AVERAGE DYNAMIC BLOOD-PRESSURE

a theoretical, historical and clinical study

by

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CONTENTS

INTRODUCTION:

The object of this paper is twofold - (1) to give a critical summary of the theoretical, experimental and clinical work which has been done bearing on the subject of oscillometry and the "average dynamic blood-pressure": (2) to give the results of records taken with a "Kymomètre" type of sphygmo-oscillometer from a series of normal and pathological patients, and any conclusions which may be drawn therefrom.

Part 1 consists of a brief historical survey of the work done by the pioneers and outstanding workers in this field, with mention of the more important instruments and apparatus used by them.

The theory of "oscillations" is considered and the average dynamic blood-pressure is discussed from its theoretical and clinical aspects, and a résumé given of the main results so far obtained.

Part 2 consists of a specification of the instrument used, and a description of the technique and technical problems of oscillometry.

From the records taken a scale of normal readings is drawn up. The records from pathological cases are compared with this scale, and the significance of deviations from normal discussed. Conclusions which may be drawn from the records obtained are summarised.

PART 1.

HISTORICAL:

In a study of oscillometry it would be out of place to enter into a full survey of the field of blood-pressure research, as much of the work done has no direct bearing on the estimation of blood-pressure by this special method. No paper on this subject would be complete, however, without a reference to the names of the pioneers who blazed the trail which made it possible for others to follow in their steps and open up the country.

Harvey (1616) with his classic demonstration of the circulation of the blood: Stephen Hales (1733) with his first experimental record of blood-pressure, and Marshall Hall (1) (1832) with his study of the contractile power of the arteries should always be remembered for the invaluable part they played in opening up this vast field of investigation.

Not until 1876 were any further serious attempts made to record blood-pressure by a clinical method. In that year Marey (2) studied the phenomenon of the oscillation of fluid in a manometer connected to a plethysmograph on the forearm and came to the conclusion that the pressure for which he obtained the greatest oscillation represented diastolic pressure of the blood. In 1880 the same worker (3) confirmed the results of his previous experiment making use of a plethysmograph which enclosed only one finger. (Fig. 1.)

In 1876 v. Basch of Vienna invented a sphygmometer consisting of a funnel covered with a thin rubber membrane and filled with water, which being placed over the radial artery transmitted the pulsations to a manometer. He only dealt with systolic pressure, which he took as being the pressure of water for which the first oscillations occurred in the manometer.

An important advance took place in 1883 when Marey (4: 5) invented his "manomètre compensateur", an instrument which made possible for the first time an experimental record of the mean effective pressure of the circulating blood, or "pression moyenne". (Fig. 2.)

In 1889 Potain (6: 7) brought out an instrument (Fig. 3) in some respects similar to that of v. Basch, but employing air instead of water as the medium through which pulsations were transmitted to the manometer. Again only the estimation of systolic pressure was attempted.

The same worker endeavoured to estimate the average dynamic blood-pressure by mathematical calculations based on records taken with Marey's sphygmograph. Though his principle is said to be exact a fallacy in his method has been exposed by Gley & Gomez (8), but in spite of that the figure he gave for the average dynamic pressure was 97mm of mercury - a figure which would be regarded as "normal" by recent workers. An attempt was made in the following year by Roy and Adami (9) to estimate both systolic and diastolic pressures by an oscillatory method. (Fig. 4.) Their instrument was a water-filled chamber applied over the radial artery, with an arrangement to allow of a graphic record being taken. They took as an index of systolic pressure the point of appearance of oscillations in the manometer, and as diastolic pressure the point at which maximal oscillations were recorded.

At the close of last century (1896-1898) we find the foundations laid for all modern methods of clinical blood-pressure estimation by the introduction of the Riva-Rocci instrument in Italy and the Hill and Barnard instrument (10) in this country. These instruments allowed of circular compression of a limb by an airfilled rubber bag supported by a silk or leather armlet.

In 1904 the subject was taken up by Erlanger (11: 12) who brought out an instrument which in principle resembled the modern Pachon oscillometer, but in design was crude and clumsy and unsuited for clinical application.

With the advent in 1909 of Pachon's oscillometer (13) - an "oscillomètre sphygmométrique à "grande sensibilité et à sensibilité constante" - this method of estimating blood-pressure advanced from the realm of experimental physiology to become a practical clinical procedure. (Fig. 5.)

Although the presence of oscillations had been noted for many years and attempts made to measure and record them, Pachon was the first to devise an instrument of sound scientific principle which was suitable for clinical application and allowed of accurate recording of oscillation amplitude. The principle of his instrument is now embodied in all types of sphygmo-oscillometer, though of course modifications and refinements have been incorporated.

In 1921 we find a revolutionary note sounded by Russell (14: 15) who enounced a theory which, if it had found support, would have shaken the foundations of all methods of clinical sphygmomanometry.

He contended that the resistance of the arterial wall was a factor of prime significance in all blood-pressure estimations, and that the finger was the only means of differentiating between the pressure required to overcome the resistance of a hypertonic arterial wall and the actual blood-pressure. His theory, however, has not found support, the main criticism as pointed out by Hill and Flack (16) being that his experiments were on the cadaver and his results did not hold good for the living subject.

In 1921 Pachon (17) and Pachon and Fabre (18: 19) conducted further experiments with the oscillometer as a result of which Pachon's former views as to the significance of the point of maximal oscillation had to be modified. In his original experiments with his "schéma circulatoire" (Fig. 6) Pachon concluded that maximal oscillations occurred where the counter-pressure was equal to the diastolic pressure of the circulation, but as a result of these later experiments performed with a modification of his schéma it was proved that maximal oscillations corresponded to a counter-pressure equal to the average dynamic pressure, or "pression moyenne."

This is the view which is accepted to-day, and which has been confirmed by the experimental work of Gley and Gomez. (8: 28) (Fig. 7.)

Within the past few years a new interest has been aroused in the subject by the work of Vaquez and others working in his laboratory. (Gley and Gomez (8: 28); Vaquez, Gley & Gomez (20; 29; 31); Vaquez & Lajoie (21); Gomez & Lajoie (22); Vaquez, Gomez & Lajoie (23); Vaquez, Kisthinios & Papaioannou (24); Gomez (25); Vaquez & Kisthinios (26); Vaquez & Gomez (27; 30).)

In the words of Vaquez (20) "L'introduction "en clinique de la notion de la pression moyenne "constitut un fait nouveau dont on ne saurait trop "souligner l'importance."

The facts and figures which they adduce certainly seem to support this contention, but much clinical work remains to be done before the true significance of the average dynamic pressure from a diagnostic and prognostic point of view will be generally accepted. It is not merely that the medical world is always slow to place too much reliance on an innovation, but rather that most of the work has been done in one school and that the method has not yet stood the test of general application, nor has it had the prolonged and intensive investigation which it merits.

Enough work has been done to prove that we are dealing with a new conception of the significance of blood-pressure, and one which may lead to a profound modification of our present methods and standards.

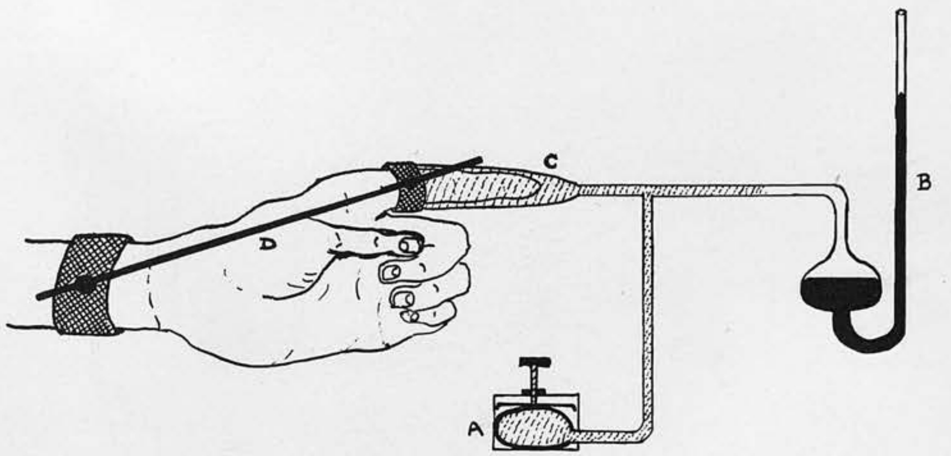
It is to be hoped that the method will at least be given a fair clinical trial so that a definite and unbiased verdict may be reached as to its true value.

INSTRUMENTS AND APPARATUS:

In the following pages a brief description will be given in chronological order of the chief instruments and apparatus which have been used in investigating the subject of oscillometry and the average dynamic blood-pressure.

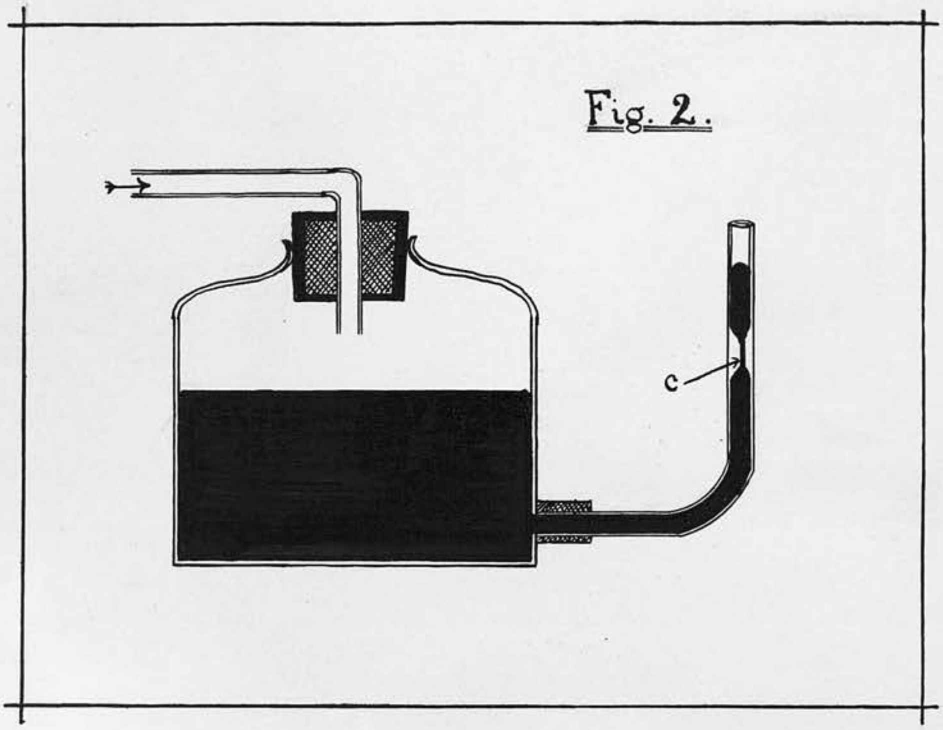
1. Marey's plethysmograph: (1880) (3.)

Fig. 1.



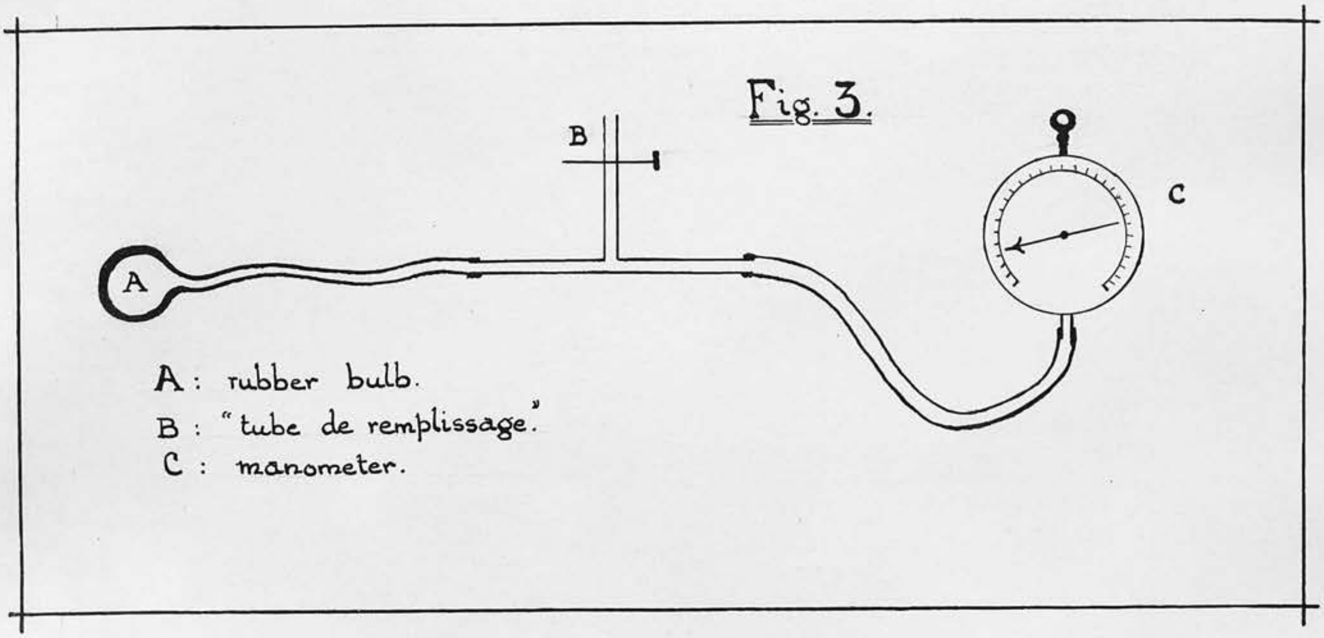
A: compressible water reservoir.
B: mercury manometer.
C: water-filled finger chamber.
D: adjustable retaining rod.

2. Marey's "manomètre compensateur": (4.)



A manometer with a wide reservoir and a column which has at its commencement a constriction (C) which prevents rapid movements of the mercury. The column of mercury remains practically stationary between Mx and Mn, indicating exactly the mean effective pressure of the system, or "la pression "moyenne."

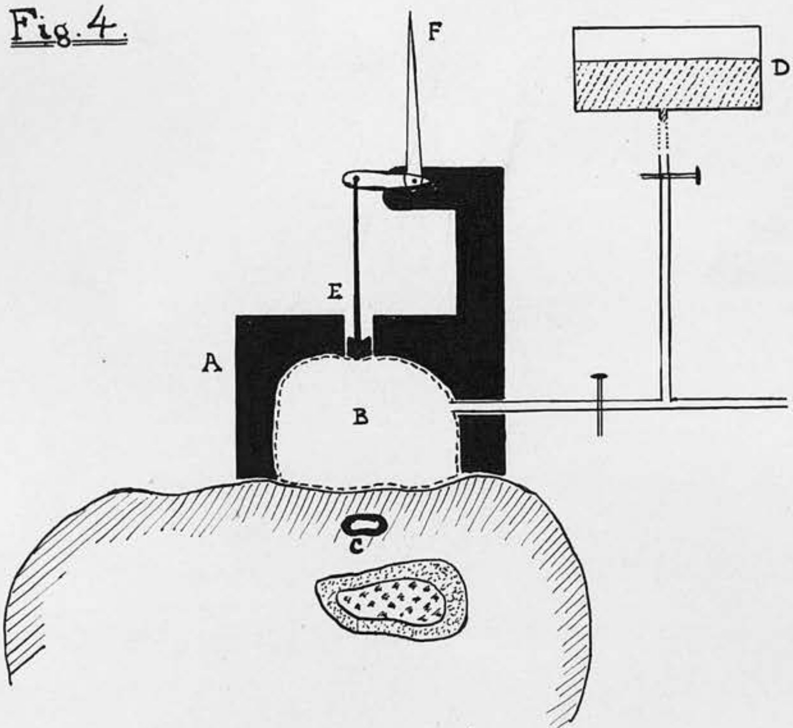
3. Potain's sphygmomanometer: (1889) (7.)



The instrument is pumped up to a pressure of about 30 mm of mercury. The bulb A is placed on the radial artery and pressure applied to it by one hand while the other hand palpates the radial artery below the bulb, and notes the point of disappearance of the pulse. The pressure on the manometer is read at this point, and indicates systolic pressure.

4. <u>Roy and Adami instrument:</u> (1890) (9.)

Fig. 4.



A: rigid box to fit over radial artery.

B: flexible water-filled bag.

C: radial artery.

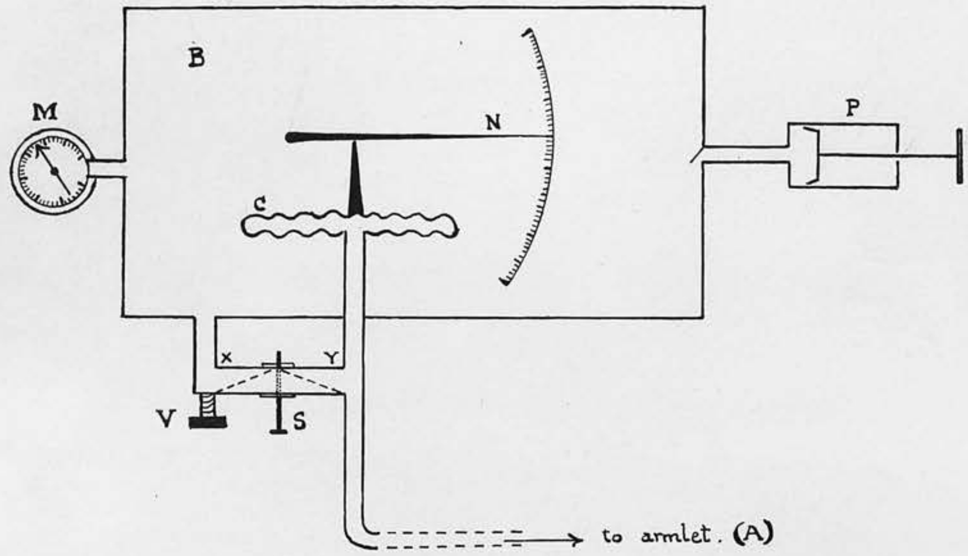
D: adjustable water reservoir.

E: button resting on bag B.

F: needle recording on smoked drum.

5. <u>Pachon's (Sphygmo-) Oscillometer:</u> (13.)

Fig. 5.



B: rigid, airtight box.

M: manometer.

C: aneroid capsule.

V: escape valve.

S: separator to cut off B & M from A & C.

N: oscillatory needle.

P: pump.

XY: rubber tubing.

