# THESIS

on

# ARTERIAL BLOOD PRESSURE

with special reference

to

...RENAL DISEASE...

# C O N T E N T S.



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## INTRODUCTION

The clinical aspects of arterial blood pressure must be studied from a practical standpoint and in a thorough and systematic way, if, in the treatment of diseases, we are to derive any real benefit from such study. We must study arterial pressure not merely by digital estimation, but also by such an apparatus as will indicate the smallest variations in blood pressure to a nicety and with certain reliance. Such an apparatus we now fortunately possess in the Sphygmometer, of which there are several varieties on the market. Of these undoubtedly the most reliable, as well as the most convenient, is Dr. C. J. Martin's[1] modification of the Riva-Rocci Sphygmometer. This instrument is quite simple and accurate.

### Description of Sphygmometer.[2]

The apparatus consists of a bag of thin rubber 1/30th of an inch in thickness, 13½ inches long and 4½ inches broad. Into the centre of one side of the bag a brass nozzle is secured by a screw and washer, as in the connection of a valve tube of a bicycle tyre. This bag is wrapped round the arm over the biceps, so that the ends overlap. Outside is applied a support of sheet lead of

somewhat/

somewhat larger dimensions, covered with cloth and secured by straps.

The interior of the bag communicates by means of a stiff walled flexible rubber tube with a four-way piece of brass. This junction is in communication on the one hand with a mercury manometer, and on the other with a rubber compressor. The tube connecting with the compressor contains a valve. The four-way of the junction is closed by a screw and leather washer, by loosening which the air in the system may be allowed to leak out.

The manometer is the ordinary U-tube variety, 11½ inches in length, provided with a scale in millimetres. The glass tube is fixed to an upright piece of wood, which latter fits into a wooden base, which, in turn, may be clamped on to a table for greater security, when in use. Each end of the glass tube is secured with rubber caps to prevent the mercury spilling.

Mode of recording Arterial Pressure.

To record arterial pressure by means of this apparatus, the limbs of the manometer should be filled with mercury, so that the surface of the mercury shall stand in each limb opposite the mark zero on the scale. The cuff or armlet should then be wrapped round the patient's upper-arm, the arm being/

being either bare or covered only by a thin layer of underclothing and thereafter it should be strapped so as to fit accurately. Now, using the left index finger for feeling the pulse, the compressor is seized with the right hand and air pumped into the apparatus until the pulse at the wrist can no longer be felt. At that point we look at the surface of the mercury and note down the number of millimetres pressure recorded. We then gradually turn the milled edge of the liberating valve and so allow the pressure in the armlet to decrease gradually until the pulse at the wrist is again perceptible to touch, and at this point we again note down the exact pressure now recorded in millimetres. This will be the maximal systolic pressure in the brachial artery at that particular time. If there is much difference between the two readings the observation should be repeated. The maximal systolic pressure for the average healthy adult ranges from 100 to 130 millimetres[3] of mercury approximately. After each observation the air is allowed out of the apparatus and the arm relieved of any venous congestion, either by elevating the limb or by friction. The pressure of the cuff should not be maintained for more than a minute or so, otherwise the venous congestion set up produces

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a little discomfort to the patient. It is important to note that in studying the effects of varying conditions, the readings must be taken uniformly with the arm on the same level as the heart and in an absolutely passive condition. To ascertain the arterial pressure in children, the cuff can be fitted on to the thigh and the pressure in the femoral artery then taken.

By means of this instrument, then, the arterial pressure in man can be recorded as easily and accurately and almost as quickly as the temperature can be taken with the clinical thermometer. It took some very considerable time however to put together such a reliable apparatus. During the last sixty years various instruments have been invented to indicate the resistance of the pulse. A dozen or so had but a short trial as they were not standardised on proper physiological lines and had many mechanical defects, and consequently could not be expected to reveal new facts in clinical work. Now, however, owing to several modifications on pre-existing apparatus, we are in possession of an instrument that we can thoroughly rely on.

Thus having briefly sketched out the nature of the apparatus I use for recording the systolic pressure/

pressure and its mode of application, I shall pass on to say something of the history of the work done on arterial pressure up to the present time.

The great pioneer in the history of the circulation of the blood was William Harvey. He was the first to discover that the blood circulated through the vessels; this was in the earlier part of the seventeenth century. But some considerable time elapsed, about a century in fact, before any further advance was made with regard to the circulation of the blood. About 1733 Stephen Hales[4] demonstrated that the blood in circulating through the vessels exerted pressure on their walls. That such a length of time should have elapsed, to us of the present day, seems rather curious, for the mere fact that a fluid circulates within certain and fixed limits presupposes the exertion of a certain amount of pressure however small. But the laws of physics as they relate to fluids were in those days in rather a primitive form, and hence it is that not until the advent of the nineteenth century was there any appreciable or real progress made in the study of blood pressure. Poiseuille[5] about the year 1828 was about the first to make use of physiological experiments affecting the force of the blood stream. To him we are really indebted

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for the introduction of the mercurial manometer of U-shape, and for devising means for preventing the coagulation of the blood in its flow through a tube connected with an open artery. His experiments on animals gained us much in his attempts to solve the problems touching the circulation from a physical standpoint. And closely following upon his heels came Ludwig,[6] who in 1847 made a most important contribution to the literature of the circulation. He, by his graphic method of recording blood pressure on a revolving cylinder or Kymographion, supplied us with most important and accurate knowledge of the mechanics of the circulation, and from his time onwards the study of the blood flow has advanced by leaps and bounds. And just about the same time the introduction of the Sphygmograph by Marey also gave an immense impetus to the study of the circulation; and although a sphygmographic tracing aids but little towards arriving at an accurate record of the arterial blood pressure, still it shows in a general way the trend of the arterial tension, and it has certainly added much to our prior knowledge of cardio-vascular conditions. In short, we can say that with the introduction of the Sphygmograph came the first endeavour to measure the arterial blood pressure/

pressure in man. For in 1855 Vierordt[7] tried to prove that the degree of pressure required to obliterate the arterial pulse at the wrist was an index of the blood pressure exerted within the walls of the artery. Ever since that time the hard and the soft pulse began to have a quantitative significance in the mind of the intelligent physician. It is true that the methods used by Vierordt and others of his time had many mechanical defects, still they have been of incalculable service in stimulating the clinical experimentalist in his endeavour to invent a more perfect apparatus for estimating the arterial tension. Up to this time the estimation of the arterial pressure was arrived at by means of a solid pad or block placed upon the artery, and in so far resembled the digital method in measuring, so to speak, the total end pressure and not the pressure per unit of surface; so that in obliterating the pulse wave we arrived at an erroneous total pressure record. In 1877 Waldenburg[8], and not long after him Verdin[8], Hoorweg[9], Cheron[8], Bloch[8], and quite recently Frey[10], brought forward different forms of apparatus for measuring the blood pressure, but they all had the same fault in that they applied the pressure to the artery through a solid medium. The next advance

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was made by Prof.von Basch[11] of Vienna. He substituted for the solid medium a fluid one about the year 1876, and so was the first to obtain a record of the pressure per unit of surface. Another respect in which he improved on the prior solid medium apparatus, was his use of a mercurial manometer for recording through a fluid medium the amount of pressure necessary to utterly compress the artery. Many and various were the modifications and small improvements which were made on von Basch's original instrument. He himself substituted for the mercurial manometer a circular spring recorder or metal manometer. Each of these two types of manometer has its champions at the present day; but there can be no doubt as to the superiority in accuracy of the mercurial one for various reasons and especially so because the spring manometer, being necessarily a delicately constructed instrument, has to be standardised frequently - at least once a year, whereas the mercurial manometer, if it be a trifle more cumbersome, still is always perfectly reliable in point of accuracy, and needs no such standardisation. Following von Basch came Potain,[12] who in 1889 made use of air instead of fluid in the compressing pad, the pressure in the pad being raised by means of a rubber compressor: in other respects their instruments were practically the same./

same. Potain may therefore be said to have been the pioneer in the air compressing method which has been gradually elaborated into the most reliable instruments we have now in our possession. The errors of von Basch and Potain's apparatus chiefly were that the pressure on the radial artery, being applied by a mere pad placed over the vessel, did not give a correct estimate of the true arterial tension, since the artery is liable to variation in position with reference to the bone, and therefore the pressure is not always applied directly over the artery and against the bone. In sketching the history of blood pressure apparatus, however briefly, it is right that the names of Dr. George Oliver[13] and Messrs. Hill & Barnard[14] should be mentioned. Great credit is due to them for their work on blood pressure and for the instruments they constructed somewhere about the year 1896, and although theirs had the same faults as the others I have already mentioned, since they both then used the fluid compressing medium, still they scored in point of compactness and convenience of application. Tigerstedt[15] enumerates the sources of error in such instruments and concludes that the absolute estimate found can have no value except as regards pressure changes in the same individual at different periods of time: they give a total over-estimation of

between 32 and 78 mm.Hg. Up to the year 1896 then, not one of the Sphygmometers which had been devised can be said to have reached any satisfactory degree of accuracy. About this year Riva-Rocci[16] and Leonard Hill[17] (it is hard to say to whom the precedence should be given) each constructed a new instrument for measuring the blood pressure, the leading feature in each being a rubber bag for encircling the arm, the bag being inflated by a rubber compressor - the old principle of the apparatus being just the same as the instrument which I now use and which I have described above. The armlet in both of these instruments, however, has been proved to be far too narrow (5 cm.). Stanton[18] and Recklinghausen[19] have shown that the broader the cuff the lower the reading on the manometer, and that the greater the circumference of the part compressed, the greater the difference in the reading. So that if we use equally narrow cuffs on the thigh and arm simultaneously, we get a much higher reading in the former than in the latter. On the other hand if we use a sufficiently wide cuff, say one of 12 cm., the readings are practically identical. And so it is now that in all trustworthy instruments, observers make use of a cuff of about 12 cm. width. I may further note that it is highly essential that the outer covering of/

of the cuff be indistensible (hence the lead-sheeting) as well as the rubber tubing stiff walled. This prevents any outward give, or loss of the actual arterial pressure (so to speak) on the part of either, and so enables the pulse wave to be transmitted in its entirety to the mercurial column. With such an instrument as this, then, the maximal cardiac systolic pressure (which for brevity I may often speak of as the arterial pressure), with which alone I am concerned in this thesis, can to all purposes be accurately estimated, the only error being a fairly constant one of about 10 mm.Hg. due to loss in transmission through the tissues overlying the brachial artery.

