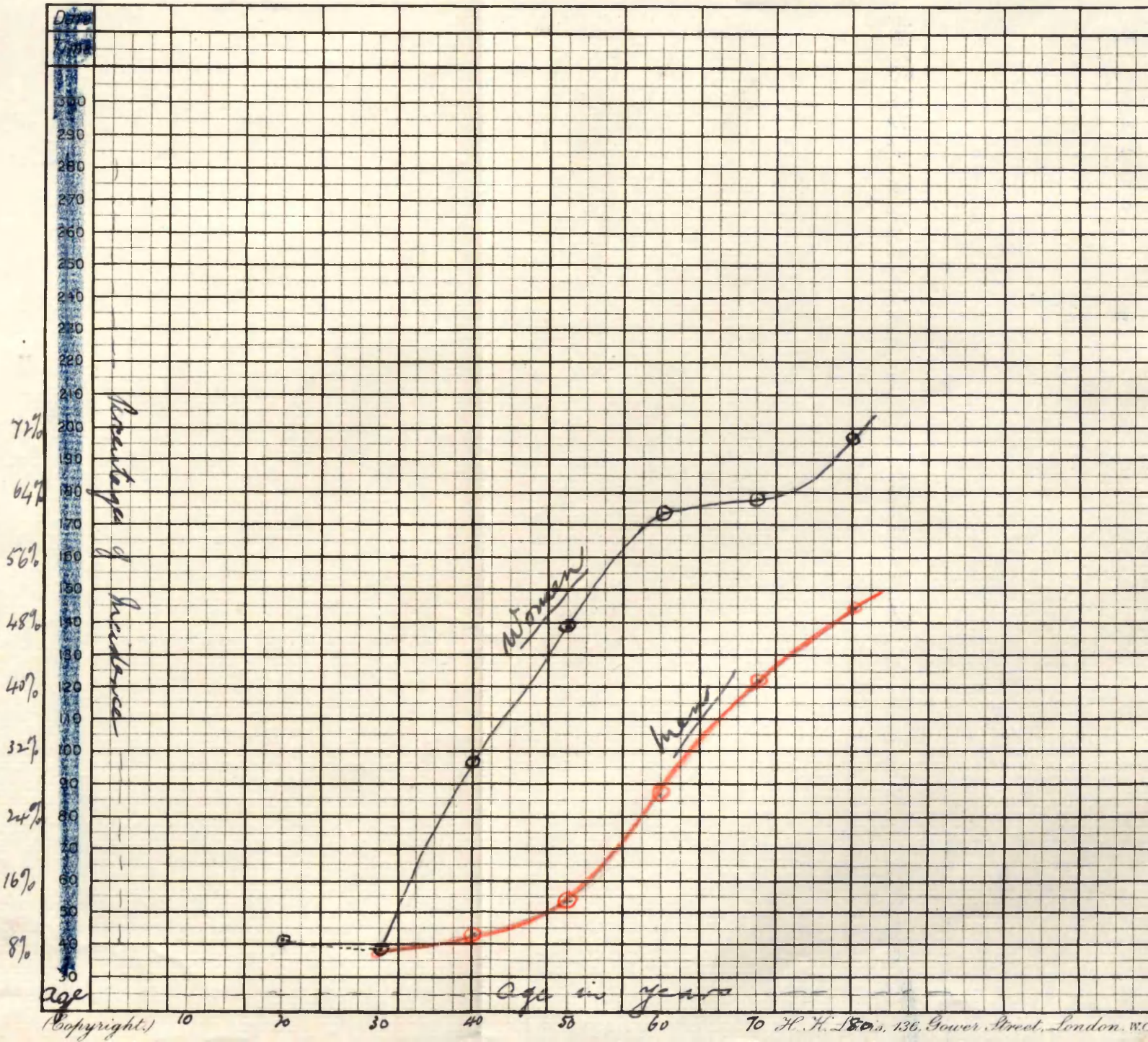
Age

Disease

Notes

High Blood Pressure

Percentage Incidence in different Decades.



in females for Boston, U.S.A. A larger number of cases would probably tend to reduce this apparently very high incidence, though it is remarkable that the figures for males bear a very close resemblance to those ordinarily accepted for large numbers of cases. (see reference 1.)

No cases of primary cardiac disease of syphilitic origin, nor of exophthalmic goitre have been included in the above cases.

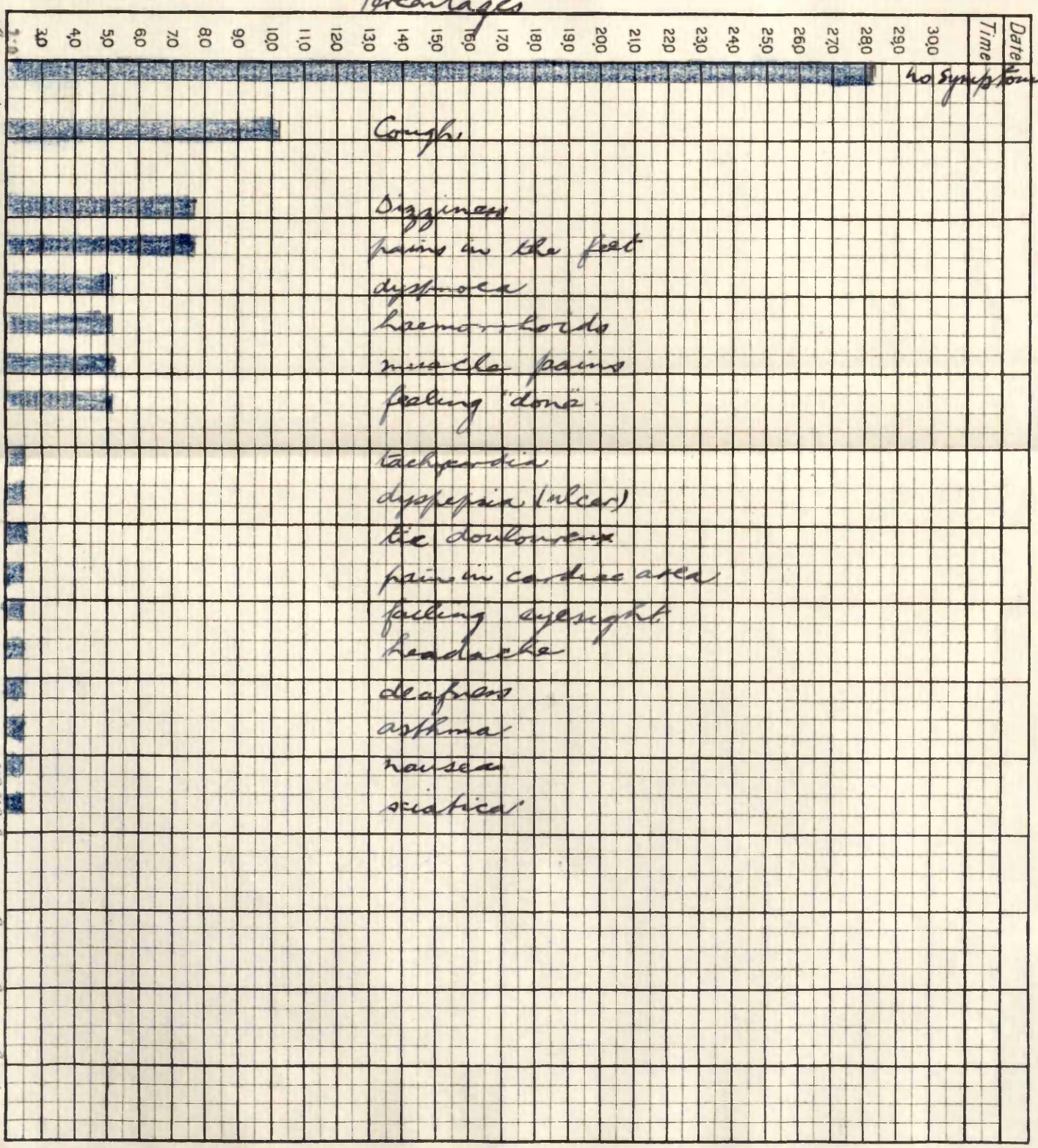
In addition to the 403 patients specified above, there were nineteen additional patients suffering from high blood pressure - eleven females and eight males. These were classified as follows:-

Disease.	Males.	Females.
Rheumatic Endocarditis.	1	6
Chronic Nephritis.	3	1
Syphilis.	1	2
Toxic Goitre.	0	1
Nephritis of Pregnancy.	0	1
Neurasthenia.	1	0
Myocarditis.	2	0

These, however, have been included merely for completeness, as this paper is intended to deal mainly with cases of essential hypertension, in so far as the condition may be daignosed clinically.

Hypertension - 39 cases

Frequency of Presenting Symptom - (men)



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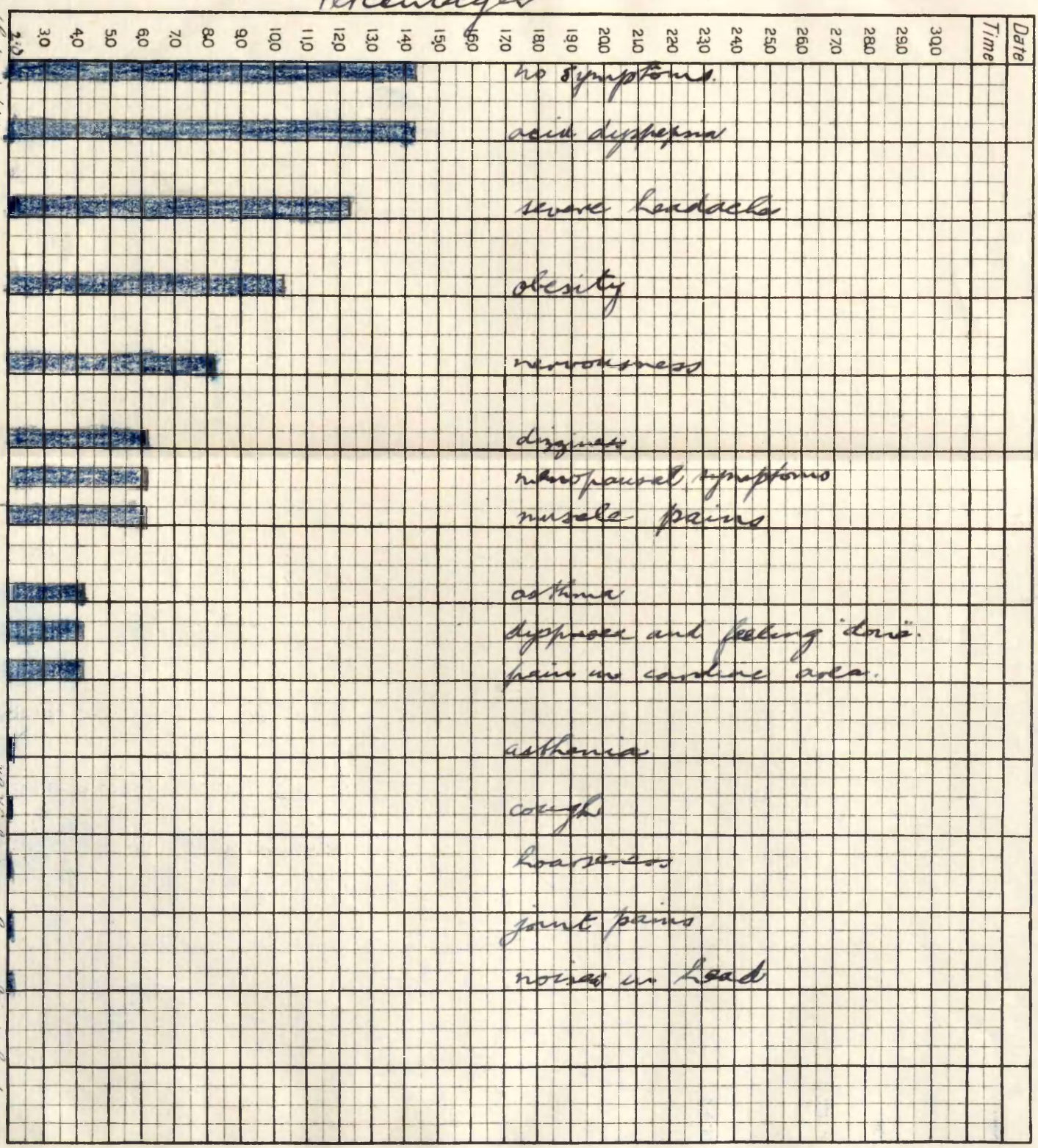
LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Hypertension – 49 cases.

Frequency of Presenting Symptom – (Women).

Percentages



(Body weight.)

LEWIS'S BLOOD PRESSURE AND PULSE CHARTS

H. K. Lewis, 136, Gower Street, London, W.C.

Frequency of Symptoms. (See chart).

The following table gives a classification of the presenting symptom in each of the eighty-eight cases of essential hypertension.

Men Total = 39 cases.

Presenting Symptom.	No. of cases.	%age of total.
No symptoms	11	28.2
Cough	4	10.2
Dizziness)	3)	7.7.)
Pains in Feet)	3)	7.7.)
Dyspnoea	2)	
Haemorrhoids	2)	5.1
Muscle Pains	2)	
Feeling 'done'	2)	
Tachycardia)		
Dyspepsia (Ulcer))		
Tic Douloureux)		
Pain in cardiac area)	1	2.5
Failing eyesight.)		
Headache)		
Deafness)		
Asthma)		
Nausea)		
Sciatica)		

Frequency of Symptoms. (See chart)

Women Total = 49 cases.

Presenting Symptom.	No. of cases.	Percen: of total
No Symptom	7	14.2
Acid dyspepsia	7	14.2
Severe headache	6	12.2
Obesity	5	10.2
Nervousness	4	8.1
Dizziness Muscle pains Menopausal symptoms	3	6.1
Asthma Dyspnoea and feeling 'done'. Pain in cardiac area.	2	4.1
Asthenia Cough Hoarseness Joint pains Npises in head.	1	2.0

The foregoing tables represent merely the frequency of the presenting symptom in the two groups of cases. On going into the history and attendant symptoms one is struck by the frequency with which the following are given a prominent place:-

1. Attacks of dizziness.
2. Nervousness.
3. Headache.
4. High colour (Face).

Obesity was a factor in 19 of the female cases (38.7%) and in only 3 of the males (7.7%).

The cases listed as presenting no symptoms usually came for some minor injury or ephemeral infective condition. These cases, taken in conjunction with the varied symptomatology already listed, make it clear that blood pressure estimation should be an integral part of every medical examination, especially when one considers the extra mortality associated with hypertension, as evidenced by the figures given in an earlier part of this paper.

Two cases (one male, one female), are suffering from old hemiplegia, from which they have recovered to such an extent as to be able to get about without mechanical support. The male patient is able to walk to the surgery from his home - a distance of about a quarter of a mile.

One patient,(first coming under observation in 1928) has died since the present series of cases was begun. She was very obese, and developed

symptoms of malignant hypertension in February 1931. Death occurred very suddenly in May 1931 from cerebral haemorrhage.

Summary of illustrative cases.

1. Symptoms.

Mrs B., age 37 years, 4 children.

Pains in back (1928) diagnosed as myositis. February 1930 - complaint of pains in the head every week for two or three years. Had severe pain in the leg two weeks previously. February 1931 had very severe, intractable pain in left sciatic region, and was in Stobhill Hospital for two months. Eye-sight had been failing fast. Headaches were very frequent and very severe, up to time of death. Losing weight rapidly. (See chart). (AT END OF THIS PAPER)

Physical Signs.

Obese, red-faced, very nervous women. During examination watched everything most apprehensively. Radials rubbery - not sclerosed. Brachial arteries barely palpable, but showing visible pulsation. Rapidly-beating loud heart. Apex beat in 6th interspace. Left border of heart one inch outside mid-clavicular line. Apical systolic murmur (not marked). Second aortic sound loud and musical.

Marked hyperaemia and oedema of both discs. Numerous haemorrhages and patches of sclerosis in retinae. Several large haemorrhages near disc in left fundus. Arteries thin and 'silvery' in appearance.

Urine - no albumen.

Patient died suddenly on 3/6/31 from cerebral haemorrhage. She said she felt as if her head was bursting-and collapsed. Two hours after death - several large haemorrhages in both fundi - oedema of discs and pupils widely dilated.

Diagnosis. Malignant Hypertension.

Note. (Daughter, aged 15 years showed B.P. 130/90).

No. 2.

Miss B., age 42 years. Domestic servant. B.P. 160/90.

Symptoms. August 1930 - painful and frequent micturition. Urine showed bacilluria, no albumen. May 1931 - nose red and inflamed, and complained that stomach seems to swell now and again.

Physical Signs. Slightly obese. ~~Acne~~ ACNE Rosacea on face. No abnormalities detected on physical examination. Second aortic sound not loud.

Diagnosis. Essential Hypertension. (See chart).

No. 3.

Mrs. C., age 30 years. B.P. 160/100. P. 90.

Symptoms. Red soft swelling above left ankle. Complaint of increase in weight for past three years. Says left ankle dislocated 9 years ago.

Physical Signs. Obese, red-faced woman. Had chilblains in 1923. Tonsillitis in 1925. Married 5 years - one child. Obesity dates from birth of baby. No abnormalities detected on physical examination. Urine showed no sugar, no albumen. X-ray of swelling was negative for bone disease.

Diagnosis. Essential Hypertension (of endocrine origin?).

No. 4.

Mrs. C., age 52 years. Skinworker. Has worked hard all her life. Husband a worry to her. He is puny and has not worked for years.

History. Phlebitis and painful varicose ulcer of leg in 1921. 1926 - varicose ulcer again discharging. 1927 - ulcer quite healed. 1929 - Vertigo and ovarian pain. Ovaries (both) removed in Samaritan Hospital (age 30 years). (See chart).

Symptoms.

Complaint of vertigo and stoutness, nervousness, depression, flushings and sweatings since operation. April 1931 - tightness in chest and severe cough. Needles and pins in fingers when working. Very short of breath; complained greatly of the cough with thick yellow sputum. Said she can't 'gather any strength'. June 1931 - feels much better after Bromide and Tonic medication, with two weeks' holiday in Largs.

Physical Signs.

Obese, worried-looking, red-faced woman. Healed varicose ulcer left leg. Visible pulsation in veins of neck. Arcus Senilis present. Apex-beat not palpable. Radials not sclerosed to palpation. Left border of heart in nipple line. Second aortic sound not loud. Fundi-arteries thin, veins engorged, some pigmentary degeneration near right disc, no haemorrhages. No râles in chest, no dullness. Urine - no sugar, no albumen.

Diagnosis.

Endocrine Dysfunction with Hypertension.

No. 5.

Miss C., age 63 years. Drapery Saleswoman. B.P. 150/94.

History.

1921 - Effusion (painful) into left knee. 1925 - Laryngitis. 1929 - Influenza and phlebitis of saphenous vein of right thigh. 1931 - Arthritis of knee-joint.

Symptoms.

Small, stout, red-faced woman. Very nervous and jerky. Painful arthritis of knee-joint which has yielded to ultra-violet ray therapy. No abnormalities detected on physical examination, except for mild sclerosis of radials.

Diagnosis.

Hypertension.

No. 6.

Mrs C., age 44 years. Weaver. B.P. 158/84.

Symptoms. June 1930 - severe headaches, 'sort of dumb feeling all over'. Takes shivering fits, which she ascribes to 'nerves'. Menstruation regular. Pains up back of neck and in legs. Haemorrhages into conjunctiva (left eye) - no pain at time of occurrence.

Physical Signs. Thin, spare, pale, nervous looking woman. Very septic teeth (extraction advised) in March 1931.

Diagnosis. Hypertension.

No. 7.

Miss D., age 37 years. Housekeeper. B.P. 214/120

History. 1925 - Chlorosis. 1929 - Colles' Fracture of left wrist.

Symptoms. Very nervous and apprehensive. April 1931 - no appetite, sourness in stomach (3 weeks), no pain, no previous stomach trouble. Considered rather 'peculiar' by previous employer. Menses absent three months.

Physical Signs. Swelling of lower abdomen gave sensation of cystic tumour in region of uterus. Sent to Samaritan Hospital, and on eve of admission for operation developed Rubella. Sent to Belvedere Hospital (3 weeks). Operation in Samaritan temporarily postponed. Arteries not sclerosed. Apical systolic murmur (soft), aortic systolic murmur, second aortic sound loud and ringing, left border of heart one inch outside nipple line. Apex-beat in 5th interspace (nipple line). Fundi show thin arteries, no haemorrhages. In May, 1931, complaint of "terrible headaches and weak turns."

Diagnosis.

Ovarian tumour and Hypertension.

(see chart).

No 8.

Miss F., age 34 years. Shop-assistant. B.P. 150/80. Weight 15 stones.

Symptoms. Deafness and noises in head.

Physical Signs. Very obese woman with large pendulous breasts. Perforation (old) in right tympanum. No other abnormalities detected on physical examination. Urine - no albumen, no sugar. Diet prescribed.

Diagnosis. Old otitis media. Obesity and Hypertension.

No 9.

Mrs F., 51 years. Charwoman. B.P. 194/106.

History. 1929 - Rheumatic pains in back and shoulders. 1930 - Pessary inserted every 2 months for old prolapse.

Symptoms. No complaint. Routine examination disclosed hypertension.

Physical Signs. Very obese, red-faced woman with vaginal prolapse. No other abnormalities detected on physical examination.

Diagnosis. Vaginal prolapse and Hypertension.

No. 10.

Miss G., age 26 years. Invoice Typist. B.P. 150/80.

Symptoms. Erythematous eruption on face and arms - never rid of it.

Physical Signs. Erythema on chest. Small red papules on arms. No other abnormalities detected.

Diagnosis. Erythema and Hypertension.

No. 11.

Miss G. Age 32 years. Dressmaker. B.P. 160/86. P. 90.

Symptoms. 1930 - Swelling and oedema of feet, and frequent micturition (one week). Back sore (one week). 1931 - Ankle swollen, bunion on big toe.

Physical Signs. Thin, apprehensive, jerky girl; urine - no sugar, no albumen, no pus. No other abnormalities detected.

Diagnosis. Vasomotor disturbance and Hypertension.

No. 12.

Mrs. K., age 20 years. Usher (Picture House).

History. Married at 18 years. B.P. 150/98.

Husband ill-treated her after one year. Left her husband and living with her father. Baby born shortly after left husband. 1930 - had tonsillitis.

Symptoms. 1931 - Feels tired and 'done' for some months past. Finds her work is too much for her. Irritating vaginal discharge since birth of baby. (Very worried over this). Says her hands and feet are swollen at night.

Physical Signs. Small, stout, worried-looking woman. Very depressed. Septic vaginal discharge with dermatitis of thighs. No other abnormalities detected. (Sent to Samaritan Hospital and now on waiting-list for cervical repair).

Diagnosis. Neurosis and Hypertension.

No. 13.

Miss M. Age 32 years. Chocolate-coverer.

Symptoms. 1929 - Swelling in neck (goitre).

Physical Signs. Pulsation in veins of neck. Goitrous swelling in mid-line. Eyes staring and prominent, marked ~~tumour~~ TREMOR on extending hands. 1930 - X-ray treatment for six months. 1931 - Attending Hospital twice a week for Ultra-Violet ray therapy. Heart enlarged ($\frac{1}{2}$" outside nipple line). Vigorously

beating 'loud' heart. No murmurs. (See chart).

Diagnosis. Toxic Goitre and Hypertension.

No. 14.

Miss McC., age 36 years. Metal-Finisher. B.P. 160/86.

History. 1928 - Septic Adenitis (axillary) from coloured blouse. Mother suffers from Hypertension for some years.

Symptoms. Buzzing in left ear (deaf for many years). Discharge sometimes from this ear.

Physical Signs. Rather nervous individual. Old perforation in tympanic membrane. Membrane acutely inflamed. No sclerosis of blood-vessels.

Diagnosis. Recurrent Otitis Media and Hypertension.

No. 15.

Mrs. McC. Age 65 years. Hair-worker formerly. B.P. 170/95. P. 90.

History. 1921 - Synovitis left knee. 1923 - Pyorrhoea Alveolaris. 1924 - Rheumatism. 1925 - Bronchitis. 1926 - Enteritis and Bronchitis. 1927 - Sugar in urine. 1930 - Feels done, pains all over, and severe cough. No sugar in urine. Nov. 1930 - Herpes Zoster, Diarrhoea and pain in shoulder.

May 1931 - Severe cough and abundant expectoration.

Symptoms. 1931 - Severe barking cough with a good deal of thick sputum. Can't sleep at night on account of cough. Very nervous woman.

Physical Signs. No palpable sclerosis of blood vessels. Second aortic sound slapping in character. Heart not enlarged to percussion. No murmurs. Skin warm and moist (perspires a good deal). Barking cough. No râles nor dullness in chest. Eyeballs slightly prominent; no enlargement of thyroid gland. Urine - no sugar, no albumen.

Diagnosis. Hypertension.

No. 16.

Miss. McG. Age 37 years. Tailoress.

6/5/31...B.P. 164/94. P. 108.

30/5/31..B.P. 190/100. P. 120.

Symptoms. Headache - noise-at night and at menstrual periods. Pains in all her joints (two weeks). Menstruation normal. Putting on weight recently. Menstruation now lasts only two days instead of four. Three weeks later, hands swollen, and stiffness in fingers, neck and arms - can't close her fist on account of swelling.

Physical Signs. Marked obesity. Very rapid heart - not enlarged. No abnormalities detected.

Diagnosis. Menopausal disturbance and Hypertension.

No. 17.

Miss McH. Age 68 years. Housekeeper.

History. 1921 - Haematemesis. 1924 - Influenza.

Symptoms. Since 1924 has had severe headaches from time to time. In 1931 (March) complaint of 'lightness' in the head, vomiting and shortness of breath. Asthmatic attacks frequent, and constant dizziness present.

Physical Signs. Thin, spare woman. Very nervous - easily startled; radials and brachials sclerosed. Visible brachial pulsation. Soft systolic apical murmur. First aortic sound reduplicated, second aortic loud and ringing. Heart not enlarged to percussion. Fundi show pigmentary degeneration round margin of left disc. Spots of pigmentation in both retinae. Veins engorged, arteries thin and wiry. No haemorrhages present. Urine - No sugar, no albumen.

Diagnosis. Hypertension and Arteriosclerosis. (see chart).

No. 18.

Miss McL. Age 21 years. Domestic Servant.

8/4/31..... B.P. 150/88. P. 96.

15/4/31..... B.P. 144/78. P. 84.