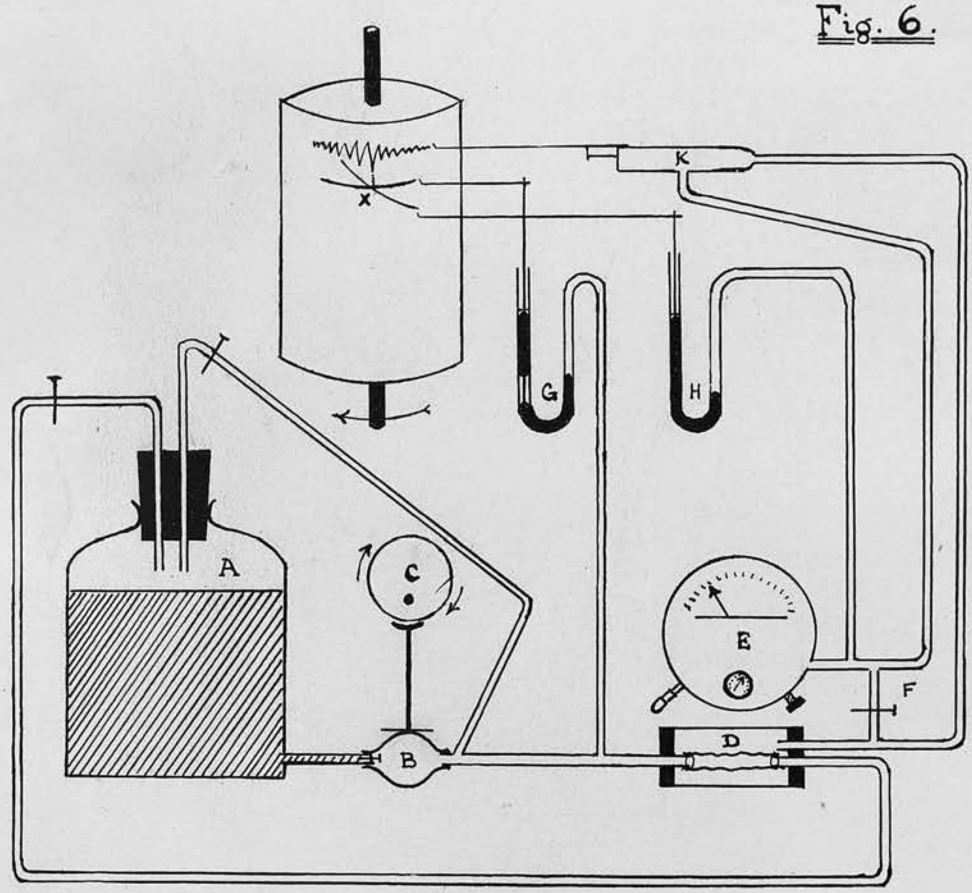
As this is the instrument on which all oscillometers are modelled an outline of its principle will be given.

When the escape valve (V) is closed and the pump is operated air is forced into the box (B), the manometer (M), the armlet (A) and the aneroid capsule (C). The walls of the capsule are thus kept in a state of equilibrium, the pressure within and without being equal: it is thus constantly in its most sensitive state, and will respond maximally to any change of internal pressure.

When the desired counterpressure, as recorded on the manometer, is reached the separator (S) is depressed, thus obliterating the rubber tube XY and closing off the box and manometer from the armlet-capsule system. Any change of pressure in the armlet is now transmitted directly to the capsule, the distortion of whose walls operates the oscillating needle (N). When the escape valve (V) is opened and the separator released the pressure is reduced throughout the instrument.

6. <u>Pachon's "schéma de circulation"</u>: (18.)

Fig. 6.



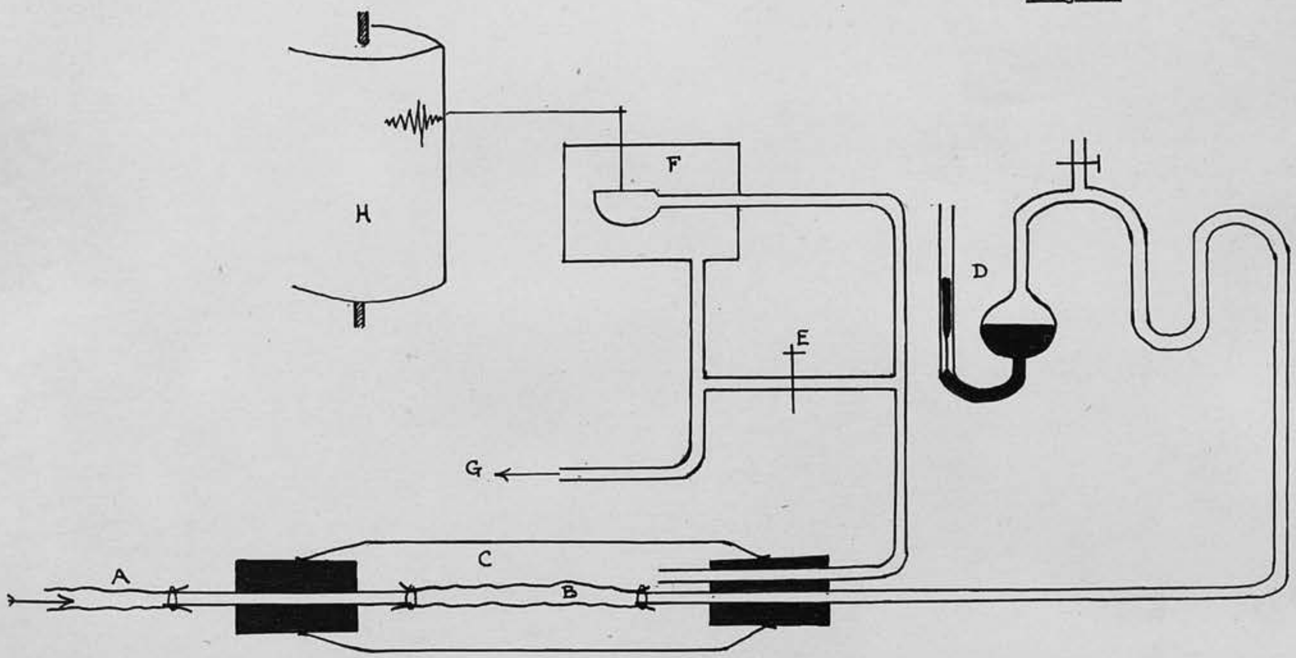
A : reservoir.
B : pump.
C : eccentric wheel driving B.
D : compression chamber.
E : oscillometer, (used simply as pressure generator.)
F : separator.
G : compensating manometer, (recording "Pression moyenne.")
H : mercury manometer, (recording fall of pressure.)
K : oscillograph.

The point X where the tracing of the decompression cuts the tracing of the pression moyenne corresponds exactly to the point of maximal oscillations, i.e. the value of the counter-pressure to which the oscillometric index corresponds represents exactly the value of the "pression "efficace" or "pression moyenne" for the circulation under examination.

7. <u>Apparatus of Gley and Gomez:</u> (8: 28.)

(Animal experiment to confirm Pachon's results.)

Fig. 7.

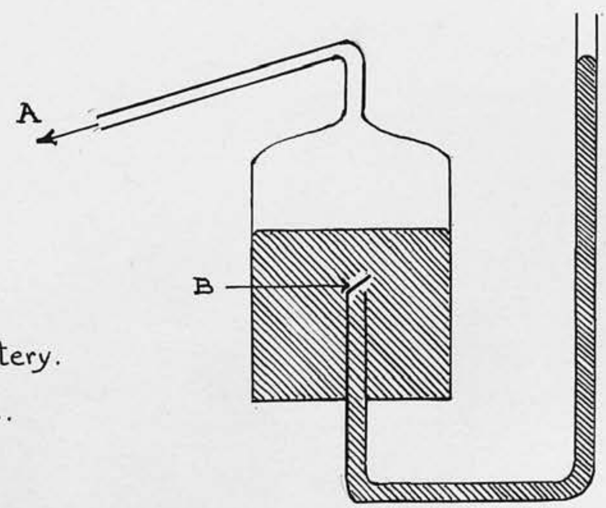


A : animal's artery.
B : segment of carotid of animal.
C : Compression chamber.
D : compensating manometer.
E : separator.
F : oscillographic capsule (Pachon-Boulitte.)
G : to oscillometer (used as pressure generator.)
H : recording drum.

The results of this experiment were identical with Pachon's - i.e. the counterpressure for which maximal oscillations were obtained corresponded exactly with the pression moyenne of the circulation as estimated by the compensating manometer.

8. "Manomètre à minima": (28.)

Fig. 8.



A: to artery.
B: valve.

This instrument was used by Gley and Gomez in the above apparatus (Fig. 7) to endeavour to confirm Pachon's finding that diastolic pressure occurs at the point where there is a "changement de pente "sur le tracé oscillométrique, et l'entrée dans une "zone terminale d'oscillations à pente faible et "uniforme."

9. Apparatus of Tetelbaum, Krinsky and Romanowa: (32.)

According to the above workers when a limb is compressed so that venous return is obstructed but arteries are still patent the venous pressure indicates the pression moyenne.

Blood is able to flow from artery to vein through the capillary bed, but not in the reverse direction. The state of affairs thus produced may be compared to a compensating manometer - the capillary bed representing the constriction in the ascending limb of the manometer.

The apparatus consists of a needle connected to a tube filled with 0.85% sodium chloride solution which communicates with a small mercury manometer.

The figures obtained for pression moyenne by this instrument correspond with those obtained by the orthodox methods, but the results given are based on a series of only 24 cases.

Even if the principle is correct, however, the method is obviously more of theoretical than practical interest.

10. "Kymomètre" of Vaquez, Gley and Gomez: (1931) (29.)

The description of this instrument will be found at the commencement of Part 2 of this paper.

THEORY OF OSCILLATIONS AND AVERAGE DYNAMIC BLOOD-PRESSURE:-

In the remainder of this paper the following abbreviations will be used:-

Mx = Systolic Pressure (Maxima.)

Mn = Diastolic Pressure (Minima.)

My = Pression moyenne.) syn-
Average Dynamic Pressure (A.D.P.)) onym-
Pression efficace (Pe.)) ous
Mean (effective) pressure.) terms.

Cp = Counterpressure.

DEFINITION OF AVERAGE DYNAMIC BLOOD-PRESSURE:

At the outset it must be emphasised that My, though it is frequently referred to as the "mean "pressure", is not the arithmetic mean of Mx and Mn in the generally accepted sense, though it is their "generalised" arithmetic mean.

The average dynamic blood-pressure may be defined according to Vaquez & Lajoie (21) as that pressure which, if applied as a constant pressure, would produce during a given time the same flow of blood through an artery as is produced by the fluctuating pressure which normally exists there - in other words it is the "mean effective pressure" of the system.

My is thus the mean of all the ordinates drawn from a sphygmographic curve to the zero line.

If the pressure curve had the form of a sinusoidal curve then My would represent the arithmetic mean of Mx and Mn; actually however we are dealing with a curve showing many irregulatities, and therefore My depends as much on the shape of the curve as on the value of its extremes.

EXPERIMENTAL DETERMINATION OF My:

In 1883 Marey made possible for the first time the recording of the "mean pressure" of a circulation when he introduced his "manomètre compensateur." This instrument, in which the oscillations of mercury in a U-tube manometer are damped down by a constriction in the ascending limb, can only be used in direct methods of pressure estimation, and so is of course unsuited to clinical use.

Prior to the introduction of this instrument the phenomenon of oscillations had been noted and attempts made to place a value on the pressure for which maximal oscillations were obtained. Marey, and later Potain, and Roy and Adami believed that maximal oscillation occurred at diastolic pressure, and we find the same view expressed as late as 1909 by Pachon in his early experiments with a "schéma "circulatoire."

The introduction of Pachon's oscillometer in that year allowed for the first time of oscillometry being used as a clinical procedure for indirect blood-pressure estimation and drew attention to the problems associated with the phenomenon of oscillations.

In 1921 Pachon and Fabre (18) conducted further experiments with the oscillometer and proved conclusively that maximal oscillations occur where a counterpressure is applied equal to the My of the system. These results have been repeatedly confirmed by other workers - Vaquez, Gley, Gomez and others - both on artificial circulations and by animal experiments, and an attempt must now be made to explain this phenomenon on theoretical grounds.

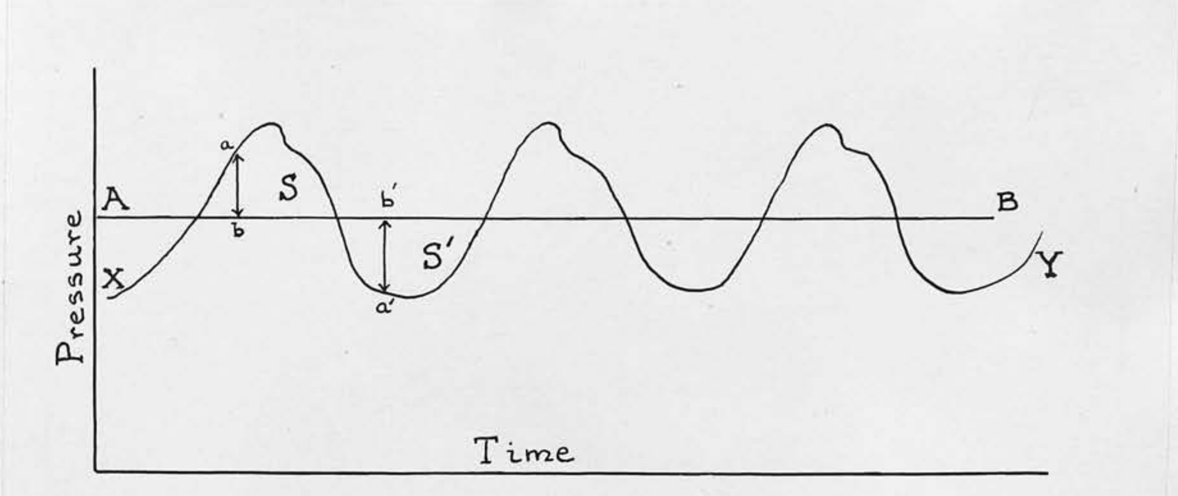
THEORETICAL DETERMINATION OF My:

Any attempt to give a complete mathematical demonstration of the fact that maximal oscillations occur where Cp = My seems doomed to failure as the problem contains such an enormous number of unassessable variables. For instance we should have to take into account such factors as friction; reflected waves, both proximal and distal; the effects of venous congestion; the reflex vaso-motor effects of compressing the artery, and many others.

It is no doubt the extreme complexity of the problem which accounts for the fact that in all the literature consulted only three attempts have been found to give a mathematical solution, (Vaquez, Gley & Gomez (31.): Pachon & Fabre (33.): Pachon (34.)) and none of them could claim to be either complete or conclusive.

In the following attempt it must be admitted that the variable factors have been entirely disregarded. The problem has been approached in the simplest way possible, and the explanation is advanced in the light of "common sense" rather than of applied mathematics.

Let XY represent a pressure curve and AB and Cp applied to the system, its value being anywhere between Mx and Mn.

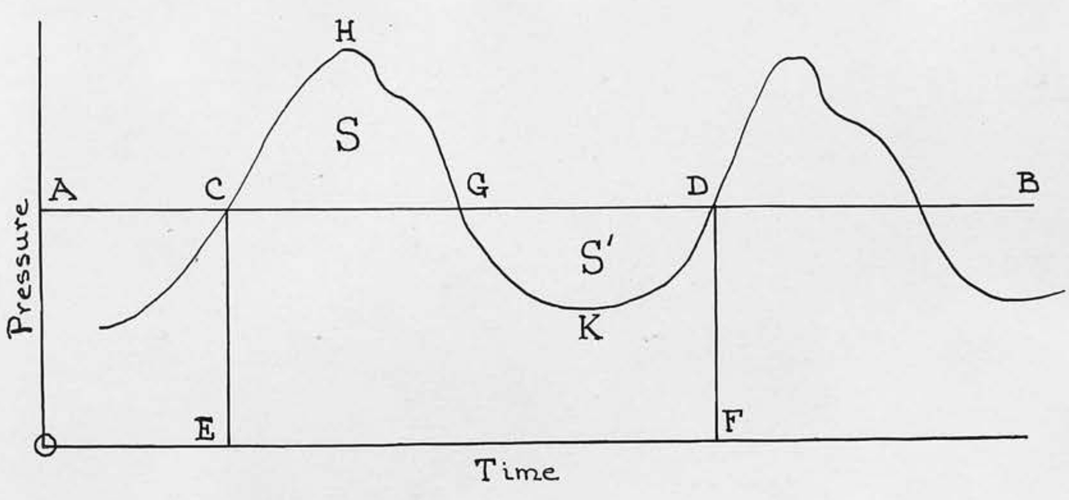


The resultant of the blood-pressure and the counter-pressure can for any moment be read by measuring the ordinate drawn from the point on the pressure curve to the counter-pressure line - e.g.

ab or a'b'. When it is above AB, e.g. ab, the force is one tending to open or distend the artery; when it is below AB, e.g. a'b', the force tends to close or compress the artery. The resultants of all the forces above AB will produce "opening" waves during the time for which they act; similarly if the forces are below AB the result will be "closing" waves.

Now the coordinates of the pressure curve being pressures and times the opening wave is proportional to the surface S and the closing wave to the surface S'. But an opening wave will result in an increase in volume of the segment of the artery and consequently an increased inflow; similarly a closing wave will result in an increased outflow, and therefore theoretically volume inflow is proportional to surface S and outflow to surface S'. But volume inflow cannot for any length of time be either greater or less than volume outflow, as it is limited by the capacity of the artery and the outflow from it. Therefore if S is greater than S', the volume inflow would be greater than the volume outflow, which is impossible. Inflow can only be equal to outflow and therefore total volume change is proportional to 2S', the remainder of the opening wave (represented by S-S') simply producing stress on the already fully distended artery. Similarly if S is less than S', volume change can only be proportional to 2S, the remainder of the closing wave (S'-S) producing stress on the already completely obliterated artery.

Where S equals S', the opening wave equals the closing wave and the total volume change is proportional to S plus S'. But only where S equals S' is the maximum possible volume inflow succeeded by the maximum possible volume outflow, thus giving a maximum volume change and maximum oscillations.



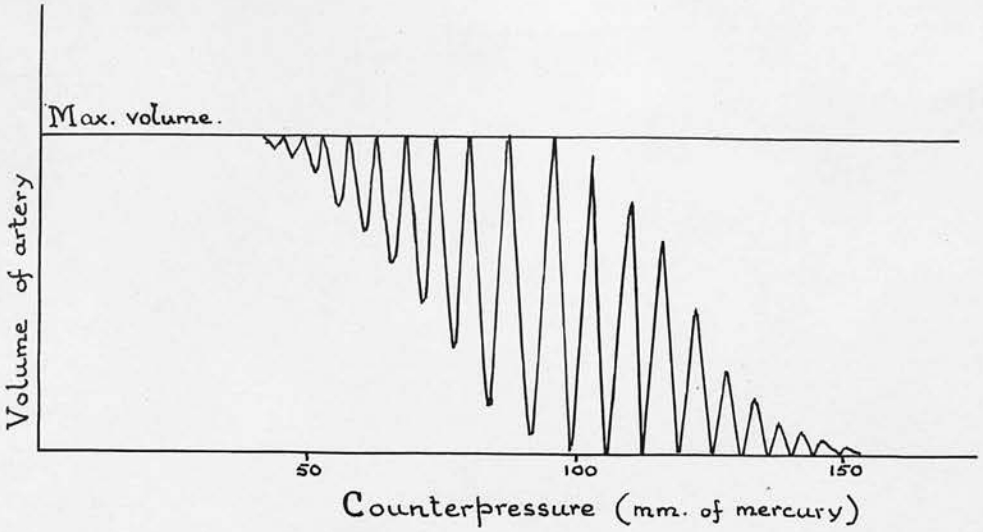
The mean ordinate (My) of the curve CHKD is the area ECHKDF/EF. If OA (or EC) is the mean ordinate then area ECHKDF equals area ECDF. Subtracting from both the area ECGKDF then area CHG equals area GKD. That is S equals S'.

But it has been shown that maximal oscillations occur where S equals S', therefore maximal oscillations occur where Cp equals My.

Maximum volume change, represented by maximal oscillations, means the change from a state of complete occlusion to a state of full distension, and it has been shown that this can only occur where Cp equals My.

If Cp is greater or less than My the volume change is less, which means that the artery is either not completely filled at each systolic phase or not completely emptied at each diastolic phase. Thus for counterpressures above My we find that the artery is completely occluded between beats and oscillations are produced by the intermittent partial distension of the occluded artery. For counterpressures below My the artery will never be completely occluded, in which case oscillations are produced by the partial occlusion of the distended artery.