## Definition of Terms.

Arterial Pressure is of three varieties :-

1. Systolic Pressure, or Maximum (cardiac) Systolic Pressure.
2. Diastolic Pressure, or Minimum (cardiac) Systolic Pressure.
3. Mean Pressure.

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1. Systolic Pressure I have already defined as corresponding to the point on the manometer at which the pulse disappears, or (better still) reappears at the wrist, when the pressure in the cuff is sufficiently raised or lowered respectively. 20

2. Diastolic Pressure. If the pressure in the cuff be lowered slowly from the systolic level by 5 mm.Hg. at a time, if possible, and the size of the oscillations on the manometer be watched, then the lowest reading at the point of maximum oscillation represents the diastolic pressure. 21 & 22

3. Mean Pressure is determined by taking the Arithmetical mean of the Systolic and Diastolic pressures. 23

## On the relative clinical significance of each variety of Arterial Pressure.

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Systolic v. Diastolic Pressure. Clifford Allbutt[24] argues strongly in favour of the Systolic (maximum) pressure being the only feasible criterion in practice. He says that "the relation of the pause to systole and diastole, the sustenance of the pulse wave, and the diastolic pressure are not well indicated by the Sphygmometer." This is absolutely true, for it is quite impossible clinically to judge with accuracy as to whether the pulsation is maximal by merely watching the fluctuations of the mercurial manometer, and in the case of rapid or irregular pulses neither the eye nor the mercury can follow the movements quickly enough; and then again, when owing to forced respiration or struggling, rapid fluctuations are present the diastolic criterion fails for a similar reason.

The systolic pressure means of judging the actual variations of systemic pressure was the original criterion of Vierordt and von Basch, and with Clifford Allbutt, I hold that it is still the best criterion that we possess. For the return of the pulse wave under compression is sharply defined, is easily detected with the finger/

finger, and the actual systolic pressure can thus be determined within half a minute, and much more expeditiously than either the mean or the diastolic. It measures the systolic end pressure in the brachial artery pretty accurately, and seeing that the brachial and the subclavian artery are in direct continuity, it gives us also the systolic lateral pressure within the subclavian and consequently a very close approximate to the systolic lateral pressure in the Aorta. Tigerstedt[25] has shown that only about $^{1}/_{160}^{th}$ of the heart's energy is expended in imparting its velocity to the blood stream, the reminder being required to overcome Aortic pressure; so that at a pressure of 160 mm.Hg. the difference between the lateral and the end pressure would amount to only 1 mm. - quite a negligible quantity in short.

Mean Arterial Pressure. Dr. John Cowan[26] in an admirable paper on blood pressure argues, on the other hand, in favour of the mean pressure being the most important guide as to the actual quality of the blood pressure. His arguments seem inconclusive, however, as he does not bring forward sufficient data to substantiate his views. Howell and Brush[27] have conclusively proved by their experiments that the mean pressure is less instructive to the clinician than either the systolic or/

or the diastolic, and that the general trend of the arterial pressure is shown either in systolic or in diastolic pressure even in extreme conditions of heart and vessel changes. Their observations and conclusions are of the utmost importance in clinical sphygmometry. Janeway[28] of New York considers that the mean pressure is not a reliable criterion to go by, and is only an approximate to the absolute mean pressure. The pulse wave he holds is always in the form of a triangle with the apex upwards, and the absolute mean pressure he maintains could be fixed only with difficulty, and even then would be found to be nearer the diastolic than the systolic pressure.

With the foregoing data, as well as from personal observations with the Sphygmometer, I think I am fully justified in adhering to the systolic criterion in my records and estimation of blood pressure, and, as I have already stated, whenever I make use of the term blood pressure in my description of clinical cases, it shall be with reference to the systolic variety, unless I expressly state to the contrary.

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## Physiological variations in Arterial Pressure.

In order to be able to appreciate the significance of alterations in arterial blood pressure in disease, the observer must, of course, be thoroughly familiar with the causes of variations in health. He will then be in a position to exclude or take account of these health variations and so be able to recognise and value the pathological indications. It would therefore be well, before going into the subject of arterial pressure from its clinical aspects, to give a brief resume of these health variations as met with, first, in the same individual, and, secondly, in different individuals.

### Variations in the same individual.

In the same individual, then, the causes which we find operating in the variations of arterial pressure are roughly speaking no fewer than fourteen. How these causes affect blood pressure may, in a general way, be seen from the following table -

1. Posture. The arterial pressure is highest in the standing, and lowest in the horizontal position.[29]

2. Gravity. The pressure is raised or lowered about 2 mm.Hg. for every inch the arm is above or below the heart level respectively.[30]

3. Sleep. During the first hours of sleep the pressure falls, but rises gradually during the morning hours.[31]

4. Rest. In the resting position there is a fall of arterial pressure.[32]

5. Exercise. With muscular exercise the pressure rises up to a certain limit and then falls even below the original as fatigue sets in.[33] Psychical conditions act in much the same way.[34]

6. Alcohol. Arterial pressure does not rise; on the contrary it falls if a considerable dose be given.[35]

7. Tobacco. Pressure rises and markedly so if strong tobacco be used - the effect lasting for about one hour after stopping smoking.[36]

8. Digestion. After meals the pressure is slightly raised,(37) and quite perceptibly so after such beverages as tea and coffee.[38]

9. Respiration. When respiration is stimulated to increased activity the pressure markedly rises.[39]

10. Temperature. Cold raises, but if prolonged sufficiently, lowers the arterial pressure. Baths of body temperature do not practically affect the pressure; both cold and hot baths raise it.[40]

11. Pulse Rate. Increase of Pulse Rate of itself probably does not alter the blood pressure, but when peripheral resistance is increased then increase in pulse rate gives rise in pressure.[41]

12. Diurnal Variation. Pressure gradually rises and attains its highest in the evening, then falls and is lowest in the early morning hours; it rises again in the forenoon and falls in the early afternoon.[42]

13. Menstruation. There is a regular fall in pressure during the menses with a return to normal about two days after cessation of the flow.[43]

14. Altitude. The pressure at first rises in going from a low to a high altit-

## Variations in different individuals.

The causes of variations of arterial pressure in different individuals on the other hand, though more limited in number, are even more important than the foregoing, since in measuring the pressure in clinical work these causes must always be kept foremost in the observer's mind and a due allowance made for each. Without a thorough knowledge of them the records of variations in diseased conditions would be absolutely valueless.

1. Sex. Arterial pressure is lower in females than in males by about 10 mm.Hg. [45]

2. Age. In childhood, under two years the average pressure is 75-90 mm.Hg.; above that age it is 90-110 mm. In the great majority of adults we have 100-130 mm. the normal; whereas over 50 years of age the mercury may run up to 145 mm. in health conditions. [46]

3. Occupation. Hard manual work causes rise in blood pressure. [47]

4. Size. Increase in weight or height is accompanied by increase in blood pressure generally. [48]

5. Temperament. Neurotic and excitable persons show higher pressures than the phlegmatic. [49]

## Arterial Pressure in Disease

It frequently occurred to me during my clinical examination of patients in my daily rounds of hospital work that, if we could follow up with an approximate degree of accuracy the trend of the arterial pressure in the course of certain forms of disease, or for that matter if we could say with fair certainty what degree of pressure the arterial tension was represented by at any particular time, it would be of great advantage to us not only in the treatment of our cases but also from a diagnostic and prognostic standpoint. Prompted by this, I began to make observations with the Sphygmometer first on healthy individuals, verifying the results handed down by various workers in the field of the physiological aspect of arterial pressure. Having thus gained a fair amount of proficiency in the use of the apparatus on normal individuals, and having satisfied myself that the health variations could be well fixed within certain limits under conditions of age, sex, occupation, posture, etc. as I briefly tabulated in the former part of this paper, I was encouraged to make further experiments in the field of disease. This for the first few months I did more or less at random in the case of any and every disease, and especially so where on fingering the pulse/

pulse, it occurred to me that the arterial tension was notably either above or below the normal. Nothing could be more convincing in proving how apt one is from digital examination to form erroneous conclusions regarding the actual state of the tension is such cases as, for example, the bounding pulse of feverish states, or again in a tortuous atheromatous or in an arterio-sclerotic radial. Now, however, with the Syhygmometer as his companion the clinician has the great satisfaction of being able to clear up any doubts he may have had concerning high or low blood pressure, as the case may be; and further still, he is taught to be more discreet and reserved in expressing off-hand an opinion as to the mm.Hg. pressure he considers the arterial wall is being subjected to. For I have on not a few occasions seen men, upon fingering the radial, make statements widely off the mark regarding the tension - men of reputed fame as clinicians, and men whomfrom years of experience and good work we have been taught to look up to as authorities on the pulse. This I say after experimental verification of the cases with my Sphygmometer. Last July I read a paper by Clifford-Allbutt in "The Hospital" in which he expresses well my views with regard to the use of the Sphygmometer as an indispensable accessory in clinical work. The gist of his statements was to the/