Symptoms. 1930 - Inflammatory dermatitis in front of knee (painful). January 1931 - Septic vesicles all over knee and thighs. Teeth very septic (extracted 21/1/31). March 1931 - Dermatitis of legs again.

Physical Signs. Rather obese, lethargic-looking girl. Small, red papules with pustular centres on both legs and thighs - later on arms. Cleared up rapidly on Calamine-Resorcin lotion and tonic treatment.

Diagnosis. Septic Dermatitis and Hypertension.

No. 19. Miss McM. Age 34 years. Library Assistant.

Symptoms. 'Wind' in stomach. Weakness of arms and legs since father's death (three weeks previously) Nursed father through long illness - Cancer of Lung). In June 1931, developed severe itch in head and face and backs of the ears.

B.P. 160/76. P. 90.

Physical Signs. Thin, spare, red-faced, very nervous woman. No sclerosis of blood vessels.

Diagnosis. Neurosis and Hypertension.

No. 20.

Mrs. R. Age 45 Years. Charwoman.

26/5/31 .....B.P. 164/94. P. 96.

6/6/31.....B.P. 150/94. P. 102.

Symptoms. May 1931 - Sciatica. June 1931 - Head very dizzy. Inclined to fall when she bends forwards. Very nervous - jumps if shouted at. Menstruation absent three months. 'Flushings' frequent. Is getting stout (1 year), used to be very thin.

Physical Signs. Very obese, red-faced, nervous woman. No abnormalities detected.

Diagnosis. Menopausal Disturbance and Hypertension.

No. 21.

Mrs. W. Age 46 Years. Mill-worker.

22/4/31.....B.P. 170/94. P. 72.

6/5/31.....B.P. 162/100. P. 78.

History. 1928 - Laryngitis. 1930 - Furuncle of face. Pain in sole of foot and up leg. Very con-

stipated. Laryngitis.

Symptoms. 1931 - Dizziness and hoarseness. Menstruation absent three years. Marked 'flushings' for some weeks. Left pupil larger than right. No other abnormalities detected. Marked obesity.

Diagnosis. Endocrine disturbance and Hypertension.

No. 22.

Mrs. W. Age 43 years. Laundry-worker.

Symptoms. Very obese woman. 1930 - Menses absent three months (always regular). Feels very short of breath. Severe cough, and pain in shoulder blade. Feet swell sometimes. Had kidney trouble three years ago. Last pregnancy - two years ago.

Physical Signs. Oedema of feet. Uterus enlarged, cervix soft. Many rhonchi in chest. Heart sounds normal. Urine - albumen +.

Diagnosis. Pregnancy and nephritic Hypertension. (Baby born at home, May 1931). (See chart).

No. 23.

Mrs. C. Age 24 years. Bag-worker. B.P. 140/104.

History. 1925 - Chlorosis. 1928 - Influenza and Tonsillitis (3 times). 1929 - Tonsillitis. P. 96.

1930 - Pregnancy and Oedema. Haemorrhoids, Joint Pains. Dec. 1930 - Normal termination of pregnancy. Feet still swollen.

Symptoms. December 1930 'took a light turn' and fell down the stairs.

Physical Signs. Very obese woman. Oedema of ankles. Slight amount of albumen in urine. No other abnormalities detected.

Diagnosis. Nephritis of Pregnancy and Hypertension.

No. 24.

Mrs. McL. Age 65 years. House duties.

History. 1926 - Severe pain in gall-bladder region. Sugar found in urine. Diagnosis of Diabetes Mellitus. Put on diet, and sugar-free for months. 1928 - Neuritic pains, urine loaded with sugar. B.P. 200/106, no albumen in urine. Glycosuria yielded readily to diet without Insulin. 1930 - urine again loaded with sugar, albumen present in urine. Glycosuria again yielded readily to dieting.

Symptoms. January 1931 - complaint of feeling 'done'. Urine contained a fair amount of sugar and albumen. B.P. 196/100. Diet again caused disapp-

earance of sugar. Severe pains in left leg. Marked old phlebitis of veins in leg, very feeble pulsation in popliteal vessels. Intermittent claudication present; leg feels cold.

Clinical Signs. Phlebitis of leg veins - veins felt like hard, knotted cords under skin. (Radials rubbery - not sclerosed). No signs of active inflammation; heart sounds very weak (patient obese). Soft systolic murmur at apex. Left border of heart in nipple line. Dyspnoeic and severe pain in leg on walking. Felt very weak. April 1931 - pain became very severe - resisted all usual analgesic remedies. Relief from intra-muscular injections of Acetylcholine. Later morphia required for relief of pain in leg.

Diagnosis. Hypertension and Vascular Endarteritis. (Sclerosis of pancreatic blood-vessels). (See chart).

No. 25.

Miss A. Age 46 years. House duties.

Symptoms. 1928 - One day felt as if she was going to fall while in the house - this sensation lasted a second or two. In the street, felt as if

the houses were moving from side to side. Bowels - regular, appetite and sleep - good. Severe occipital headaches. Menstruation regular (3 days). Very irritable for some weeks. B.P. 190/98. Felt very nervous. 1929 - Fainting turn in house. Felt very alarmed. Menses still present - slightly irregular. Sick after meals; bowels regular. Weight = 108.5 lbs. May 1930 - Menses present for nine days. Feels weak, and thinks her heart is bad. Epistaxis during menses. Does not feel her brain able for work of managing the house. Headache still present at times.

Physical Signs. Red-faced, nervous, dyspeptic looking woman. Irritable heart. No sclerosis of blood vessels. Heart not enlarged. Second aortic sound not markedly loud. Urine - no sugar, no albumen.

Diagnosis. Menopausal disturbance and Hypertension. (See chart).

No. 26.

Mrs. C. Age 72 years. House duties.

Symptoms. 1930 - Widespread petechiae on both lower limbs up to hip-joint. Weakness and great

nervousness (feeling as of some impending catastrophe). very easily annoyed for two months past. Appetite poor. (Symptoms date from sudden death of husband after operation). Bowels regular. Pains in knee-joints. March 1931 - very itchy dermatitis developed from sulphocyanate therapy - red splotches all over skin. March 1931 - feels a great nervousness over her. April 1931 - not well all week. Very nervous and fatigued.

Physical Signs. Bronchitis and radials tortuous and sclerosed. Second aortic sound ringing in quality. Fundi - no neuroretinitis, veins engorged, no haemorrhages. Left border of heart in nipple line, no murmurs. Urine - no sugar, no albumen.

Diagnosis. Hypertension and Arteriosclerosis.

No. 27

Mrs. C. Age 49 years. Teacher. B.P. 180/110.

Symptoms. 1930 - Threatened appendicitis. 1931 - Bilious attacks and terrible headaches (6 years) In bilious attack, first gets headache, then nauseated. Bowels sometimes constipated. Menstruation ceased (2 years). Feels the cold very much.

Physical Signs. No tenderness in gall-bladder region, nor in appendix region. No sclerosis of blood-vessels. Heart not enlarged. Urine - no sugar, no albumen.

Diagnosis. Menopausal disturbance (?) and Hypertension.

No. 28.

Mrs H. Age 64 years. House duties. B.P. 180/120.

Symptoms. Headaches and dizziness (ten years). Very constipated.

Physical Signs. Very obese woman. Sclerosis of radials - not marked. No other abnormalities detected.

Diagnosis. Hypertension.

No. 29.

Mrs. H., age 76 years. House duties. B.P. 16/5/31...210/84. P. 80. 22/5/31...B.P. 200/84,P.90.

Symptoms. Hoarseness (2 weeks). Voice normal previously. Feels a great weakness and can't take her food, (one month). Sleeps well; rises sometimes at night to micturate. Very constipated. No previous illnesses. Feet swell a little when walking, and is compelled to walk very slowly because of tiredness

and shortness of breath.

Physical Signs. Radials sclerosed and tortuous. Second aortic sound loud and slapping (not pure). Apical systolic murmur. Left border of heart in mid-clavicular line. Corrigan's pulse; larynx - swelling and redness of vocal cords, with pus (from pharynx) lying in between them.

Diagnosis. Hypertension, arteriosclerosis and Laryngitis.

No. 30.

Mrs K., age 74 years. House duties. B.P. 200/100.

Symptoms. Hemiplegia in 1925. Right eye painful and sight defective. Severe headaches (for years), and very bad pain in hemiplegic lower limb (much swollen). Can't sleep because of headaches and pain in leg.

Physical Signs. Neuro-retinitis left eye. Thin arteries. No retinitis in right eye. Radials rubbery - not sclerosed. Left border of heart in mid-clavicular line. Second aortic sound loud and slapping. No murmurs. Urine - albumen +. Tongue coated. Hemiplegic leg swells up unless massaged,

skin in pre-tibial area yellow and discoloured. No pulsation in vessels of foot. Slight return of power in leg, arm almost completely restored to normal.

Diagnosis. Nephritis and Hypertension.

(See chart).

No. 31.

Mrs. M. Age 62 years. House-duties. B.P. 184/100.

Symptoms. January 1931 - Heaviness in stomach after food and sore head. Stomach swells after food, subject to indigestion; a feeling like 'wind' around her heart makes her feel 'useless'. April 1931 - feeling of load on head, then comes into her eyes, (thinks it is 'wind' coming up from her stomach). Feet swell a little at times.

Physical Signs. Obese woman. Radials and brachials hardly palpable. Visible carotid pulsation. Heart sounds rather weak. Left border of heart ½" outside mid-clavicular line. Second aortic sound loud. No murmurs. Fundi - no haemorrhages, arteries thin, veins rather engorged. Visible pulsation in retinal veins. Slight pigmentary degeneration at right margin of right disc. Urine - no sugar, no albumen.

Diagnosis. Hypertension and Dyspepsia. (Subjectively feels much better on KCNS therapy, though it has not reduced the blood pressure. (See chart).

No. 32.

Miss M., age 56 years. Teacher. B.P. 280/130.

Symptoms. 1929 - Finds it difficult to walk on account of pain in the knees. Feels 'terribly tired' on slight exertion. Short of breath. Weight = 13st. 7lbs. Appetite good. Bowels regular. Bad headaches at night, and also when she rises in the mornings.

Physical Signs. Radials and brachials rubbery. Heart enlarged. Systolic murmur at aortic area. Extra-systoles fairly frequent. Ankles and pre-tibial areas very oedematous. Fundi not examined. Urine - trace of albumen, no sugar, no casts. Marked obesity.

Diagnosis. Hypertension and cardiac failure. (See chart).

No. 33.

Miss P. Age 53 years. Housekeeper. B.P. 203/105.

Symptoms. 1931 - Long-standing varicose ulcer (10 years). Takes dizzy 'turns' and feels very faint. Frequent headaches.

Physical Signs. Very obese, red-faced, apprehensive woman. Heart slightly enlarged to percussion. Apex beat not palpable; aortic systolic murmur. Second aortic sound loud and slapping. Reduplication of first pulmonic sound. Radials rubbery, brachials tortuous. Fundi - arteries thin. Obliteration of veins (in places) where arteries cross over. Slight haemorrhage along line of a blood-vessel in left retina. Urine - a trace of albumen, no sugar.

Diagnosis. Hypertension. (See chart).

No.34.

Rev. R.P. Age 65 years. Clergyman. B.P. 190/105 (1929).

Symptoms. 1920 - Typhoid Fever. 1929 - discomfort after meals, and headaches from time to time. 1930- On light diet for dyspepsia. May 1930- Took a dizzy feeling and staggering as he came in for consultation. Sent home in taxi and kept in bed for a month. Feeling of numbness developed in right thumb, and at corner of mouth. Blood pressure

fell to 120/92 mm. Hg. (He had been very worried over some parish matters for some months previously). Went for three months leave of absence, and felt very well on his return. May 1931 - again felt occasional dizziness and headache, with dyspeptic symptoms. (Bismuth and Bromide mixture prescribed, with rest in bed for 2 hours in afternoons). June 1931 - B.P. 158/94, feeling very well. Plays eighteen holes of golf twice a week.

Physical Signs. Slight sclerosis of arteries. No obesity. Second aortic sound not very loud. Soft systolic apical murmur ; left border of heart in nipple line. Fundi - arteries very thin. Remains of old (slight) haemorrhage in right retina. Discs normal. Urine - no sugar, no albumen.

Diagnosis. Hypertension and arteriosclerosis. (See chart).

No. 35.

Mr. G. Age 64 years. Hammerman (1926). B.P. 260/120.

Symptoms. Injection of absolute alcohol for trigeminal neuralgia, in 1927. Later had lumbago. 1930 - Carbuncle of neck. March 1931 - Neuralgia

of face again started, especially when he starts to eat. Rises two or three times at night to urinate. Sometimes slightly incontinent; always constipated. Has had chronic bronchitis for some years. April 1931 - says pain in jaw and face is like the sting of a nettle. June 1931 - Neuralgia almost gone.

Physical Signs. Brachials and radials thick and rubbery. Visible pulsation in brachial (whole length of artery) - artery not tortuous. Second aortic sound loud and ringing. No murmurs. Cardiac area increased downwards and $\frac{1}{2}$" to the left of mid-clavicular line. Very slight pitting in legs. Prostate slightly enlarged. Fundi - arteries very thin, no haemorrhages. Urine - no sugar, no albumen.

Diagnosis. Prostatic Hypertrophy and Hypertension. (See chart).

No. 36.

Mr. McK. Age 59 years. Shoemaker. B.P. 260/130.

Symptoms. May 1931 - Feels quite weak and 'done' (one month). Says he feels that he needs a rest from his work. Has never had any serious ill-

nesses. Severe cough in the mornings. Feels a gurgling in his throat at night, and becomes short of breath. June 1931 - short of breath on going upstairs. (Never felt this before). Has felt a great nervousness and shaking of his head (for three years) whenever he becomes excited. Can't read small print.

Physical Signs. Rather obese, red-faced man. Arteries rubbery, not sclerosed. Second aortic sound loud. No murmurs; left border of heart in nipple line, apex-beat in sixth interspace. Fundi- arteries very thin and tortuous in places. Veins congested. Large masses of pigment at right margin of both discs. No haemorrhages. Urine - albumen, no sugar, a few casts.

Diagnosis. Hypertension and Nephritis. (see chart).

No 37.

Mr. W. Age 70 years. Engineer (1921). B.P. 230/105.

Symptoms. 1921 - Bronchitis. 1928 - Sciatica March 1929 - Left eye destroyed by an ulcer. 1930 - pains up back of neck and head. Discharge from eyes,

and post-nasal discharge. Pain across forehead. Pains in back of head, and up the left side of head. Buzzing in ears. Cataract developing in remaining eye. (Fundus not visible). April 1931 - Frequent soreness and dryness in throat. Very severe, hard cough; throat feels raw, especially at night. Greatly bothered with 'sciatic' pains in right hip and thigh.

Physical Signs. Pharynx congested. Mucous membrane looks atrophic. Radials and brachial tortuous and sclerosed. Second aortic sound loud and ringing. Left border of heart $\frac{1}{4}$" outside nipple line. Apex-beat in sixth interspace. Cataract in right eye (Empty left socket). Urine - albumen +, no sugar.

Diagnosis. Arteriosclerosis, Hypertension and Nephritis. (Galactose Tolerance = abnormal curve). (See chart).

No. 38.

Mr. O.B. Age 68 years. Miner. B.P. 200/110.

Symptoms. Jan 1931 - Severe accident in pit. Breast-bone broken and skull dented (no fracture). Says sight is leaving his eyes. March 1931 - took

a weak turn. Severe pain in head and down his neck. April 1931 - severe pain in head and in neck muscles, when he lies down at night. May 1931 - severe pain in head - can't sleep.

Physical Signs. Dent on crown of skull. Radials and brachials sclerosed and tortuous. Visible pulsation in brachials and in neck. Heart enlarged downwards - apex beat in sixth interspace. Second aortic sound loud and musical; no murmurs. Fundi- arteries thin. Slight old haemorrhages and pigmentary degeneration at outer margin of right disc.

Diagnosis. Hypertension and Arteriosclerosis. (See chart).

No. 39.

Mr. McG. Age 73 years. Labourer. (1920). B.P. 190/94.

Symptoms. 1925 - varicose ulcer on right leg. 1930 - ulcer healed, leg swollen. March 1931 - pain in leg and toes. 4th May 1931 - severe pain across toes of right foot. 28th May 1931 - incipient gangrene of foot and leg, (sent to Hospital). June 1931 - returned home, gangrene did not develop.

Physical Signs. Healed varicose ulcer of right

leg. Skin in vicinity of ulcer shows marked yellowish discolouration and eczema (dry). Radials and brachials sclerosed and tortuous. Visible brachial pulsation. Second aortic sound slapping in character. Systolic murmur at apex. Apex-beat in sixth interspace. Left border of heart in nipple line. Fundi - not visible, owing to keratitis from burn with chemicals in his youth. Urine - no sugar, no albumen. In May 1931, right foot and leg livid and discoloured up to knee.

Diagnosis. Varicose ulcer, Hypertension, Arteriosclerosis, Incipient Gangrene. (See chart).

No. 40.

Mr. McG. Age 64 years. Slater. B.P. 270/128.

Symptoms. 1929 - Bronchitis and Influenza. March 1931 - Bronchitis and severe cough. Feels quite well otherwise.

Physical Signs. Radials and brachials tortuous and sclerosed. Visible brachial pulsation. Left border of heart ½" outside nipple line. Apex-beat best felt in fourth interspace. Second aortic sound loud and musical. First sound at apex weak-not pure. Fundi - arteries very thin, veins en-

gorged. Slight haemorrhages along lines of veins in right fundus. Constriction of veins by arteries. Slight, dark pigmentary ring round left optic nerve. Urine - not examined, (patient did not return).

Diagnosis. Hypertension and Arteriosclerosis.

No. 41.

Mr. W. Age 65 years. Labourer. B.P. 180/110.

Symptoms. Left Hemiplegia in January 1929. Six months later fell in house and fractured femur of hemiplegic leg. November 1930 - had a slight 'shock' . Injury to right eye several years ago, no sight in it since. Always very constipated. Very cross and irritable. Feels well when bowels kept regular by aperients.

Physical Signs. A certain amount of return of function in hemiplegic lower limb. No power in arm. Marked arteriosclerosis of arteries. Left border of heart in nipple line. Soft systolic murmurs at apex. Second aortic sound loud. Fundus not examined. Urine - albumen +, no sugar, a few casts.

Diagnosis. Nephritis, Hypertension and Arteriosclerosis. (See chart).

No. 42.

Mr. McG. Age 71 years. Labourer. B.P. 190/100.

Symptoms. Heart enlarged 1" outside nipple line. Very many dropped beats, unequal in force. Pulse about 80 per mm. Heart sounds loud at apex, weak at aortic area; apex-beat in 6th interspace. Radials and brachials sclerosed and tortuous. Fundi - arteries thin, a few small haemorrhages in right retina. Urine - no sugar, no albumen. (Digitalis and KCNS prescribed - 2 weeks later, blood pressure had fallen from 190/100 to 168/90. Left border of heart in nipple line).

Diagnosis. Hypertensive Cardiac disease, and Arteriosclerosis.

No. 43.

Mr. R. Age 68 years. Labourer. B.P. 234/100. P. 66.

Symptoms. 1930 - Right sciatic pain. Large right hydrocele of testicle. Prostate moderately enlarged. 1931 - Feels fairly well. No pain now.

Physical Signs. Radials and brachials sclerosed and tortuous. Visible brachial pulsation. Second aortic sound loud and muscial. First aortic sound

very faint. Well-marked systolic murmur at apex. Apex-beat not palpable. Left border of heart ¼" outside nipple line. Fundi - arteries very thin, veins tortuous and congested. No haemorrhages. Area of degeneration (?) in right disc near its centre. Urine - albumen +, no sugar, no casts. Galactose tolerance test showed liver dysfunction. Van den Bergh negative.

Diagnosis. Enlarged prostate, Nephritis, Hypertension, Arteriosclerosis.

No. 44.

Mr. R. Age 49 years. Garage Proprietor. B.P. 180/118. P. 84.

Symptoms. Deafness in left ear. Hypertension discovered on routine examination. No symptoms referable to blood pressure. Feels quite well except for deafness (deaf).

Clinical Signs. Rather obese, slight pallor. Wax in both ears. Left drum inflamed. Radials and brachials rubbery, not sclerosed. Visible brachial pulsation. Heart not enlarged to percussion. No murmurs; second aortic sound loud. Fundi - Veins engorged, arteries thin. Some pigmentary degenera-

tion at edge of left disc. High refraction in right eye. Urine - not examined. (Patient not seen since March 1931).

Diagnosis. Otitis (mild) and Hypertension.

No. 45.

Mr. M. Age 74 years. Miner (1920) B.P. 190/140. P. 74.

Symptoms. 1925 - Hemiplegia (left side). 1930 - slight shock. January 1931 - rambling and incoherent in speech at times. Very slight return of function in hemiplegic limbs. April 1931 - died with uraemic symptoms.

Clinical Signs. Hemiplegia (left side). Confined to bed since 1925. Intelligent, but inclined to ramble in his speech at times. Oedema. Heart enlarged, 1" outside nipple line. Soft systolic murmur at apex. Apex-beat in 6th interspace. Urine - contains albumen, no sugar. Marked arteriosclerosis of blood vessels.

Died in uraemic coma.

Diagnosis. Arteriosclerosis, Nephritis, Hypertension, Uraemia.

No. 46. Mr. C. Age 56 years. Tram-driver.

30/4/31 .....B.P. 162/96. P. 84.

30/5/31 .....B.P. 148/90. P. 84.

Symptoms. Pain in stomach one hour after food, followed by vomiting (9 months). Eruption on trunk composed of raised red papules, some of which contain pus. This dermatitis becomes worse after food, and usually comes out at this time of the year (March 1931). When he lies on his right side, especially after a meal, he feels a choking sensation, and is compelled to jump up. Says his weight is increasing.

Clinical Signs. Markedly obese, red-faced, apprehensive man. Radials and brachials hardly palpable. No cardiac abnormalities detected. Fundi - normal. Urine - no sugar, no albumen.

Diagnosis. Obesity and Hypertension.

(Note: Bismuth and Bromide mixture, with instructions for diet, reduced the B.P. as above).

Prognosis.

The gloomy prognosis as indicated by life insurance statistics would hardly seem to be borne out in cases coming under medical treatment, and amenable to instructions. Case No. 1 illustrates the fatal progression of malignant hypertension, uninfluenced by therapy. This syndrome has been emphasized, largely by American and German observers, as occurring in comparatively young people, with a hopeless prognosis. The ordinary hypertensive cases, without complications, may be kept in a state of well-being for many years, provided they are intelligent enough to take heed of the warning symptoms and seek medical advice. At the same time, prognosis must always be guarded, especially when there is a family history of apoplexy. Fatal, or at least, disabling cerebral haemorrhage is apt to come with disconcerting suddenness even in those apparently well. Nephritic hypertension, too, seems to predispose to sudden cerebral accident.

I would emphasize that in an intelligent, commonsense patient, coming under medical treatment, hypertension holds out a comparatively good prognosis.

Treatment.

All cases in this series have been treated primarily with a view to the reduction of blood pressure. Some authorities do not approve of this, but I prefer to regard high blood pressure as the manifestation of some unknown, active toxic factor, and hence to be combatted. In most cases (about 95%), a reduction of from 50-100 mms. Hg. cause no discomfort, usually the reverse, in fact. Where a reduction in blood-pressure does cause symptoms of distress, no further attempt in this direction need be made.

In a more enlightened community, the treatment of hypertension will come more into the domain of preventive medicine. A yearly medical overhaul of every person in the country (as is now practised by many large firms for their employees) will lead to detection of hypertensives (and potential hypertensives), at an early stage. These patients may then be kept under constant supervision, and given advice as to regulation of their working hours, necessity of freedom from worry, (where possible) and avoidance of over-eating. Such persons require

more rest than normal people, and should be advised to rest in the afternoon (or evening), especially when they feel very tired, short of breath, dizzy, irritable and subject to headache. These warning symptoms seem so slight to the unenlightened layman as not to be worth noticing, but expert advice, given with discernment (not necessarily mentioning blood pressure), would lead to earlier treatment and more hopeful results.

Patients with a tendency to obesity should be urged to limit their carbohydrate intake especially. When persuaded to weigh themselves at regular intervals, they soon begin to take a pride in any reduction of their weight. As noted in the illustrative summaries of cases, obesity is a much more common factor in women than in men. It is worth remarking on the ignorance displayed by even quite intelligent patients as to the primary cause of obesity. It takes quite a good deal of argument and demonstration to convince many of them that excessive carbohydrate ingestion in excess of their energy requirements is usually the most important factor in their increased adiposity. Those of them who are con -

cerned about their over-weight usually look for some drug to 'work the charm', and are quite disappointed with such prosaic advice as reduction of food: There is still a good deal of room for improvement in the instruction of laymen in the fundamentals of biology and hygiene.

Drugs.

For established cases of hypertension in this series, the most useful drugs, in order of efficaciousness, for reducing blood pressure have been:-

i. Acetylcholine.

ii. Potassium Sulphocyanate.

iii. Bromides and Bismuth Subnitrate.

(i) Acetylcholine.

The preparation used was 'Acecoline' supplied by the Anglo-French Drug Co. The sterile product is supplied in sealed ampoules accompanied by ampoules of redistilled water for its solution, and may be given by subcutaneous or intramuscular injection. In case No. 24 (see chart), eleven consecutive daily injections of 0.20 grm. reduced the blood pressure from 170/100 mm. to 140/82-90 mm. With the blood-pressure at 140 mms. (Systolic) the

patient felt very weak, but at the level of 150 mm. (Systolic) felt quite well. Incidentally this patient suffered from severe, intractable pain in the leg, resembling that seen in Bürgher's disease. The injection of 0.20 grm Acecoline invariably gave relief for about four hours. (The only other drug that was of use in this condition was Morphia). Three months after the treatment the blood pressure was 160/90 (original level in 1928 = 200/107 mm.Hg.). As already noted, intravenous injection of this drug is extremely dangerous, because of the sudden, large fall in blood pressure. Hence, in intramuscular injection, the needle of the syringe is withdrawn a little distance after introduction of the needle, in order to make sure that the latter is not in a vein.

(2) Sulphocyanates.

Both the sodium and potassium salts have been used in this series, the latter being preferred on the grounds that the sodium ion has been stated to increase muscle and nerve irritability in hypertension. In most cases a fall of blood-pressure was produced, and in practically every case a feeling of

well-being and relief from symptoms was produced, even where no decrease of pressure was produced. (See No. 31).