Vaquez, Gley and Gomez (31) point out that this change in the starting point of the oscillations can be represented diagrammatically thus:-



SIGNIFICANCE OF My:

With a constant pulse rate the outflow of blood from a vessel in unit time is directly proportional to the mean effective pressure of the circulation, provided that the peripheral resistance remains constant. Moreover the work performed by the heart is proportional to the outflow of blood in unit time from the vessels. Thus there is a direct relationship between the work of the heart and My, and Vaquez and Lajoie (21) emphasise that any elevation of the latter is going to throw extra strain on the cardiac muscle. As a result of this it has been stated, with clinical and radiographic confirmation (Vaquez, Gley and Gomez (20); Vaquez and Gomez (27); Gley and Gomez (35);) that the size of the left ventricle always corresponds to the height of My. They are strongly of the opinion that the "pression moyenne" plays a primary role in the etiology of cardiac hypertrophy and of arteriosclerosis.

Theoretically there can be little comparison between the effect of the A.D.P. and that of systolic pressure on the work of the heart. The systolic pressure is only momentary; the A.D.P. is the expression of the sustained effort imposed on the heart. It is emphasised by Vaquez and Gomez (27) that a slight elevation of the A.D.P. will much more readily cause cardiac hypertrophy than even excessive bursts of high pressure in a case of systolic hypertension.

Similarly with the arteries, we cannot gain an accurate conception of the stress to which they are constantly subjected by measurement of the momentary extreme pressures. Systolic pressure measures the initial stress but gives no indication of the constant hydraulic load which they have to bear throughout the cardiac cycle - "mauvais "critérium, la pression maxima ne pouvant caractéri- "ser spécifiquement un régime vasculaire dont elle "représente seulement un très court moment." (Vaquez, Gley and Gomez (20).) It is My which gives us a clearer understanding of what the heart is doing and of the state of the circulation in the various organs than any other measurable incident of the cardiac cycle. (Vaquez and Lajoie (21).) "La pression moyenne est donc l'élément "essentiel qu'il importe de connaître si l'on veut "avoir une idée du régime vasculaire." (Vaquez, Kisthinios and Papaioannou (24).)

RÉSUMÉ OF CLINICAL RESULTS:-

Normal values for My:

One of the striking characteristics of My is its constancy in health even under circumstances which may profoundly modify the extreme pressures. (Vaquez and Lajoie (21); Vaquez, Kisthinios and Papaioannou (24); Lajoie (36).) Naturally we find observers differing slightly in the figures which they accept as normal, but we may take it that in youth 80 - 90 mm. is normal, and that with advancing years there is a slight elevation of My, perhaps up to 110 mm. We find the extreme figures quoted as 70 in youth up to 120 in old age. The latter figure would not be accepted as normal by Lajoie (36) who regards any My of over 110 as definitely pathological.

His figures which may be taken as fairly generally accepted are as follows:-

Age:	10 - 25 years.	25 - 50 years.	Over 50 years.
My :	80 - 90 mm.	90 - 100 mm.	90 - 110 mm.

It has been found by the workers quoted above that My is identical for the sexes, and that such physiological conditions as digestion, menstruation or pregnancy which may modify the extreme pressures cause no change in My.

The level of My has been found to be constant throughout the body in the recumbent position, and it is not even influenced by the Klemperer experiment where one arm is immersed in hot water and the other in cold water and readings taken on each arm.

My and exercise:

In the healthy subject My remains almost constant after exercise. If a rise is found it is never more than 10 mm., and as this is within the range of observational error it is not comparable with the coincident rise in Mx, which may be as much as 50 mm.

In hypertensive subjects without signs of cardiac insufficiency results are comparable with those obtained from healthy subjects; My may rise 10 mm., but returns to normal within two minutes.

In hypertensive subjects with signs of cardiac insufficiency My may rise 20, 30 or 40 mm. remaining up for as much as 5 minutes and only gradually returning to normal.

Gomez and Lajoie (22) conclude that the presence in a hypertensive subject of only slight change in systolic pressure after exercise, accompanied by a considerable rise in the level of My, furnishes not a presumption but a definite sign of cardiac insufficiency.

An increase in My after exercise in a person who has a cardiac affection but no subjective or objective symptoms of decompensation is regarded as a premonitory sign of ensuing cardiac failure. (Lajoie (36).)

<u>My and "hypertension":</u>

It must not be imagined that every case with an elevated Mx will show a corresponding rise of

My, as it is well known that such transient circulatory disturbances as are caused by exercise, emotion &c. may cause a profound change in the extreme pressures. In every old-standing case of hyperpiesis, however, it is found that My is above normal, and that its elevation corresponds with the severity of the condition. Vaquez and Lajoie (21) state that the hypertrophied left ventricle, its dilatation, the decompensation as the disease progresses and the patient's objective and subjective symptoms are always in proportion to the state of the average pressure; these symptoms and clinical signs always being less pronounced in patients with a high systolic pressure but with a slightly elevated or normal average pressure.

My and aortic incompetence:

It is well known that cases of pure aortic incompetence may suffer little inconvenience from their affection, in spite of a gross increase of "pulse pressure". It is found that in such cases, so long as the valvular lesion is compensated, My remains practically normal. (Vaquez, Gley and Gomez (20); Vaquez and Lajoie (21); Vaquez and Kisthinios (26); Gley and Gomez (35); Lajoie (36).)

<u>"Hypertension moyenne solitaire"</u>:

By this term is meant an increase in My without gross or constant alteration of the extreme pressures, and with or without symptoms suggestive of hypertension. The condition is described by Vaquez, Gley and Gomez (20): Vaquez and Lajoie (21): Vaquez and Gomez (27: 30) and Lajoie (36).

They state that it may commence at a relatively early age (e.g. 25-30 yrs.) and lie dormant for many years, giving rise eventually either to a sudden cardiac failure, cerebral haemorrhage &c., or merging gradually into a manifest hypertension.

The existence of this condition would account for those cases where either the symptoms or the clinical and radiological signs suggest a more advanced stage of hyperpiesis than is evidenced by the systolic pressure. In these cases it would be found that the severity of the symptoms and signs would correspond to the extent of elevation of My.

It is partly with a view to recognising this condition that measurement of My is stressed as being of the utmost importance in every Life Assurance examination.

(Lian and Zamfir (38) state that they never come across cases of "hypertension moyenne solitaire" which they regard as clinical curiosities.)

My in Eclampsia and Glaucoma:

Reference is found to these conditions in papers by Vaquez and Lajoie (21): Vaquez & Gomez (27) and Gley and Gomez (35).

Their findings are that in normal pregnancy there is no alteration of My, but that eclampsia causes a marked elevation, the prognosis of the condition depending on the level of My much more than on the readings of the extreme pressures.

The majority of cases of glaucoma are also found to have a My above normal limits.

My and hypotension:

In hypotension My remains normal, and therefore there is no modification in the circulatory regime.

"Voilà pourquoi, conformément à ce que nous "avons toujours pensé, l'hypotension n'est pas une "entité morbide." (Vaquez, Gley and Gomez (20).)

PART 2.

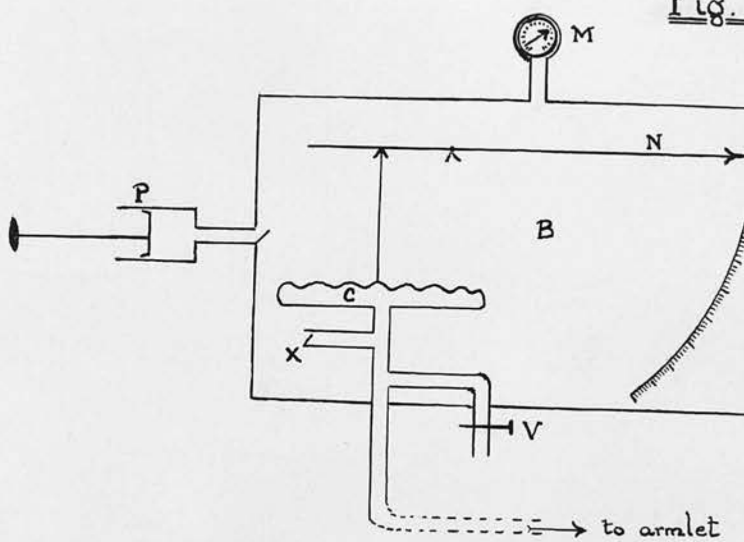
SPECIFICATION OF INSTRUMENT USED:

(Figs. 9 & 10 P.37)

The instrument used throughout this series of observations has been the "Kymomètre" of Vaquez, Gley and Gomez. (29.)

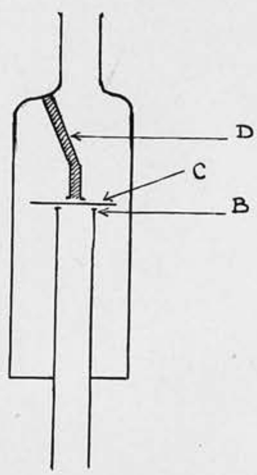
Brought out in 1931 this instrument is a modification of the Pachon oscillometer and has the advantage that the oscillating needle automatically starts from zero on the scale, whereas in Pachon's instrument oscillations might range between any two points on the scale. A further advantage is that the separator has been replaced by a valve allowing air to pass from the box into the capsule and armlet but not in the reverse direction. The function of this valve is to keep the pressure in the box automatically equal to the lowest pressure in the capsule - i.e. diastolic pressure. When the walls of the capsule are in this state of equilibrium the needle registers zero on the scale, or can be brought into that position by means of a small adjusting screw.

Fig. 9.



B : rigid, airtight box.
C : aneroid capsule.
M : aneroid manometer.
N : oscillatory needle.
P : pump.
V : air escape valve.
X : automatic valve.

Fig. 10.



Automatic Valve:

On the seating B formed by the open end of a metal tube rests a valve C formed from a thin, rigid mica membrane. A metal abutment D limits the extent of its opening. The apparatus being vertical the weight of the valve keeps it on its seat, and consequently it only needs a force equal to the weight of the mica to open the valve - this weight of course being negligible.

Mechanism of the instrument:

When air is pumped into the box B with the escape valve closed it passes into the manometer M, and also through the valve X into the armlet - capsule system. At any given pressure, which is registered on the manometer, the wall of the capsule C is in a state of equilibrium, and so is in its most responsive condition to any change in pressure.

When the pressure in the armlet is increased by pulsation of the underlying artery the valve X closes and the wall of the capsule is distorted. This is translated into movement of the oscillating needle and the amplitude of oscillations for that particular counterpressure is read.

On releasing the escape valve V, the valve X opens, and so pressure is reduced throughout the whole system.

Advantages of the "Kymomètre" over the original Pachon instrument:

1. It is more compact, and in every respect a more finished production.
2. There is only one control - viz. the escape valve, whereas on the Pachon instrument, when fitted with a Gallavardin armlet (39) there are three, viz. the escape valve, the separator and the armlet tap. This makes for ease and rapidity of operation.
3. The oscillating needle automatically starts at zero on the scale, so that it is only necessary to read the upper limit of its excursion.

TECHNIQUE OF OSCILLOMETRY:

In taking records with this instrument the rules and precautions common to all bloodpressure estimations should of course be observed.

The patient is made comfortable in a recumbent or reclining posture, and reassured as to the triviality of the procedure. If it is a novel experience to him the armlet should be inflated as a preliminary in order to accustom him to the unusual sensation.

The armlet is applied firmly as high up on the arm as convenient, so as to be well above the bend of the elbow, making sure that the brachial artery is covered by the bag. The arm is supported in a completely relaxed condition in such a way that the armlet is on the same level as the heart.

Air is now rapidly pumped into the instrument until the pressure registered on the manometer is considerably above the expected systolic pressure. With a stethoscope applied to the brachial artery just above the elbow the pressure is reduced 10 mm. at a time. When the counterpressure is still considerably above systolic pressure the needle will commence to oscillate, registering the so-called "supra-maximal oscillations", which are due to the pulse wave impinging on the upper edge of the armlet. (This phenomenon is to a considerable extent abolished by the use of Gallavardin's double armlet.) (39.)

The record of oscillation magnitude against counterpressure is now commenced, and continued for each 10 mm. fall of pressure down to zero.

Mx is taken, as usual, as the counterpressure for which the first audible "click" in the stethoscope occurs. It is advisable that this point should be checked by noting the palpable reappearance of the radial pulse either at this counterpressure or at the pressure for which the next reading is taken, 10 mm. lower. (MacWilliam & Melvin (40); MacWilliam (41: 43); Melvin & Murray (42).)

With further reduction of the counterpressure the oscillations increase until, in normal cases, a maximum is reached somewhere between 110 and 70 mm. - usually about 90 mm.

An abrupt fall in the magnitude of the oscillations now usually occurs, corresponding with a gradual reduction in the intensity of the auscultatory sounds.

Mn is taken as occurring at the commencement of the 4th phase of the Korotkoff sounds as described by MacWilliam & Melvin (40) and Melvin and Murray (42) i.e. the point at which a clear loud thud gives place to a dull, muffled sound.

When the pressure has been reduced to zero the observation is concluded by noting the pulse rate.

OBSERVATIONS ON THE TECHNICAL PROBLEMS OF OSCILLOMETRY:

1. Recording the amplitude of oscillations:

When first using an oscillometer the observer is at once struck by the fact that the amplitude of the oscillations for any counterpressure is far from constant. This, of course, is due to the respiratory fluctuation, and it is essential that the observer adopt at the outset a definite system of recording the oscillations.

Throughout this series it has been the rule to observe the oscillations over at least two respiratory phases for each counterpressure and to record the maximal excursion of the needle as the oscillometric index for that pressure. It appears to be of no consequence whether the largest or the smallest oscillation is recorded, provided that respiration is of normal depth and that the same system of recording is adhered to throughout.

Although this is one of the first problems which confronts the observer it is remarkable that in all the literature consulted it has only been referred to twice.

Eldahl (44) points out that the oscillatory amplitude varies with the respiratory phase, being smaller during inspiration and greatest during the last part of expiration. Roy and Adami (9) in 1890 made the following observation regarding estimation of systolic and diastolic pressures:- "It "need hardly be said that both the maximum and the "minimum pulse-pressures are influenced by the res-"pirations, so that the true maxima and minima of "the arterial pressure are the pressures of the "apices of the pulse waves at the summits of the "respiratory rises, and of the bases of the pulse "waves at the bases of the respiratory oscillations "of the arterial tension."

This dictum has been borne in mind in estimating systolic and diastolic pressures. If, during decompression, at a certain level only one beat out of every five or six is audible in the stethoscope, that point is taken as the level of systolic pressure, although all the beats may not be audible until further decompression has taken place. Similarly with diastolic pressure: if at a certain counterpressure <u>any</u> of the beats come through as a loud sound we must decompress further until <u>all</u> the sounds are dull and muffled before we can say that the true diastolic level has been reached.

It must be remembered that where extra systoles are present care must be taken to disregard these adventitious beats and for recording purposes to attend only to the regular normal beats. In cases of auricular fibrillation it is obviously impossible to place any of the three levels of blood pressure with any degree of accuracy.

2. <u>Variations in tightness of armlet:</u>

In order to obtain comparable results it is naturally desirable that the armlet should be applied as nearly as possible in the same manner in every case. It has been shown however (Fig.13) that the reading for My is not affected by the tightness of the armlet, though the tighter the cuff the greater is the amplitude of the oscillations throughout the scale.

In an article dealing with the interpretation of the oscillogram Blavier (46) states that the amplitude of the oscillometric index varies proportionately with the tightness of the cuff and with the amount of air required for its inflation to any given pressure. He concludes that unless a special armlet is used in which volume of air as well as pressure is known it is fallacious to draw any conclusions from the mere amplitude or form of the oscillations.

3. Rate of decompression:

Exception might be taken to the technique described on account of the fact that deflation is not a gradual process, but is done in steps of 10 mm. at a time, thus giving a possible error of 9 mm. for all the readings. Admittedly there is this margin of error, but it does not appear to be of great importance. It must be remembered that systolic and diastolic pressures are not constant for the individual, and a larger error than this might be produced by some trivial act on the part of the patient such as momentary holding of the breath, slight muscular contraction or some transient emotional disturbance.

So labile are the extreme pressures that we find the belief expressed by Vaquez, Kisthinios and Papaioannou (24) that it would only be by repeating the estimation of the extreme pressures every minute or two for several consecutive days, avoiding all the known causes of "accidental" rise of pressure, that we could be sure of having an exact basis for estimation.

In contrast with this characteristic of the extreme pressures it is found, as has already been mentioned, that the mean pressure - in health at least - is practically a constant for the individual, though in "normal" cases it may lie anywhere between 70 and 110 mm. - a range of 40 mm. Here again, then, the margin of error of 9 mm. is not of great significance unless the reading lie at one extreme of the normal limits.

In assessing the mean pressure it must be borne in mind that the magnitude of the oscillations is readily upset by slight movements or respiratory irregularities on the part of the patient which might easily cause an error of 10 mm. in placing the level of the greatest oscillations.

It is not uncommon to find bloodpressure recorded after this fashion:- 132/93/77. It would appear from what has just been said that the patient's vascular condition could be assessed equally well if his pressures were recorded as

130/90/80 - an approximation admittedly, but one which gets us as near to the truth as possible.

4. "Plateau" curve:

Where for two or more consecutive counterpressures the same maximal oscillation is obtained the curve when plotted assumes the form of a plateau, and no exact figure for the mean pressure can be given. This was a serious difficulty when a narrow armlet was in vogue, but is seldom encountered with an armlet of about 14 cm. (Vaquez & Lajoie (21): Vaquez, Gley & Gomez (31).)

Occasionally two counterpressures are found to give the same oscillatory reading, but in such a case it may be presumed that the maximal oscillation would occur somewhere between these two counterpressures - again an error of less than 10 mm.

On the few occasions where a plateau has been found consisting of more than two points a further estimation after tightening the armlet has always overcome the difficulty. By tightening the armlet the amplitude of the oscillations is increased, so allowing of a more exact differentiation of their amplitude.

5. <u>Plotting the oscillometric curve:</u>

The observations having been completed, a permanent record of the case is made by plotting a curve in which the ordinates represent oscillatory magnitude and the abscissae counterpressures in mm. of mercury. Fig. 10A is a typical normal curve and shows the method which has been adopted of keeping the records of this series. The curve obtained after exercise is superimposed on the original curve, so that any variation may be noted at a glance.