the effect that the day is not far gone when the physician found it just as hard to tell approximately what his patient's temperature was as he does to-day the degree of rise and fall of blood pressure. In those days no thermometer was necessary. Every man could surely tell whether or not, and to what extent, fever was present, and he who could not must indeed be behind the times and quite devoid of clinical perception. At the present time there are those who hold a parallel opinion with regard to arterial pressure. They say that he who cannot with his finger tell the degree of arterial pressure must be, to say the least, unworthy of the age in which he practises medicine. We now know that those who expressed such an opinion concerning temperature must have been a trifle presumptuous- capable men as many of them without a doubt have been. And before many years have passed I venture to prophesy that the same can be said of the present physician who is so confident in his powers of determining the arterial pressure with the aid of the finger alone. Oliver and Janeway, whose works on blood pressure are well known, endorse these sentiments. To use the words of the latter: "Five minutes trial of the Sphygmometer will convince the most sceptical that his previous judgments, based on his supposedly trained sense of touch, were often fallacious. High tension/

tension was certainly recognised before the introduction of the Sphygmometer, but so was fever before the days of clinical thermometers. In Medicine accuracy of observation is the first step towards a correct diagnosis, without which nothing but bald empiricism is possible." It is only, however, after considerable experience with this instrument that one begins to realise, and is impressed by, its sphere of usefulness. He has to bear in mind, as he records the blood pressure, the various physiological factors at work, and these he must eliminate to make his observations of clinical significance in the diagnosis or treatment of his cases. This, at first, he will find tedious and difficult work, but with patience, time, and scientific zeal and accuracy, his efforts will be certainly reassuring, as well as highly instructive. He begins to rely on the Sphygmometer as a new and ready means of clearing up circulatory problems, as well as a valuable assistance in differential diagnosis and in the treatment of diseases.

In this Infirmary, which is a general hospital and which contains about 900 beds, consisting mostly of acute diseases in medicine and surgery, I have had during the last three years ample opportunity and plenty of good material for studying blood pressure in its relation to disease from an experimental/

experimental standpoint. After considerable discrimination of cases, I was led to limit my observation records to cases of Nephritis. This selection I made after due consideration, because in this disease most of all was I struck by the inestimable value of arterial pressure records in diagnosis, prognosis, and treatment.

## Renal Disease.

In Kidney Disease I have found the Sphygmometer most useful on many occasions, and I shall now give a tabulated list of results of blood pressure observations in fifty cases of acute and chronic nephritis and cirrhotic kidneys. In all these forms of renal trouble, there is, generally speaking, an appreciable and variable rise of blood pressure, the rise being the more marked the more chronic the disease. In the acute form of nephritis, as in all other acute diseases, sphygmometric observations are more satisfactory than in the subacute and chronic forms, since in the latter conditions the circulatory system becomes more or less involved/

involved and throws difficulties in our path which have to be carefully reckoned with and made allowance for. I have, therefore, included in my table of analysis the cardio-vascular changes present in each case, as well as other remarks bearing upon or influencing the pulse tension.

Table I. Renal Cases.

| Case. | Age. | Occupation. | Disease. | Mm.Hg.* | Remarks. | Result. |
|---|---|---|---|---|---|---|
| 1 | 30 | House Wife | Acute Nephritis | 140-120 | Blood and epitelial casts and much albumen in urine. Heart slightly enlarged. 2nd Aortic sound much accentuated. | Cured. |
| 2 | 35 | " | " | 170-110 | Uraemic coma and fits. Suppression of urine, which contained much blood. Cardiac enlargement to nipple line. | " |
| 3 | 8 | School Girl | " | 130-90 | Scarlatinal nephritis. Marked cardiac hypertrophy with mitral systolic bruit - Chronic endocarditis. | " |
| 4 | 37 | House Wife | " | 140-110 | Suppression of urine. Moderate oedema. Blood casts in urine. Heart and vessels normal. | " |
| 5 | 21 | Dom.Servant | " | 150-110 | Uraemic fits.Pericarditis. Vide infra,Case 5, p.33. | " |
| 6 | 29 | Dom.Servant | " | 145-120 | Blood,epithelial and granular casts.Cardiac enlargement to nipple line. Mitral stenosis, (presystolic thrill). | Greatly improved. |
| 7 | 36 | Labourer | " | 140-120 | Uraemic stupor - no fits. Early arteriosclerosis. Chronic bronchitis and emphysema | Cured. |

* The figures in this column represent respectively the highest and the lowest systolic (arterial) pressure observed in taking routine blood pressure measurements of each Case.

| Case. | Age. | Occupation. | Disease. | Mm.Hg. | Remarks. | Result. |
|---|---|---|---|---|---|---|
| 8 | 11 | Schoolboy | Acute Nephritis | 120-95 | Much albumen with blood and epithelial casts in the urine. Mild case. | Cured. |
| 9 | 28 | Sail-maker | " | 140-115 | Alcoholic. Oedema of lower extremities. Heart enlarged to ½ inch outside nipple line. | " |
| 10 | 34 | Carter | " | 150-125 | Uraemic fits with coma. Arterio-sclerosis. Heart slightly enlarged. Oedema of face, scrotum, legs. | " |
| 11 | 31 | Labourer | " | 130-120 | Haematuria slight. Heart and vessels normal. | " |
| 12 | 40 | Porter | " | 155-125 | Haematuria. Marked cardiac hypertrophy to ½ inch outside & 1½ inch below the nipple. Aortic regurgitation. Radial artery tortuous and atheromatous. | " |
| 13 | 20 | Sailor | " | 130-100 | Heart & vessels normal. Haematuria. Mild case. | " |
| 14 | 38 | Labourer | " | 160-120 | Uraemic fits, coma, oedema. Post Mortem showed large congested kidneys. Heart slightly enlarged with no organic disease. | Died |
| 15 | 22 | Labourer | " | 120-100 | Haematuria. Mild case. Cardio-vascular system normal. | Cured. |
| 16 | 32 | Porter | " | 140-125 | Heart enlarged to nipple line. Early arterio-sclerosis. Mitral systolic bruit. Alcoholic in habits. | " |
| 17 | 34 | Plumber | " | 145-110 | Mild case. Blood & epithelial casts in urine. Heart & vessels normal. | " |
| 18 | 29 | Labourer | " | 145-125 | Heart enlarged slightly. Chronic bronchitis. | " |
| 19 | 16 | Errandboy | " | 140-110 | Scarlatinal nephritis. Marked oedema of face & ankles. Haematuria. | " |
| 20 | 25 | Labourer | " | 135-110 | Alcoholic. Heart & vessels normal. Uraemic stupor - no fits. Haematuria. | " |

| Case. | Age. | Occupation. | Disease. | Mm.Hg. | Remarks. | Result. |
|---|---|---|---|---|---|---|
| 21 | 40 | Plasterer | Subacute or Chronic Nephritis | 165-130 | Had two previous attacks of Nephritis. Beer drinker. Cardiac enlargement. Albumen and granular casts in urine. | Discharged much relieved. |
| 22 | 52 | Labourer | " | 175-140 | Pale, pasty complexion. Albuminuria & polyuria. Large heart. Been treated in this hospital before for Nephritis. | " |
| 23 | 35 | Sailor | " | 190-135 | Urine high coloured - S.G.1020. Albumen, trace of blood. Cardiac enlargement ½" outside nipple. 2nd attack. | " |
| 24 | 46 | Labourer | " | 200-120 | Vide infra - full notes of this case & P.M. p.41. | Died. |
| 25 | 44 | Labourer | " | 170-135 | Albumen plentiful. Tube casts. No blood in urine. 40-70 oz. passed daily. Early cardio-vascular changes. | Discharged improved. |
| 26 | 25 | Butcher | " | 160-120 | Aortic regurgitation. Had rheumatism 10 Yrs. ago. Apex beat on nipple line. Much albumen with tube casts. | " |
| 27 | 57 | Labourer | " | 165-130 | Face puffy and pasty. Scanty urine, 30-40 oz.per diem. Hyaline & granular casts. No blood in urine. | " |
| 28 | 53 | Labourer | " | 180-125 | Subject to slight gouty attacks. Arterio-sclerosis and cardiac hypertrophy. Urine S.G.1022. 30-50 oz.daily. | " |
| 29 | 61 | Painter | " | 140-90 | In extremis on admission. Vide infra P.M. notes, etc. p.45. | Died. |
| 30 | 47 | Labourer | " | 180-140 | Slight arterio-sclerosis and cardiac enlargement. Oedema of face and legs. Uraemic asthma. Slowly improved. | Discharged improved. |
| 31 | 55 | Butcher | " | 190-130 | Much albumen with trace of blood. Passed moderate quantity of urine S.G.1014. Had 2 attacks of gout. | " |

| Case. | Age. | Occupation. | Disease. | Mm.Hg. | Remarks. | Result. |
|---|---|---|---|---|---|---|
| 32 | 56 | Bricklayer | Subacute or Chr.Nephrit-is. | 150-140 | Puffy, pasty face. Urine scant, albumin-ous with trace of blood. Apex beat on nipple line. Attacks of asthma. | Discharged improved. |
| 33 | 54 | Carter | " | 170-120 | Large heart with apical systolic bruit. Heavy beer drinker. Cirrhotic liver. Urine S.G. 1010. about 40 oz. daily. Albumen, blood. | " |
| 34 | 22 | Dom. Servant | " | 150-130 | Uraemic dyspnoea. Urine scant first, then free. S.G. 1014. Albumen. Trace of blood. 2nd attack. | " |
| 35 | 29 | House Wife | " | 190-120 | In Hospital for 5 months. Became finally water-logged & died in uraemic coma. P.M. showed large kidneys, 11 oz. each. | Died. |
| 36 | 30 | " | " | 180-140 | Slight arterio-sclerosis & cardiac hypertrophy. Uraemic stupor with asthma-tic attacks. Gradually improved & took her discharge. | Improved. |
| 37 | 31 | " | " | 160-140 | 2nd attack. Oedema of face and lower extremities. Chronic bronchitis. Heart and vessels normal. Albuminuric retin-itis. | " |
| 38 | 66 | " | " | 165-110 | Died 5 days after admission. Had uraemic stupor & marked dyspnoea at intervals. Much albumen. P.M. show-ed large flabby pale kidneys ($10\frac{1}{2}$-11 ozs). Capsule strips in most parts. Heart 16 ozs. | Died. |
| 39 | 45 | Plumber | Granular Contracted Kidneys. | 190-170 | Heart much enlarged. Tortuous, atheromat-ous arteries. Urine S.G. 1004. about 80 ozs. daily. Hyaline casts. | In statu quo |