Messrs. Parke Davis supply the sodium salt as 'Elixir Sod.Sulphocyanatis', but for panel patients the potassium compound may be prescribed in a mixture, and is really to be preferred. Acute nephritis is a contra-indication to the drug. Furthermore, it must be used with caution, and in small doses, as it is apt to give rise to dermatitis with severe itching, and to inflammation of mucous membranes. Two patients in this series developed slight toxic symptoms from the ingestion of sulphocyanate, (see Nos. 26 and 36). In these however, the dose was too large (gr. iv t.i.d.). In case No 26 dermatitis and severe itching appeared, while in case No 36 vomiting, diarrhoea, headache and itching of the skin appeared - both after one week's medication. However, both patients had been warned to stop the medicine on the appearance of itching of the skin, though No. 26 actually developed a widespread blotchy, very irritating dermatitis. After this experiment it was considered more advisable to give small doses

gr i tid. for one week, gr i bid. for one week, gr i daily for one week, followed by one week free from medicine. The simple precaution of warning the patient to cease from the drug on the appearance of itching of the skin, with weekly medical inspection at first, would seem to be sufficient to avoid severe toxic manifestations.

Even cases with chronic nephritis appear to react well to the drug. (See charts Nos. 26 and 30). (3) Bromides and Bismuth Subnitrate seem to have a calmative effect on the nervous dyspepsia which is sometimes a potent factor in causing distress of mind in hypertensives. Chart No. 24 illustrates the fall in arterial pressure induced by this simple medication, though in this case it was combined with rest in bed for two hours in the afternoon. Incidentally rest in bed has been found to reduce blood pressure very materially in this series of cases, but the fall is transient - the blood rises rapidly again when the patient rises and undertakes his daily duties.

Bromides alone, or combined with Thyroid Extract, in the nervous crises of the menopausal

period, with raised arterial pressure, seem to be sufficient to tide many patients over this difficult period. A verbal reassurance as to the transitory effects of the symptoms is very necessary in order to dispel any alarm at their disconcerting nature.

Hydrotherapy has not been found to be a treatment of election in an industrial practice.

General Conclusions.

The findings in the present series of 403 consecutive cases indicate a high incidence of high systolic blood pressure in females of 27.5% and in males of 17.3%. The findings for high diastolic blood pressure are approximately the same as the foregoing. Taking the figures for the different decades it was found that in a working-class population there was a three to one preponderance of high blood pressure in females as compared with males in the periods 30 - 60 years.

The severity of the disease largely depends on the state of the blood-vessels and the integrity of the heart-muscle. It is notoriously difficult to estimate prognosis because of the risk of sudden fatal haemorrhage, heart failure or cessation of kidney function. Most of the cases under consideration have responded quite well to treatment, but a high diastolic pressure is always of disquieting moment, and calls for very guarded prognosis. It was found that eleven out of twelve patients with marked high blood pressure showed impaired liver function as judged by the galactose tolerance test.

With suitable therapy the disease does not seem to progress materially even in advanced cases, apart from those showing the syndrome of Malignant hypertensive neuro-retinitis. Untreated hypertensives apparently supply the major proportion of sudden deaths from cardiac failure and cerebral and renal vascular accidents.

The manifestations of the disease are protean, while it has been shown that of the 403 cases examined, 28% of males and 14% of females complained of no symptoms referable to the cardiovascular system. In a general way the following were the commonest findings, either alone or, more frequently, in combination:

a. Attacks of dizziness.

b. Nervousness.

c. Headaches.

d. High-coloured complexion.

e. Obesity (most marked in females).

The safest way of diagnosing the disease is obviously to record the blood-pressure in all patients coming for examination.

What can we do for the man affected?

It becomes increasingly obvious, even to the uninitiated, that rest and freedom from worry where possible, are cardinal points in the treatment of hypertension. Again and again one sees a rapid rise of blood-pressure, even in patients under treatment, as a result of some anxiety state or pressure of work. The results are frequently disastrous. It would seem as if the unstable vasomotor system of hypertensives is incapable of responding to any extra mental stress or strain, hence the primary and fundamental necessity for freedom from these noxious agents.

Granted cessation from work, there are several combinations of sedative drugs that may be invoked to dull down the nervous tension so often apparent in these patients. Sedative medication, combined with rest in bed for a week or more, is found to have a salutary effect in lowering the pressure, with consequent increase in well-being, and less risk of cardiovascular accident. Unfortunately, as the blood pressure is not permanently lowered by these measures, it is usually necessary

to have recourse to drugs with a more specific action and the writer believes that in the Thiocyanates and Acetylcholine we have two powerful agents for reducing blood pressure. It is his belief that in the absence of our knowledge of a definite etiological agent, an attempt should always be made to lower the blood pressure - permanently, as far as may be possible. The subjective reaction of the patient will be the most important guide as to the extent the pressure should be reduced.

In addition, hypertensives should have explained to them that they need frequent supervision, that they must live their lives at a lower level of nervous tension than heretofore, and that frequent rest-periods are indicated. Those cases of hypertension coming under observation at an early age are more amenable to treatment, before the almost inevitable cardiovascular degeneration has set in. The treatment of such cases really enters more into the domain of preventive medicine. They should be advised as to a suitable occupation involving little nervous tension, and come frequently for medical supervision. It goes without saying that all

hypertensives require a thorough overhaul of all the systems of the body, in order to put them in the best possible condition for combatting their disability.

There is no doubt that the treatment of hypertension is now on sound scientific lines. The cardiovascular disabilities consequent on permanent hypertension may be greatly reduced by appropriate reduction of blood-pressure, and its maintenance at an optimum level by frequent supervision.

It is a significant fact that, in the writer's experience, most of the sudden fatal vascular accidents have been observed in cases of untreated (or unrecognised) hypertension.

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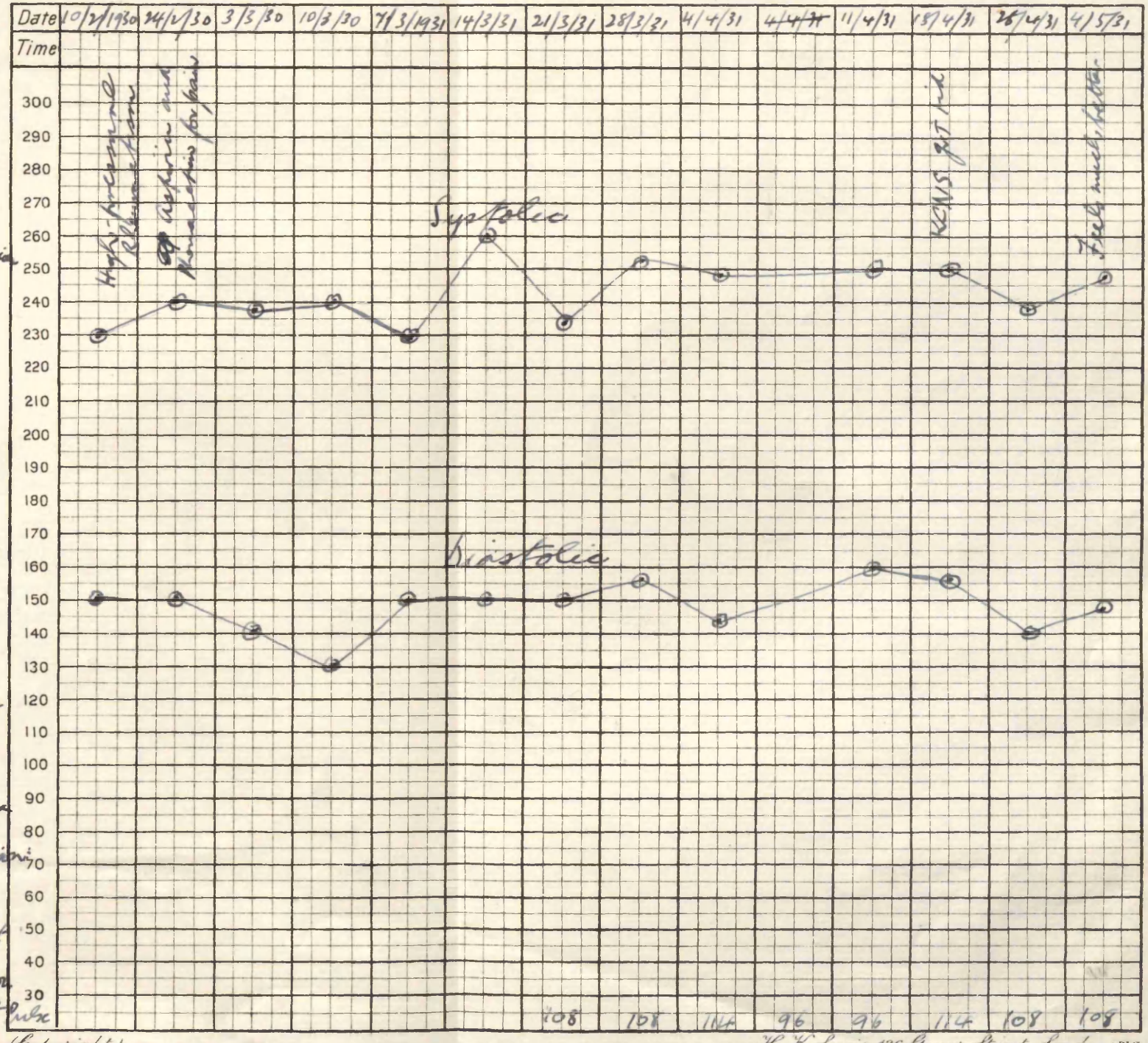
.....oOo.....

Case No. 1.

no. 1 (a)

LEWIS'S BLOOD PRESSURE AND PULSE CHART.



Name

mrs. B.

Age 37 years.

Disease

malignant Hypertension

Notes

no family history of Hypertension, nor of cerebral accident.

Four sisters living and well.

Patient illustrates

(1) High-pressure Rheumatism

(2) Fatal Syndrome of malignant hypertension:

(a) Neuro-Retinitis

(b) Rapid loss of weight

(c) Persistent Hypertension

(d) Rapidly progressive fatal course.

H. K. Lewis, 136, Gower Street, London W.C.

Case No. 1. (continued).

no. 1 (contd) (b)

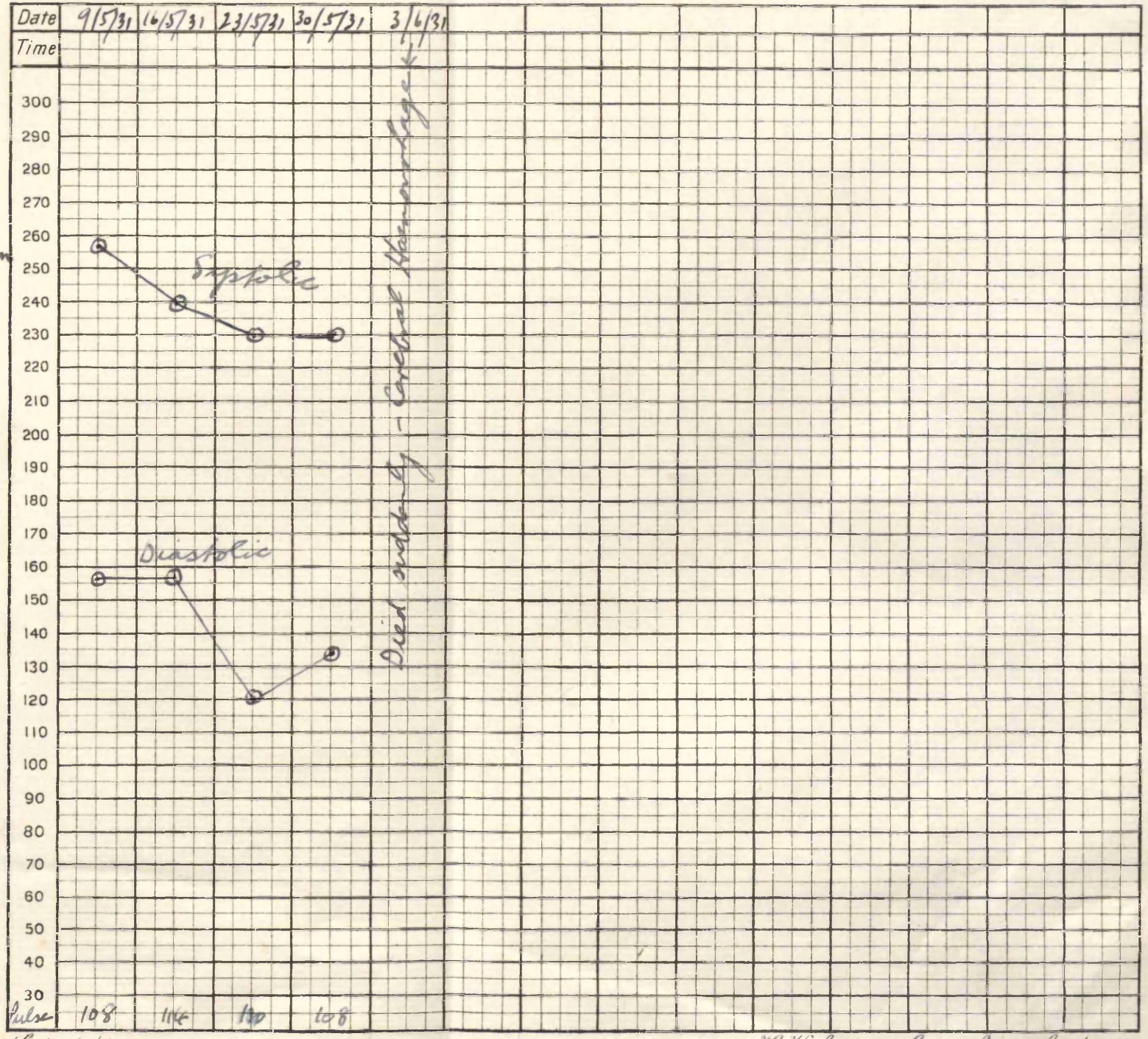
Name Mrs. B.

Age 37 years.

Disease Malignant Hypertension

Notes

LEWIS'S BLOOD PRESSURE AND PULSE CHART.



H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 2.

no. 2

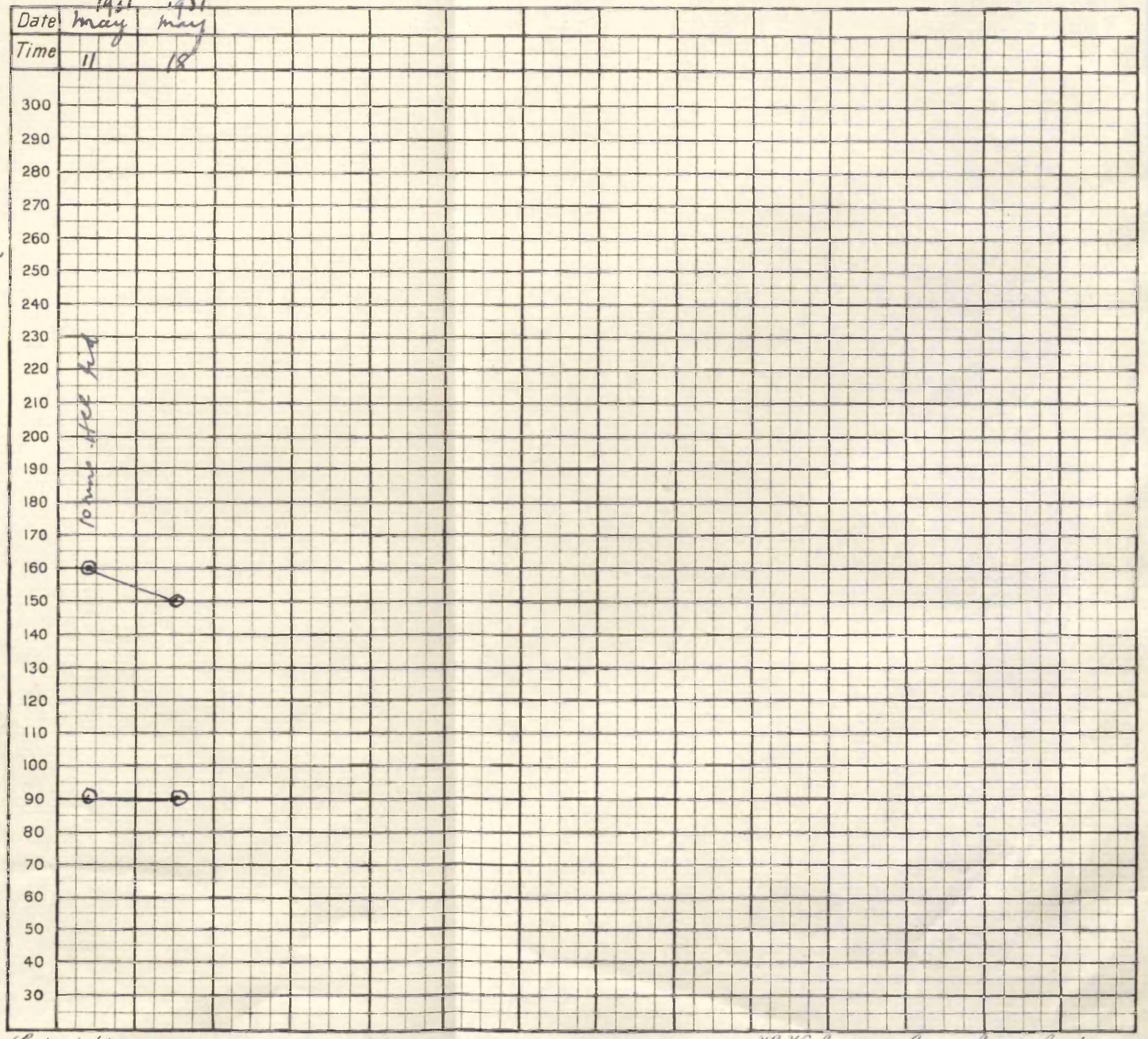
LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Name
miss. B.
(Domestic Servant.)

Age 41 years.

Disease
Essential Hypertension

Notes



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Case No. 4.

no. 4.

LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Name

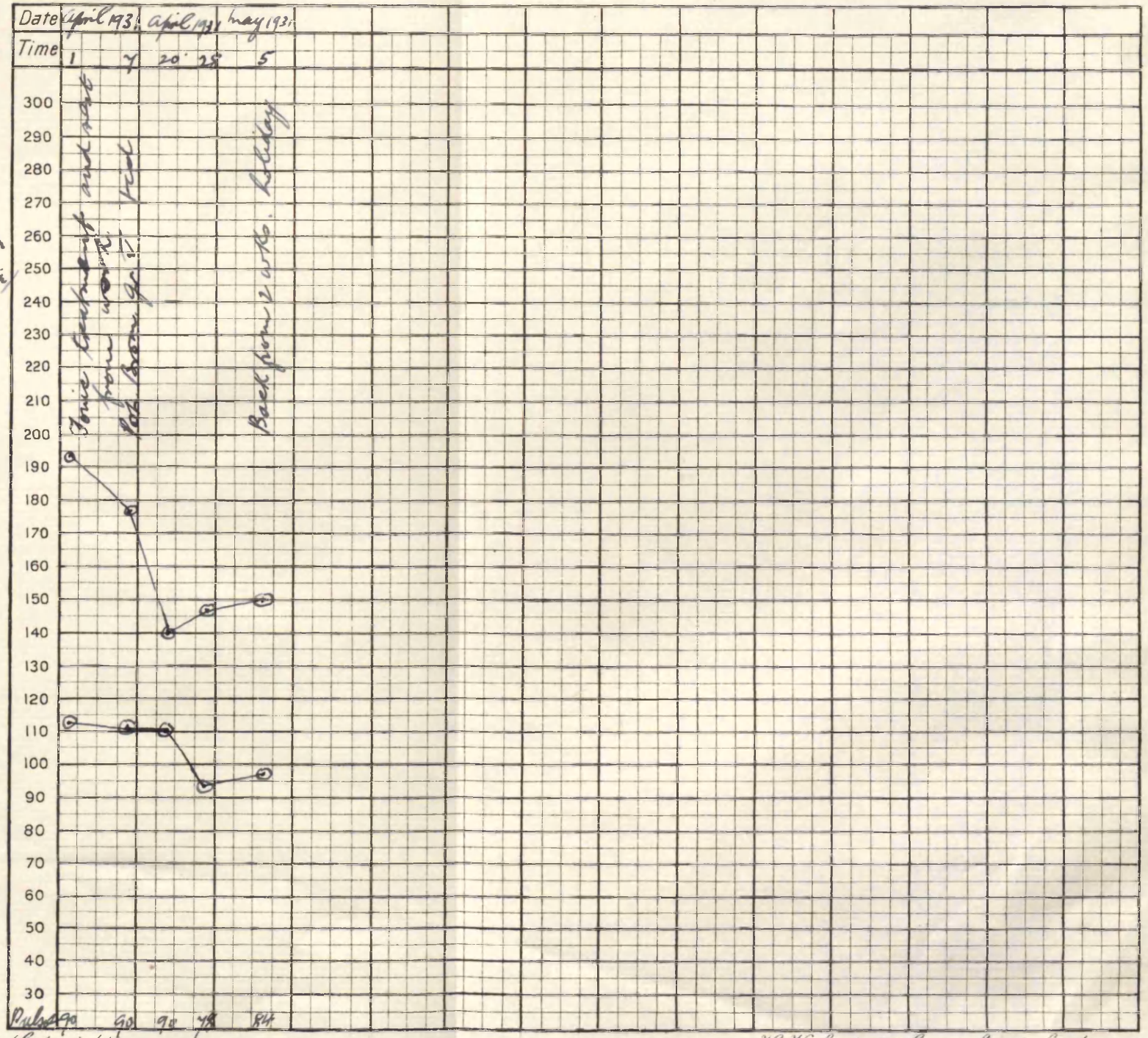
Mrs. B.

Age 52 years

Disease

Essential Hypertension
(Endocrine Dyspunchia)

Notes



H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 7.

no. 7.

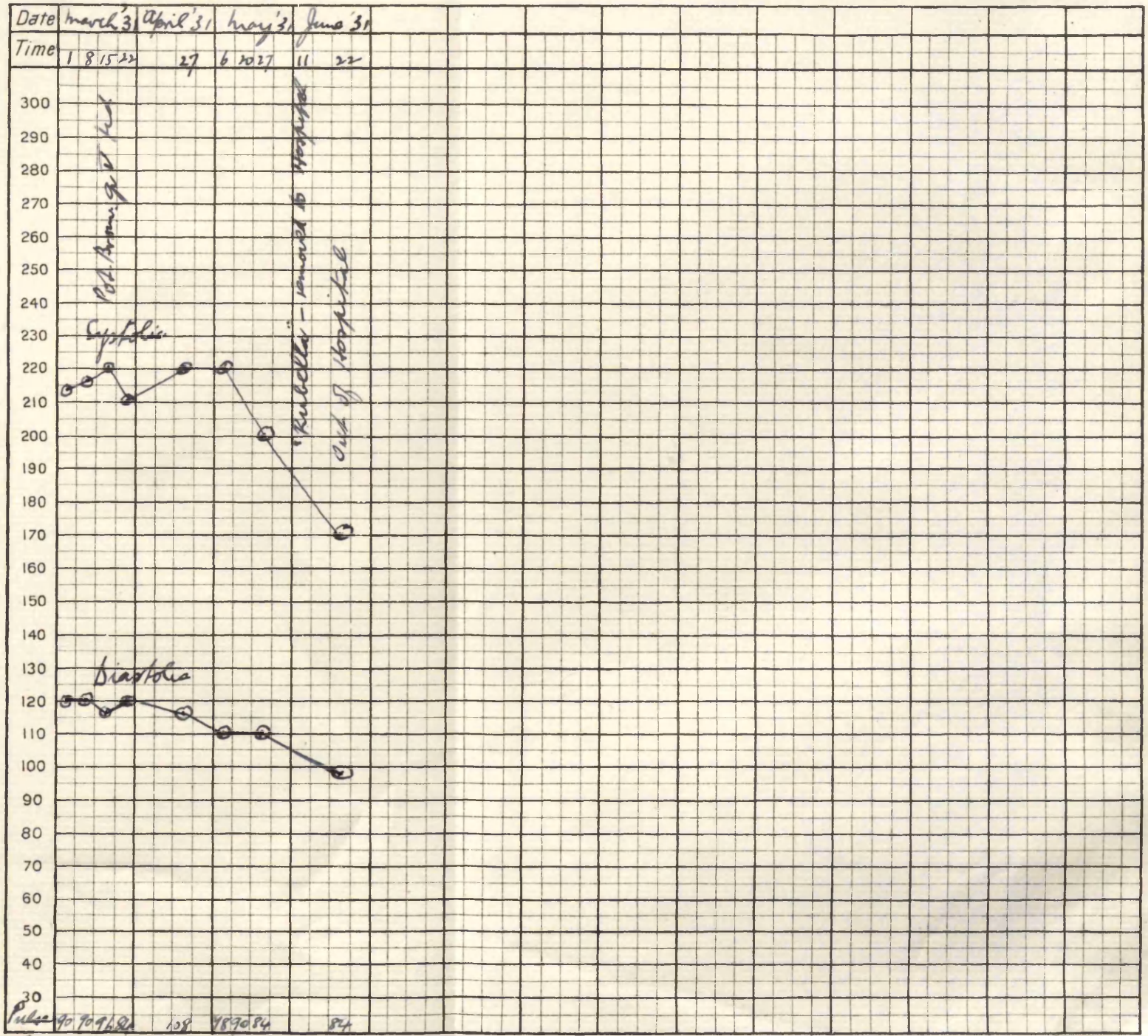
LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Name
miss. B.

Age 37 years.

Disease
Ovarian Tumour and Hypertension

Notes
marked fall of B.P. probably to be attributed to 11 days enforced rest in bed.