6. <u>Interpretation of the oscillometric curve:</u>

Reading from the right of the curve it is seen that the oscillations are at first of small amplitude, corresponding to the phase of "supra-maximal" oscillations. These excursions of the needle are caused not by blood passing under the armlet but by the impingement of the pulse wave on the proximal edge of the armlet. By using a Gallavardin armlet (39) this phase is practically done away with, as the supra-maximal pulsations impinge on a portion of the armlet which is cut off from the remainder of the instrument, so that the first oscillation of any

1/12/33 Robert R—. aet. 23 <u>Normal</u>. (Student)

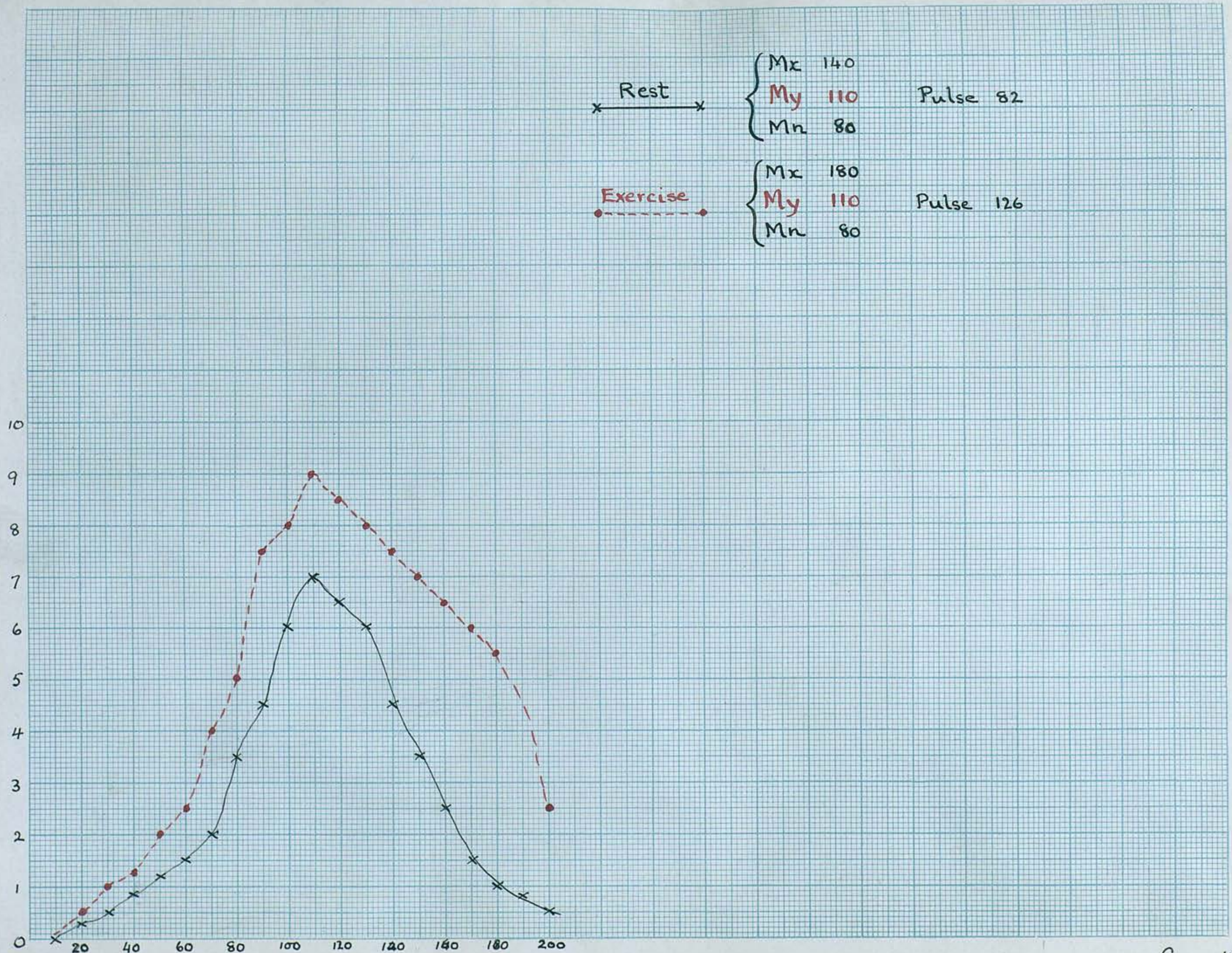


Fig. 10^A

Case 45

appreciable magnitude is produced by an actual flow of blood under the armlet, and therefore occurs at the level of systolic pressure.

The first point to be decided is whether on the ordinary type of curve there is any constant characteristic feature which indicates the level of Mx.

It has been stated, and apparently accepted without comment by the majority of observers, that on the oscillometric curve there is a definite angle where supra-maximal oscillations are replaced by true oscillations, and that this angle therefore represents the level of Mx. Doubt has been cast on this statement by Gallavardin (39) who states that in recent years it has been the custom to place systolic pressure below the point where supra-maximal oscillations merge into increasing oscillations to a point indefinitely fixed in the increasing range.

In the present series it was found that although angles were frequently present on the curves they did not occur with any regularity or uniformity. In only 5 cases did Mx as estimated by the auditory method coincide with the angle on the curve.

It can therefore be concluded that there is no constant point on an oscillometric curve which can be taken as an index of systolic level.

Following the curve from right to left we find that oscillations increase rapidly, but not uniformly to the point of maximal oscillation, or Maximal Oscillometric Phase. (M.O.P.) - a term recommended by Miller and Chester (37). The significance of this point will not be touched on here as it is dealt with in some detail at a later stage.

The summit of the curve having been passed it is usual to find a rapid fall away in the amplitude of the oscillations, giving a steep descent to the curve, which however tends to flatten out for the lowest counterpressures - though even this feature is not constant. It is remarkable that what may be taken as a typical oscillometric curve bears a close resemblance to a normal sphygmogram.

It should be stated however that no two individuals yield a curve of exactly the same shape, though there is a remarkable constancy in the shape of the curve for the same individual, even for records taken at wide intervals of time and under varying conditions. (Fig.12).

It remains to be seen whether there is any characteristic feature in the curve which indicates the level of Mn.

Pachon (13) in 1909 stated that the pressure indicated by the manometer at the point of maximal oscillation is the minimal pressure of blood in the artery. He however was working on a "schéma "circulatoire", and his results were not confirmed clinically.

In later experiments conducted with Fabre (18) in 1921 his views were changed and he found that on an oscillogram Mn occurs "au début d'une zone "de pulsations d'un caractère particulier" which present (1) "une pente à peine existente ou seule- "ment faible; (2) une amplitude nettement moindre "et une faible différenciation", and he affirms that a similar point is to be found on an oscillometric curve.

In 1931 Gley and Gomez (28) by animal experiments, using a "manomètre à minima" confirmed these results, though later Gomez (25) in a clinical study was also compelled to modify his views. He found that the oscillometric curves obtained were of three types:-

(1) where the change of slope is evident and Mn can be exactly placed:

(2) where the curve is too gradual or in steps so that any attempt to locate Mn would be fallacious:

(3) where there is a steady fall from My to zero.

Out of 300 cases he found type 1 curve in 27% and types 2 and 3 in 73%, concluding that in the majority of cases Mn cannot be read with any degree of certainty. He explains the discrepancy in the results of his two experiments by the fact that in the original animal experiments segments of vein, and not artery, were used.

In the present series of 252 cases type 1 curve was found in 17% and types 2 and 3 curves in 83% of cases. In only 13 cases (i.e. 5.2%) did Mn as estimated by the auditory method coincide with the angle on the curve; in the great majority of cases it lay at least 20 mm. higher.

These results are in accordance with Gomez' (25) conclusions when he states that in the great majority of cases any attempt to read the level of diastolic pressure from an oscillometric curve is fallacious.

As in the case of Mx, so with the estimation of Mn we have to rely on the older and more generally accepted auditory method.

In the words of MacWilliam and Melvin (40) "In the cases where the Erlanger or the Pachon "apparatus gives dubious or unreadable indications, "the auditory method has always in our experience "given a definite verdict. It is greatly superior "to the palpatory and sphygmographic methods that "have been proposed. We are satisfied that all the "evidence as to diastolic pressure gained by the "other methods, and much more also, can be better "obtained by the quick and simple auditory method."

Lastly, in the interpretation of the oscillometric curve, are there any features by which a "normal" curve can be distinguished from a pathological curve, or does any pathological state yield a curve peculiar to that condition?

It has been stated that the "normal" curve bears a close resemblance in shape to a normal sphygmogram, but we do find curves from perfectly normal subjects where this resemblance is not obvious, and moreover curves from pathological subjects frequently assume this "normal" shape.

Obviously in hyperpietics there is bound to be a widening of the curve, and in cases where Mx, My and Mn are all elevated there will be a complete shift to the right.

Apart from these changes no characteristic features have been found which, from a study of the curve, would enable the subject to be classed as normal or pathological, or to be suffering from cardio-vascular disease or any other specific pathological condition.

It would thus appear that the oscillometric curve is unnecessary, all that requires to be noted being the three levels Mx, My and Mn.

______...______

RESULTS OF PRESENT INVESTIGATION.

In the present series records have been taken from 252 individuals, many of whom were examined repeatedly, either with a view to comparing their pressures under varying circumstances, or in order to keep a record of their pressures during the course of an illness or pregnancy.

CHOICE OF CASES:

Many of the cases were chosen because they were believed to be normal subjects, and a scale of normal pressures has been drawn up from their records. Others were chosen in order to compile a series of cases of some particular type of illness.

The remainder are records taken in a routine fashion in general practice in cases where there was some indication that an estimation of the blood pressure might be of assistance in diagnosis, prognosis or treatment.

CLASSIFICATION OF RECORDS:

1. NORMAL:

A. RECORDS FROM NORMAL CASES AT REST:

This group consists of records from male and female "normal" cases in three age groups - viz. 10 - 25 years, 25 - 50 and over 50 years.

B. RECORDS FROM ARM AND ANKLE:

In a group of 20 normal cases readings were taken with the cuff applied firstly in the usual situation over the brachial artery, and secondly at the ankle, in order to ascertain whether the site of application of the cuff influenced the reading obtained for My.

C. NORMAL PREGNANCY:

Records were taken from 24 patients at varying stages of normal pregnancy. Monthly records were kept in one case throughout a normal pregnancy.

D. RECORDS FROM THE SAME PATIENT AT DIFFERENT TIMES.

A demonstration of the fact that every person appears to have a characteristic oscillometric curve, although readings may be taken at wide intervals of time and under varying conditions.

E. VARIATION IN THE TIGHTNESS OF APPLICATION OF THE ARMLET:

Evidence is produced that variation in the tightness of application of the armlet affects the amplitude of the oscillations throughout, but does not affect the reading obtained for My.

F. SIGNIFICANCE OF OSCILLATORY AMPLITUDE:

From a study of (a) the oscillatory increase after exercise and (b) those cases showing an oscillatory amplitude of over 15 scale divisions, an attempt is made to assess the significance of oscillatory amplitude.

G. EXERCISE IN HEALTHY SUBJECTS:

The response of My to exercise was recorded in 22 normal young male adults.

2. PATHOLOGICAL:

A. EXERCISE IN HYPERTENSIVES:

11 Patients suffering from varying degrees of

hypertension were submitted to exercise tests to compare the response of My to that obtained in normal subjects.

B. <u>ARTERIOSCLEROSIS:</u>

14 Patients were included in this group, all having certain signs and symptoms in common, and all having a Mx of 150 or over.

C. <u>AORTIC INCOMPETENCE:</u>

Readings were taken from 7 patients suffering from this valvular lesion in order to verify the statement of various observers that so long as the valvular lesion is compensated My remains practically normal.

D. <u>CHRONIC INTERSTITIAL NEPHRITIS:</u>

Records were kept in 11 cases of this disease as being one particularly liable to cause cardiovascular disturbance.

E. <u>RIGHT-SIDED VALVULAR LESIONS AND CHRONIC MYOCARDIAL DAMAGE:</u>

The level of My was recorded in 14 patients who constituted this group.

F. <u>MALIGNANT DISEASE AND CHRONIC DISEASES CAUSING PROLONGED CONFINEMENT TO BED:</u>

The influence of these factors on the level of

My was recorded in a group of 19 patients in the Longmore Hospital.

G. <u>LOBAR PNEUMONIA:</u>

Records were kept in 3 cases of lobar pneumonia throughout the illness; of these cases one proved fatal.

H. <u>SPINAL ANAESTHESIA:</u>

This condition was chosen as being one liable to have a disturbing effect on blood-pressure, and records were kept in 19 cases in which the level of anaesthesia was at least up to the 11th dorsal segment.

I. <u>ANALYSIS OF CASES (1) WITH ELEVATION OF My, AND (2) WITH ELEVATION OF Mx BUT NORMAL My.</u>

J. <u>MYOCARDIAL INFARCTION:</u>

Records are presented from one case of myocardial infarction as being a case of especial interest.

Finally a summary of the results obtained is given, and an attempt is made in the light of these findings to assess the significance of My and to indicate the importance of its being recorded in every blood-pressure estimation.

1a. <u>NORMAL SCALE</u>: (Tables 1 - 6)

In selecting "normal" cases only those were included who at the time of examination felt, and on examination appeared to be in good health; who had no cardiac lesion, and who gave no history of any illness which might cause a cardio-vascular disturbance. In order to accord with the general trend of opinion no case showing a resting Mx above 150 was regarded as normal.

An arbitrary age classification was made following that employed by Lajoie (36) viz. 10 - 25 years; between 25 and 50, and over 50 years.

In the first two age groups there is no difficulty in obtaining "normal" records, but in the third group - i.e. patients over 50 years, very few records were obtained.

This is accounted for firstly by the fact that neither in general practice nor in hospital do we encounter many patients who according to the definition adopted could be classed as normal; secondly a considerable number of records taken from elderly people who appeared to be in good health and gave a satisfactory history could not be included on account of the fact that their resting Mx was above 150. This bears out the conclusion of Thomson and

Todd (45) who state that there is no "normal" blood pressure (i.e. systolic and diastolic) in old age.

Male:

In the first age group the average My for the 31 cases was 90; the range being from 70 - 110. In the second group the average of 29 cases was 89, with a range of 70 - 100, while in the third group of 8 cases the average My was 98 with a range of 80 - 110.

Female:

The first age group contained 23 cases showing an average My of 85, the range being from 80 - 90. The second group of 12 cases had an average My of 88 with a range of 80 - 100, while in the third group of 9 cases the average My was 96 with a range of 80 - 110.

These results correspond closely with the figures given by Lajoie, the only difference being that in this series the readings for males are in each age group very slightly higher than those for females. In both sexes there is seen to be a slight increase in My with advancing years, but the extremes of "normal" values correspond to Lajoie's figures - viz. 70 - 110.

Table I.

NORMAL (MALE): AGE I0 - 25 years:

Case	Age	Mx	My	Mn	Case	Age	Mx	My	Mn
24	22	I30	90	70	I03	I7	I30	80	70
25	20	I30	I00	70	I04	I7	I30	90	80
26	2I	I30	90	80	I54	I4	I00	80	60
27	I9	I30	I00	70	I58	I5	I30	I00	60
28	I9	I30	90	70	232	I8	I30	80	60
32	I8	I20	90	60	237	I8	90	70	60
35	22	I20	90	70	238	I9	I40	90	70
40a	22	I40	I00	60	243	20	I30	80	60
4I	2I	I30	80	60	246	I9	II0	90	60
42	2I	I40	I00	60					
44	24	I30	I00	70					
45	23	I40	II0	80					
52	I7	I40	I00	70					
53	I9	I00	90	70					
54	I7	I20	90	70					
56	2I	I30	90	60					
57	2I	I30	I00	70					
67	20	I30	90	70					
7I	24	I30	90	60					
72	I9	I00	80	50					
73	20	I40	I00	70					
78	I7	II0	70	50					

AVERAGE My: 90

(Range: 70 - II0)

NORMAL (MALE): AGE 25 - 50 years:

Table 2.

Case	Age	Mx	My	Mn
7	29	I30	I00	70
8	30	I40	I00	80
33	34	I00	90	60
37	3I	I20	90	70
43	26	II0	80	80
55	25	I30	90	70
58	49	I30	I00	70
65	39	I50	I00	80
68	26	I50	I00	80
70	29	I30	I00	70
I08	32	I20	90	80
I55	47	I2o	90	70
I59	33	I20	90	70
I85	29	I50	I00	70
I92	39	I20	80	60
202	32	I20	80	60
204	50	I40	90	60
206	38	I50	I00	70
209	42	I30	90	70
2I9	26	I00	70	50
228	30	I00	70	60
229	27	I40	90	80

Case	Age	Mx	My	Mn
230	40	I20	80	70
23I	28	I50	I00	70
233	30	I20	80	60
234	32	I30	80	70
236	33	II0	80	60
240	32	I40	I00	80
24I	32	I30	90	80
244	49	I30	90	70
247	26	II0	90	60
248	3I	II0	80	60

AVERAGE My: 90

(Range: 70 - II0)

NORMAL (FEMALE): AGE IO - 25 years:

Table 4.

Case	Age	Mx	My	Mn	Case	Age	Mx	My	Mn
84	I8	I30	80	60	2I3	II	I20	90	70
I63	22	II0	90	60	225	23	I20	80	60
I64	I4	I20	80	60					
I68	23	I20	90	70					
I74	22	II0	80	60					
I77	I5	I30	90	70					
I78	23	I00	80	50					
I79	22	I40	90	70					
I82	23	II0	80	60					
I83	I8	I30	80	70					
I84	22	I30	90	70					
I87	23	I40	90	70					
I89	20	I20	80	60					
I90	24	I30	90	70					
I95	I8	I20	90	70					
I96	2I	I30	90	50					
I97	I5	I00	80	60					
I98	I8	II0	90	70					
I99	I9	I20	90	70					
200	I8	I00	80	70					
20I	I8	I30	80	60					

AVERAGE My: 85

(Range: 80 - 90)

NORMAL (FEMALE): AGE 25 - 50 years:

Table 5.

Case	Age	Mx	My	Mn
II	36	II0	80	60
46	33	I20	I00	70
I33	30	I20	90	70
I42	32	I30	90	70
I76	28	II0	80	70
I9I	28	II0	80	70
I93	26	I20	90	60
205	45	I50	I00	80
208	49	I50	90	70
2I0	25	I20	80	60
2I2	29	I40	I00	70
2I5	32	I30	90	60

AVERAGE My: 89

(Range: 80 - I00)

NORMAL (FEMALE): AGE over 50 years:

Case	Age	Mx	My	Mn
63	68	I40	I00	70
85	50	I00	80	60
96	66	I50	II0	80
I40	60	I30	90	80
I70	62	I30	I00	70
I75	52	I40	90	70
I86	90	I50	II0	90
2I7	66	I40	90	70

AVERAGE My: 96

(Range: 80 - II0)

Records from this series shown in tabular form:-

	10 - 25 years	25 - 50 years	Over 50 years
My - MALE	90	89	98
My - FEMALE	85	88	96

1b. RECORDS FROM ARM AND ANKLE: (Table 7)

In a group of 20 young male adults records were taken at rest from arm and ankle, the patient lying on a couch. Owing to the weakness of the sounds at the ankle in several cases neither Mx nor Mn could be placed accurately.

In 14 cases there was a change in the level of Mx within the limits of +20 mm. to -20 mm.

Only 6 cases showed a change in Mn, and in no case did such change exceed 10 mm. either higher or lower.

My remained unchanged in 15 cases: the remainder all showed a change of only 10 mm. - practically a negligible alteration.

From these results we may conclude that in the recumbent position the site of application of the cuff has no appreciable effect upon the reading obtained for My.

RECORDS from ARM and ANKLE:

Table 7.