| Case. | Age. | Occupation. | Disease. | Mm.Hg. | Remarks. | Result. |
|---|---|---|---|---|---|---|
| 40 | 64 | Labourer | Granular Contracted Kidneys. | 265-230 | Bovine heart -apex 1½ in.below and outside nipple. Mitral incompetence. Polyuria 70-80 ozs. daily.Died of cardiac syncope. No P.M. | Died. |
| 41 | 71 | Cooper | " | 205-140 | Large heart.Advanced arterio-sclerosis. Vessels hard and tortuous. Urine S.G.1008. about 70 ozs. daily. Trace of albumen. | Relieved. |
| 42 | 61 | Labourer | " | 220-180 | Oedema of feet.Mitral incompetence. Marked dyspnoea. Polyuria. P.M.- kidneys small granular,3½-3 ozs.each. | Died. |
| 43 | 55 | Labourer | " | 210-160 | Was in this Hospital 3 mths. with recurrent dyspnoea,polyuria, headaches,angina pectoris.Autopsy showed very small contracted kidneys 2 - 3 ozs. each. Capsule does not strip. Heart large - weighed 19 ozs. | Died. |
| 44 | 42 | Labourer | " | 240-200 | Polyuria.Albuminuria with hyaline casts. P.M.showed heart = 16 ozs. Small interstitially contracted kidneys, 3 ozs.each. | Died. |
| 45 | 56 | Clerk | " | 190-175 | Has been in hospital for last two years for kidney trouble.Nocturnal asthma. Passes much urine of low S.G. with albumen. | In statu quo. |
| 46 | 64 | Labourer | " | 210-110 | Died one week after admission. Had frequent attacks of dyspnoea & angina, and latterly had apoplectic stroke two days before death. See Notes p..49. | Died. |

| Case. | Age. | Occupation. | Disease. | Mm.Hg. | Remarks. | Result. |
|---|---|---|---|---|---|---|
| 47 | 38 | Housewife | Granular Contracted Kidneys. | 210-150 | Has chronic bronchitis; continual headaches. Marked attacks of asthma. Polyuria (70 ozs. daily). Albumen & blood occasionally. | In statu quo. |
| 48 | 48 | " | " | 200-180 | Heart dilated. Mitral incompetence. Chronic emphysema. Urine - pale, S.G.1004, about 60-70 ozs. daily. | Relieved. |
| 49 | 45 | " | " | 220-205 | Anemic and emaciated. Puffy face and ankles. Cardiac hypertrophy with mitral insufficiency. P.M. heart weighed - 21 ozs. Kidneys contracted R - 3 ozs. L - 4 ozs. | Died. |
| 50 | 47 | " | " | 260-110 | Nocturnal asthma. Occasional vomiting. Headaches. In Hospital for 3 weeks. Died of cardiac insufficiency. See Notes, p.52. | " |

## Acute Nephritis.

In differentiating between a simple case of primary acute nephritis and one of a subacute or an exacerbation of a chronic nephritis, the Sphygmometer comes prominently forward as an aid in diagnosis. As the above table shows, I have failed to find any very appreciable rise in the arterial pressure in uncomplicated cases of acute Bright's disease. In the works of recent observers we find difference of opinion on this point, Shaw[50] and Carter,[51] for example, saying that they could find no increase in arterial tension, while Bary[52] and Buttermann[53] hold that there is usually a moderate degree of hypertension, the latter recording cases that have reached up to even 170 mm.Hg.(12 cm.). The term Acute Nephritis is, I am inclined to think, very frequently applied to cases that are in reality an acute exacerbation of a chronic nephritis, or at least to cases that have had one or two former attacks of the disease in a more or less mild form but where no history of a previous attack could be got from the patient, and I am led to come to the conclusion that it is in such cases as these that the pressure reaches such high figures as Buttermann reports. On the other hand, with suppression of urine and acute uraemic manifestations we do get a temporary sudden rise of pressure, which gradually disappears as the renal functions/

functions are re-established. This was very well shown in Case 5 (Table I), which is so typical of what I have generally met with in Acute Nephritis cases that I think I cannot do better than give a detailed account of the case. I select this case in particular also, because though the clinical symptoms were so severe, yet the degree of oedema was comparatively slight - so slight as not to interefere materially with Sphygmometric observations. For it is a fact well known amongst observers that where the oedema is at all appreciable, blood pressure measurements ought not to be attempted, a slight oedema of the tissues of the arm increasing the reading by 10-20 Mm.Hg.

With special reference to the application of the Sphygmometer in the treatment of Acute Nephritis I shall have a word to say after I have sketched out the clinical history of the following case.

Case 5 - Table I.

The patient, M.F., a woman of 21, was admitted to Hospital on Nov.12,1905, complaining of severe headache, vomiting, and pain in the back. Her previous health had always been good, had no illness of any form except measles, from which she made perfect recovery. Present illness commenced 10 days before admission with chilliness, vomiting, and pain in the head. The cause of her illness she attributed to going/

going about in wet clothes.

Nov.12. On admission the symptoms were as above stated - headache, backache, and vomiting. The eyelids were puffy and she had slight oedema of the ankles. The tongue was coated, breath offensive, bowels constipated, with great thirst. The pulse was frequent and small with moderately high tension; no cardiac hypertrophy; slight accentuation of aortic 2nd sound; no murmurs. Oedema of lungs was supervening, with occasional moist cough.

Urine - Sp.Gr.1025, was scanty, acid, high-coloured and smoky, with copious urates and loaded with albumen. Dysuria present. Microscopic examination showed granular, epithelial,and blood casts.

She was put on the following treatment, four hourly -

R. Potass. Acet. gr.X.
Spt. Ether. nit.
Tr. Hyoscyam. āā M.XXX.
Aq. Chloroform. ad ½ oz.

The loins were dry-cupped and Linseed Poultices applied 4-hourly.

Nov.13. Patient is rather collapsed this morning; face pale and puffy; breathing laboured and hurried. Bowels were well moved. Severe occipital headache with frequent vomiting. (Bism.Carb. gr. X given, with hot fomentations to epigastrium). Is very restless in the evening, tossing about the bed and moaning. Skin not acting. Pupils dilated,with dimness/

dimness of vision. Rapid irregular pulse..

R. Digitalin gr. $\frac{1}{100}$, hypoderm.
R. Vapour Bath - good result.

Nov.14. Vomiting still persists, though not so urgent. Ophthalmoscopic examination shows intense albuminuric retinitis. Vision has gradullay failed since yesterday and patient is now quite blind.. Has had little or no sleep since admission. Urine very scanty; skin dry. Muscular twitchings present. Chloral Hydrate gr. XXX administered per rectum at noon. Dosed on and off for about three hours. In evening is again restless; twitchings of right arm and face. Vapour bath given - fair result.

Nov.15. Suppression of urine. Marked oedema of lungs and face. Severe dyspnoea at intervals. Great thirst - tongue brown and dry - sordes about the teeth. Stomatitis starting. Has vomited several times - treatment as before. Bowels acted well after enema. Loins again dry-cupped and Vapour Bath given - good result.

Nov.16. Condition in statu quo this morning, but later the pulse becomes rapid and strained, and a soft pericarditic rub can be made out on auscultation - gallop rhythm present. Has not passed urine for 12 hours. Complains of headache. Had severe epileptiform convulsion lasting about 3 minutes. Vapour Bath given and chlor.Hydr. gr.XXV per rectum.

Nov.17. Condition in extremis. Suppression of urine/

urine, Frequent uraemic twitchings. No Vapour Bath given owing to collapsed condition. No vomiting. Pericarditic rub hardly audible.

R. Strychnin gr. $\frac{1}{30}$ )
Digitalin gr. $\frac{1}{100}$ ) hypoderm.

Nov.18. Dyspnoea urgent. Had another fit lasting about 4 mins. Vapour Bath given - patient suddenly collapsed. Cheyne-Stokes' respirations: Comatose. Rallied after mustard poultice to praecordia, and strychnin and Digitalin hypoderm. Profuse sweating after the bath.

Nov.19. Slight improvement this morning. Pupils not so dilated and can see a little. Face flushed and anxious, at times. Rather drowsy all day; has had some sleep. Skin and bowels acting better. Passed 30 ozs. of urine last 24 hours, containing much blood and almost solid with albumen on boiling. No twitchings - no pericarditic rub.

Nov.20. Condition of patient shows slight improvement as regards sight, pulse, and diuresis.

Nov.21. Pulmonary oedema subsiding. Tongue not so dry. Still very drowsy. Pupils medium, sight improving - Pulse tension lower - diuresis freer . Condition altogether much better than yesterday.

---

From this date onwards patient made an uninterrupted recovery, and there was no oedema, pulmonary/

pulmonary or subcutaneous, by the 1st December, on which date she passed the normal quantity of urine. Heart sounds clear and regular. Retinitis passing away and sight good. She was fed throughout on a milk diet, barley water, and Benger's Food.