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Case No. 13.

no. 13

LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Name

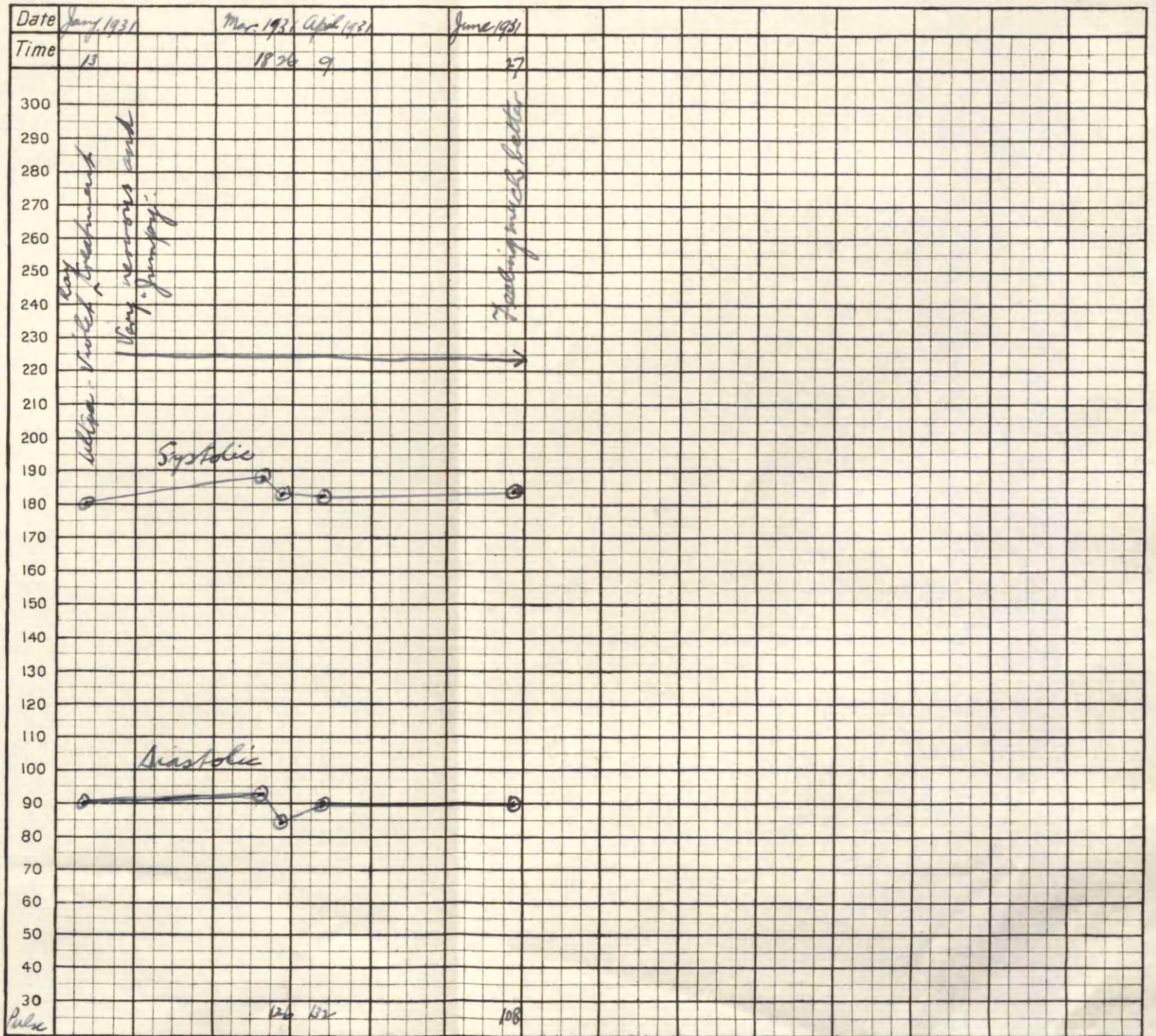
Miss M.

Age 32 years.

Disease

Toxic Goitre and Hypertension

Notes



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Case No. 17.

no. 17

Name

miss h^e H.

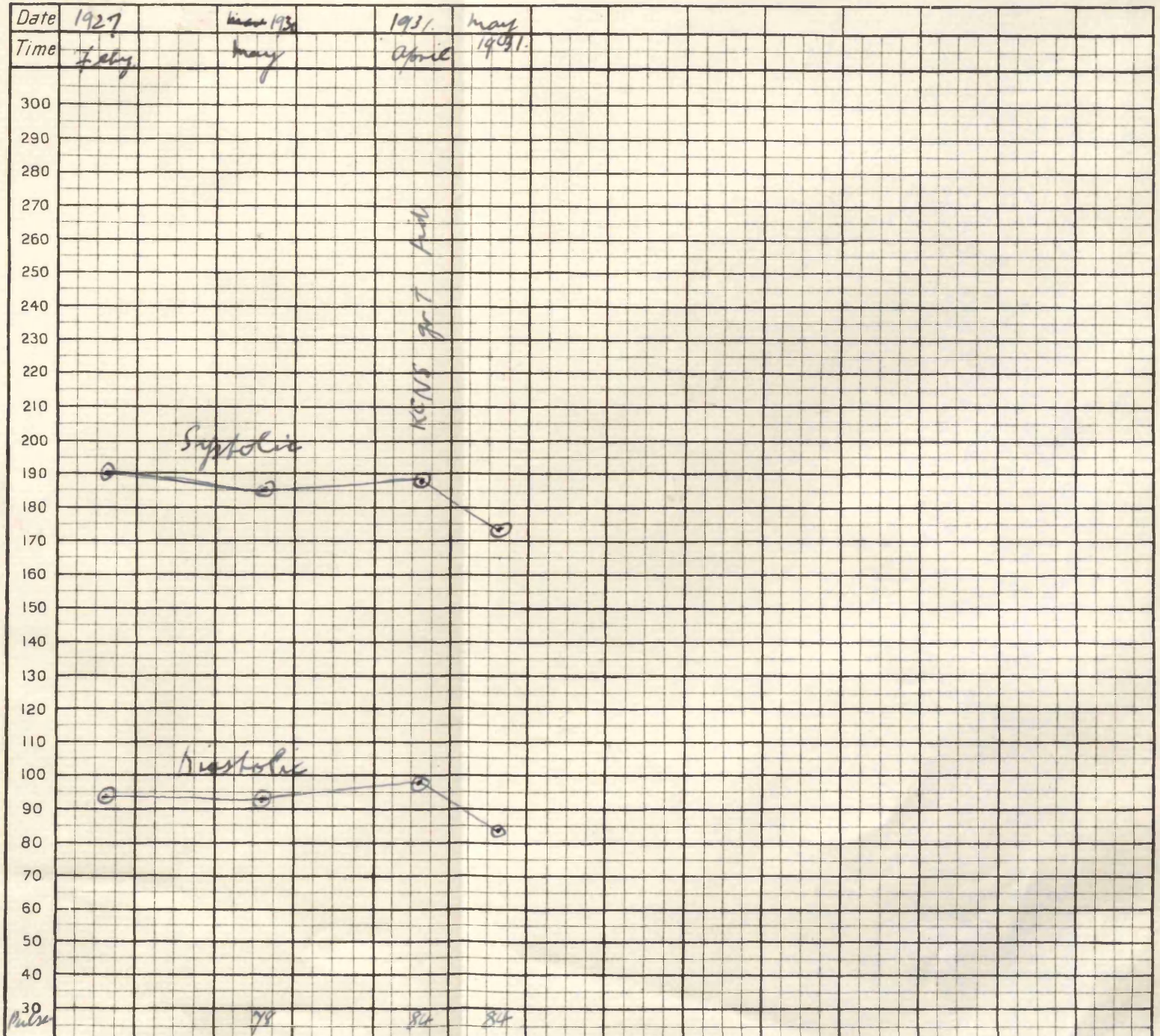
Age 68 years.

Disease

Hypertension

Notes

LEWIS'S BLOOD PRESSURE AND PULSE CHART.



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Case No. 22.

no. 22

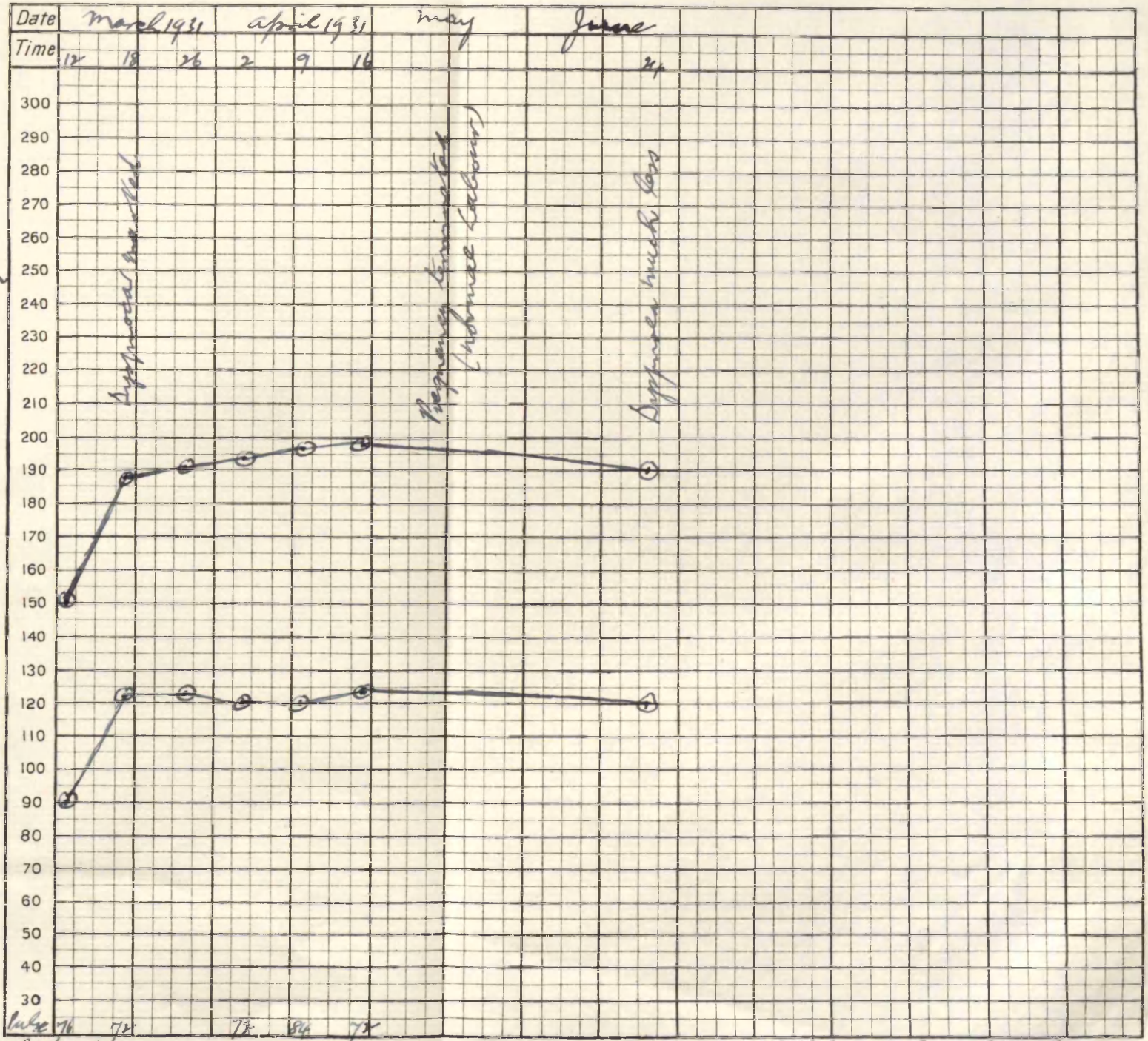
LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Name
Mrs. W.

Age 43 years

Disease
Pregnancy and Leptitic Hypertension

Notes



H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 24.

No. 24

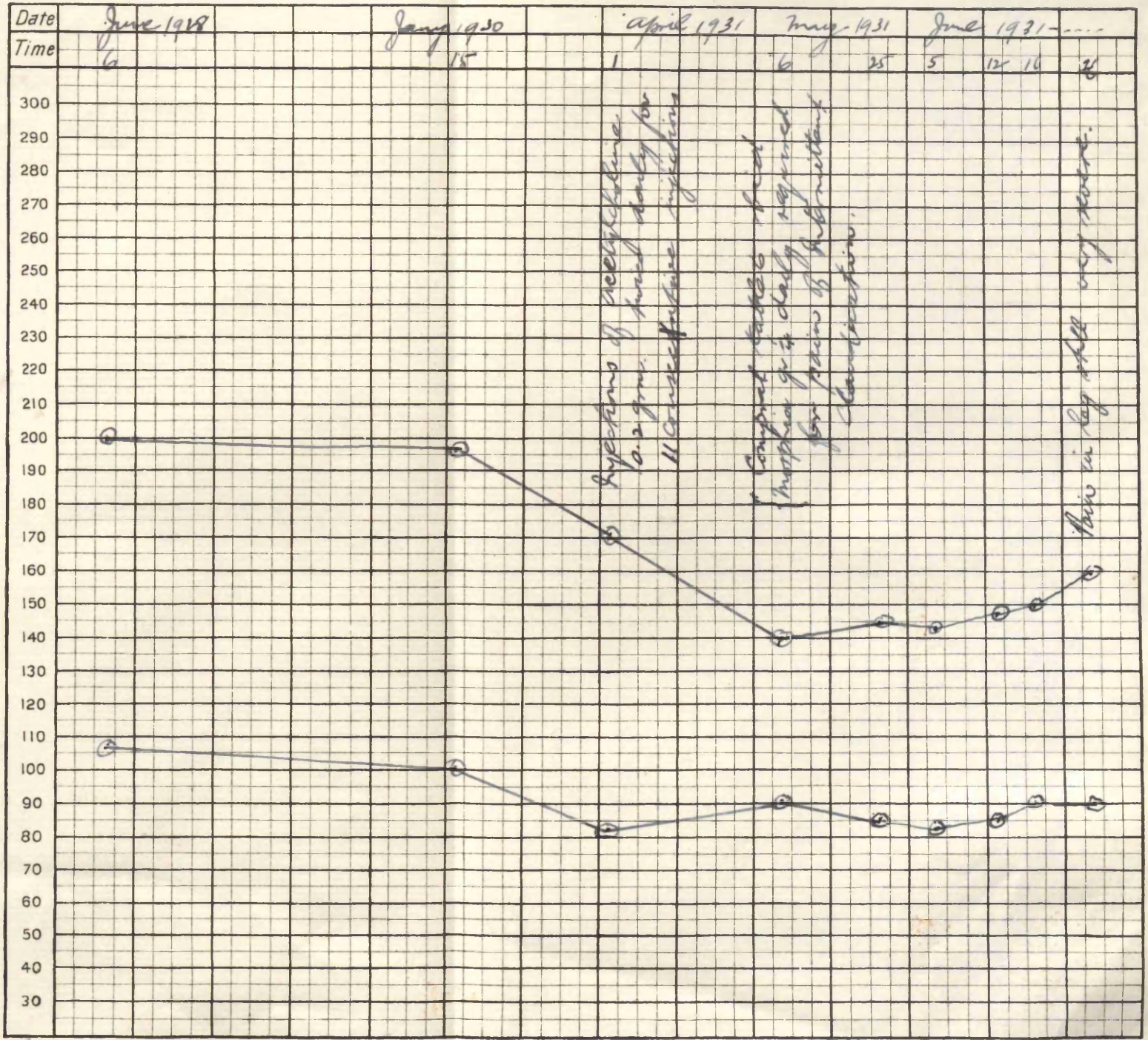
Name Mrs. McG.

Age 65 years.

Disease
Hypertension and Endarteritis.

Notes
Patient felt very weak when B.P. reduced to 140/90 as a result of injections of acetylcholine.

LEWIS'S BLOOD PRESSURE AND PULSE CHART.



H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 25.

no. 25.

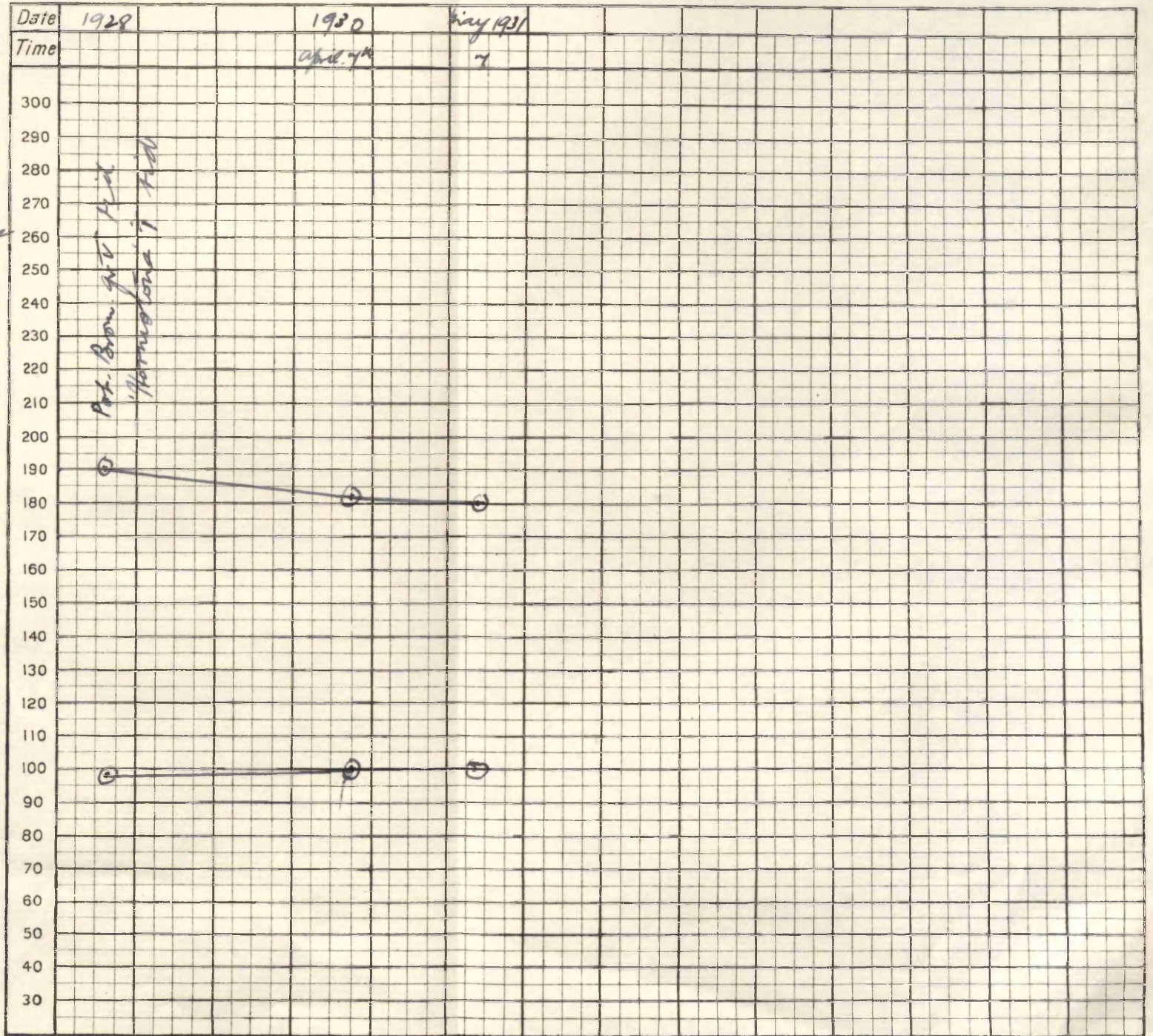
LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Name
Miss A.

Age 46 years.

Disease
Menopausal disturbance and Hypertension.

Notes



H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 26.

no. 26.

Name

Mrs. C.

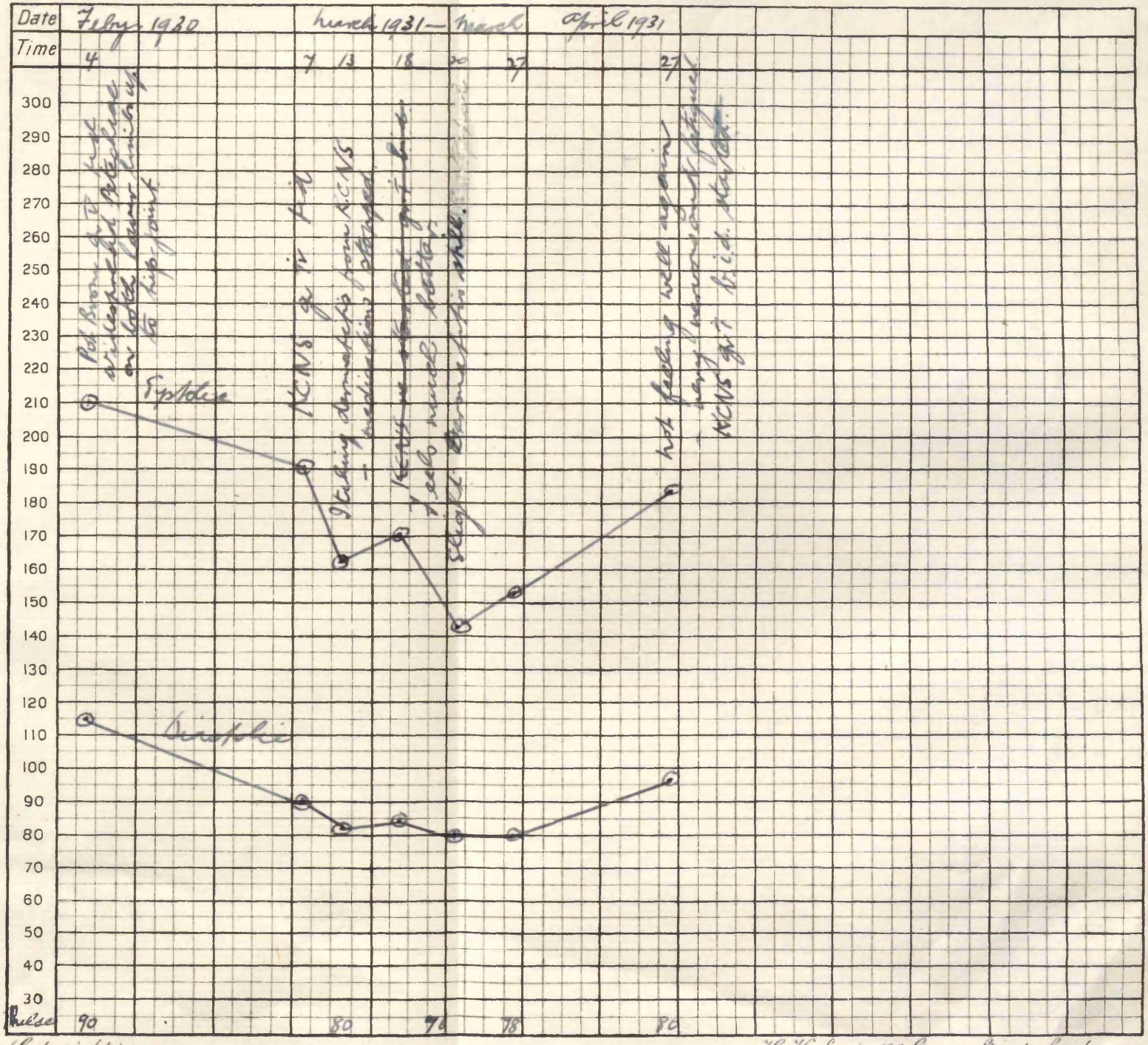
Age 72 years.

Disease

Hypertension.

Notes

LEWIS'S BLOOD PRESSURE AND PULSE CHART.



H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 30.

no. 30

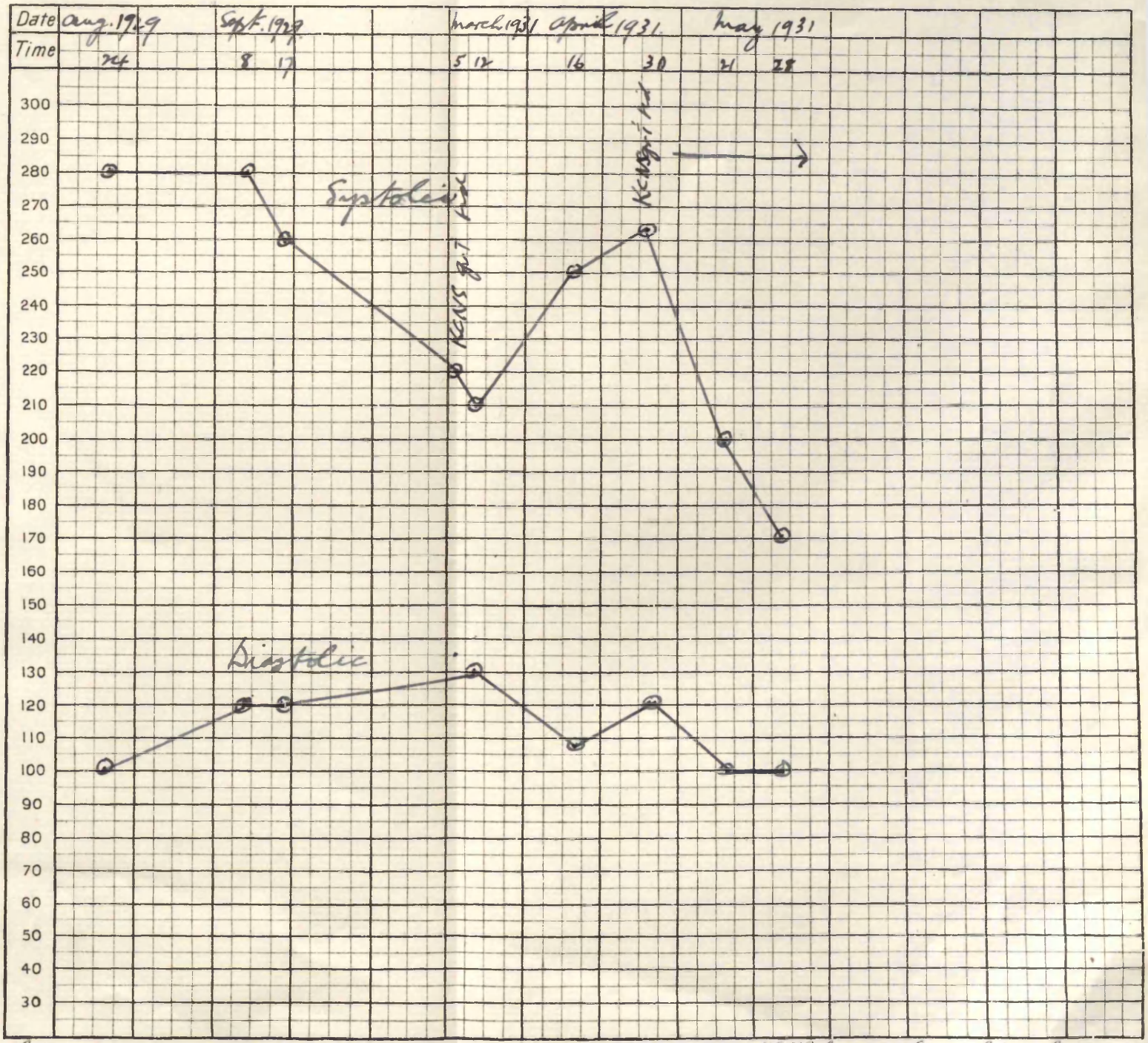
LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Name Mrs. K.