Case	Age	Mx Arm	Mx Ankle	Change in Mx	My Arm	My Ankle	Change in My	Mn Arm	Mn Ankle	Change in Mn
24	22	I30	I40	+I0	90	90	-	70	?	?
25	20	I30	I30	-	I00	I00	-	70	70	-
26	2I	I30	I50	+20	90	90	-	80	70	-I0
27	I9	I30	I30	-	I00	I00	-	70	?	?
28	I9	I30	?	?	90	80	-I0	70	?	?
32	I8	I20	I40	+20	90	90	-	60	?	?
33	34	I00	II0	+I0	90	90	-	60	60	-
35	22	I20	I30	+I0	90	90	-	70	70	-
40a	22	I40	I30	-I0	I00	I00	-	60	70	+I0
4I	2I	I30	I20	-I0	80	80	-	60	60	-
42	2I	I40	I20	-20	I00	I00	-	80	70	-I0
43	26	II0	I00	-I0	80	80	-	70	60	-I0
44	24	I30	I20	-I0	I00	I00	-	70	80	+I0
45	23	I40	I40	-	II0	I00	-I0	80	80	-
52	I7	I40	I20	-20	I00	90	-I0	70	70	-
53	I9	I00	?	?	90	I00	+I0	70	?	?
54	I7	I20	?	?	90	90	-	70	?	?
55	25	I30	I20	-I0	90	90	-	70	60	-I0
56	2I	I30	I20	-I0	90	80	-I0	60	60	-
57	2I	I30	I20	-I0	I00	I00	-	70	70	-

1c. NORMAL PREGNANCY: (Table 8)

For the purposes of these records "normal "pregnancy" was taken as meaning that the patient showed no albuminuria, had no oedema, showed no gross elevation of systolic pressure and had no complications in the way of hyperemesis &c. Three cases, viz. 23, 49 and 60 had Mx of 150, 150 and 160 mm. respectively, but as they were all very nervous subjects and presented no other abnormality they were regarded as normal cases.

The average My for the 24 cases was 88 mm. - a perfectly normal figure. The range of My was between 70 and 110 mm. - again within normal limits.

The records in this group were taken at any stage of the pregnancy from the second to the ninth month.

Monthly records were kept of one patient throughout her pregnancy - (Fig. 11). It was found that My remained constant and that the shape of the curve remained practically unchanged throughout.

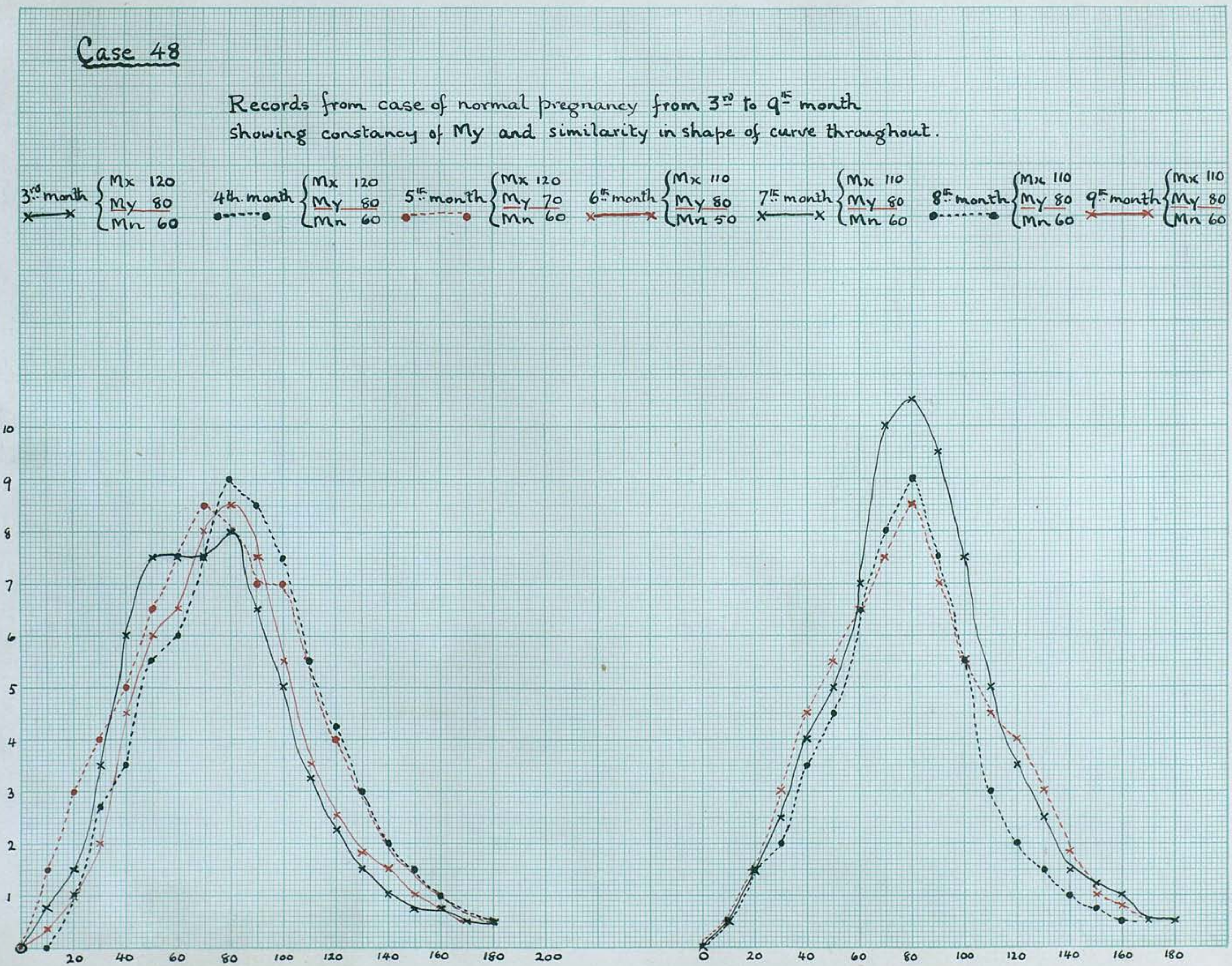
From these records it would appear that a normal pregnancy does not affect the level of My.

NORMAL PREGNANCY: Table 8.

Case	Age	Mx	My	Mn
2	29	I40	90	70
I6	29	I40	90	80
I7	30	I40	I00	80
23	30	I50	I00	90
38	30	I40	II0	70
48	35	I20	80	60
49	33	I50	I00	80
60	24	I60	II0	90
64	32	II0	90	60
66	36	I20	70	60
87	34	I20	90	70
9I	28	I20	90	60
I02	23	I20	90	70
II2	24	II0	80	60
II4	2I	I30	90	70
I32	36	I30	90	70
I43	23	II0	80	70
I46	26	I20	70	60
I47	22	I40	80	70
I48	32	I40	90	80
I69	28	I25	90	60
I72	36	I20	80	70
I73	25	I20	70	60
22I	20	I20	80	50

AVERAGE My: 88

(Range: 70 - II0)



Case 48
Records from case of normal pregnancy from 3rd to 9th month
showing constancy of My and similarity in shape of curve throughout.
3rd month {Mx 120 My 80 Mn 60
4th month {Mx 120 My 80 Mn 60
5th month {Mx 120 My 70 Mn 60
6th month {Mx 110 My 80 Mn 50
7th month {Mx 110 My 80 Mn 60
8th month {Mx 110 My 80 Mn 60
9th month {Mx 110 My 80 Mn 60
10
9
8
7
6
5
4
3
2
1
20
40
60
80
100
120
140
160
180
200
0
20
40
60
80
100
120
140
160
180

Fig. 11.

1d. RECORDS FROM THE SAME PATIENT AT DIFFERENT TIMES: (Fig. 12)

In the course of this investigation many patients were repeatedly examined and it became obvious that every individual has a characteristic oscillometric curve to which he remains true even if records are taken at wide intervals of time or under varying conditions.

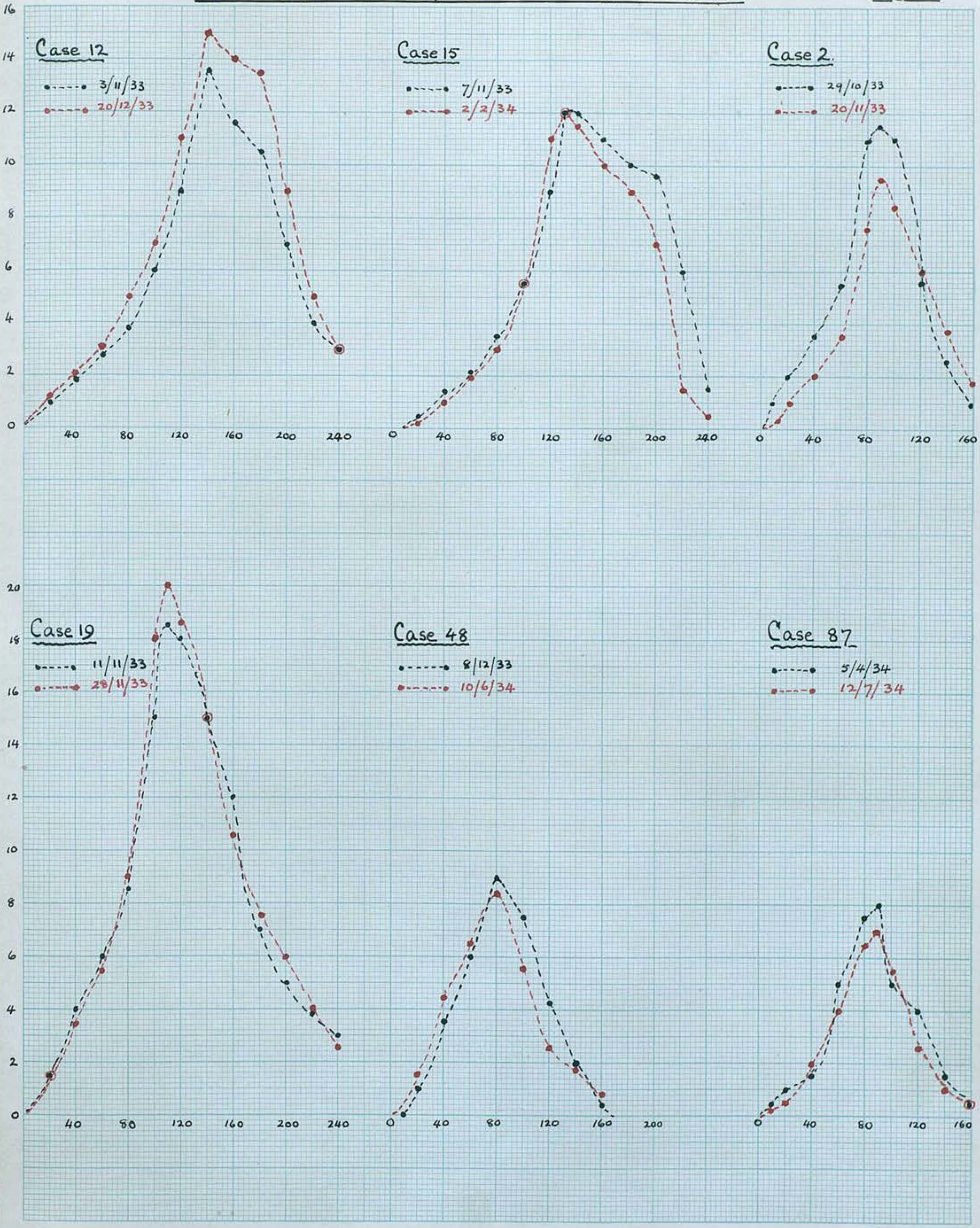
Fig. 12 shows curves from 6 patients taken at intervals ranging from 3 weeks to 6 months. The constancy in the type and shape of curve is obvious.

The same feature is illustrated in Fig. 14, (exercise in healthy subjects) where after exercise the shape of the curve remains unaltered, though the oscillatory amplitude is increased throughout.

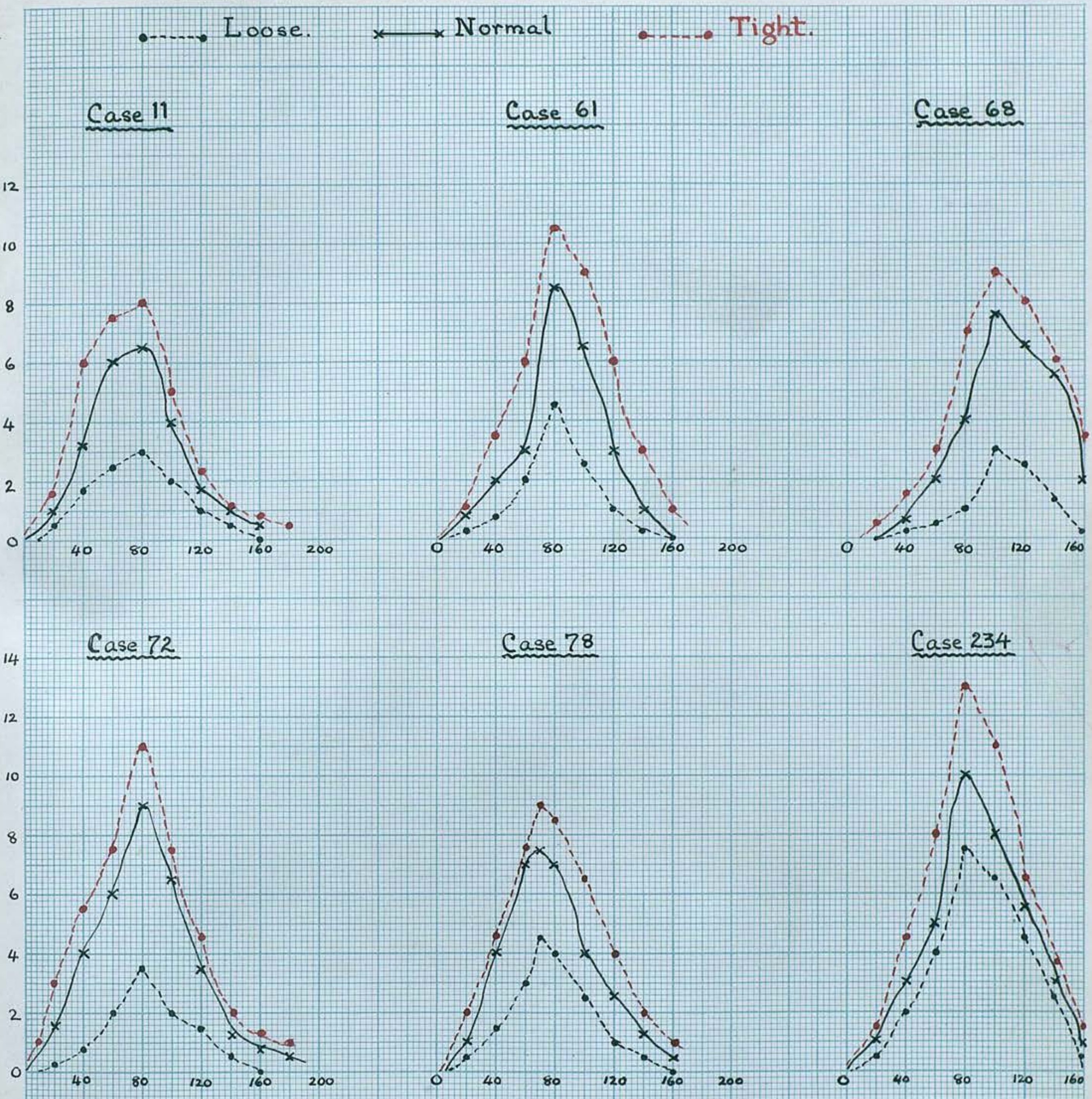
No great significance is attached to this feature as so far no curves characteristic of any particular condition have been found, and the conclusion has been arrived at that plotting of the oscillometric curve is unnecessary - all that requires to be known being the three levels Mx, My and Mn.

Records from same patients at different times

Fig. 12.



Varying tightness of Armlet. Fig. 13.



Increasing tightness of armlet:-

1. Increases amplitude of oscillations throughout.

2. Does not affect level of My.

to differentiate between them with any degree of accuracy, particularly if there is a well marked respiratory fluctuation, and thus the placing of My becomes uncertain on account of the production of a plateau curve. In such cases it is always advisable to repeat the readings after a tighter application of the armlet, when the greater amplitude of the oscillations produced allows of their more exact differentiation and an accurate location of My.

CONCLUSION:

Increasing the tightness of application of the armlet:-

(1) Increases the amplitude of the oscillations throughout.

(2) Does not affect the reading obtained for My.

1f. SIGNIFICANCE OF OSCILLATORY AMPLITUDE: (Tables 9, 10 & 11.)

It has been shown that oscillatory amplitude increases throughout with increased tightness of the armlet. Blavier (46) states that the amplitude of the oscillometric index varies proportionately with the tightness of the cuff and with the amount of air required for its inflation.

It will be shown (Section lg.) that after exercise there is an increase in oscillatory amplitude throughout the scale.

It will be seen from Table 9 that the increase in oscillatory amplitude bears no relationship to increase in Mx or P.P. or to alteration in My or Mn.

In these cases it is assumed to be due to the fact that exercise calls for increased cardiac output with an increase in stroke-volume which will cause greater distension of the artery with each pulse wave.

Table 10 consists of those cases showing unusually large oscillations - only oscillations of more than 15 scale divisions were considered in this group. Here again we find that oscillatory amplitude bears no direct proportion to the level of Mx, Mn, My or P.P. It is seen that of the 16 cases 15 are male, and that the majority of them were suffering from diseases known to affect the state of the arterial walls. Clinically none of them suffered from gross thickening or hardening of peripheral arteries, and the pulse in all cases was typically of a "bounding" nature - quite distinct from the tense, hard pulse of the advanced arterio-sclerotic.

Table 11 shows the cases of arteriosclerosis, chronic interstitial nephritis and of aortic incompetence in the series, and from this it is obvious that these diseases of themselves do not give rise to an increase of the O.I.

It is suggested that in the early stages of arteriosclerosis when the elasticity of the larger vessel walls is not yet impaired, the rising blood pressure results in increasing distension of the peripheral arteries with the higher pressure wave, giving rise to an increase of the O.I. As the vessel wall becomes harder and loses its elasticity so are the pulse waves damped down, with the result that in the advanced case of arteriosclerosis in spite of high pressures we find a relatively immobile arterial wall and a correspondingly low O.I.

Results:

The O.I. was found to be higher than normal:-

1) In direct proportion with the tightness of application of the armlet.
2) Where there was increase in the stroke-volume of the cardiac output, as for instance after exercise.
3) Where high blood pressure was acting on an elastic arterial wall, as in the early stages of arterio-sclerosis.

Table 9 .

INCREASE in OSCILLOMETRIC INDEX RESULTING from EXERCISE:

Case	Increase in Mx	Alteration in Mn	Alteration in My	Increase in P.P.	Increase in O.I.
24	20	+IO	-	IO	I½
25	IO	-IO	-	20	3
26	IO	-IO	-	20	I
27	IO	-IO	-	20	2
28	IO	-IO	-	20	2½
32	IO	+IO	-	-	½
33	20	+IO	IO	IO	3
35	20	-IO	-	30	3½
40a	60	-	-	60	6½
4I	50	-	IO	50	4
42	30	-IO	IO	40	5
43	20	-	IO	20	3
44	30	+IO	-	20	3½
45	40	-	-	40	2
52	40	-IO	-	50	5
53	40	-	IO	40	2½
54	30	-IO	-	40	½
55	20	-IO	IO	30	3
56	40	-IO	-	50	3½
57	30	-	-	30	3
78	30	+IO	-	20	3½

Table IO.

CASES with OSCILLOMETRIC INDEX OVER I5 SCALE DIVISIONS:

Case	Sex	O.I.	Mx	My	Mn	P.P.	Disease
I	M	I9	2IO	I20	IOO	IIO	Arteriosclerosis.
6	M	I7	200	80	70	I30	Aortic incompetence
I2	M	I5	2IO	I40	I20	90	Arteriosclerosis.
I9	M	I8	I70	IIO	90	80	Arteriosclerosis.
40	M	2I	220	I60	I40	80	Chr. inter. neph.
5I	M	I7	I90	I40	I20	70	Chr. inter. neph.
62	M	I5	220	I30	IOO	I20	Chr. inter. neph.
8I	M	I9	I40	80	60	80	Neurasthenia.
IIO	M	I9	I70	IOO	80	90	Arteriosclerosis.
III	M	I5	I20	90	880	40	Myocard. infarct.
II3	M	I9	I30	80	70	60	Chr. inter. neph.
I38	M	I7½	I70	IOO	80	90	Arteriosclerosis.
I44	M	I6	200	I30	IOO	IOO	Aortic incompetence
I85	M	I6	I50	IOO	70	80	Normal.
223	F	I5½	I40	70	20	I20	Aortic incompetence
24I	M	I8½	I30	90	80	50	Normal.