I append charts showing temperature, pulse, respirations, arterial pressure, etc,, which speak for themselves.

I should like to add, before concluding my remarks on Acute Nephritis, that in taking routine blood pressure measurements of such cases, the Sphygmometer once more comes to our aid and acts like a danger signal in pointing out, as it undoubtedly does, the advent of uraemic urgent symptoms which call for immediate treatment for lowering the arterial pressure, for warding of uraemic convulsions, and for reducing the extreme degree of over-work on the part of a heart already far too much overstrained. "Forewarned is forearmed" is a sound old proverb, and I cannot help emphasising the fact that we cannot dispense with an instrument which so clearly points out to us the probable invasion of such a dreaded and too often fatal symptom as uraemic eclampsia.

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See Table I. Case 5. page 26

Chart 1.

(Mill Road Infirmary)
or

# WEST DERBY UNION INFIRMARY. Liverpool.



**Patients Name** M. Fisher ... Aged 21 .. (Dom. Servant) A2 ... Ward.

Acute Nephritis

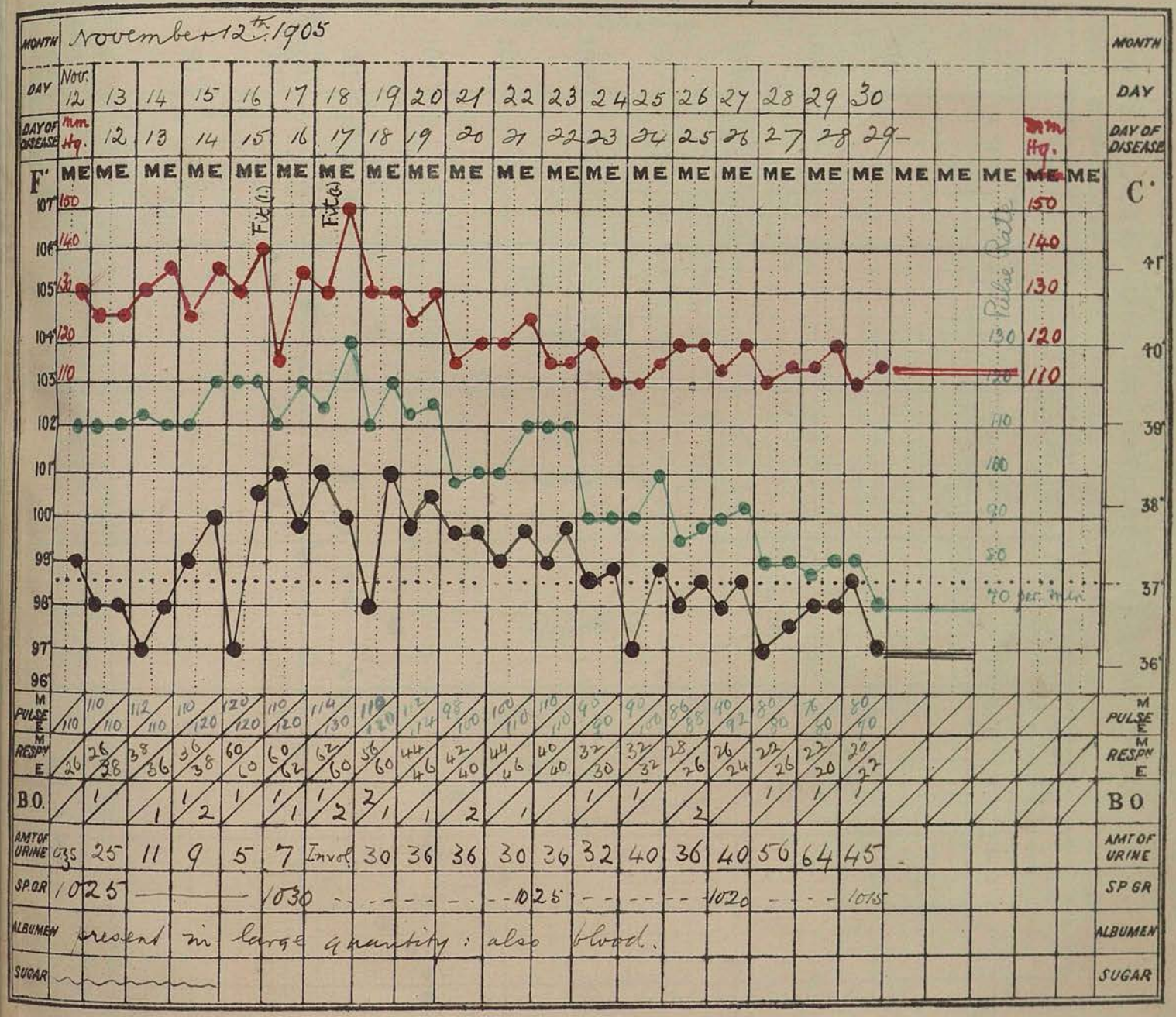



Chart Showing 1. Blood Pressure (arterial) in Red
2. Pulse Rate . . . . in Green
3. Temperature . . . . in Black.

Note the parallel movement of Pressure & Pulse.

(Mill Road Infirmary, or)

See Table I. Case 5. page 26

# WEST DERBY UNION INFIRMARY, LIVERPOOL.

Name M. Fisher (Dom. Servant) Age 21 Disease Acute Nephritis. Admitted Nov. 12th 1905

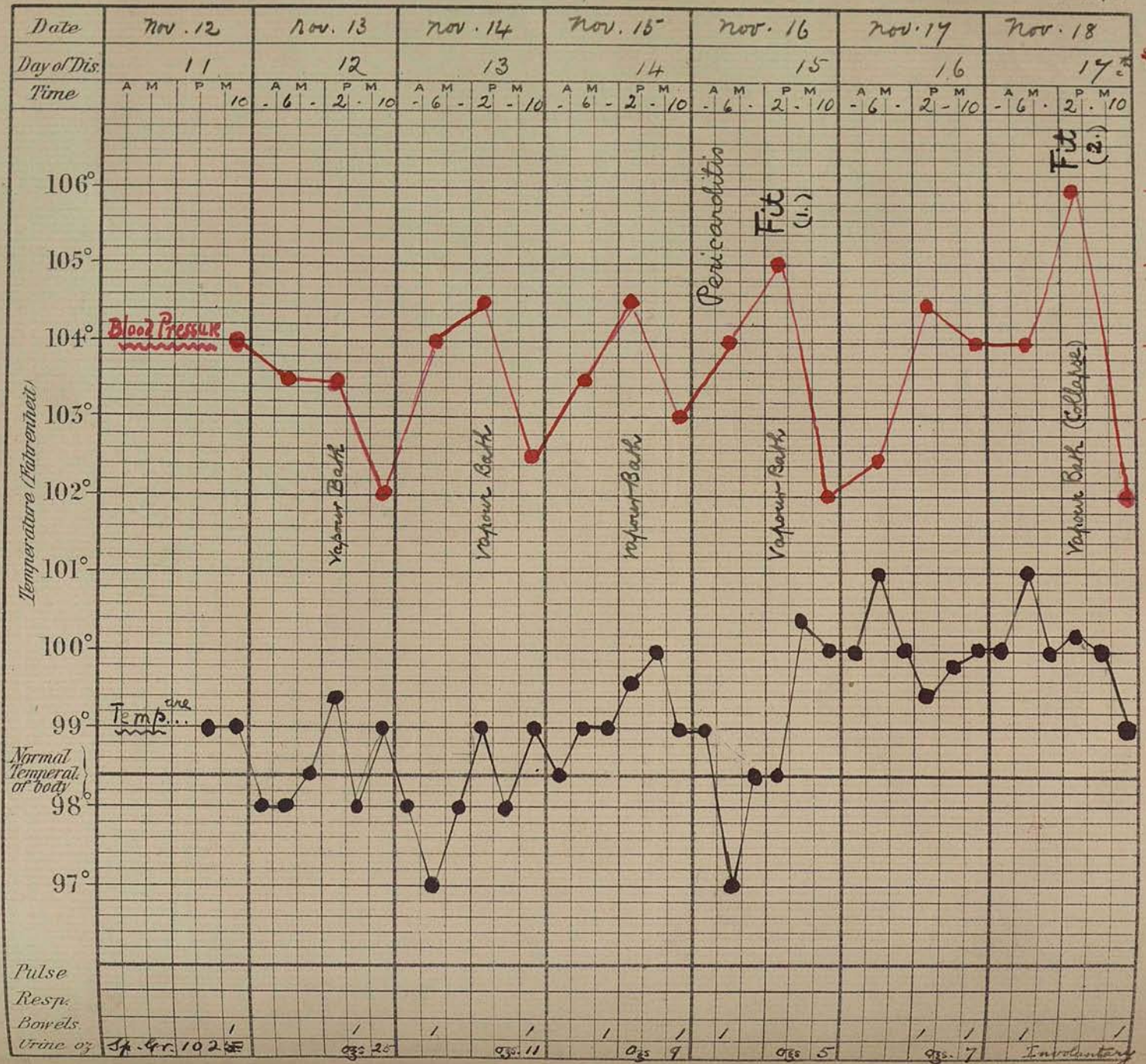
Chart 2.

Notes of Case

Diet. etc.

See pages 33-37 for ① Treatment ② Diet, & ③ Notes of Case

Result Cured.