Age 74 years.

Disease Nephritis and Hypertension

Notes Hemiplegia in 1925.



H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 31.

no 31

LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Name

Mrs. M.

Age 62 years

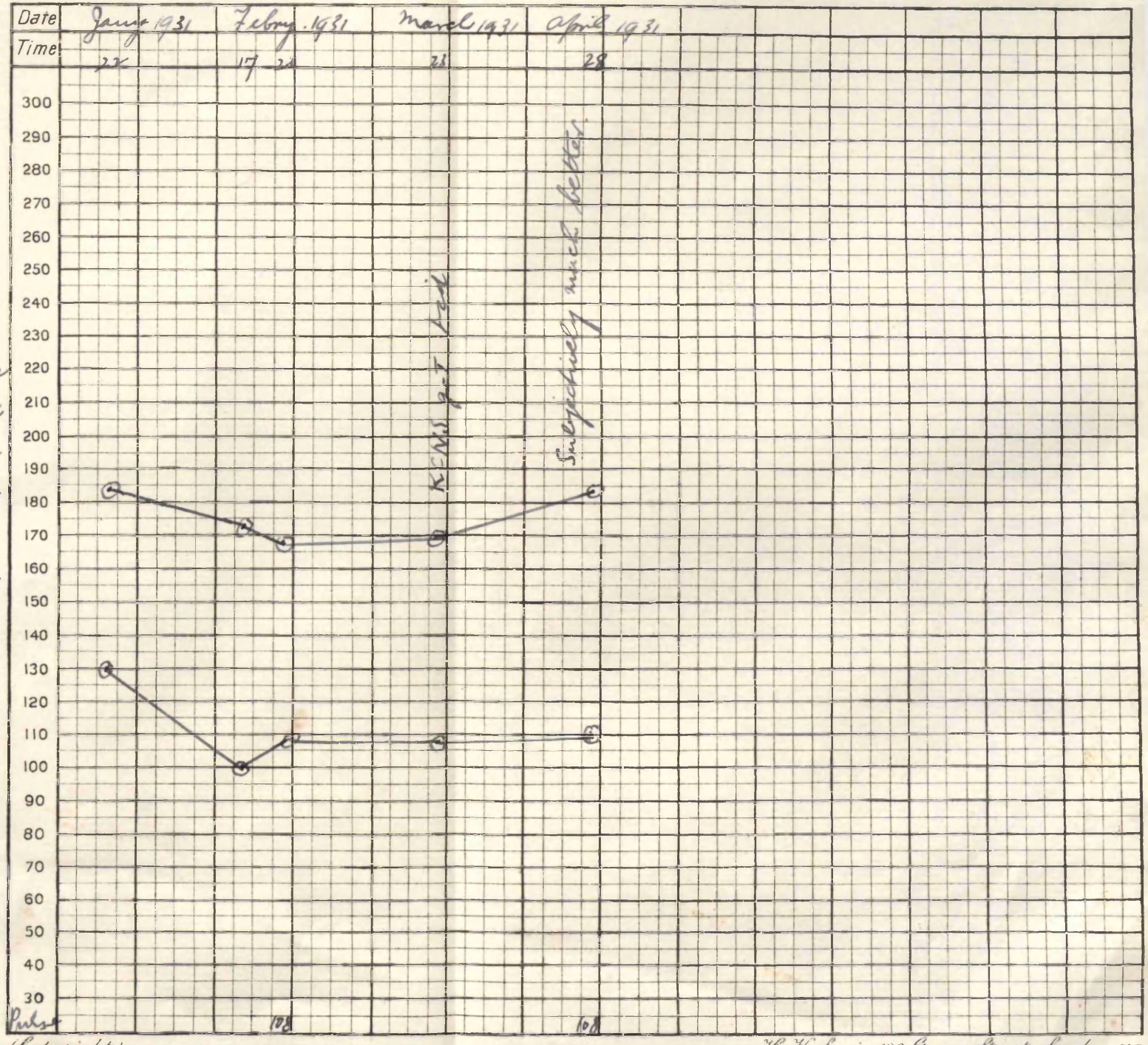
Disease

Hypertension

Notes

Galactose tolerance test showed marked liver dysfunction.

Van den Bergh test = { delayed direct / positive indirect



H. K. Lewis, 136 Gower Street, London, W.C.

Case No. 32.

no. 32

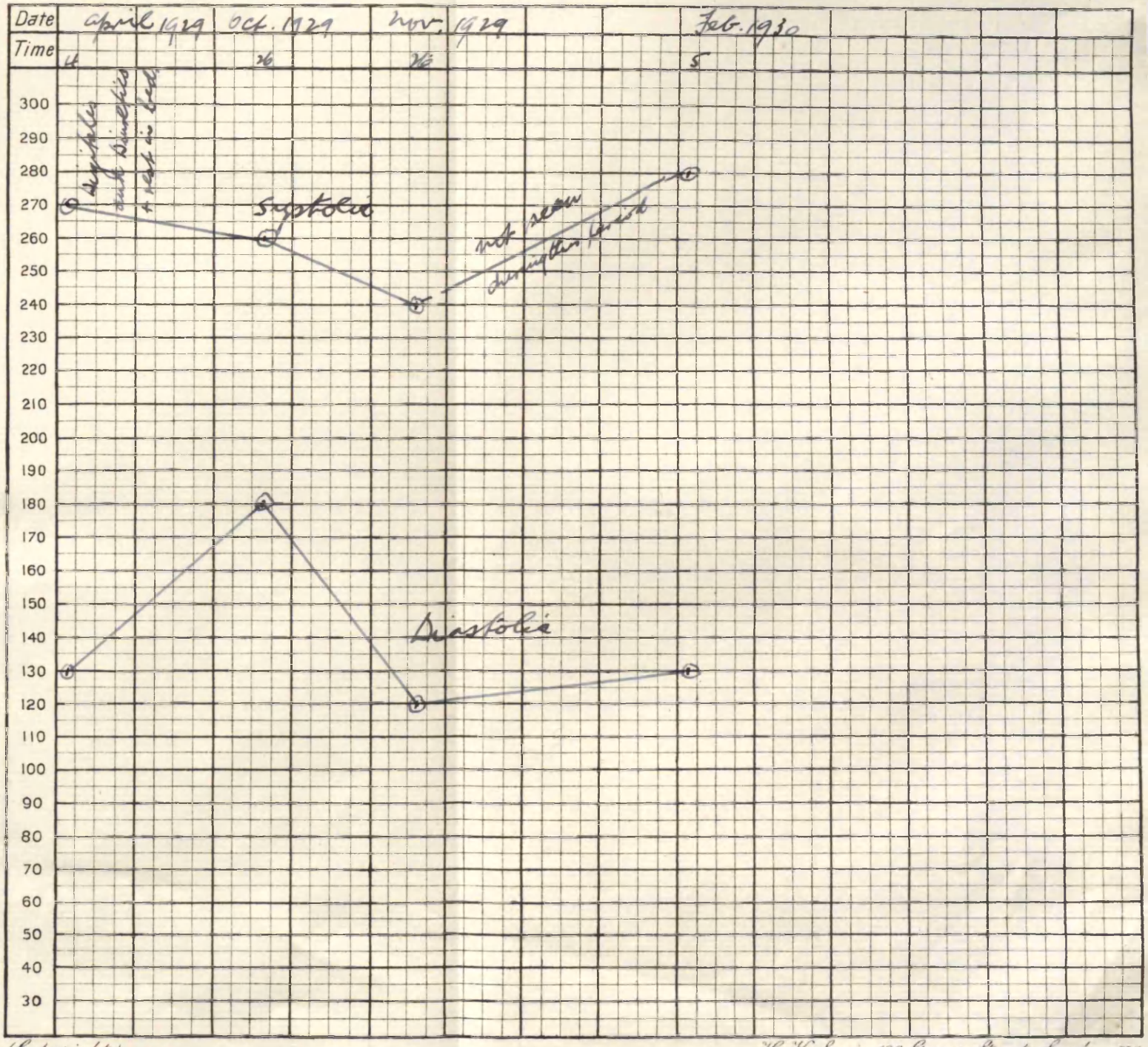
LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Name
Miss M.

Age 54 years

Disease
Hypertensive Cardiac Disease

Notes



H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 33.

no. 33

LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Name

Miss P.

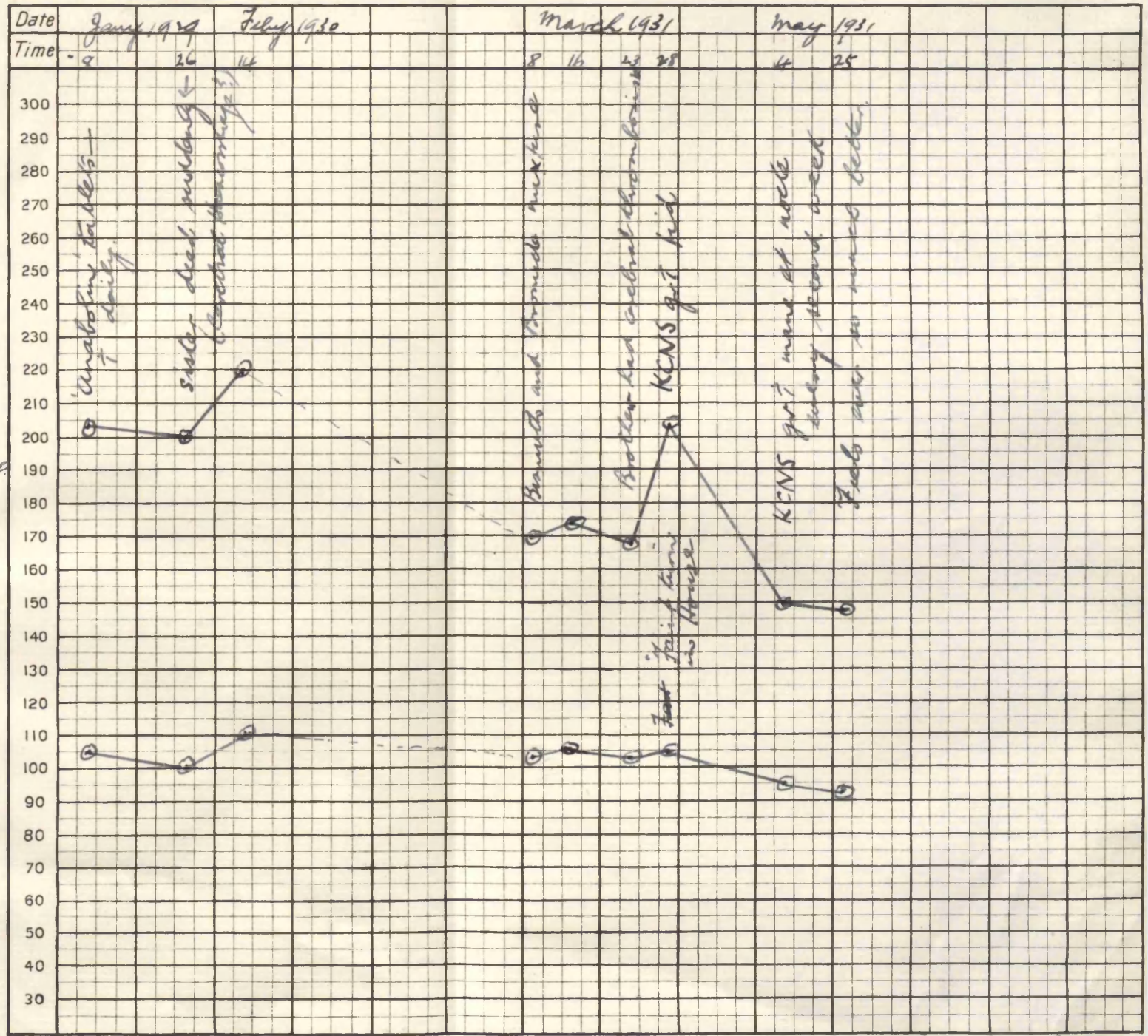
Age 53 years.

Disease

Hypertension

Notes

Family history of death from Cerebral Haemorrhage

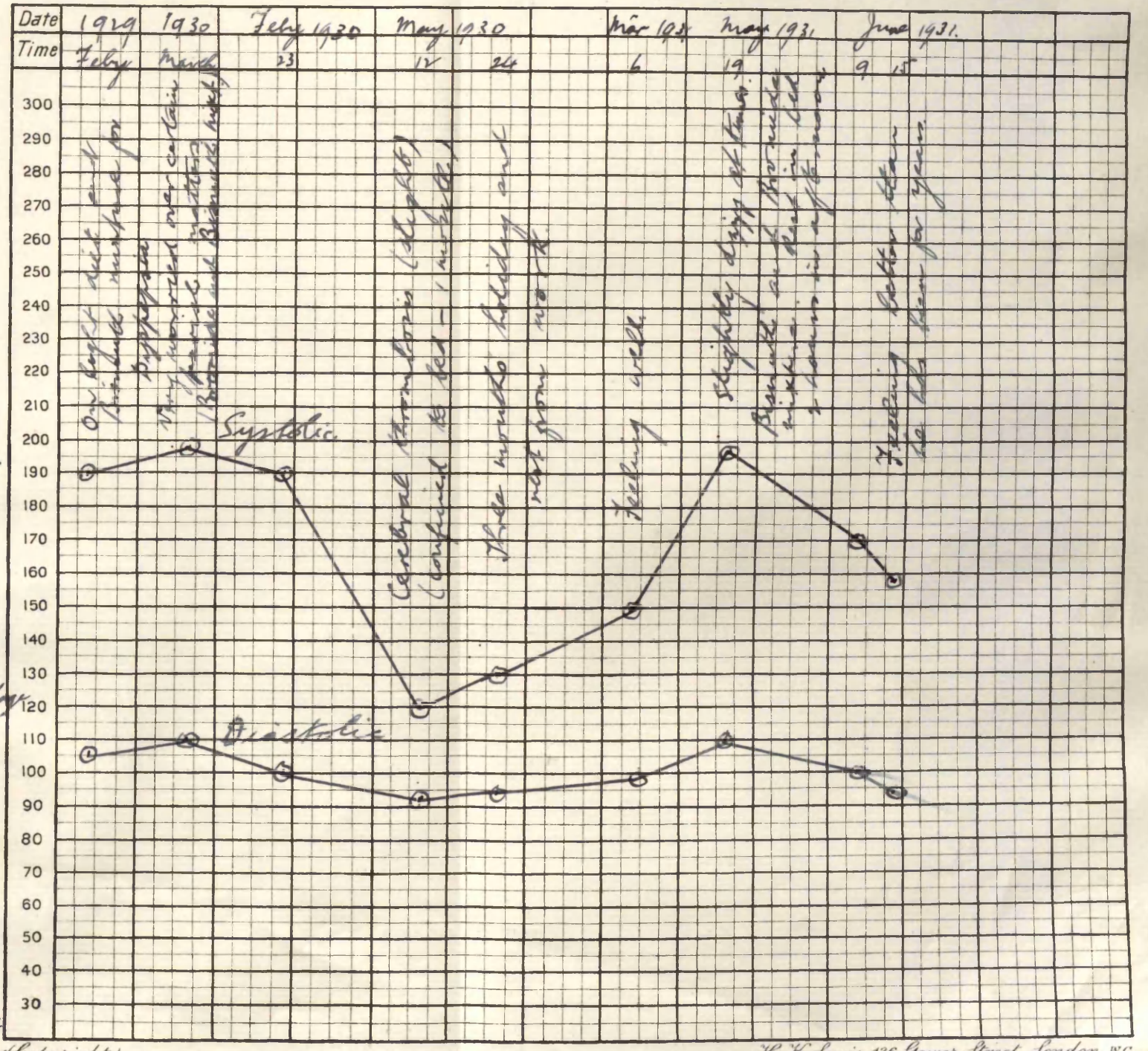


H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 34.

no. 34

LEWIS'S BLOOD PRESSURE AND PULSE CHART.



Name

Rev. R. P.

Age 65 years.

Disease

Hypertension

Notes

Family history of death from Cerebral Haemorrhage. (~~see no. 33 – sister~~).

① Father died of old age

② mother died of post-partum haemorrhage

③ Brother has high B.P.

④ one sister and one brother died suddenly.

⑤ Sister has high B.P. (no. 33).

⑥ another sister has acid dyspepsia

⑦ Hopley has spasm & haemorrhages.

H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 35.

no. 35

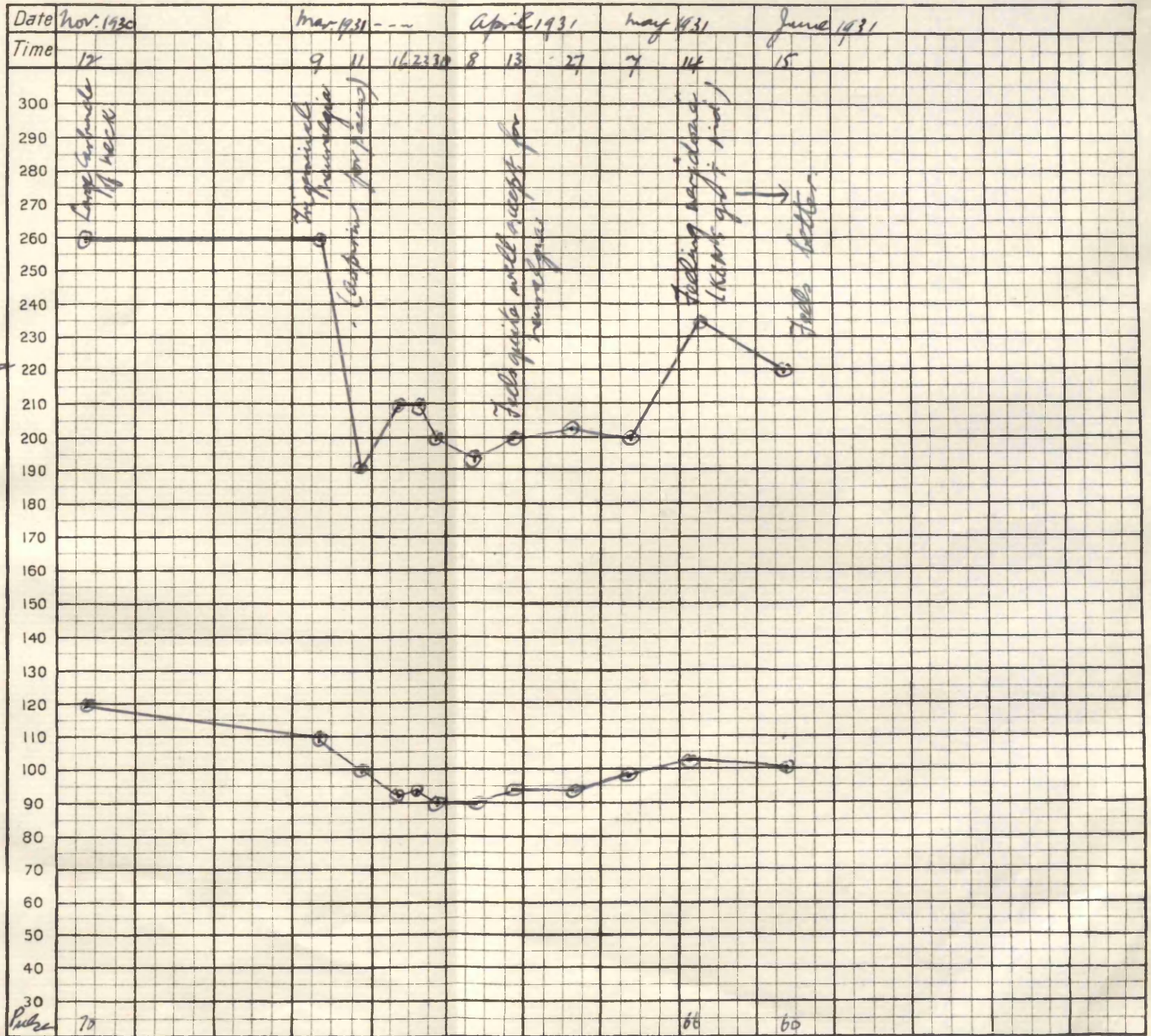
LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Name
Mr. B.

Age 64 years

Disease
Enlarged Prostate and hypertension

Notes
Galactose tolerance test showed normal curve. Van den Bergh negative.



H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 36.

no. 36

LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Name

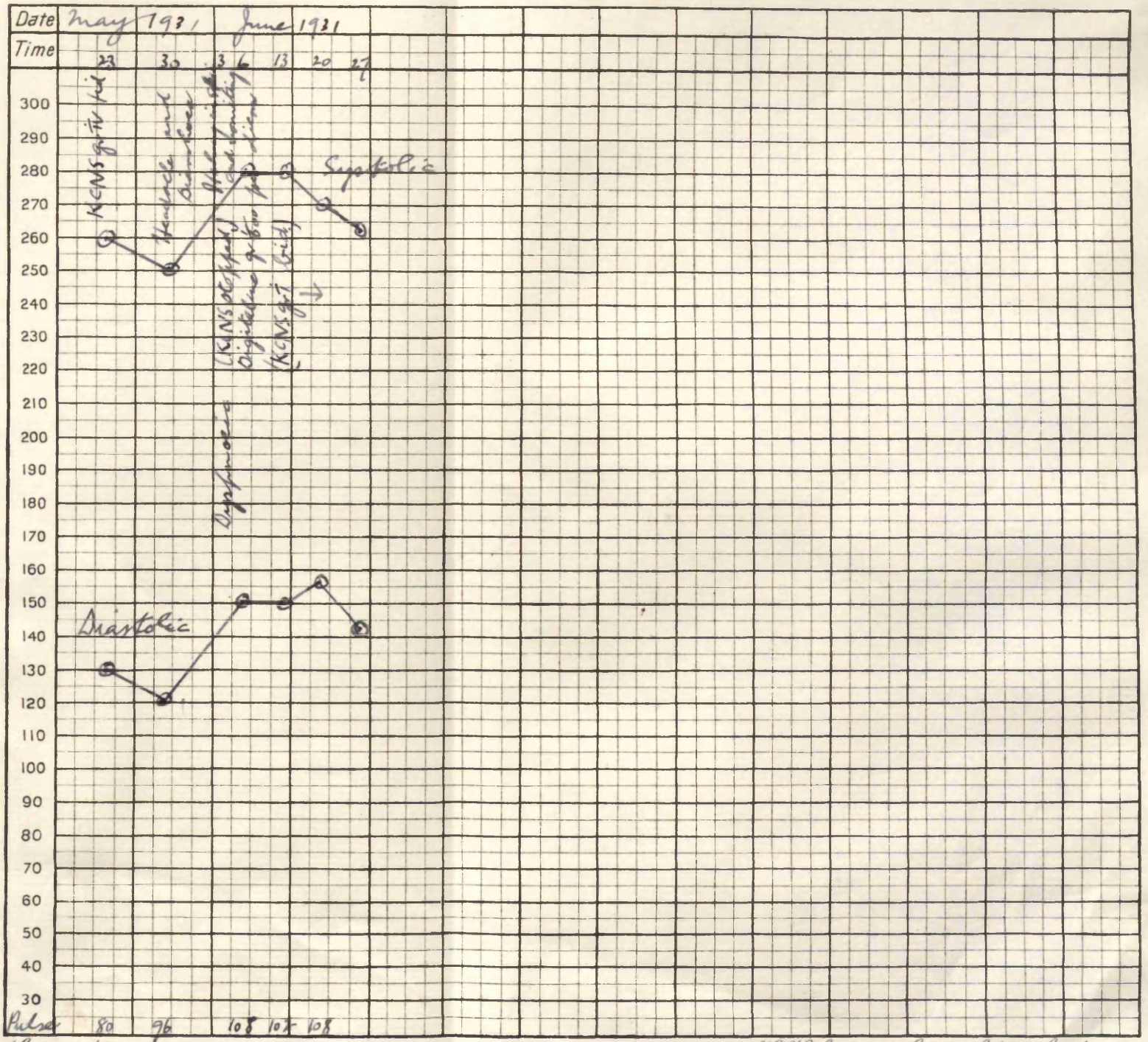
Mr. Mrs K.

Age 59 years.

Disease

Hypertension and nephritis.

Notes

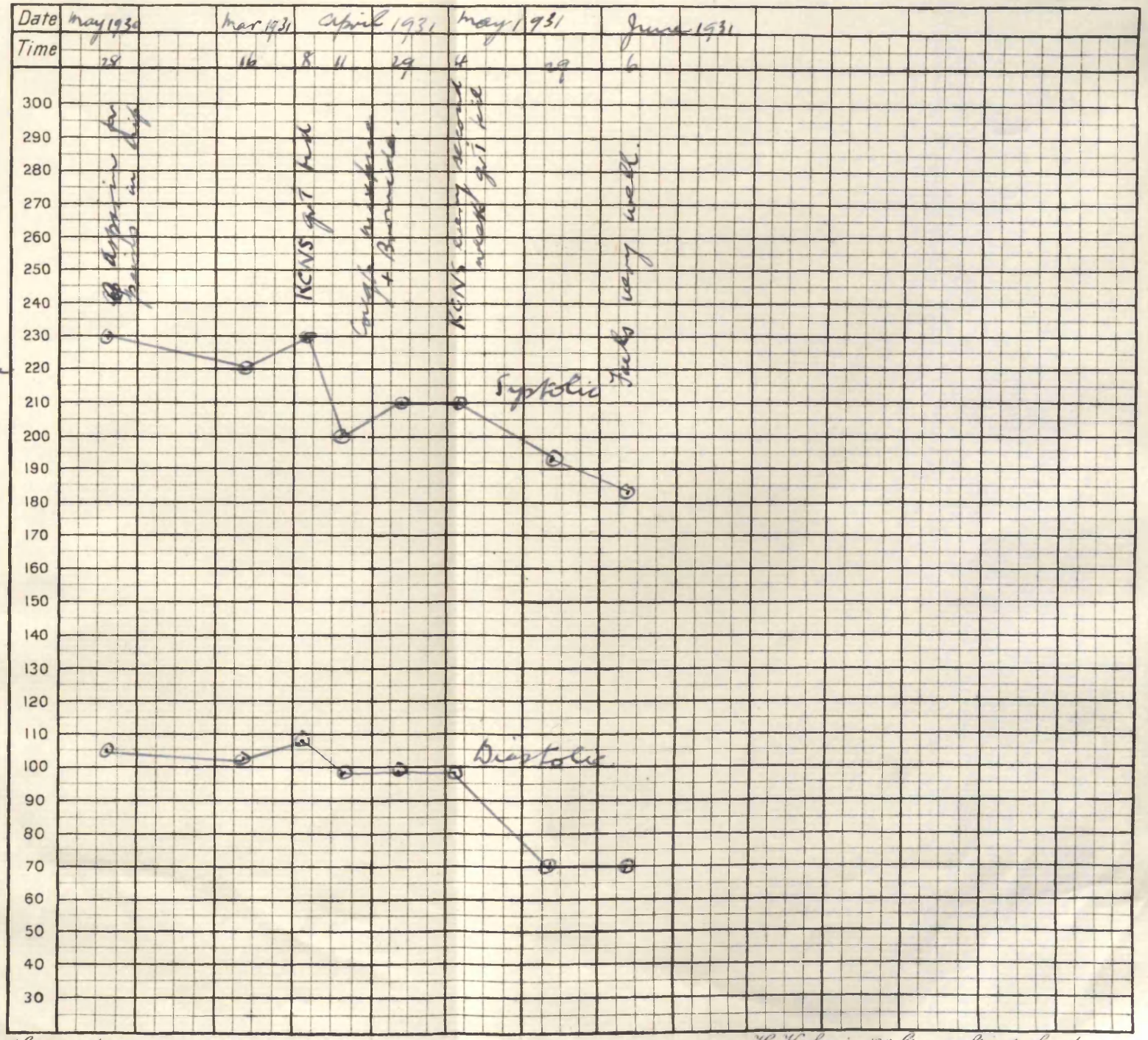


H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 37.

no. 37

LEWIS'S BLOOD PRESSURE AND PULSE CHART.