Table II.

O.I. in ARTERIOSCLEROSIS, CHR. INTER. NEPH. and AORTIC INCOMPETENCE:

Case	Mx	My	P.P.	O.I.	
1	210	140	110	15	
12	210	140	90	13½	
19	170	120	70	18	
21	170	100	80	13	
76	180	120	80	13½	
82	160	120	60	9	
88	210	120	110	14	ARTERIOSCLEROSIS
105	170	140	80	13	
110	170	100	90	19	
138	170	100	90	17½	
139	160	80	80	12	
181	210	120	110	12½	
203	210	160	80	6½	
218	180	110	100	13½	
9	220	120	120	8½	
115	230	130	130	13	CHRONIC INTERSTITIAL
40	220	160	80	21	NEPHRITIS
51	190	140	70	17	
62	220	130	120	15	

(over

Table II (contd.)

Case	Mx	My	P.P.	O.I.	
93	220	170	80	11	CHRONIC INTERSTITIAL NEPHRITIS
101	170	120	90	13	
113	130	80	60	19	
123	180	120	70	8½	
157	210	180	50	6½	
171	240	180	100	11	
6	200	80	130	17	AORTIC INCOMPETENCE
18	130	70	70	8½	
39	160	90	100	12½	
59	160	90	90	10½	
144	200	120	100	16	
162	190	110	100	13	
223	160	70	140	14½	

1g. EXERCISE IN HEALTHY SUBJECTS: (Table 12 & Fig.14)

In a group of 22 healthy young male adults records were taken at rest and again after exercise, the armlet having been left in place. The exercise consisted of marking time "at the double" with knees up for 50 steps - sufficient to cause in every case definite increase in pulse and respiration rate. The results are shown in Table 12 and some typical curves reproduced in Fig. 14.

From the table it will be seen that in every case there was a rise in Mx ranging from 10 - 60 mm. (average 27 mm.)

My was unchanged in 16 cases, and in the remainder the change was never greater than 10 mm. - in two cases 10 mm. lower and in four cases 10 mm. higher.

Mn showed little alteration, and in no case was the change more than 20 mm.

On examining the curves it was found that the oscillatory amplitude was increased throughout after exercise, so that in 21 cases the record when plotted lay in a curve completely outside the curve representing readings at rest. In one case a single reading on the second curve lay within the first curve.

EXERCISE in HEALTHY SUBJECTS:

Table I2.

Case	Age	Mx		Change in Mx	My		Change in My	Mn		Change in Mn
		Rest	Exer.		Rest	Exer.		Rest	Exer.	
24	22	I30	I50	+20	90	90	-	70	80	+I0
25	20	I30	I40	+I0	I00	I00	-	70	60	-I0
26	2I	I30	I40	+I0	90	90	-	80	70	-I0
27	I9	I30	I40	+I0	I00	I00	-	70	60	-I0
28	I9	I30	I40	+I0	90	90	-	70	60	-I0
32	I8	I20	I30	+I0	90	90	--	60	70	+I0
33	34	I00	I20	+20	90	80	-I0	60	70	+I0
35	22	I20	I40	+20	90	90	-	70	60	-I0
40a	22	I40	200	+60	I00	I00	-	60	60	-
4I	2I	I30	I80	+50	80	90	+I0	60	60	-
42	2I	I40	I70	+30	I00	90	-I0	80	70	-I0
43	26	II0	I30	+20	80	90	+I0	70	70	-
44	24	I30	I60	+30	I00	I00	-	70	80	+I0
45	23	I40	I80	+40	II0	II0	-	80	80	-
52	I7	I40	I80	+40	I00	I00	-	70	60	-I0
53	I9	I00	I40	+40	90	I00	+I0	70	70	-
54	I7	I20	I50	+30	90	90	-	70	60	-I0
55	25	I30	I50	+20	90	I00	+I0	70	60	-I0
56	2I	I30	I70	+40	90	90	-	60	50	-I0
57	2I	I30	I60	+30	I00	I00	-	70	70	-
78	I7	II0	I40	+30	70	70	-	50	60	+I0
I85	29	I40	I70	+30	I00	I00	-	70	80	+I0

Fig. 14.

Effect of exercise on healthy subjects.

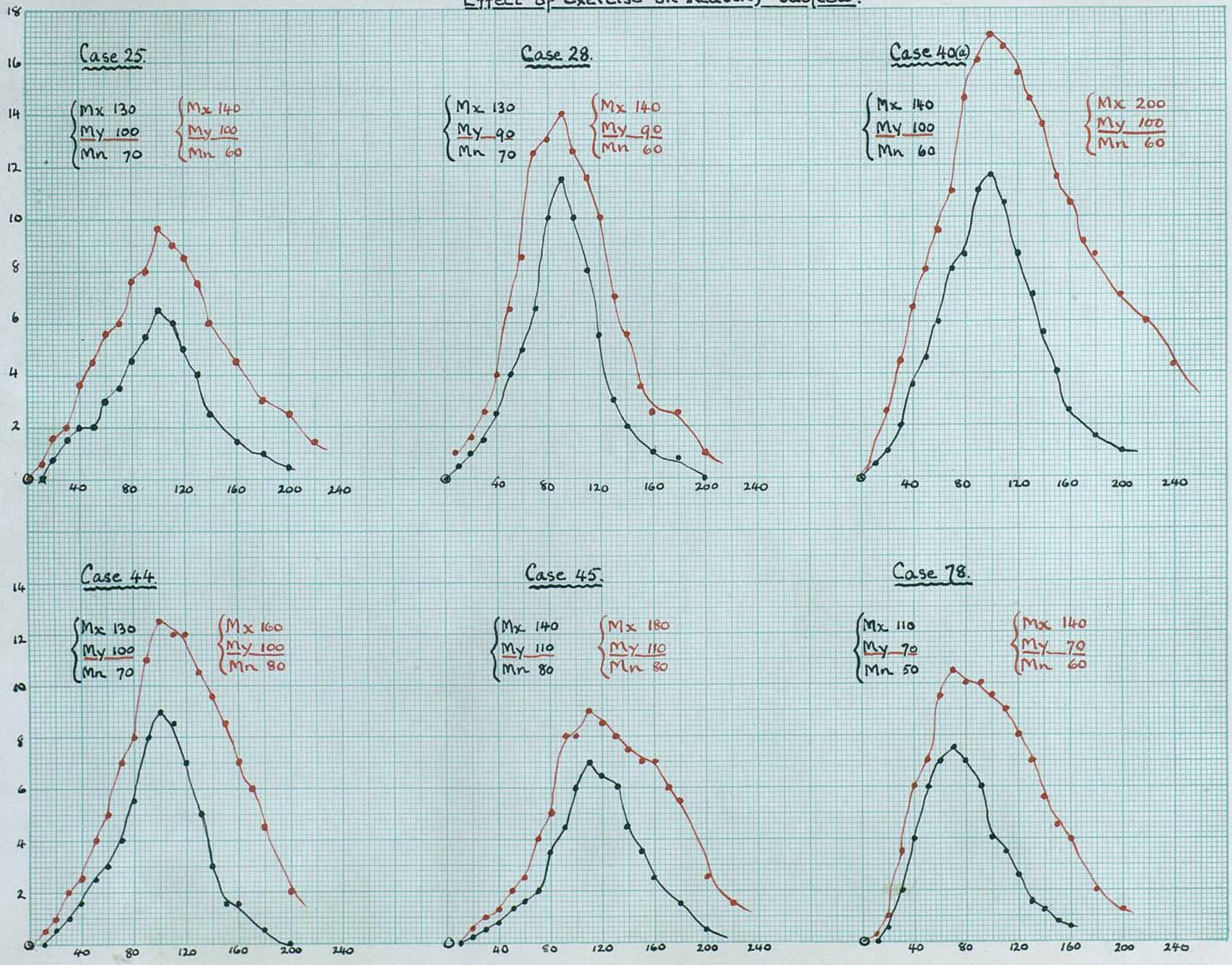


Fig. I4.

The conclusion to be drawn from this group is that in the healthy subject :-

(1) Exercise produces an increase of oscillatory amplitude throughout.

(2) My rising remains unaltered - the change of 10 mm. in a few readings being on the borderline of observational error.

2a. <u>EXERCISE IN HYPERTENSIVES:</u> (Table 13 & Fig. 15)

Exercise tests in these subjects were naturally more difficult than in normal cases firstly on account of the fact that many of the cases were unfit for any form of exercise, and secondly because many of them were unwilling to cooperate. Further, owing to the varied tolerance of the cases the exercise could not be standardised; patients in bed were asked to sit up and lie down as fast as possible until definite dyspnoea was experienced; ambulant cases were given either the "high stepping" or "toes touching" exercise, again until dyspnoea or some other warning symptom was experienced.

11 Patients with Mx above 150 mm. were submitted to these tests (Table 13) - the age range being from 27 - 79 years, and the resting Mx ranging from 160 - 230 mm.

In every case Mx rose from 10 - 40 mm. after exercise - a change similar to that occurring in the healthy subject. My rose in 10 cases and remained unchanged in one. The response of Mn was variable, rising in 4 cases, remaining unaltered in 5 cases and falling in 2 cases. In no case was the increase or decrease more than 20 mm.

Of the 11 cases 5 have died, 5 are alive and one is untraced. Of the cases who are alive 4 showed an My rise of only 10 mm. after exercise; of them, two (107 & 160) are in fair health; case 15 is threatening uraemia, and case 88 suffers from severe angina. The case (62) who showed a rise of 30 mm. is now bedridden with a hemiplegia.

Four of the cases who died showed a rise of 20 - 30 mm. after exercise.

Case 188 who showed a rise of only 10 mm. died of broncho-pheumonia.

From a study of the table it is seen that of the cases who have survived the average age was 62 and the average increase of My 14. In contrast with this those who died (excluding case 188 who died of an intercurrent infection) showed an average My increase of 27.5, whilst their average age was only 54.

EXERCISE in HYPERTENSIVES: Table 13.

Case	Sex	Age	Mx Rest	Mx Ex.	Change in Mx	My Rest	My Ex.	Change in My	Mn Rest	Mn Ex.	Change in Mn	Result
1	M	61	180	190	+10	130	160	+30	120	110	-10	Dead
12	M	60	210	230	+20	140	160	+20	120	120	-	Dead
15	M	67	230	260	+30	130	140	+10	100	110	+10	Alive
62	M	61	220	240	+20	130	160	+30	100	120	+20	Alive
69	M	27	160	200	+40	120	120	-	90	110	+20	?
88	M	78	200	210	+10	110	120	+10	100	100	-	Alive
107	F	44	170	210	+40	110	120	+10	90	100	+10	Alive
144	M	51	200	220	+20	130	160	+30	100	90	-10	Dead
160	F	59	160	180	+20	100	110	+10	90	90	-	Alive
188	M	79	170	190	+20	110	120	+10	90	90	-	Dead
203	F	43	210	240	+30	160	180	+20	130	130	-	Dead

EXERCISE in HYPERTENSIVES.

Fig. 15.

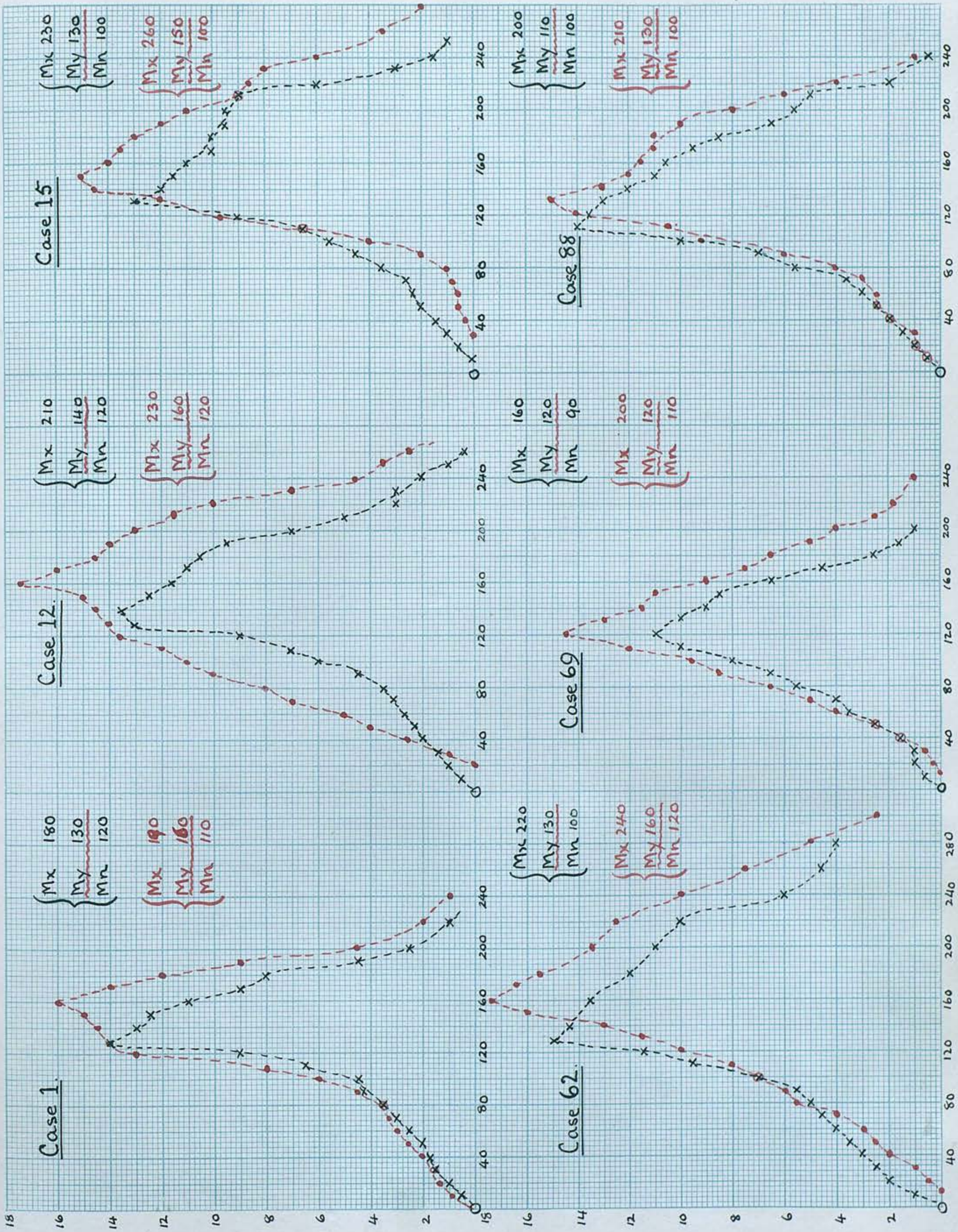
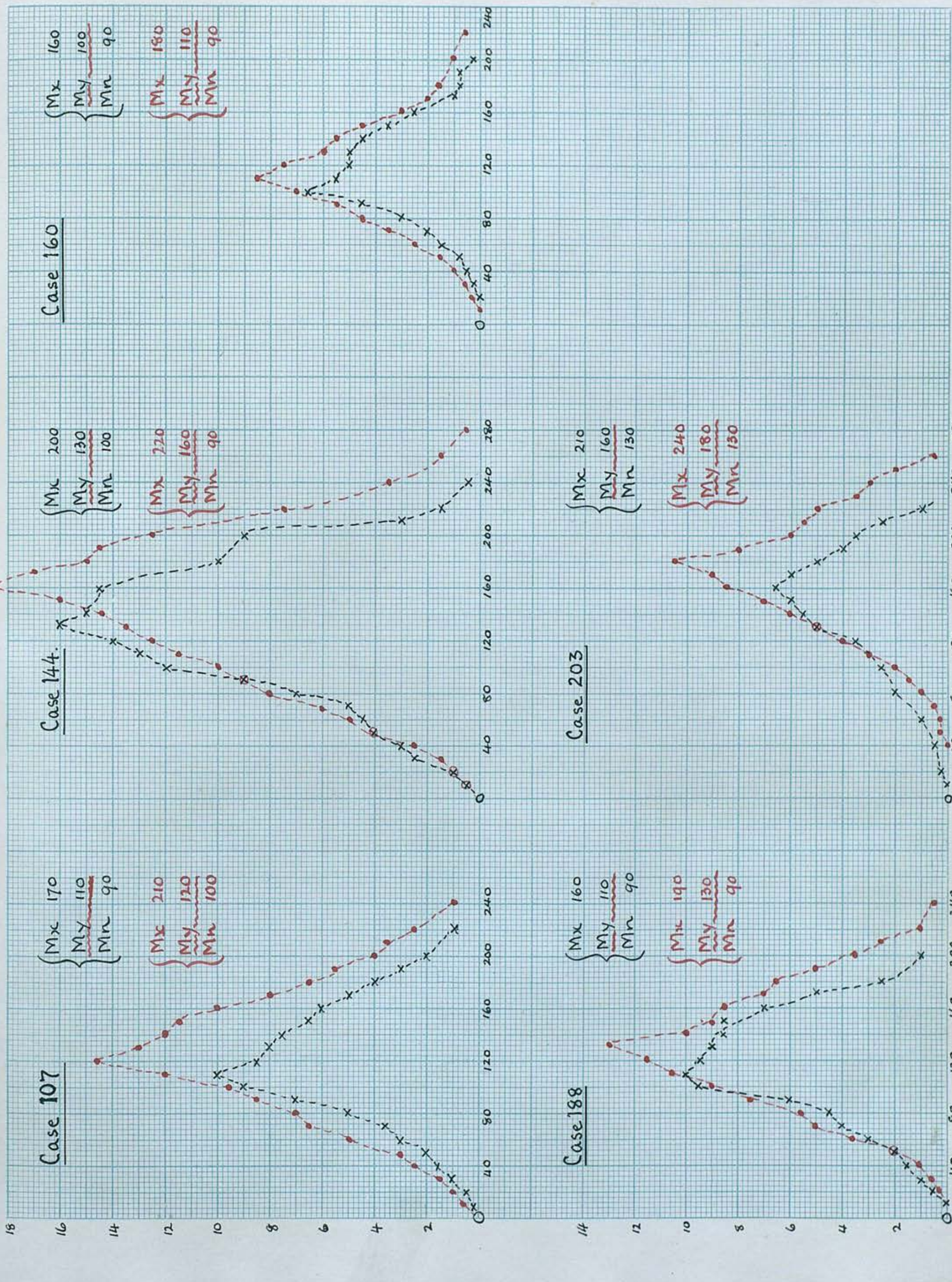


Fig. I5 (contd.)


Case 107
Mx 170
My 110
Mn 90
Mx 210
My 120
Mn 100
Case 144.
Mx 200
My 130
Mn 100
Mx 220
My 160
Mn 90
Case 160
Mx 160
My 100
Mn 90
Mx 180
My 110
Mn 90
Case 188
Mx 160
My 110
Mn 90
Mx 190
My 130
Mn 90
Case 203
Mx 210
My 160
Mn 130
Mx 240
My 180
Mn 130

From these figures it would appear that any rise in My after exercise must be regarded seriously, while the gravity of the prognosis corresponds fairly closely to the amount of the rise.