## Subacute and Chronic Tubal Nephritis.

In this stage of renal disease, when the symptoms are of a mild form and when the patient passes a moderate quantity of urine containing a slight amount of albumen, the arterial pressure may occasionally oscillate about the normal limits. This we especially find in the better-class patients whose cardio-vascular system has suffered little or no damage through the previous attacks of nephritis, since they have had all the care and attention that medical skill and good nursing could provide. Where, however, the opposite order of things prevails, as it too vividly does in the case of the poor, and where the bodily frame is sorely handicapped as it far too often is with the effects of alcohol, syphilis and exposure, the cardio-vascular mechanism seldom escapes and there is a general hyperplasia of the heart and vessel walls which shows itself most strikingly in increased arterial tension. And so it is that amongst the class of patients we get in this Infirmary, one almost invariably finds a certain degree of hyper-tension pari passu with subacute nephritis. The figures I have given in the above table confirm this statement.

Another class of cases in which we usually find hypotension in the Amyloid Kidney, where

cardiac/

cardiac hypertrophy invariably fails and where consequently the arterial pressure is usually normal, or at times even subnormal. Again, in marked cardiac insufficiency and other complicating diseases such as pulmonary phthisis and in cachectic conditions, we frequently have hypotension running hand in hand with serious long-standing renal disease.

With regard to the prognosis of cases suffering from chronic nephritis, if we exclude granular contracted kidneys, hypertension - especially when it is of a progressive nature and associated with marked cardio-vascular disease - appears to be of very serious import. The following case is a striking illustration of this, as the blood pressure chart so well shows.

Case 24. Table I.

Patient, G.R., aged 46, was admitted into Hospital complaining of headache, dizziness, pain in the back and swelling of the feet and face. He had shortness of breath on slight exertion and a troublesome cough for some months back. Ten years ago he was treated for a similar complaint and was then "cured" in about three months.

He had marked enlargement of the heart with an accentuated aortic 2nd sound and mitral systolic soft murmur. The apex beat was 1 inch outside and 1½ inches below the nipple. The urine (Sp.Gr.1012) showed/

showed a great quantity of albumen and a slight trace of blood, with granular epithelial and fatty casts. The pulse tension did not feel high, but upon the Sphygmometer being applied it was found to be 170 Mm.Hg.

Treatment was administered as noted on the chart below, but the condition of the patient gradually became worse. Three days after admission he suddenly began to show signs of mental aberration; he became restless, talked noisily to himself, and had delusions of persecution. Paroxysmal uraemic dyspnoea supervened, with anxious expression and pale puffy face. The following day he complained of severe stabbing pain in the cardiac region, and upon auscultating he was found to have undoubted pericardial friction. The same night he had a sharp attack of epistaxis, which recurred at intervals during the next two days. On the 5th day the arterial pressure had fallen very considerably and the patient died of gradual cardiac failure the 6th day after admission. The terminal hypotension of impending cardiac dissolution is vividly shown in this Blood Pressure Chart (see page 44).

Post Mortem Notes.

At the autopsy both kidneys were found to be considerably enlarged, the left weighing 11 ozs. and the right 10 ozs. The capsule was somewhat thickened/

thickened and adherent at places. The surface was rather pale and mottled. On section the cortex was a little thickened, firm, dense and translucent, with well marked congestive areas at the bases of the pyramids. There was a general increase of interstitial connective tissue. Three small cortical cysts of the size of a pea were present in the left kidney.

The Heart was much enlarged (18 ozs.). It showed recent pericarditis on the anterior aspect of the visceral pericardium where there was a small deeply congested area covered over by a thin layer of plastic lymph. The mitral orifice was slightly dilated but there was no valvular disease. There were small atheromatous patches on the 1st part of the aorta.

Liver weighed 5½ lbs.,was much enlarged and had chronic venous congestion. Capsule was slightly thickened.

Spleen also enlarged, weighed 12 ozs., was soft, darkish red, and pulpy; no perisplenitis.

Lungs had hypostatic congestion in both posterior bases. The right weighed 2 lbs.,4 ozs., and the left 1 lb.,12 ozs. There were no pleural adhesions, but there was marked chronic emphysema of the anterior borders.

Brain was quite normal.

Arteries. Save the small atheromatous patches above referred to, no atheroma was discoverable elsewhere after minute and careful examination, but there was general thickening of the arterial walls from well-advanced arterio-sclerosis.

(Mill Road Infirmary, or)

(Case 24. Table 1.)

# WEST DERBY UNION INFIRMARY, LIVERPOOL.

Name George Robinson Age 46 Disease Chronic Parenchymatous Nephritis Admitted 31-1-1906.

Chart 3.

**Notes of Case**

Diet. etc.

Milk diet + weak hot tea. -

Jan. 31.
R. Pulv. Jalap. Co. 3ʒ
nightly if necessary.

R. Potass. Acetat gr. XV
Liq. Ammon. Acet 3ʒʒ
Spt. Aether. Nit. mXXX
Syr. Aromat. mXX
Aq. Camphor ad. 3ʒ
every 4 hours.

Feb. 1. Dry Cupping of loins.

Feb. 3. Do. .. .

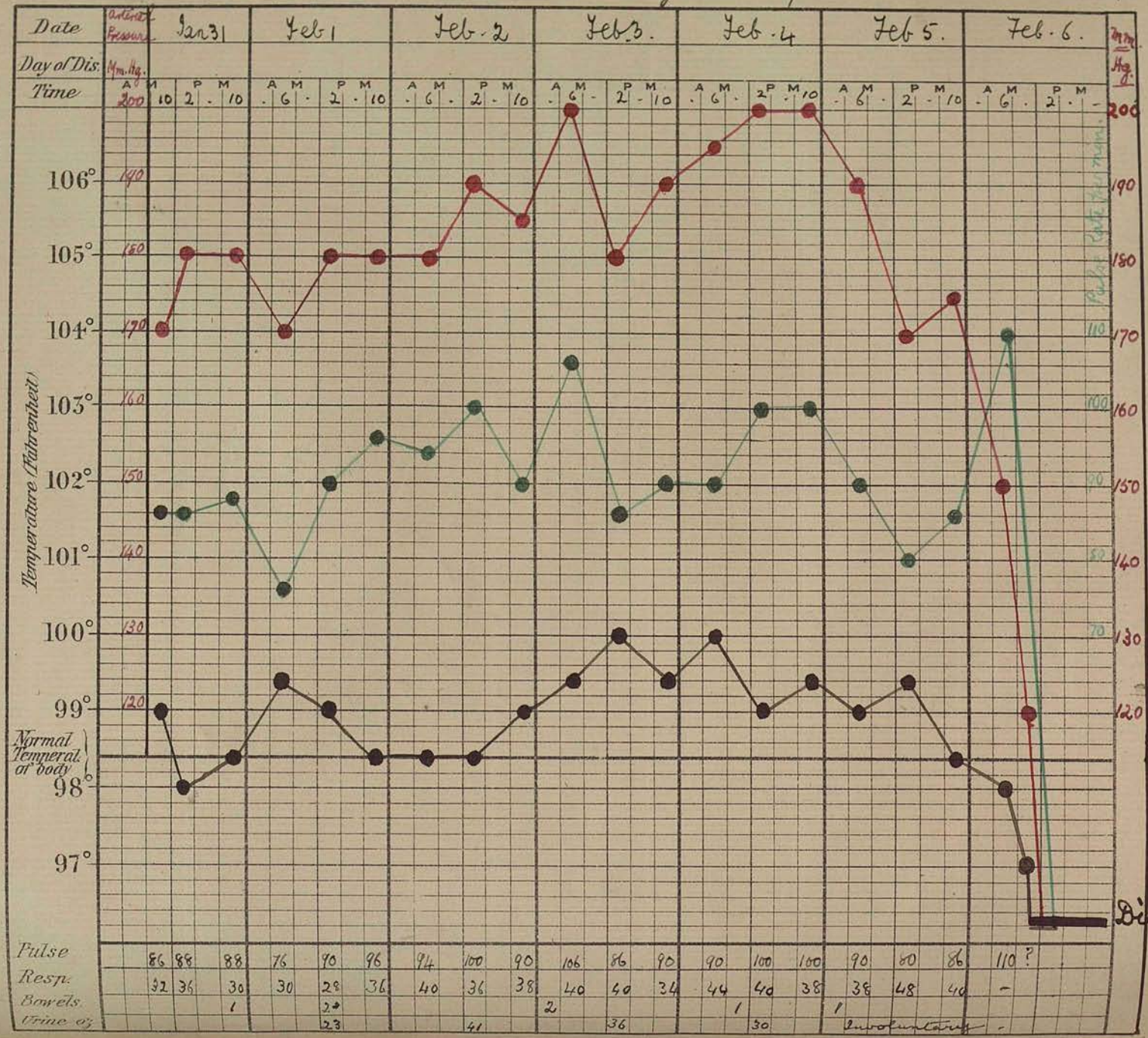
" R. Vapour Bath
for 15 mins.

R Hyoscin Hydrobromid
gr. 1/200 ..
Hypoderm.

Feb. 4. R. Mustard to
Praecordia.

Feb. 5. R. Strychnin gr 1/50
Digitalin gr 1/100
Hypoderm.
4 Hourly.

Result Died



Case 29. Table I.

This patient, who was a painter, aged 61, was admitted into hospital in a critical condition. On examination he was found to have chronic emphysema with numerous moist and sibilant rhonchi in both lungs. The heart was much hypertrophied and had mitral insufficiency. The pulse was irregular, of low tension, and the radial wall felt thickened and slightly tortuous. The urine, sp.gr.1015, was scanty and contained much albumen but no blood; it also contained fatty and hyaline casts microscopically.

He had been in indifferent health for the previous five months, but only lately took to bed. He complained mostly of difficulty in breathing, troublesome cough, weakness and pain in the loins, and dizziness, and had been passing but very little urine. He had two slight attacks of lead colic 10 years ago.

On admission he showed marked pallor and puffiness of the face, oedema of feet, and was suffering from severe uraemic asthma and evident cardiac failure. He developed Cheyne-Stoke's respirations, passed into a state of coma and died the following day - treatment being of no avail.