Name

Mr. H.

Age 70 years.

Disease

Hypertension and nephritis.

Notes

Galactose tolerance test showed impaired liver function.

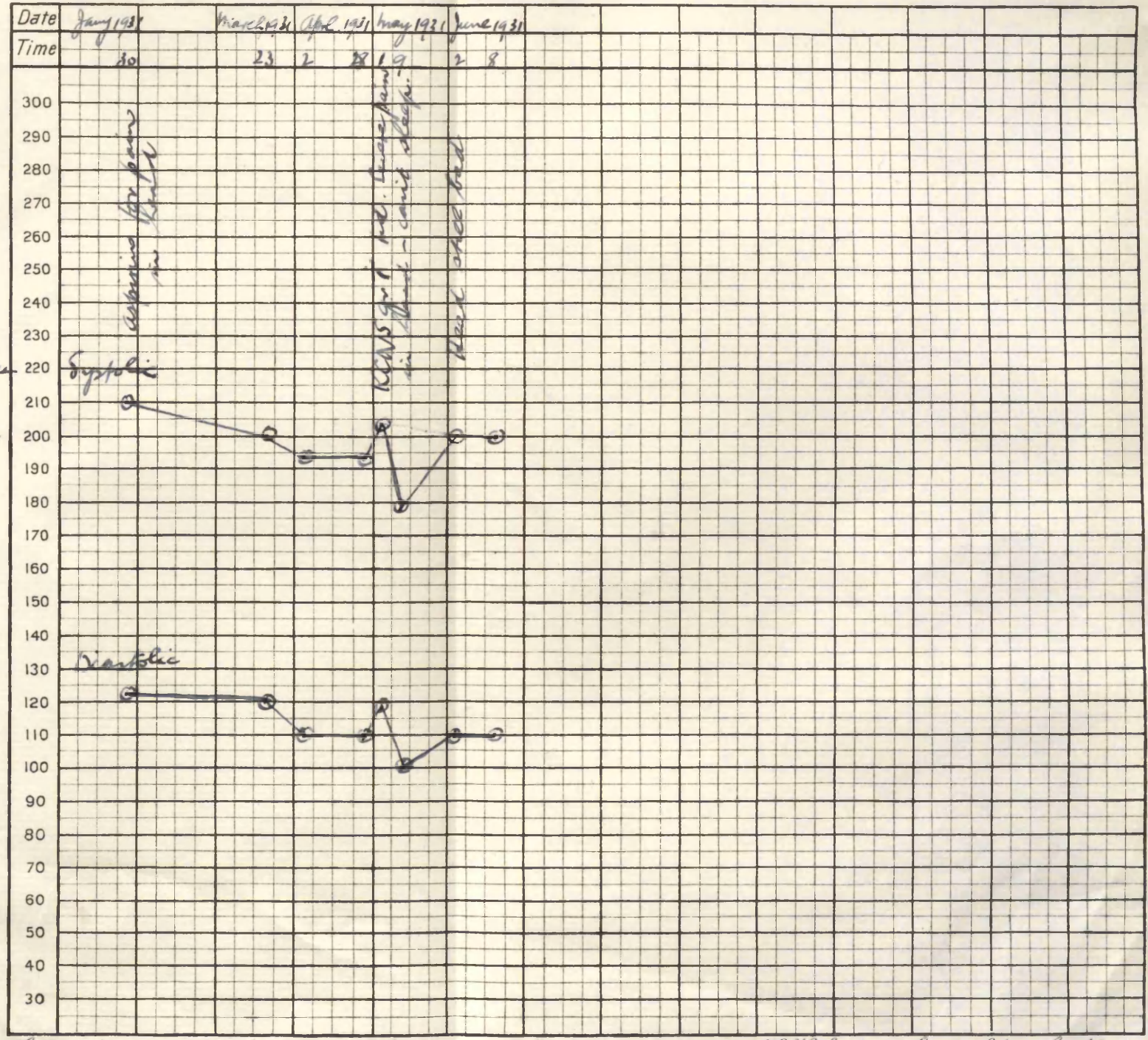
Van den Bergh negative.

H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 38.

no. 38

LEWIS'S BLOOD PRESSURE AND PULSE CHART.



Name

Mr. O.B.

Age 68 years.

Disease

Hypertension

Notes

Galactose tolerance test showed liver dysfunction Van den Bergh negative.

H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 39.

no. 39.

LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Name

Mrs. Mc G.

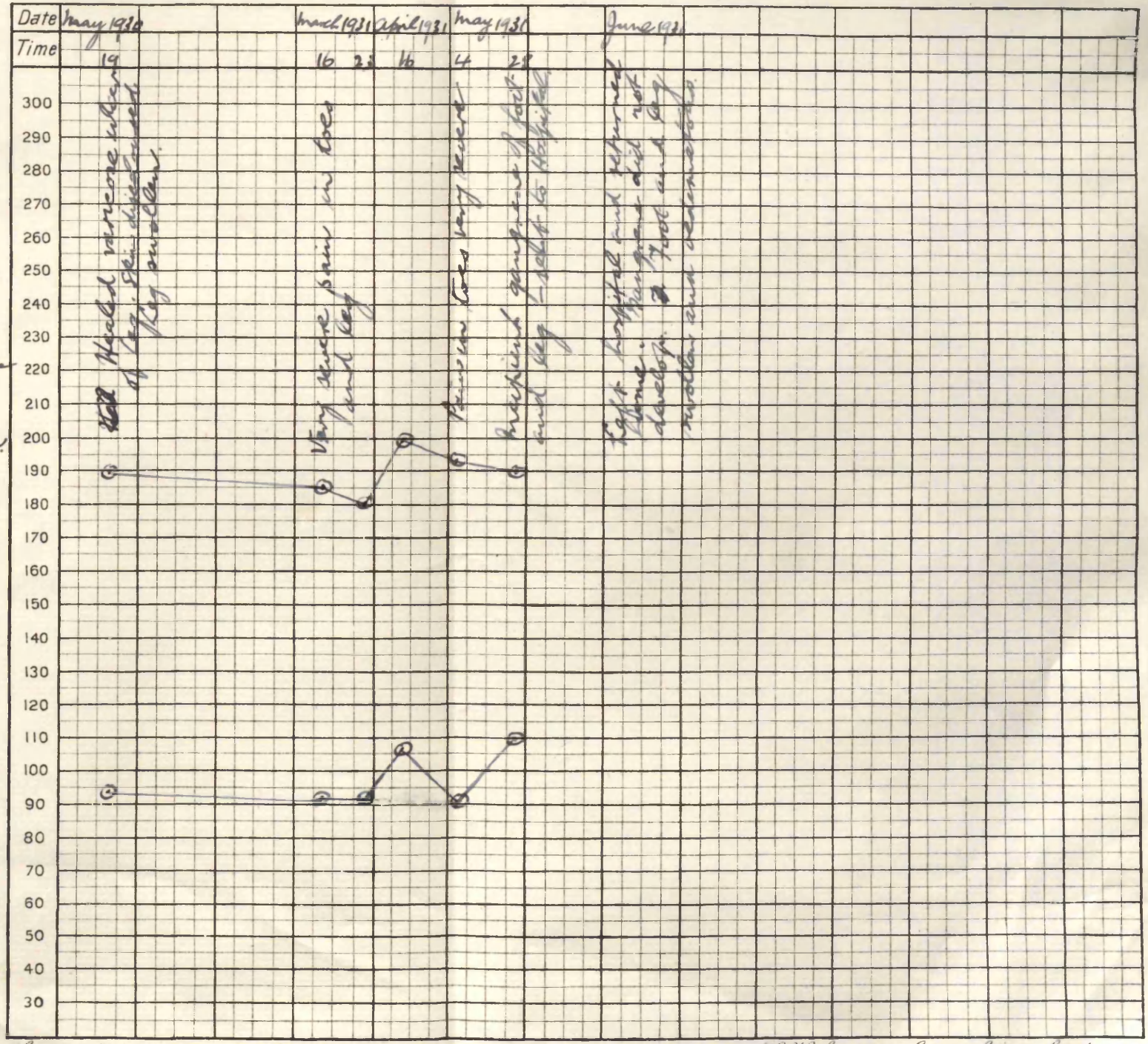
Age 43 years.

Disease

Hypertension and Varicose Ulcer.

Notes

Galactose tolerance test showed liver dysfunction. Van den Bergh negative.



H. K. Lewis, 136, Gower Street, London, W.C.

Case No. 41.

no. 41

LEWIS'S BLOOD PRESSURE AND PULSE CHART.

Name

Mrs. W.

Age 67 years

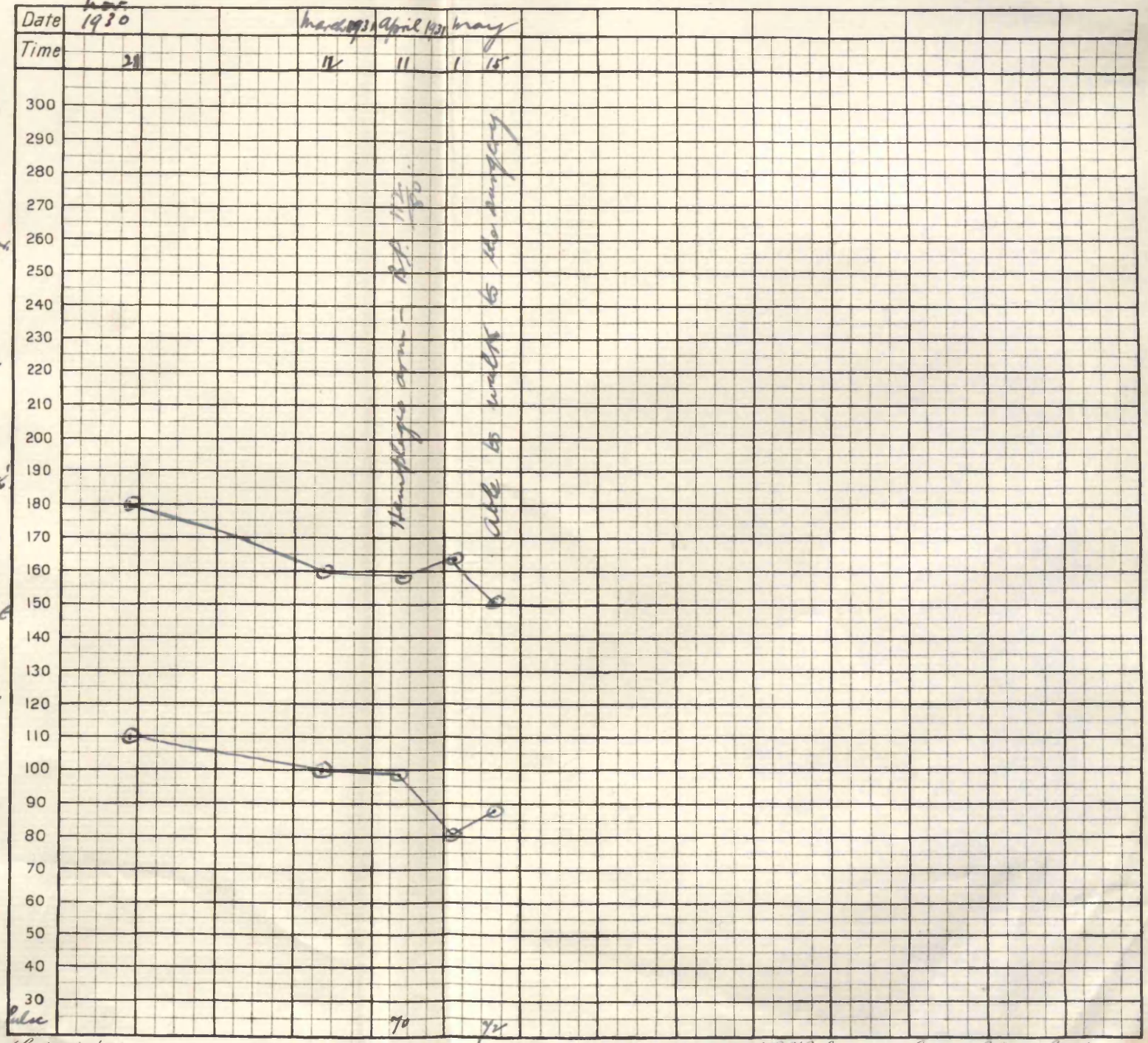
Disease

Nephritis, Hypertension and Arteriosclerosis

Notes

1929 – Left Hemiplegia
" – fracture of femur.

1930 – A slight 'shock' (mouth twisted, and incoherent speech).
No sight in right eye as a result of an accident.



H. K. Lewis, 136, Gower Street, London, W.C.

LEWIS'S BLOOD-SUGAR CHART

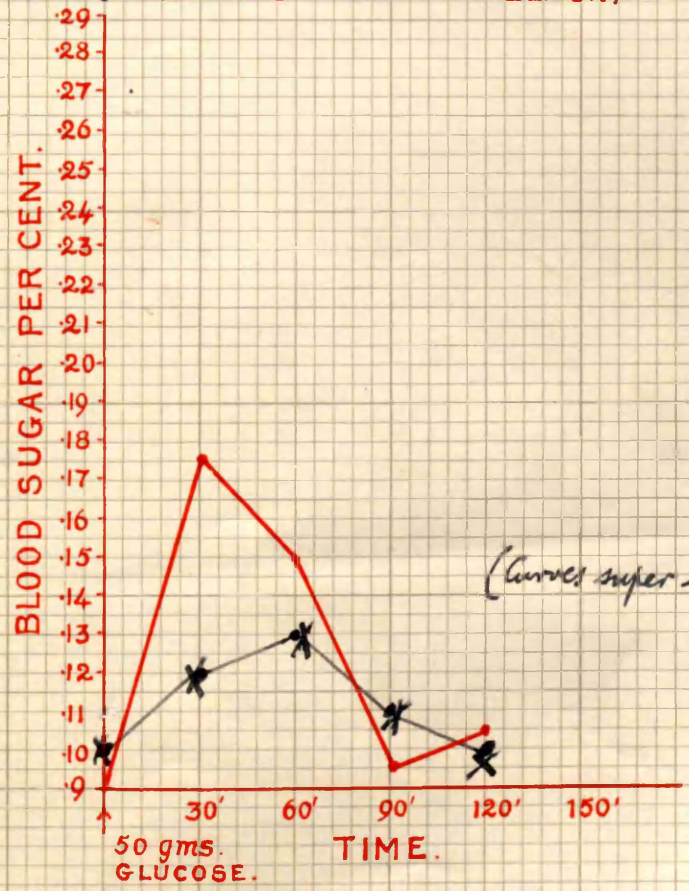
Control (1)

Name Mrs. L. Age 35 yrs.

B.P. 124/72 Gastric Ulcer.

SUGAR TOLERANCE CURVE.

40 gms. Galactose } AVERAGE NORMAL PATIENTS — RED. — BLACK X—X—X



(Curves super-imposed.)

Urine. No albumen, no casts, no urobilinogen, no urobilin

Blood Van den Bergh negative

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

(2)

LEWIS'S BLOOD-SUGAR CHART

Name Mrs. McC. Age 52 yrs.

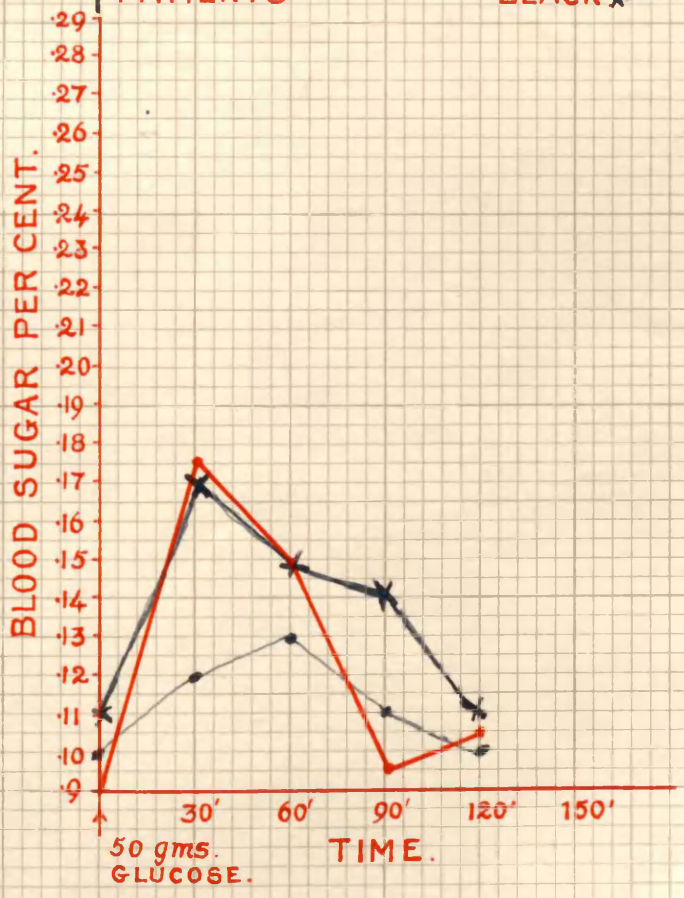
B.P. 160/120 Hypertensia

SUGAR TOLERANCE CURVE.

40 gms. Galactose { AVERAGE NORMAL PATIENTS

RED ~~RED~~

BLACK x



Urine: albumen ± trace
no casts, no corpuscles
no crystals
Van den Bergh's negative

Blood

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

(3) Control

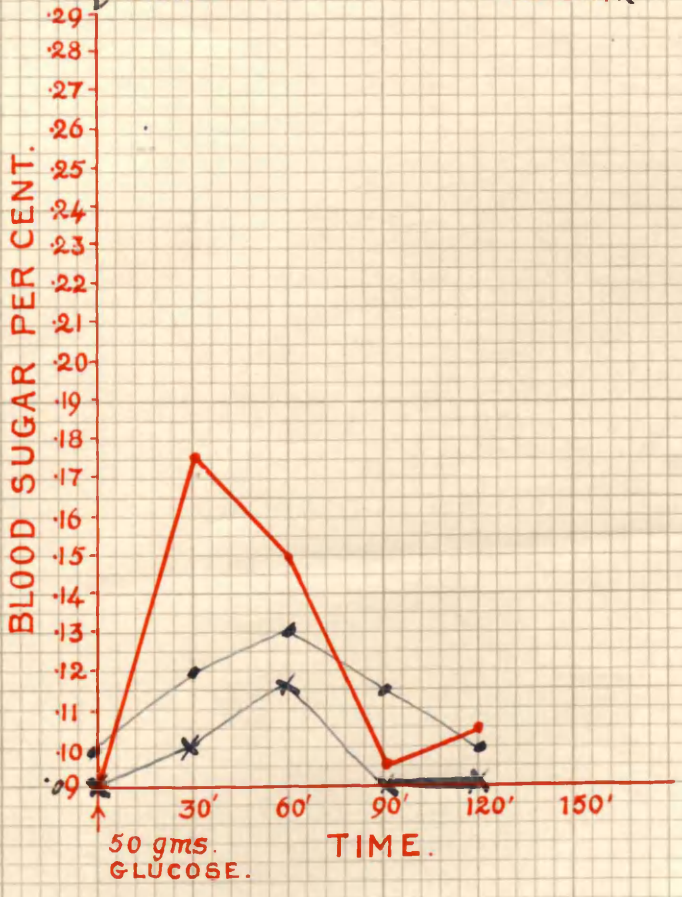
LEWIS'S BLOOD-SUGAR CHART

Name J. T. Age 18 yrs.

BP 110/68. Normal.

SUGAR TOLERANCE CURVE.

40 gms Galactose } AVERAGE NORMAL PATIENTS — RED. — BLACK. ×—×—×



Urine: no albumen, no casts
no urobilinogen, no urobilin
Blood: Van den Bergh negative

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

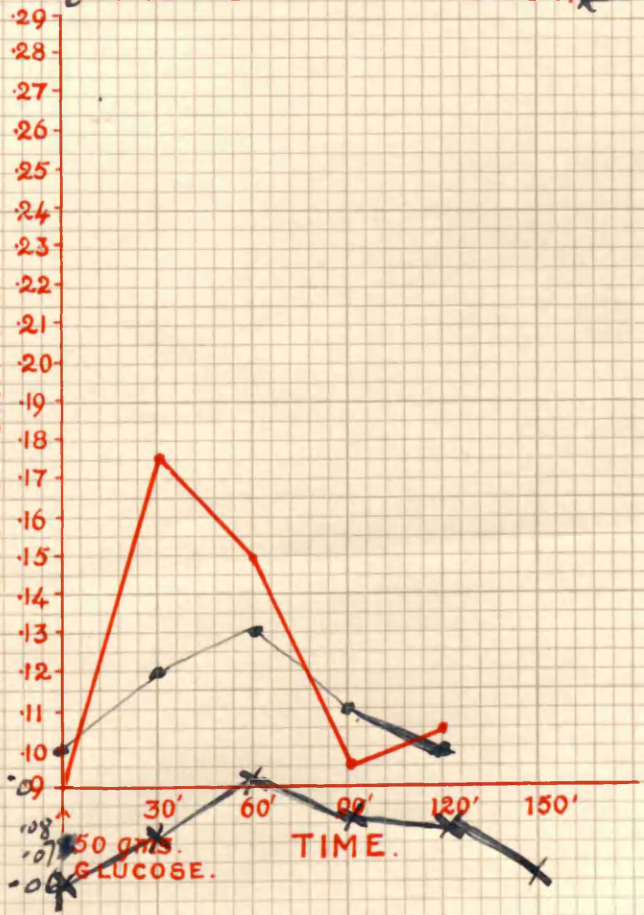
LEWIS'S BLOOD-SUGAR CHART

(4) Control Name Mr S Age 34 yrs.

B.P. 129/72 (Disseminated Sclerosis)

SUGAR TOLERANCE CURVE.

40 gms. Galactose { AVERAGE NORMAL PATIENTS — RED. BLACK.



Urine No albumen, no casts
No urobilinogen, no urobilin
Blood Van den Bergh negative

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

Control

(5)

LEWIS'S BLOOD-SUGAR CHART

Name hr. S. Age 45 yrs.

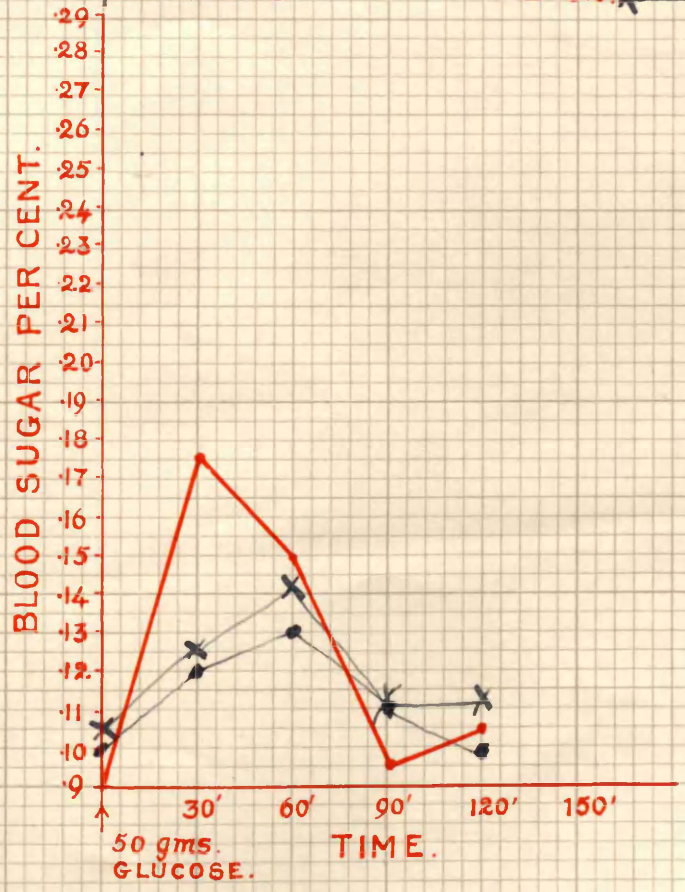
BP 130/84 Mediastinal Tumour.

SUGAR TOLERANCE CURVE.

40 gms. Galactose { AVERAGE NORMAL PATIENTS — RED. BLACK. ×—×—×

Urine. No albumen, no casts, no urobilinogen, no urobilin. Van den Bergh negative.

Blood



London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

LEWIS'S BLOOD-SUGAR CHART

(6) Control

Name Mrs Murdoch Age 51 yrs.