2b. ARTERIO-SCLEROSIS: (Table 14)

14 cases came under this group as having certain features in common, viz. all had palpable thickening of peripheral arteries and all except one (139) showed clinical evidence of cardiac hypertrophy without valvular defect. All complained of dyspnoea on exertion.

Mx was elevated in every case, but Mn only showed gross elevation in one case (139).

The average My was 119 - ranging from 80 - 160.

The average O.I. was slightly above normal, but the curves showed no characteristic features.

Of the 14 cases 6 died within 5 years.

Case 1 died of coronary thrombosis and cases 12, 105 & 203 of cerebral haemorrhage - these being the 4 cases with gross elevation of My. The other 2 deaths were due to cerebral syphilis (161) and an intercurrent infection (21). One case was untraced.

Of the remainder, who are alive, none showed

ARTERIO - SCLEROSIS:

Table I4.

Case	Age	Mx	My	Mn	Dyspnoea.	Precordial pain.	Cardiac hypertrophy.	Vertigo.	Claudication.	Result.
I	6I	2I0	I40	I00	+	+	+	−	−	D
I2	60	2I0	I40	I20	+	+	+	+	−	D
I9	64	I70	I20	I00	+	+	+	+	−	A
2I	67	I70	I00	90	+	−	+	+	−	D
76	60	I80	I20	I00	+	−	+	+	−	A
82	60	I60	I20	I00	+	−	+	+	+	A
88	78	2I0	I20	I00	+	++	+	+	−	A
I05	58	I70	I40	90	+	+	+	+	−	D
II0	62	I70	I00	80	+	+	+	+	−	?
I38	64	I70	I00	80	+	+	+	−	−	A
I39	67	I60	80	60	+	+	−	−	−	A
I6I	65	2I0	I20	I00	+	−	+	+	−	D
203	43	2I0	I60	I30	+	−	+	+	−	D
2I8	64	I80	II0	80	+	−	+	−	+	A

an My higher than 120, although Mx in one case was as high as 210; (this patient suffers from severe angina pectoris.)

Of those who have died the average age was 59 and the average My 133, while though the average age of those who survived was 65 their average My was only 110.

Any conclusion that may be drawn from such a small group is that the gravity of the prognosis depends on the level of My rather than of Mx.

2c. <u>AORTIC INCOMPETENCE:</u> (Table 15 & Fig. 16)

A small group of 7 cases whose ages ranged from 28 - 70 years. Four cases - 6, 39, 144 & 223 - had clinically no other valvular lesion. Cases 2 & 59 had also mitral incompetence, while case 162 was a "double aortic." All were ambulant patients and though their symptoms varied considerably Mx was elevated in all cases except one (2), the average being 171 mm. Mn averaged 67, so that the average "pulse-pressure" was 104. My averaged 90 - quite a normal figure.

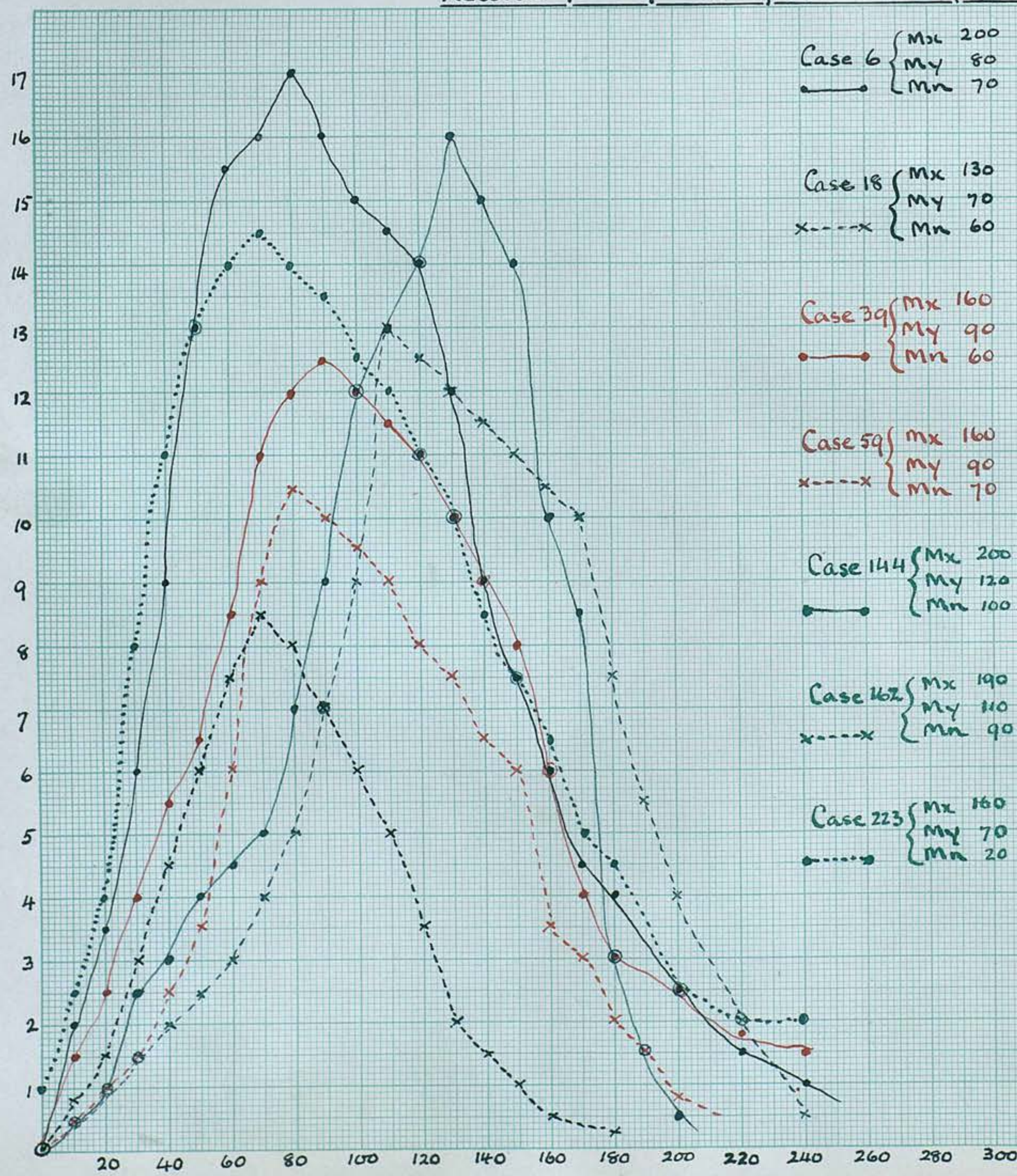
This bears out what has been stated by various observers (Vaquez, Gley & Gomez (20): Vaquez &

AORTIC INCOMPETENCE:

Table 15.

Case	Age	Sex	Mx	My	Mn	P.P.	
6	52	M	200	80	70	130	Pure aortic.
18	28	M	130	70	60	70	Aortic & mitral incomp.
39	31	M	160	90	60	100	Pure aortic.
59	70	F	160	90	70	90	Aortic & mitral incomp.
144	51	M	200	120	100	100	Pure aortic.
162	62	F	190	110	90	100	Double aortic.
223	25	F	160	70	20	140	Pure aortic.

Records from 7 cases of Aortic Incompetence.



More similarity was found between the curves in this group than in any other.

All the curves were of types 2 or 3, and all showed a very rapid fall away from My.

It should be noted, however, that this type of curve is in no way pathognomonic of aortic incompetence.

Fig. 16.

Lajoie (21): Vaquez & Kisthinios (26): Gley & Gomez (35): Lajoie (36)) that so long as the valvular lesion remains compensated there is practically no change in My.

On plotting the curves of these cases more similarity was found in this group than in any other. All the curves were of types 2 and 3, and all showed a very rapid fall away from My. (Fig. 16) It should be noted, however that this type of curve is in no way peculiar to cases of aortic incompetence.

2d. <u>CHRONIC INTERSTITIAL NEPHRITIS:</u> (Table 16 & Fig. 17)

In 11 cases of this disease of varying severity records were taken, the range of ages being from 39 - 67 years.

Two cases - 157 & 171 - were in advanced uraemia and died within a few days.

Two cases - 15 & 62 - are alive to-day, though both are invalids. Five others have died since the records were taken 7 years ago. Two cases are untraced.

As was to be expected Mx was above normal in almost every case, the average being 203. Excluding case 113 where the patient was profoundly collapsed as a result of prolonged diarrhoea and vomiting the average Mx was 210.

The average My for the whole group was 140 - or again excluding case 113 the average was 145.

Mn averaged 115 for the whole group, or 119 for the same 10 cases. The average O.I. was above normal, but ranged from $6\frac{1}{2}$ - 19 divisions. This variation was reflected in the curves which showed no characteristic feature - in fact the striking point was the wide variation in the type of curve found. (Fig. 17)

There was a noticeable lack of correspondence between the heights of Mx and My; for instance case 157 showed a Mx of 210 with My 180, while case 15 had Mx 230 and My only 130.

The two patients of this group who have survived both showed gross elevation of Mx - 230 and 220 respectively - but both had an My of only 130 - little above normal.

The moribund cases both showed the greatest elevation of My - viz. 180.

If any conclusion may be drawn from such a small group of cases it would be that the prognosis - making allowance for such factors as the profound collapse in case 113 - depends upon the height of My and not upon the height of Mx.

CHRONIC INTERSTITIAL NEPHRITIS:

Table I6.

Case	Age	Sex	Mx	My	Mn	Result	
9	6I	M	220	I20	I00	?	(Cerebral symptoms & (precordial pain.
I5	67	M	230	I30	I00	A	(Dyspnoea; vertigo; (tinnitus.
40	47	M	220	I60	I40	D	Dyspnoea; headache.
5I	59	M	I90	I40	I20	?	(Dyspnoea; epigastric (pain.
62	6I	M	220	I30	I00	A	(Dyspnoea;precordial (pain; insomnia.
93	48	F	220	I70	I40	D	Uraemia.
I0I	56	M	I70	I20	80	D	Ruptured aneurysm.
II3	34	M	I30	80	70	D	(Dyspnoea; collapse; (severe D.& V.
I23	65	M	I80	I20	II0	D	(Bedridden; mental (confusion.
I57	50	M	240	I80	I40	D	(Uraemia; died two (days later.

CHRONIC INTERSTITIAL NEPHRITIS. Fig. 17.

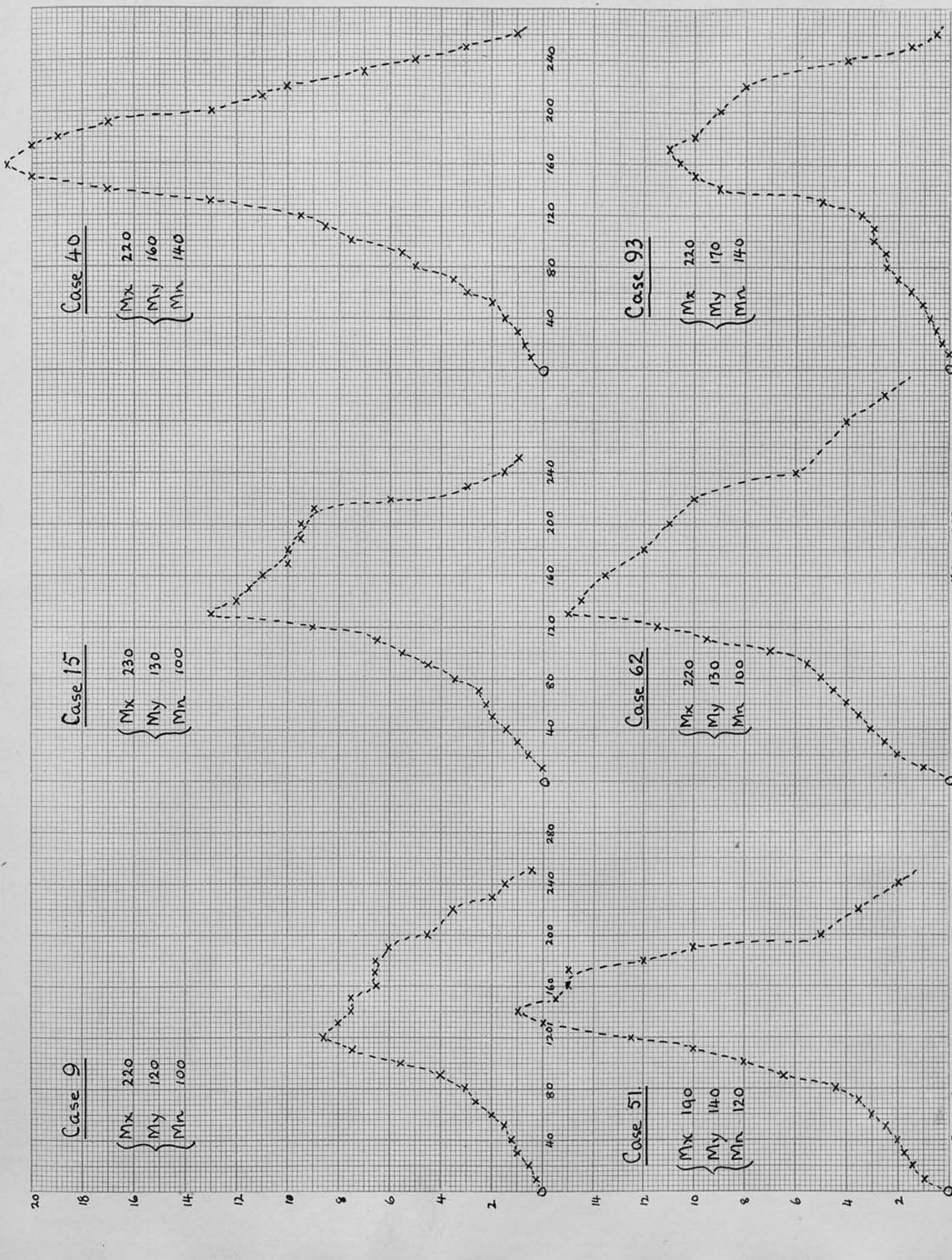
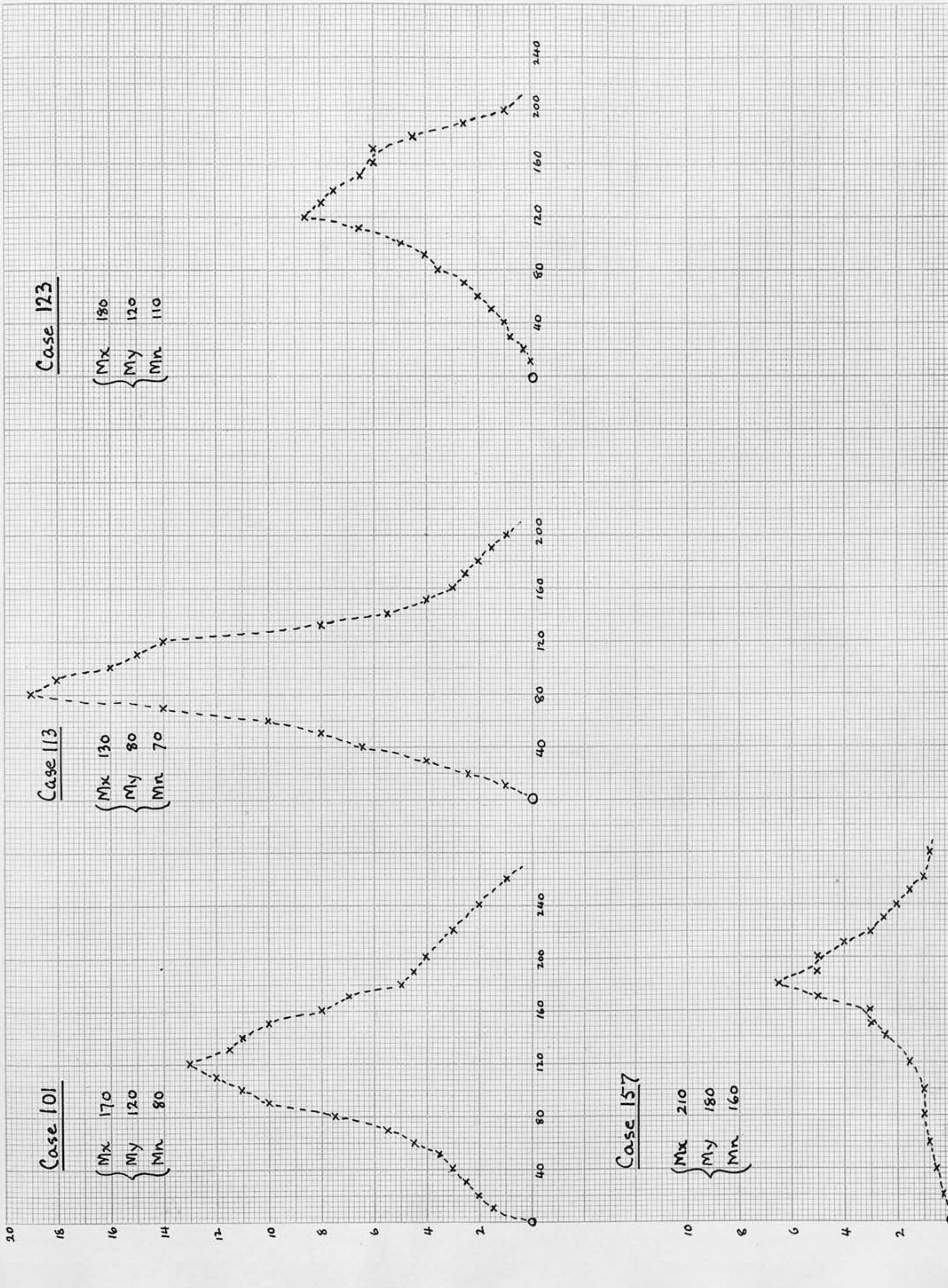


Fig. I7 (contd.)


Case 101
Mx 170
My 120
Mn 80
Case 113
Mx 130
My 80
Mn 70
Case 123
Mx 180
My 120
Mn 110
Case 157
Mx 210
My 180
Mn 160

2e. RIGHT-SIDED VALVULAR LESIONS AND CHRONIC MYOCARDIAL DAMAGE: (Table 17)

This group consists of 14 cases of right-sided cardiac valvular disease or chronic myocardial damage. In some cases the lesion was compensated while in others any degree of congestive heart failure was present. In no case was there evidence of arterio-sclerosis or left-sided heart failure.

The average My for the group was 96, ranging from 80 to 120 - i.e. within normal limits.

The figures are set out in Table 17, and from these records it would appear that right-sided heart failure of any degree or valvular lesions of the right heart cause no departure from normal in the level of My.

2f. MALIGNANT DISEASE & CHRONIC DISEASES CAUSING PROLONGED CONFINEMENT TO BED. (Table 18)

In this group the records were taken from patients in the Longmore Hospital - some in the cancer wards and some in the general wards where such lingering conditions as disseminated sclerosis, rheumatoid arthritis &c. are treated. Excluded from this group were any cases with an obvious cardiac lesion or cases of illness which might be expected to cause cardio-vascular disturbance.