The arterial pressure on admission was 120 Mm.Hg., and after six hours treatment with large doses of Strychnine and Digitalis it was 140 Mm.

Six/

Six hours later it fell to 90 Mm. No further observations were possible.

Post Mortem Notes.

Kidneys. Right = 9½ ozs.

Left = 10 ozs.

Both kidneys showed thickened, pale, and mottled cortex. The capsule was adherent in most parts and the right cortex contained four small clear-walled cysts.

Heart (18 ozs.). The left ventricle was much thickened and the mitral orifice dilated, admitting three fingers easily.

Arteries. The aorta had several small patches of atheroma in the first part of the arch, and the coronary arteries on cardiac section much thickened, but no atheroma noticeable.

Liver was much congested with thickened capsule opaque in parts. It weighed five pounds.

Lungs were both oedematous at their bases, and both apices and anterior borders were emphysematous.

There was nothing further worthy of note.

## Granular Contracted Kidneys.

## (Chronic Interstitial Nephritis).

In no form of disease have I met with such persistent high arterial pressure as in this variety of Nephritis. It is essentially a disease of kidneys, heart and arteries combined, and the more advanced the disease the more hyperplasia of the heart and vessel walls do we find. How exactly such hyperplasia comes about has been a much disputed question, and I do not know that we are to-day much nearer a solution of the problem than they were in the days of Bright. He gave as the cause of the cardiac hypertrophy in Nephritis some altered condition of the blood which he maintained might either abnormally stimulate the heart, or increase the resistance in the capillaries and small vessels. The whole question touching the marked hypertrophic changes which we find in the heart and arteries in renal diseases and their relationship to each other is an intensely interesting one, but I cannot do more than merely refer to it in this paper. Equally interesting is the relationship of arterial hypertension to arteriosclerosis. In recent times there has been a keen discussion on this latter question, and the general consensus of opinion now seems to be that arteriosclerosis, in nephritic cases at least, is not (as used/

used to be considered) the cause, but rather the effect of arterial hypertension. But even allowing that this is so, what then is the cause of the hypertension? This question is still more difficult to answer, and there has been such a conflict of opinions that we may well leave the answer sub judice. Cowan gives a good and interesting critical exposition of the various views in "The Practitioner" of August, 1904. That permanent hypertension does exist in cirrhotic kidney and that concurrently with it there is a marked degree of cardiac hypertrophy and arterio-sclerosis is a fact which, though already well recognised, is still more firmly established by the above tabulated statistics (vide pp.26-31). This condition of things, however, is not consistent with even comparative health for any length of time. "A hypertrophied heart is not as good as a normal one," as the old dictum says; neither are thickened arteries as reliable as normal ones. They are deprived of their elastic distensibility and the continual overstretching of their walls by a rising and already far too high internal pressure sonner or later leads to degenerative changes and local weakening and apoplexy - the occurence of which as a common termination to interstitial nephritis has been well established, as the following case still further proves:

Case 46. Table I.

Patient, a labourer,, aged 64, on admission to hospital had marked pulmonary oedema, a very much enlarged heart with mitral regurgitation, hard and thickened radials and oedema of the ankles. The urine sp.gr.1015, was scanty, high coloured, contained a large quantity of albumen, a trace of blood, and granular and hyaline casts. He had been in bad health for the past two years and had been treated twice for Kidney disease previous to that time.

Five days after admission he had a severe attack of cerebral haemorrhage, passed into a state of coma, with flushed face, slow, full and tense pulse, stertorous breathing, conjugate deviation, and flaccid limbs. Soon the coma deepened, Cheyne-Stoke's breathing and respiratory failure set in with cyanosis, the pulse quickened, the temperature rose, the blood pressure gradually fell, and the patient died about 24 hours after the apoplectic seizure.

Post Mortem Notes.

At the autopsy both kidneys showed advanced chronic interstitial nephritis, each weighing $3\frac{1}{2}$ ozs. The capsule was thickened and adherent, and when stripped revealed an irregular granular surface. The cortex on section was tough, firm

and/

and much atrophied, and contained several small cysts

The Heart was vastly hypertrophied, weighing 32 ozs: the left ventricle was dilated and the mitral orifice measured 3½ inches in diameter. The mitral valves were thickened and had small patches of atheroma at their bases.

The Arteries were generally much sclerosed and thickened, patches of atheroma being met on the first part of the aorta. The arteries at the base of the brain were hard and tortuous.

The Liver (46 ozs.) was about normal in size and was of the nut-meg variety. Capsule thickened.

The Spleen (5 ozs.) was rather firm, with opaque thickened capsule, and had two old fibrous infarcts.

The Lungs both showed marked oedema with emphysema, but other wise showed no abnormality.

The Brain (50 ozs.) had extensive haemorrhage in the right basal ganglia, which had ruptured into the right lateral ventricle.

The following blood pressure chart (Chart 4), belonging to the above case, is interesting for the following reasons :-

(1) Effect of nitroglycerine & vapour baths on pressure.

(2) The moderate degree of arterial pressure, accounted for by cardiac insufficiency.

(3) Rise in pressure following cerebral haemorrhage.

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(Mill Road Infirmary, or)

# WEST DERBY UNION INFIRMARY, LIVERPOOL.

Chart 4.
(Case 46 Table I.)

Name G. Paynter ; Labourer Age 64 Disease Chronic Interstitial Nephritis Admitted 1. 12. 05

**Notes of Case**

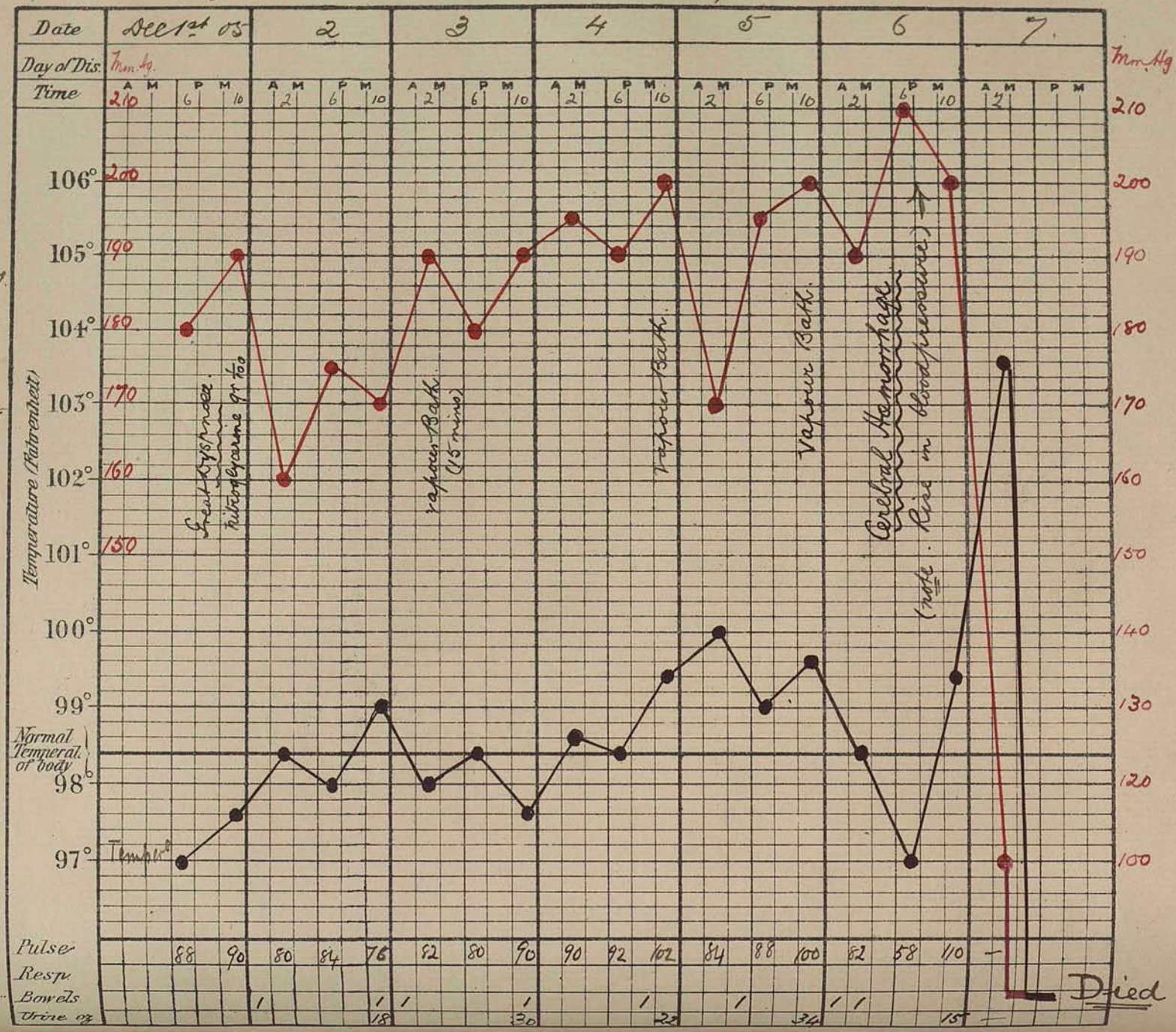
Diet. etc.
Milk Diet.

R. Caffein. citr. gr V.
morning & evening.

R. Tinct. Digitalis m. 10
Spt. Ether. Nitr. m
Liq. Strych. Hydrochlor. m V
Aq. Chloroform. ad 3ß.
4 hourly.

R. Pulv. Salapae Co. 3ß
pro re nata.

Result Died.