B.P. 134/78. Bronchitis

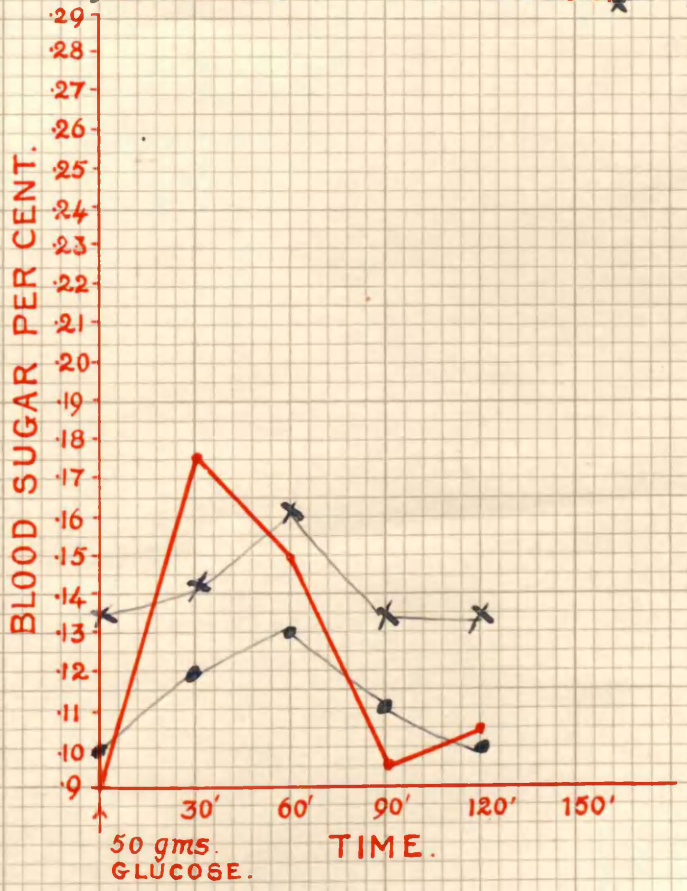
SUGAR TOLERANCE CURVE.

40 gms Galactose

AVERAGE NORMAL PATIENTS

~~RED~~

BLACK



Urine. No albumen, no casts, no urobilin, no urobilinogen.

Blood. Van den Bergh negative.

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

LEWIS'S BLOOD-SUGAR CHART

Control (1)

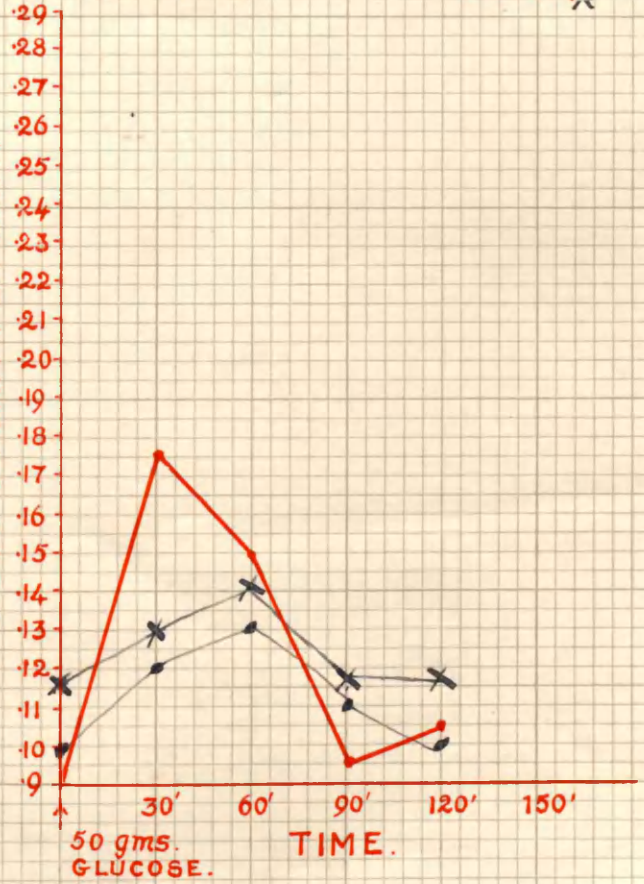
Name Mr. B Age 5-7 yrs.

B.P. 136/82 Subacute Rheumatism.

SUGAR TOLERANCE CURVE.

40 gms. Galactose { AVERAGE NORMAL PATIENTS

~~RED~~ BLACK



Urine no albumen; no casts
no urobilinogen; no urobilin
Blood Van den Bergh negative

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

LEWIS'S BLOOD-SUGAR CHART

Control ®

Name Mr. A. Age 27 yrs.

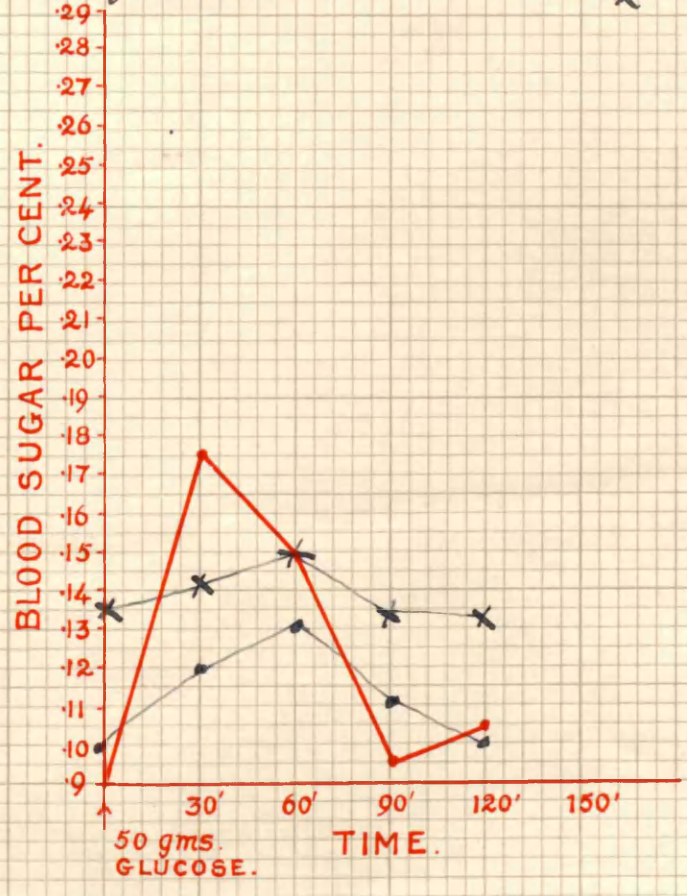
BP 138/80. Renal Calculus

SUGAR TOLERANCE CURVE.

40 gms. Galactose { AVERAGE NORMAL PATIENTS

~~RED~~

BLACK



Urine – albumen +; Blood +; last no casts, urobilinogen +, no urobilin

Blood Van den Bergh ? (not done)

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

(9) Control.

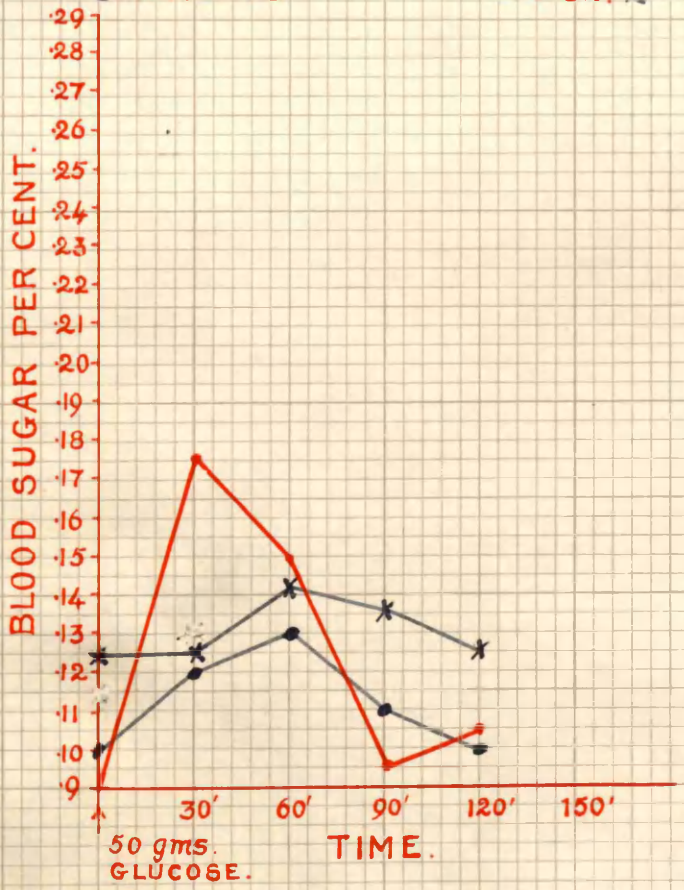
LEWIS'S BLOOD-SUGAR CHART

Name Miss H. Age 18 years.

B.P. 110/66. Mucous Colitis.

SUGAR TOLERANCE CURVE.

40 grms. Galactose { AVERAGE NORMAL PATIENTS — RED. BLACK. ×—×—×



Urine. Slight reduction of Fehling's soln. Not enough for quantitative estim.? No urobilinogen, no urobilin.

Blood. Van den Bergh negative.

London: H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

(10)

Control

LEWIS'S BLOOD-SUGAR CHART

Name hw. B Age 46 yrs

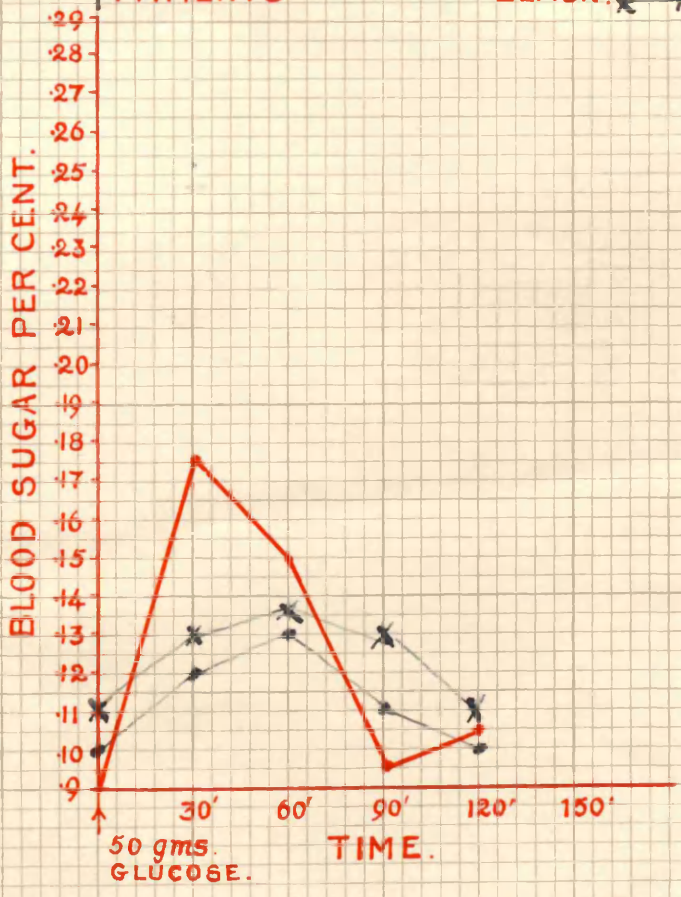
B.P 140/82. Endocarditis

SUGAR TOLERANCE CURVE.

40 gms. lactose

AVERAGE NORMAL PATIENTS

~~RED~~ BLACK.



Urine. No albumen, no casts
No urobilinogen, no urobilin

Blood. Van den Bergh negative

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

(11) Control

LEWIS'S BLOOD-SUGAR CHART

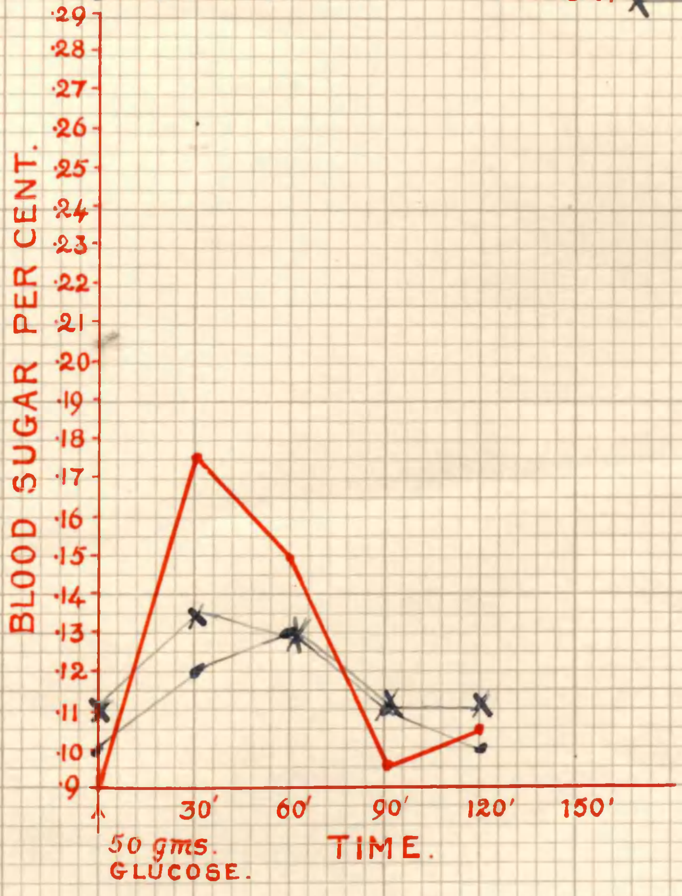
Name Mr. M. *Age* 26 yrs.

B.P. 114/78. normal

SUGAR TOLERANCE CURVE.

40 gms. of dextrose { AVERAGE NORMAL PATIENTS — ~~RED.~~ •—•—• BLACK. ×—×—×

Urine no albumin, no casts
no urobilinogen, no urobilin
Blood Van den Bergh negative



London : H. K. Lewis & Co. Ltd., 136, Gower Street. W.C. 1

(12)

LEWIS'S BLOOD-SUGAR CHART

Name Mr. H. H. Age 31 yrs.

BP 230/150 Acute nephritis

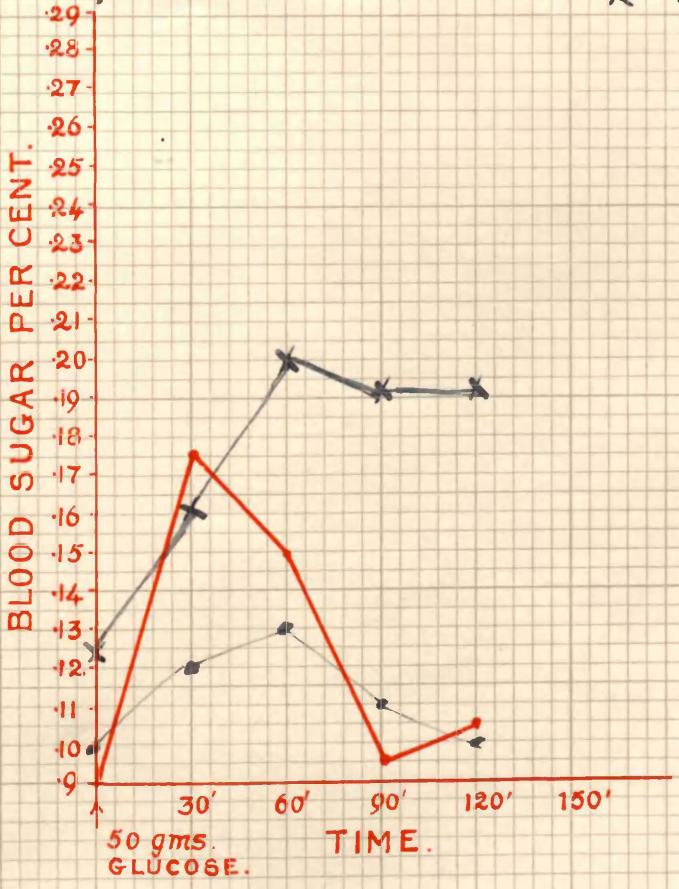
SUGAR TOLERANCE CURVE.

AVERAGE NORMAL PATIENTS

RED

BLACK

40 gms. Glucose



Urine — albumen ++, casts numerous no urobilinogen, no urobilin

Blood Van den Bergh ± negative

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C. 1

(13)

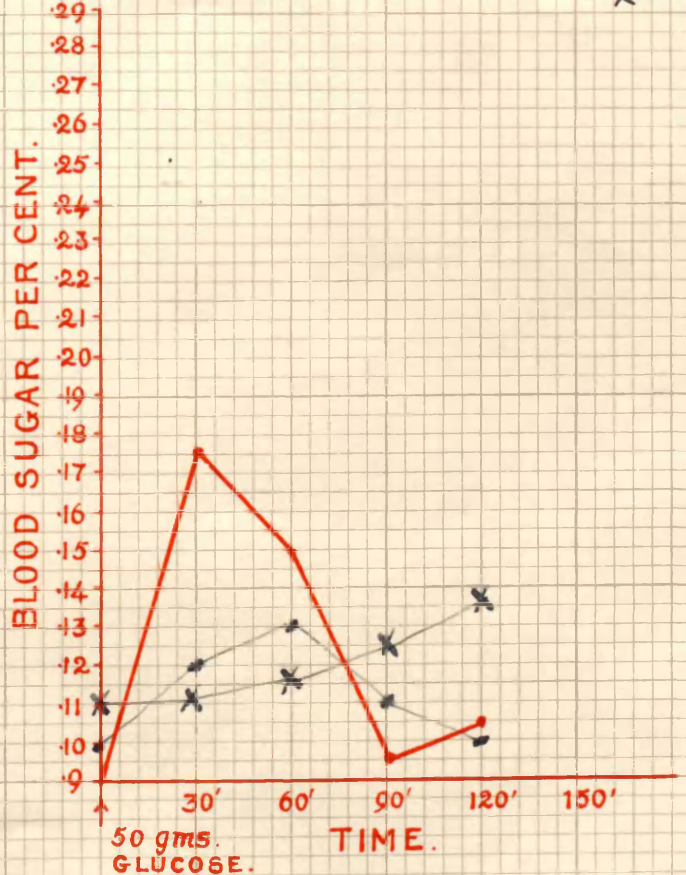
LEWIS'S BLOOD-SUGAR CHART

Name Mr. P .... *Age* 60 yrs.

B.P. $\frac{210}{130}$. Uremia. (Enlarged Prostate)

SUGAR TOLERANCE CURVE.

40 gms. of Galactose { AVERAGE NORMAL / PATIENTS — ~~RED.~~ ●—●—● BLACK. ×—×—×



Urine. Albumen and casts ++
no urobilinogen, no urobilin

Blood Van den Bergh ? (not tested)

London : H. K. Lewis & Co. Ltd., 136, Gower Street. W.C. 1

(14)

LEWIS'S BLOOD-SUGAR CHART

Control

Name: Mr. M. — Age: 27 yrs.

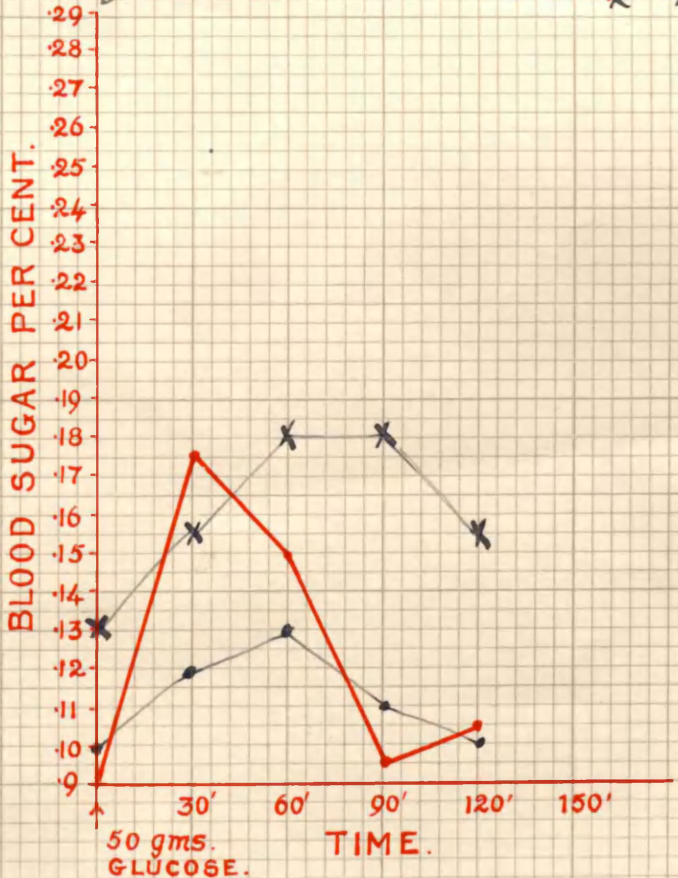
B.P. 114/76 — Diabetic

SUGAR TOLERANCE CURVE.

40 gms. Galactose { AVERAGE NORMAL — ~~RED~~. ●—●—●
{ PATIENTS — BLACK. ✕—✕—✕

Urine: no albumen, no casts, no urobilinogen, no urobilin

Blood: Van den Bergh negative



London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C. 1

(15)

LEWIS'S BLOOD-SUGAR CHART

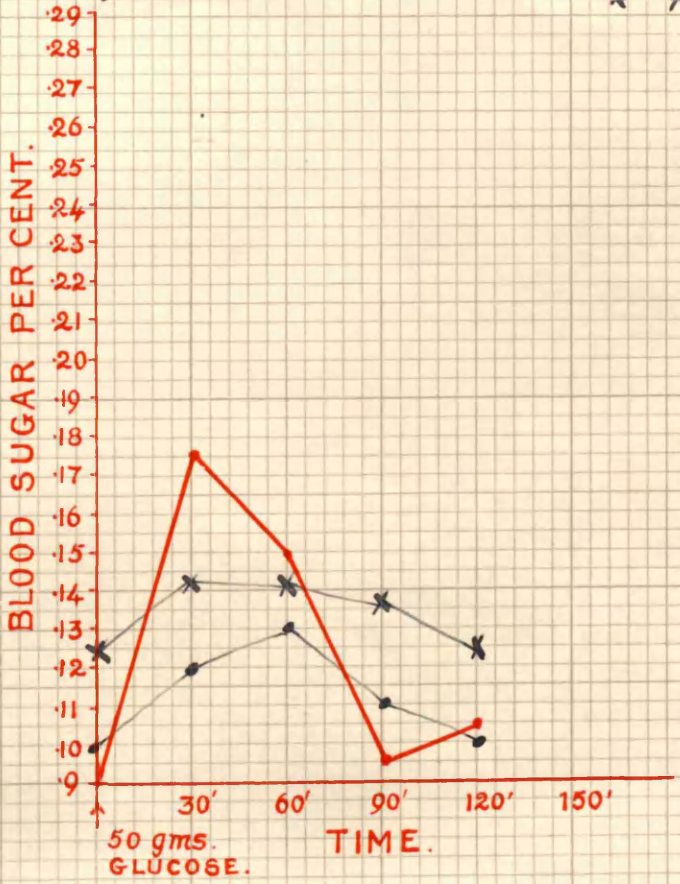
Name Mr. G. Age 65 yrs.

B.P. 200/110. Enlarged Prostate + Hypertension

SUGAR TOLERANCE CURVE.

40 gms. Galactose { AVERAGE NORMAL PATIENTS

~~RED~~ BLACK



Urine Albumen - trace
no casts, no erythrocytes
no urobilin
Blood Van den Bergh negative

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

(16)

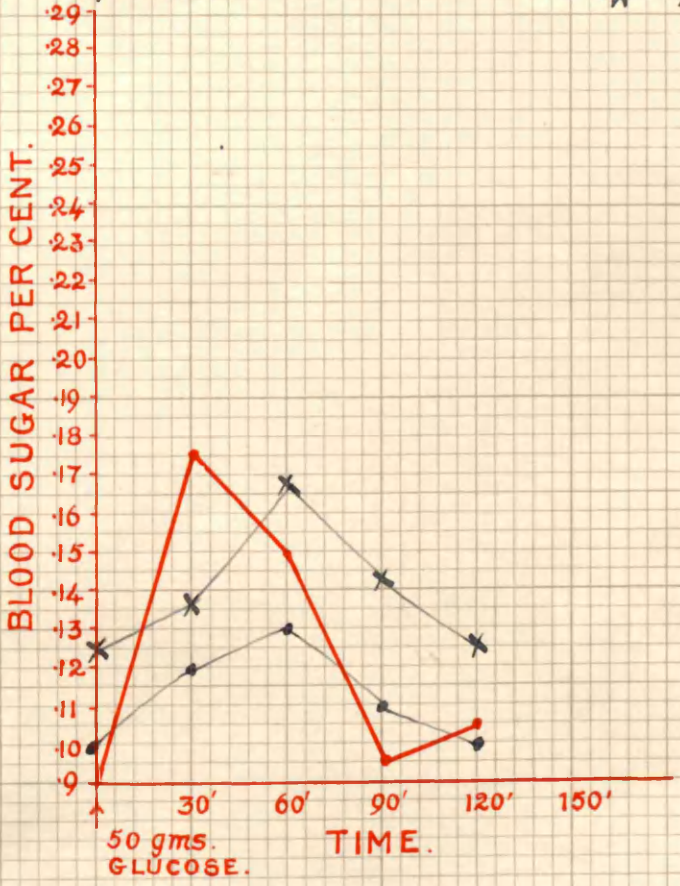
LEWIS'S BLOOD-SUGAR CHART

Name Mrs H. Age 70 yrs.

BP 210/110 Hyperpiesia

SUGAR TOLERANCE CURVE.

40 gms. Galactose { AVERAGE NORMAL PATIENTS — RED. — BLACK.



Urine albumen ++, no casts
no urobilinogen no urobilin
Blood Van den Bergh negative

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C. 1

(17)

LEWIS'S BLOOD-SUGAR CHART

Name Mrs M. Age 50 yrs.

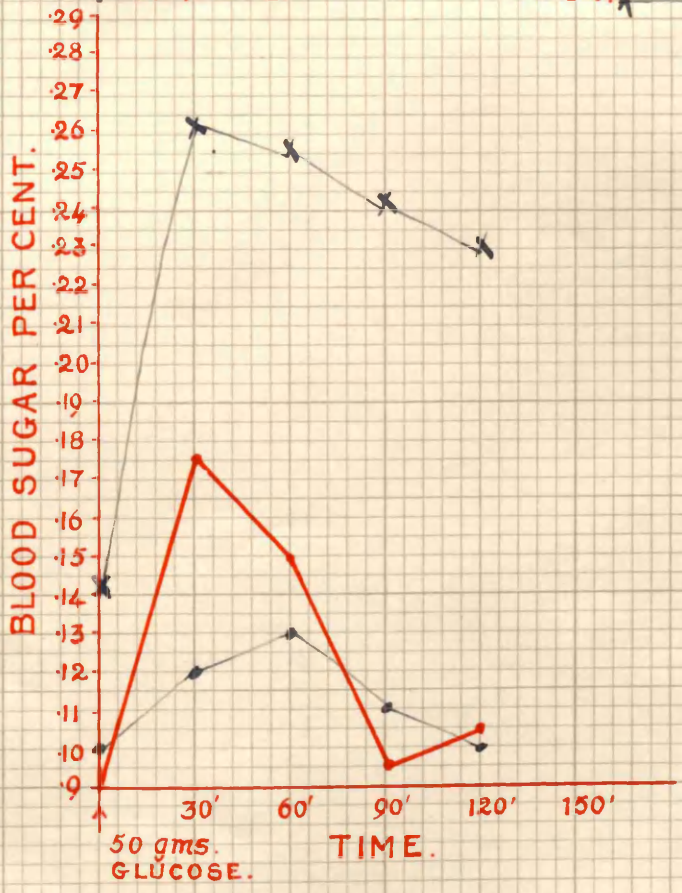
BP. 190/120 Hypertension

SUGAR TOLERANCE CURVE.