As an interesting case of chronic interstitial nephritis with extreme and persistent hypertension, the following is worthy of being recorded on account of the recurrence of severe attacks of pulmonary oedema with distressing dyspnoea invariably at night, the patient getting striking relief from Nitroglycerine. The case is of still further interest in showing the unequal struggle between an overtaxed hypertrophied heart and arterial hypertension, followed by inevitable complete failure of cardiac compensation and sudden dissolution - so well shown by the terminal descent of the pressure curve on Chart 5 (p.56).

Case 50.Table I.

H., B., housewife, aged 47, was admitted into this Hospital in November 1905, complaining of great difficulty in breathing, especially at night, severe headaches, dizziness and dimness of vision, occasional vomiting, and frequent attacks of palpitation. She had been treated here some 12 months ago for a similar but milder attack, when she remained an in-patient for about a fortnight, and took her discharge in a condition fit to resume her daily work. At that time her arterial pressure recorded 180 to 200 mm.Hg., and the apex beat was about $^{3}/_{4}$ in. outside, and one inch/

inch below the nipple. She had a faint mitral systolic murmur but compensation was well established. Since then the heart had considerably hypertrophied, the apex in Nov.1905 being at least 1 inch outside and 1½ inches below the nipple, the arterial pressure recording 260 mm.Hg. - about the highest measurement I have met with in Nephritis. Her radial artery was much thickened, tortuous and whip-cord like to palpitation. The nocturnal attacks of orthopnoea were most distressing for the first two nights after admission and were accompanied by severe pulmonary oedema, headache, and occasional vomiting. She had also considerable oedema of both ankles. Ophthalmoscopic examination revealed albuminuric retinitis, white patches being scattered irregularly round the disc whose margin appeared somewhat swollen.

Under nitrites, potassium iodide and vapour baths she made a wonderful recovery and in a week's time she was in comparative comfort and making satisfactory progress. A fortnight after admission, however, she relapsed into her former dyspnoeic condition and all treatment was of no avail. Cardiac failure set in with irregularity and loud blowing apical systolic bruit. The pulmonary oedema and the oedema in the ankles became more marked; she developed Cheyne-Stoke's breathing and died after being/

being in a semicomatose condition for 12 hours. There were no uraemic fits.

Post Mortem Notes.

Kidneys. Both kidneys were exceedingly small and hard, each weighing 2 ozs. The capsule was thickened, opaque, and firmly adherent to the cortex, and on being torn away left a pale very granular surface. On this surface small pin-head sized cysts were seen. On section the cortex was pale, tough, and seemed to have very little parenchyma left - only a thin layer of sclerosed tissue.

Heart. Heart weighed 22 ozs. The left ventricular wall being enormously thickened. There was no organic disease of the valves, but the mitral orifice was dilated to 2 ins. diameter.

Arteries. There were several small atheromatous patches on the first part of the aorta, but no atheroma visible elsewhere. The radials, the cerebrals, and the splanchnic arteries were found to be much thickened on section.

Lungs. Lungs were both extensively oedematous, the right weighed 28 ozs. and the left 26 ozs; both apices were emphysematous.

There/

There were no signs of pleurisy.

Liver. Liver weighed 63 ozs: had chronic venous congestion and capsule was thickened.

Spleen. Spleen = 6 ozs. Capsules opaque and thickened: tissue firm and dark red.

Brain. Brain = 45 ozs. Normal.

---

The following blood pressure chart from the above case is interesting in showing the value of Nitro-glycerine and Vapour Baths in reducing the arterial pressure, the reduction being attended by a marked improvement in the symptoms; further it points out the bad prognostic significance of a hypertension that baffles therapeutic treatment.

Chart. 5.

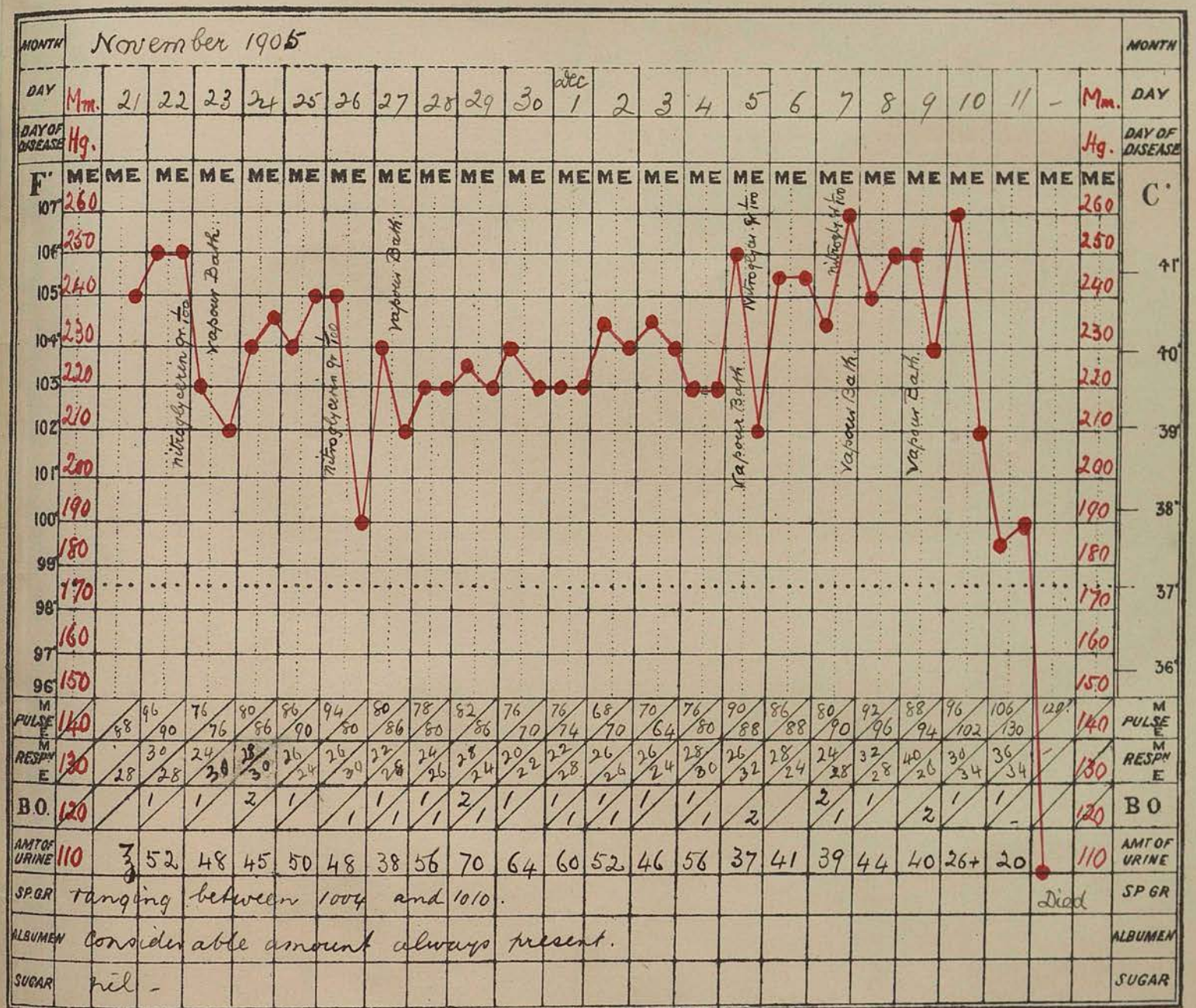
(Mill Road Infirmary, or)

56.

# WEST DERBY UNION INFIRMARY.

Case 50 - Table I.

*Patients Name* Hannah, Blanche; Aet. 47. A2. *Ward.*



## SUMMARY

To briefly recapitulate, the chief conclusions which may be drawn from the foregoing remarks on blood pressure measurements in renal disease may be summed up under the following heads.

Diagnosis.

(1) The Sphygmometer is a valuable accessory for the clinician in clearing up any doubt that may exist regarding the actual degree of arterial pressure.

(2) In differentiating between a case of primary acute nephritis and one of a subacute or an exacerbation of a chronic nephritis, blood pressure observations are most useful.

(3) In diagnosing the advent of acute uraemia the blood pressure chart may be said to be almost, if not quite, as essential as the temperature chart in a case of fever.

(4) So diagnostically important are routine blood pressure records in cirrhotic kidney disease that given a case with a systolic pressure over 200 Mm.Hg., the existence of this disease must be disproved by repeated examination before making any other diagnosis.

Further/

Further in cirrhotic kidney, examination of the urine may be negative and there may be such a degree of obesity or of pulmonary emphysema as to make percussion of the heart impossible. Here the application of the Sphygmometer will certainly assist us in arriving at a correct diagnosis.

Prognosis.

(1) In cases of nephritis where the arterial pressure has been permanently high and is gradually increasing in spite of treatment, even if the patient be enjoying a moderate degree of health, our prognosis must be a guarded one, especially if there be any sign of cardiac insufficiency.

(2) A high and increasing blood pressure is of grave significance where we have good reasons for suspecting cerebral arterial disease, for cerebral apoplexy is a common termination of chronic Bright's disease.

(3) The Sphygmometer is of great prognostic value in predicting uraemic eclampsia - a grave complication of nephritis.

(4) When the pressure curve takes a sudden downward/

downward course in a case of long-standing hypertension, the immediate prognosis is decidedly bad, indicating onset of cardiac failure.

Treatment.

In the treatment of nephritis, blood pressure charts are visible proof of the progress of the case, and as such are, I consider, an invaluable aid to the physician. Any sudden rise or fall in pressure is graphically shown on the chart, and this puts the physician in a position frequently to administer timely remedies. Such therapeutic interference is specially indicated, for example, in the dangerous hypertension which so frequently leads to cerebral haemorrhage , in the hypotension of cardiac insufficiency, or again in the hypertension which is the precursor of uraemia.

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