40 gms. Galactose } AVERAGE NORMAL PATIENTS

~~RED.~~ BLACK.

Urine: no albumen, no casts
no urobilinogen, no urobilin
Blood Van den Bergh ∫ delayed direct
positive indirect



London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

(18)

LEWIS'S BLOOD-SUGAR CHART

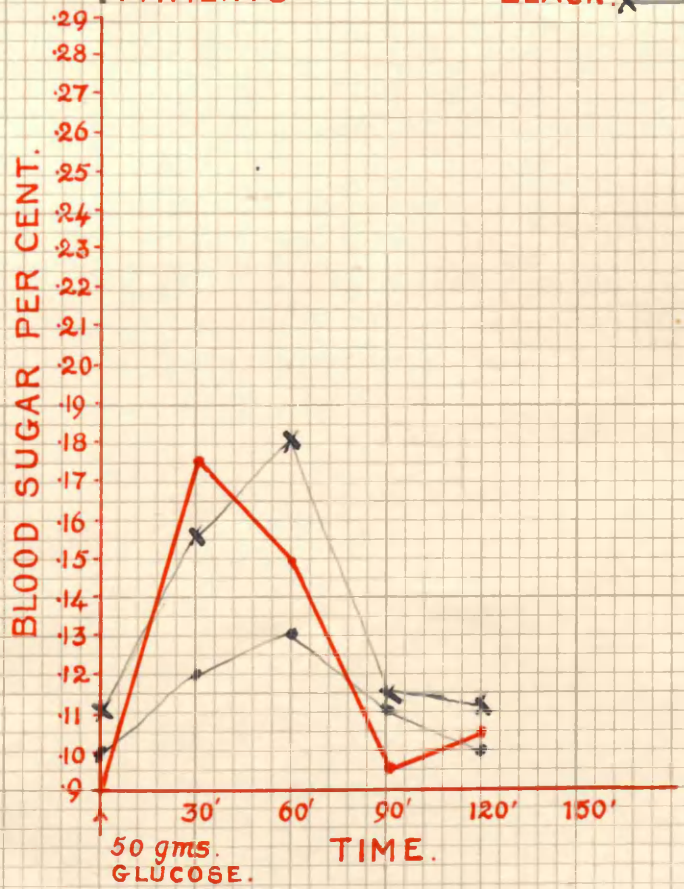
Name Mrs. R. Age 75 yrs.

B.P. 220/195 Hyperpiesia

SUGAR TOLERANCE CURVE.

40 gms. galactose { AVERAGE NORMAL PATIENTS

~~RED.~~ BLACK.



Urine albumen 0, no casts
no urobilinogen, no urobilin
Blood Van den Bergh negative

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

(19)

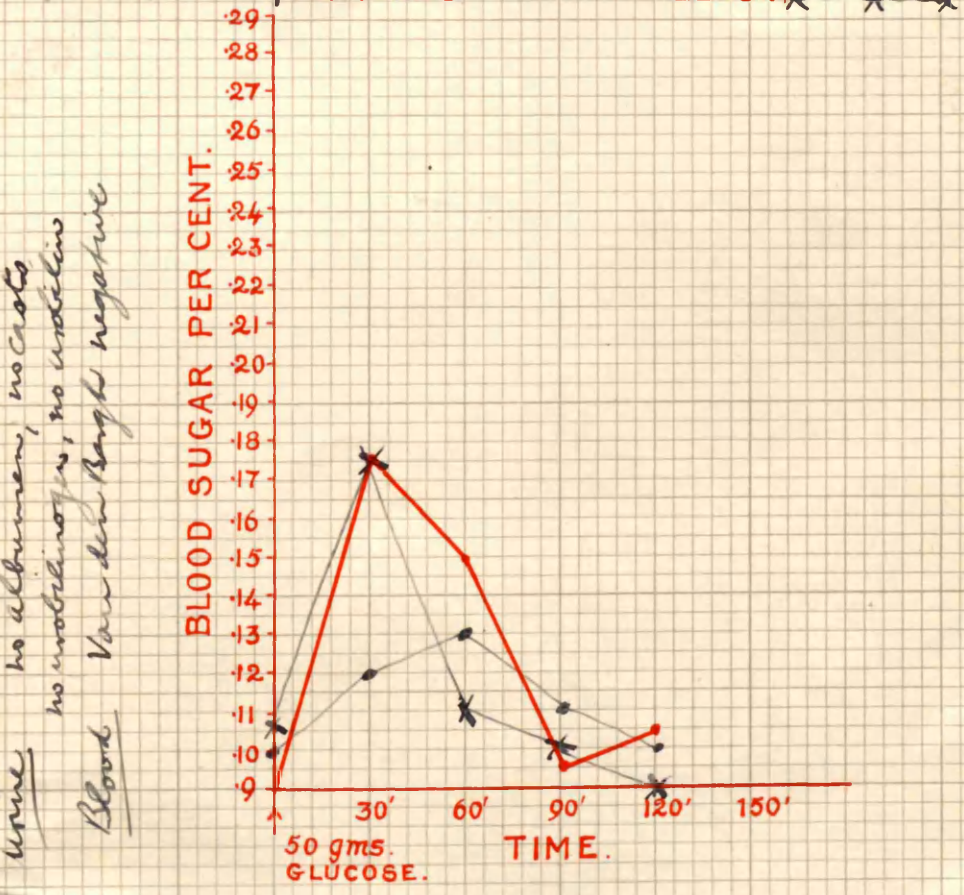
LEWIS'S BLOOD-SUGAR CHART

Name Mr. Wm J. Age 71 yrs.

BP 190/112 Hyperpiesia

SUGAR TOLERANCE CURVE.

50 gms. Glucose | AVERAGE NORMAL PATIENTS | ~~RED~~ BLACK



Urine: no albumen, no casts, no urobilinogen; no urobilin; no ketones

Blood: Van den Bergh negative

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(20)

LEWIS'S BLOOD-SUGAR CHART

Name Mrs. H. D. Age 5[illegible] yrs.

B.P. 200/136 Endocarditis + Hyperpiesis

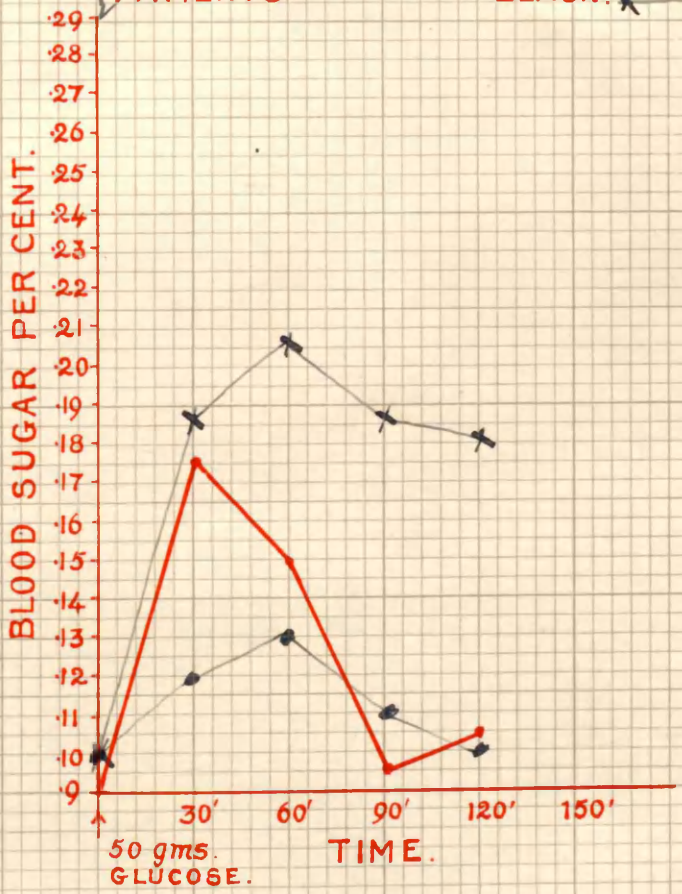
SUGAR TOLERANCE CURVE.

40 gms. Galactose

AVERAGE NORMAL PATIENTS

~~RED~~.

BLACK.



Urine: albumen +, casts = a few
no urobilinogen; no urobilin
Blood Van den Bergh negative

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

(21)

LEWIS'S BLOOD-SUGAR CHART

Name Mrs S Age 65 yrs.

B.P. 232/125 Hypertension

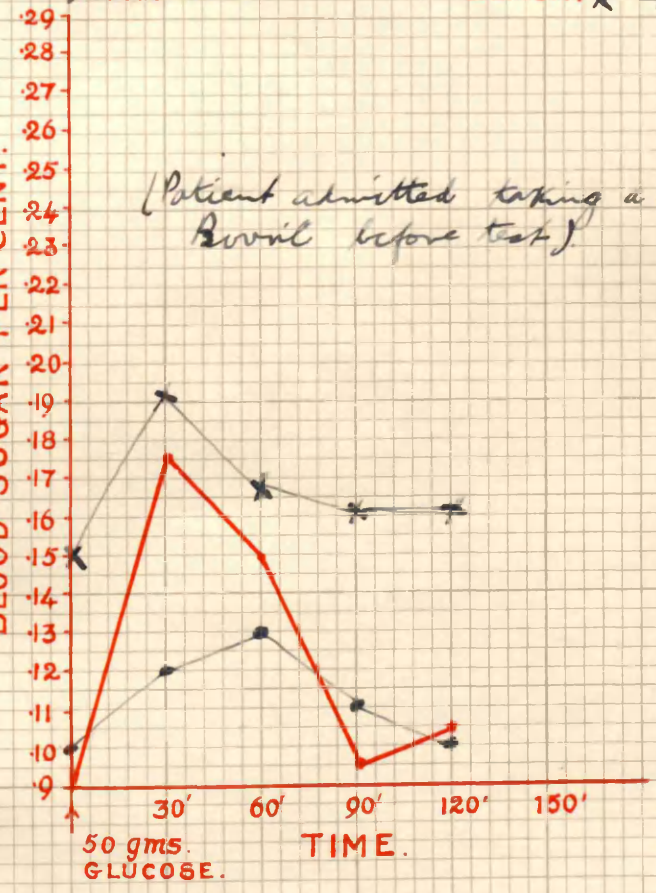
SUGAR TOLERANCE CURVE.

50 gms. dextrose

AVERAGE NORMAL PATIENTS

~~RED~~

BLACK.



(Patient admitted taking a cup of Bovril before test.)

Urine: no albumin, no casts, no urobilinogen, no urobilin

Blood Van den Bergh negative

Bacilluria + (B. Coli)

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C. 1

(22)

LEWIS'S BLOOD-SUGAR CHART

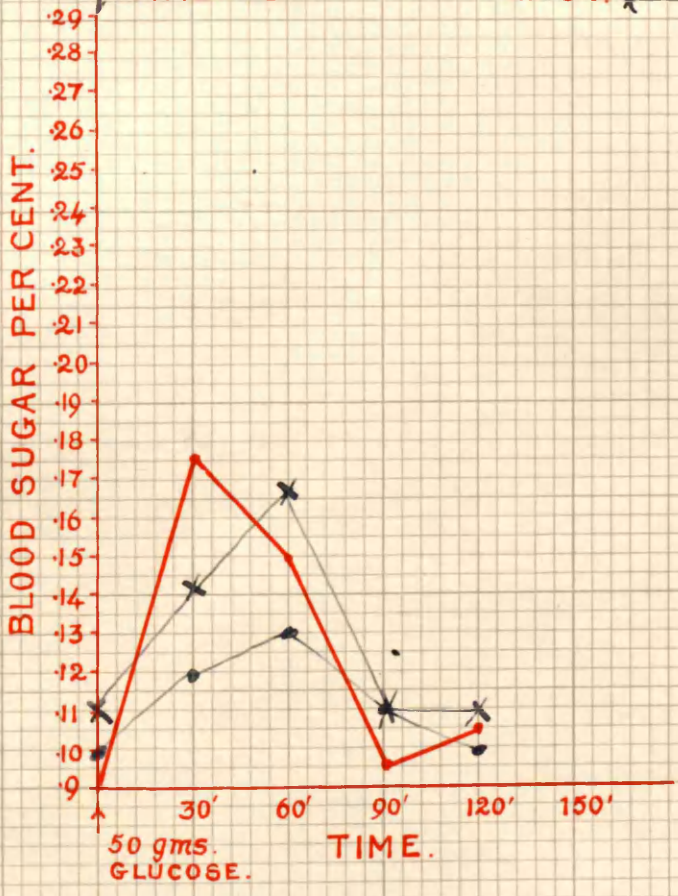
Name Miss D. Age 48 yrs.

BP 236/130 Hyperpiesia

SUGAR TOLERANCE CURVE.

40 gms. Galactose { AVERAGE NORMAL PATIENTS

~~RED~~. BLACK.



Urine: No albumen, no casts
no urobilinogen, no urobilin
Blood Van den Bergh } delayed direct positive indirect

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

(13)

LEWIS'S BLOOD-SUGAR CHART

Name Mrs. B. Age 66 yrs.

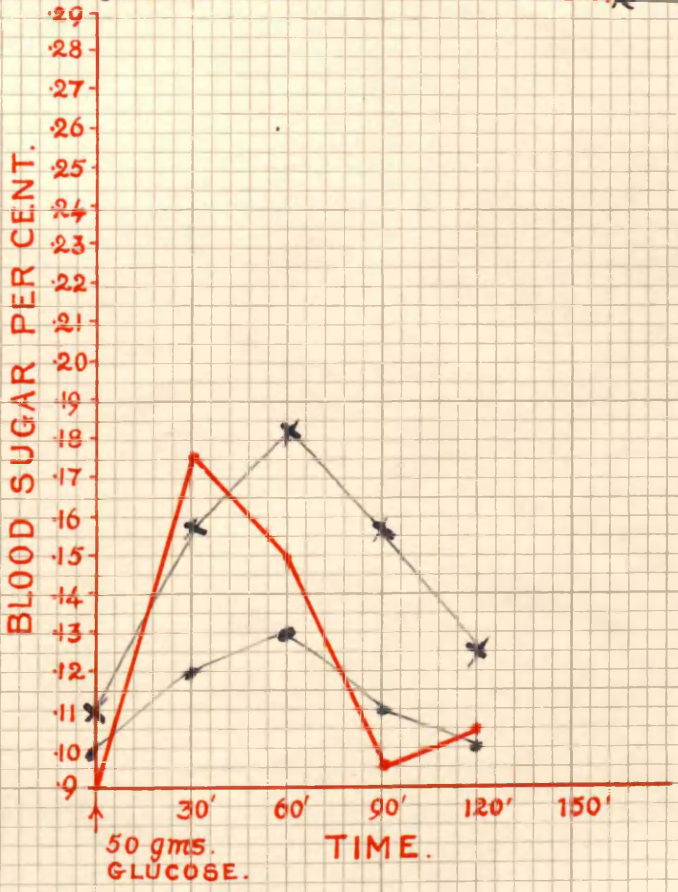
B.P. 190/112. Hyperpiesia

SUGAR TOLERANCE CURVE.

40 gms. Galactose } AVERAGE NORMAL PATIENTS

RED. ——●——●——●——

BLACK. ——×——×——×——



Urine: no albumen, no sugar, no urobilinogen, no urobilin

Blood: Van den Bergh, negative.

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

(24)

LEWIS'S BLOOD-SUGAR CHART

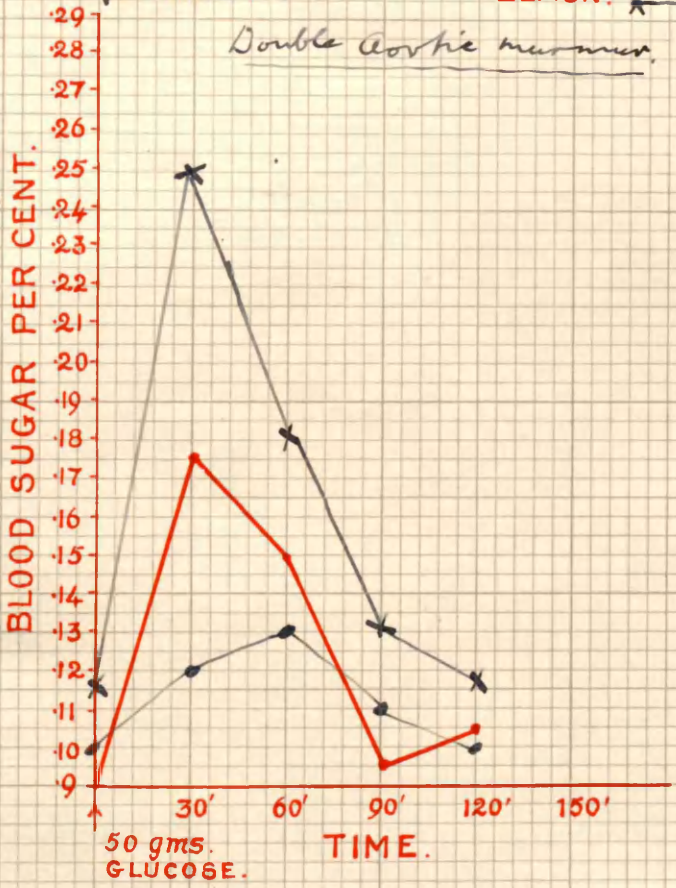
Name Mrs. B. Age 51 yrs.

B.P. 190/110. Hypertension + Angina Pectoris

SUGAR TOLERANCE CURVE.

40 gms. Galactose { AVERAGE NORMAL PATIENTS — RED. BLACK.

Double aortic murmur.



Urine: no albumen, no casts, no urobilinogen, no urobilin

Blood: Von den Bergh negative Wassermann + + (1929).

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

(25)

LEWIS'S BLOOD-SUGAR CHART

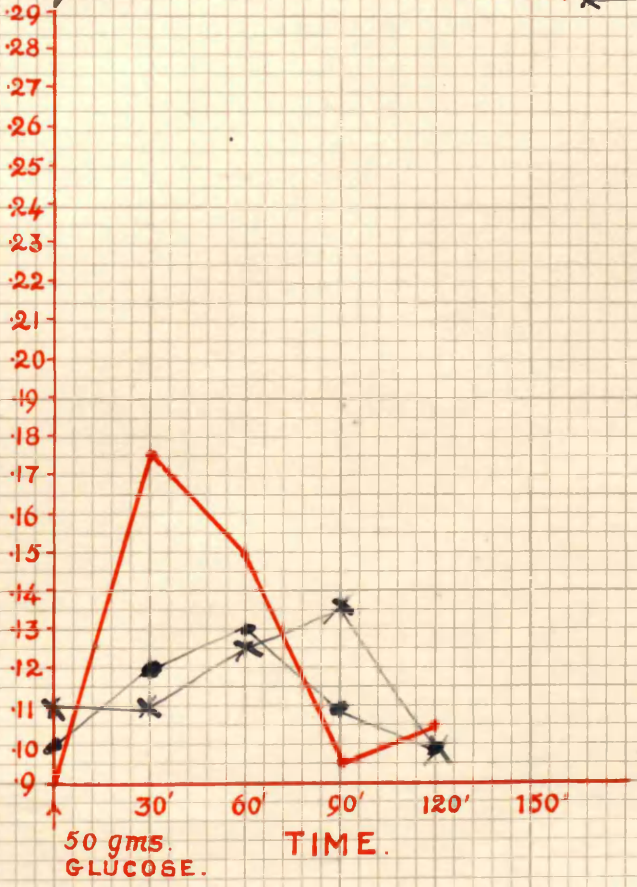
Name Miss Mc M. Age 70 yrs.

B.P. 210/130. Hypertension

SUGAR TOLERANCE CURVE.

40 gms. Galactose

AVERAGE NORMAL PATIENTS ~~RED.~~ BLACK.



Urine: No albumen no casts no urobilinogen, no urobilin

Blood: Van den Bergh negative

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

(26)

LEWIS'S BLOOD-SUGAR CHART

Name Mrs. Mc C. *Age* 56 yrs.

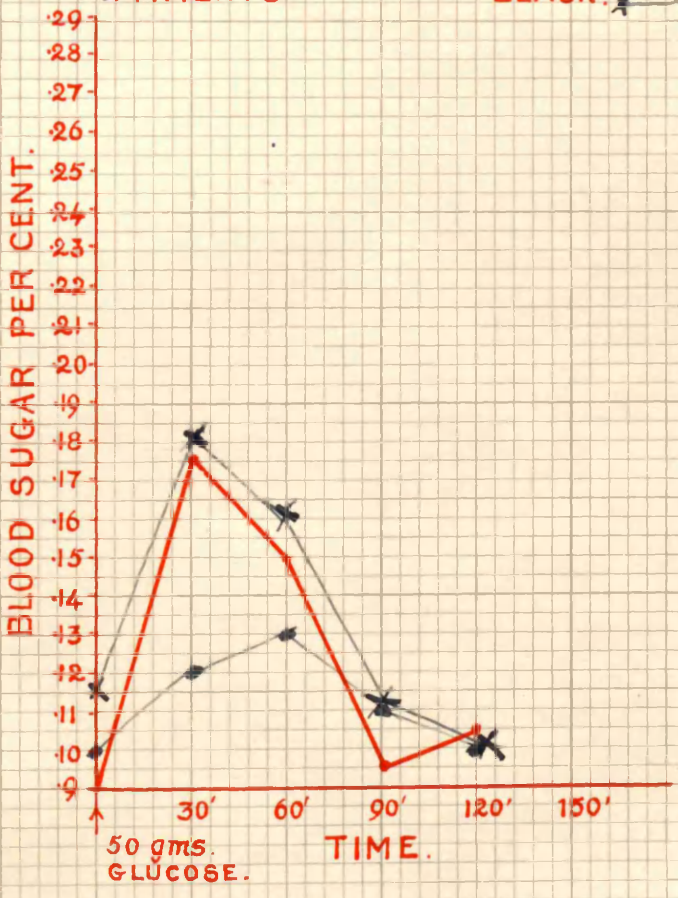
B.P. 186/112 Hyperpiesia

SUGAR TOLERANCE CURVE.

40 gms. Galactose {AVERAGE NORMAL ~~RED~~. / PATIENTS BLACK.

Urine no albumin, no casts, no urobilinogen, no urobilin

Blood Van den Bergh negative



London : H. K. Lewis & Co. Ltd., 136, Gower Street. W.C. 1

(27)

LEWIS'S BLOOD-SUGAR CHART

Name Mr. H. Age 54 yrs.

BP 185/112. Hyperpiesia.

SUGAR TOLERANCE CURVE.

40 gms Galactose } AVERAGE NORMAL PATIENTS ~~RED~~ BLACK.

Urine. albumen: trace, no casts
no urobilin or bile, no urobilin
Blood Van den Bergh ~~delayed direct~~ positive indirect

BLOOD SUGAR PER CENT.

·29 ·28 ·27 ·26 ·25 ·24 ·23 ·22 ·21 ·20 ·19 ·18 ·17 ·16 ·15 ·14 ·13 ·12 ·11 ·10 ·9

30′ 60′ 90′ 120′ 150′

50 gms. GLUCOSE. TIME.

London : H. K. Lewis & Co. Ltd., 136, Gower Street, W.C.1

(28)

LEWIS'S BLOOD-SUGAR CHART

Control

Name Mr. J. Age 66 yrs.

B.P 120/90 Arteriosclerosis

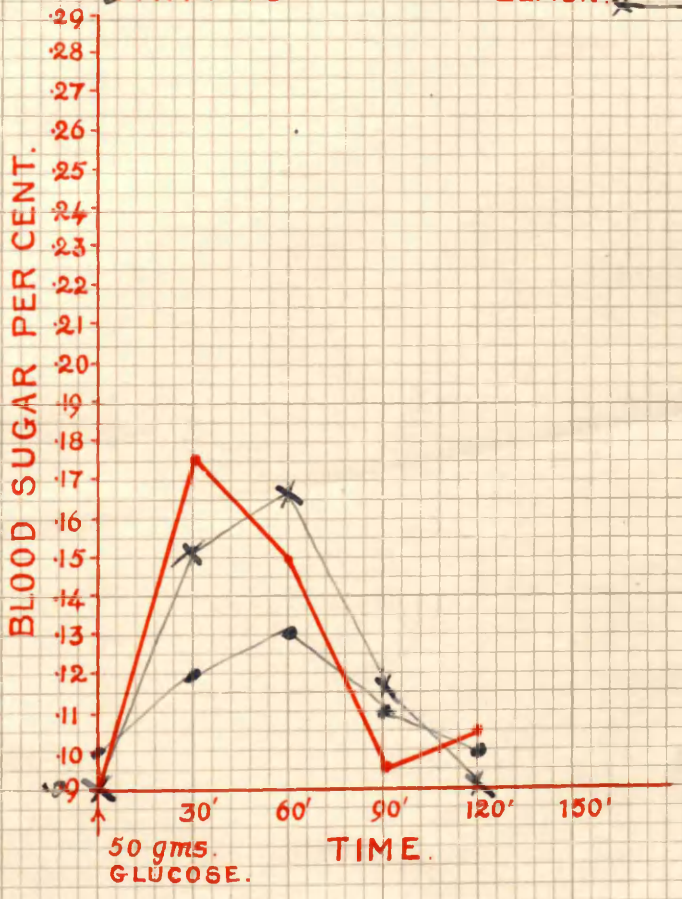
SUGAR TOLERANCE CURVE.

40 gms galactose

AVERAGE NORMAL PATIENTS

~~RED~~

BLACK



Urine No albumen, no casts
no urobilinogen, no urobilin

Blood Van den Bergh negative

London : H. K. Lewis & Co. Ltd., 136, Gower Street. W.C. 1