RIGHT - SIDED VALVULAR LESIONS and CHRONIC MYOCARDIAL DAMAGE:

Case	Age	Sex	Mx	My	Mn	
5	59	M	I60	I00	80	Chronic myocarditis.
22	57	M	I30	II0	60	Coronary stenosis of 5 years' standing.
69	27	M	I60	I20	90	Mitral incompetence.
80	30	M	I30	80	60	Rheumatic mitral stenosis.
94	48	M	I05	90	70	Chronic bronchitis and myocarditis.
99	56	F	I30	90	70	Chronic myocarditis with early heart failure.
I00	42	M	I40	II0	90	Chronic myocarditis with emphysema.
III	48	M	I20	90	80	Myocardial infarction one year ago.
I50	57	M	I40	I00	70	Influenzal myocarditis.
I5I	34	M	I40	90	70	Rheumatic mitral stenosis.
I60	59	F	I60	I00	90	Chronic myocarditis.
I80	34	F	II0	90	80	Mitral stenosis with congestive heart failure.
2I4	62	F	I00	80	60	Congestive heart failure with ascites.
2I6	77	F	I50	I00	80	Senile heart failure.

MALIGNANT DISEASE &c:

Table I8.

Case	Age	Mx	My	Mn	
83	57	I20	100	80	Advanced carcinoma of breast.
8I	5I	90	80	60	Carcinoma of stomach.
II9	70	I90	I20	I00	Paraplegia.
I2I	70	I80	II0	90	Tabes.
I23	68	I80	I40	I00	Chr. rheumatoid arthritis.
I25	60	II0	80	60	Diabetes.
I26	48	I50	I00	80	Chr. fibroid phthisis.
I27	49	90	80	60	Disseminated sclerosis.
I28	3I	I50	I20	I00	Fractured spine.
I29	6I	I40	I00	80	Disseminated sclerosis.
I30	34	II0	90	70	Disseminated sclerosis.
I35	72	I80	I20	I00	Carcinoma of rectum.
I36	58	I80	II0	90	Epithelioma of jaw.
I37	82	I40	I00	80	Carcinoma of rectum.
I4I	69	I80	II0	90	Chr. T.B. hip.
I45	56	II0	I00	80	Cerebral tumour.
I49	59	2I0	I70	I40	Carcinoma of rectum.
I52	73	I30	I00	70	Carcinoma of tongue.
I53	68	I20	90	70	Carcinoma of oesophagus.

Results are shown in Table 18 and point to the fact that neither malignant disease nor prolonged confinement to bed appear to influence the factors which would normally produce change in the pressure levels.

For instance we find a case (149) of advanced carcinoma of the rectum with pressures of 210/170/140 and a case of advanced rheumatoid arthritis (124) with pressures of 180/140/100.

On the other hand we find a case of carcinoma of the stomach (89) and a case of disseminated sclerosis (127) each with pressures of 90/80/60.

From this it would appear that neither malignant disease nor chronic diseases causing prolonged confinement to bed of themselves cause any change in My.

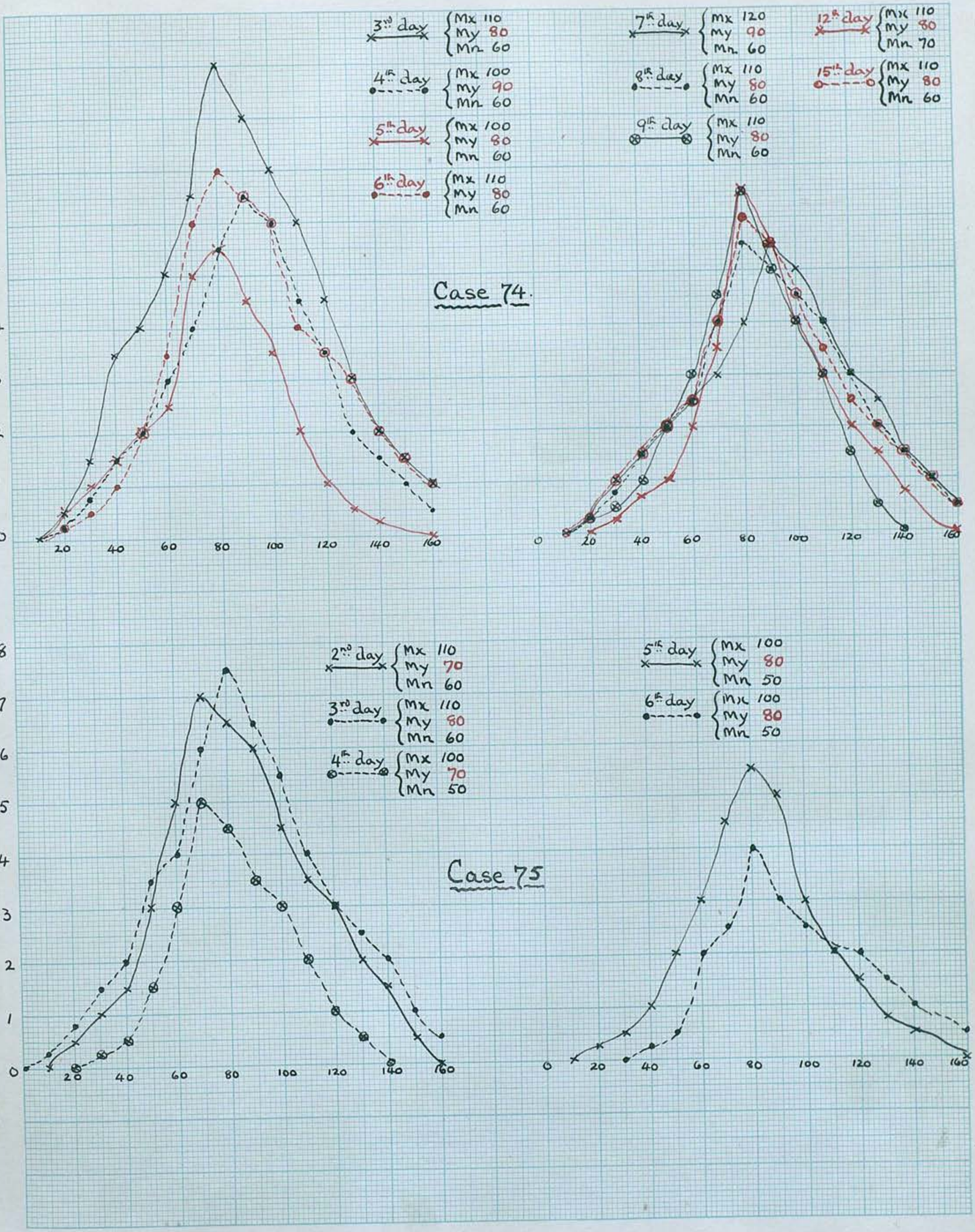
2g. LOBAR PNEUMONIA: (Fig. 18)

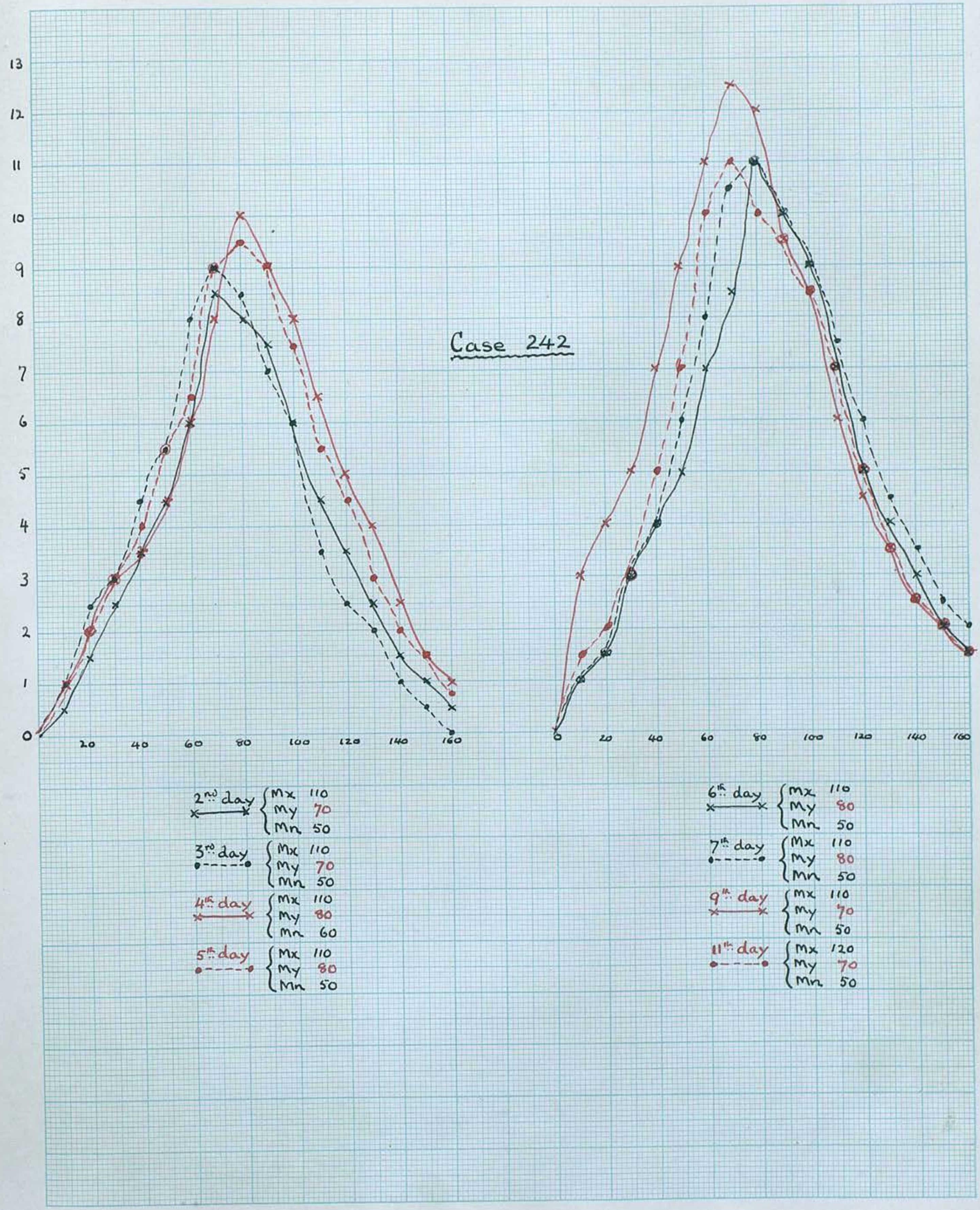
Records were kept throughout the illness in three cases of right lower lobe pneumonia.

Cases 74 and 242 made complete recoveries; case 75 died on the 7th day of the illness following a pseudo-crisis on the 6th day.

It is obvious that no conclusions can be drawn from such a small group, but it is worthy of notice

LOBAR PNEUMONIA. Fig. 18.




Case 242
0
1
2
3
4
5
6
7
8
9
10
11
12
13
20 40 60 80 100 120 140 160
0 20 40 60 80 100 120 140 160
2nd day Mx 110 My 70 Mn 50
3rd day Mx 110 My 70 Mn 50
4th day Mx 110 My 80 Mn 60
5th day Mx 110 My 80 Mn 50
6th day Mx 110 My 80 Mn 50
7th day Mx 110 My 80 Mn 50
9th day Mx 110 My 70 Mn 50
11th day Mx 120 My 70 Mn 50

that in all cases My ran a practically level course throughout - 10 mm. being the greatest deviation recorded - while in the fatal case there was no change in My on the day before death to indicate any deterioration in the patient's condition.

In all cases the level of My was within normal limits throughout the illness.

2h. SPINAL ANAESTHESIA: (Tables 19 & 20)

Included in this group were 19 cases, in 15 of which Stovain was the anaesthetic employed, and in 4 Neocaine.

Dosage: Stovain: 1.2 - 2 c.c.

Neocain: 0.I. - 0.15 gm.

Sex: All the patients in this group were male.

Age: 18 - 49 years.

Premedication: All patients of this group were given an intramuscular injection of Ephedrin gr $\frac{1}{2}$ - gr i about $\frac{3}{4}$ hour before operation. Only one case (239) required ephedrin during operation on account of signs of collapse.

Nature of operation: The operations - except the three cases of stone in the ureter - were all of a type unassociated with any great degree of shock, and were all performed on patients in a reasonably good state of health.

Level of anaesthesia: Only cases where anaesthesia was complete up to the level of the 11th dorsal segment were included in this group.

RESULTS:

A. NEOCAINE:

This anaesthetic was only used in four cases. Of these two - (228 and 240) showed practically no alteration in the three levels, in fact case 240 actually showed a rise of My of 10 mm. after 15 minutes, though there was a fall of 10 mm. in My after 25 min. The other two cases showed a marked fall of both Mx and My after 15 minutes. Case 239 showed signs of collapse after 25 min. but rallied after the administration of Ephedrin gr $\frac{1}{2}$ intramuscularly. Case 241, though showing even a more pronounced fall of both Mx and My - 130/90/80 to 80/60/50 - did not exhibit the same signs or symptoms of shock, and rallied as the operation proceeded.

On account of these disturbing falls of pressure Neocaine was given up in favour of 5% Stovain with glucose.

B. STOVAIN: 15 cases.

The response of Mx and Mn was variable. In several cases there was a transient rise of Mx after 10 - 15 minutes - probably largely as a result of apprehension. In the majority there was a slight fall in Mx and Mn, though only in one case (250) did the fall of Mx exceed 20mm. In this case (stone in the ureter) there was a fall from 120/90/70 to 80/60/50 after 15 minutes and to 70/60/40 after 30 minutes. There did not however appear to be a corresponding deterioration in the condition of the patient, and he rallied spontaneously, reaching 100/80/60 after 45 minutes.

This was the only case in which My showed a variation of more than 10 mm.; any change present in the remainder came within the range of observational error.

CONCLUSIONS:

The only conclusion which may legitimately be drawn from these records is that where the patient suffers from shock - whether as a result of the anaesthetic used or as a result of the operative procedure - all three levels of blood pressure are affected, though the signs and symptoms exhibited by the patient do not correspond to the absolute falls of pressure.

Spinal Anaesthesia – Néocaine

Table 19.

Case	Age	Operation	Premedication (Ephedrin gr)	Dose of Néocaine (grm.)	Preoperative B.P.	Blood Pressure readings after administration of Anaesthetic: 5min	10	15	20	25	30	35	40	45	50	55
228	30	Hernia.	j	0·12	100 70 50		100 70 60		100 70 50		100 70 60					
239	26	Stone in ureter.	j	0·15	100 80 60			80 60 50		※		80 60 50		100 80 60		
240	32	Knee Cartilage.	ß	0·1	140 100 80			150 100 80		140 90 70						
241	32	Knee Cartilage.	j	0·1	130 90 80			80 60 50		100 70 60						

※ Intramuscular injection of Ephedrin gr ß on account of signs of collapse.

— Spinal Anaesthesia — Stovain. —

Table 20.

Case.	Age.	Operation.	Premedication.	Dose of Stovain.	Preoperative B.P.	Blood Pressure readings. after administration of Anaesthetic. 5 min	10	15	20	25	30	35	40	45	50	55	60.
			Ephedrin gr	c.cm.													
229	27	Knee Cartilage	i	1·2	140 90 80			130 90 70		120 90 70		110 80 60					
230	40	Hernia	i	1·4	120 80 70			100 70 50			100 70 60		100 80 60				
231	21	Hernia	i	1·4	150 100 70			170 100 60						110 90 40			
232	18	Hydrocele	i	1·4	130 80 60			100 70 50			100 70 40			110 70 40			
235	21	Appendix	i	1·8	100 80 60		120 80 70		120 80 70								
236	33	Hernia.	i	1·6	110 80 60		100 80 60		100 80 60			100 80 60					
237	18	Knee Cartilage	i	1·5	90 70 60		90 70 60		90 70 60								
238	19	Knee Cartilage	i	1·5	140 90 70			130 90 60			130 80 50						
243	20	Knee Cartilage	i	1·4	130 80 60			120 90 50			120 90 50						
244	49	Hernia	i	2	130 90 70		110 80 70		100 80 70		100 80 70						
245	24	Hernia	i	2	110 90 60			100 90 60		110 90 60			110 90 70				
246	19	Appendix	ss	2	110 90 60		130 90 60		100 90 50		100 90 60						
248	31	Hydrocele	ss	2	110 80 60		130 90 70		130 90 70		120 90 70		120 90 60		110 90 60		110 90 70
250	27	Stone in Ureter	i	2	120 90 70			80 60 50			70 60 40			100 80 60			
251.	25	Stone in ureter.	ss	2	140 90 60		140 90 60			130 90 50			140 90 50			140 90 50	

21. ANALYSIS OF CASES (1) WITH ELEVATION OF My: (2) WITH ELEVATION OF Mx BUT NORMAL My: (Tables 21 & 22)

The first 200 cases of the series - i.e. records taken between the years 1933 and 1936 - were chosen in order to make a comparison between cases showing elevation of My and those showing elevation of Mx with a normal My.

Into the first group (Table 21) came 20 cases, My ranging between 130 and 180 mm. All showed an elevation of Mx and in most cases Mn was also above normal.

It was found however that there was no proportion between the extent of elevation of My and of Mx. We find one case, for instance, (15) with Mx of 230 and My of only 130 mm., while another case (105) shows an My of 140 and Mx of only 170 mm.

Looking at the outcome of these cases whose records were taken between 4 and 7 years ago we find that of the 20 cases 15, or 75% are now dead: 4, i.e. 20% are alive, but one of these is moribund and two are bedridden. One case out of this group is untraced. In the cases which are now dead the average My was 151, while in those which are still alive it was 133 mm. On the other hand the average Mx for those who have died was 205, while for those who survived it was 225.

In the second group (Table 22) consisting of 35 cases only those were included in which Mx was 160 or over and My was not above 120 mm. The range of Mx was between 160 and 220 mm. Of these cases 14, or 40% are now dead; 16, or 46% are known to be alive; 5 cases are untraced.

Relative prognostic significance of increases in Mx and My:-

	Dead	Alive	Untraced
Cases showing elevation of My (average age 55)	75%	20%	5%
Cases showing elevation of Mx but Normal My (average age 58)	40%	46%	14%

From these results we may conclude that in a case of hypertension the gravity of the prognosis depends upon the elevation of My to a far greater extent than upon the elevation of Mx.

Table 2I .

CASES WITH My above I20:

Case	Age	Sex	Mx	My	Mn	Result	
I	6I	M	2I0	I40	I00	D	(Died 6 months later of (myocardial infarct.
4	30	F	2I0	I40	90	A	Toxaemia of pregnancy.
I2	60	M	2I0	I40	I20	D	(Art.sclerosis;died 2 (yrs.later of cer.haem.
I4	59	F	I90	I30	I00	?	Neurasthenia.
I5	67	M	230	I30	I00	A	(Arteriosclerosis; old (lead poisoning.
40	47	M	220	I60	I40	D	Chr. inter. nephritis.
5I	59	M	I90	I40	I20	D	"" "" "" "" ""
62	6I	M	220	I30	I00	D	"" "" "" "" ""
90	56	M	I80	I30	II0	D	Chronic myocarditis.
93	48	F	220	I70	I40	D	Chr. inter. nephritis.
I05	58	M	I70	I40	90	D	Arteriosclerosis.
II6	64	F	240	I30	II0	A	Hemiplegia.
I20	44	M	2I0	I60	I30	D	Encephalitis.
I24	68	F	I80	I40	I00	D	Chr. rheumatoid arth.
I44	5I	M	200	I30	I00	D	Aortic incompetence.
I49	59	F	2I0	I70	I40	D	Carcinoma of rectum.
I57	50	M	2I0	I80	I60	D	Chr. inter. nephritis.
I66	65	F	220	I60	I40	A	Hemiplegia.
I67	60	F	220	I30	II0	A	Cong. heart failure.
I7I	39	F	240	I80	I40	D	Uraemia.

Table 22.

CASES with Mx above I50 at REST, and My NORMAL:

Case	Age	Sex	Mx	My	Mn	Result	
3	78	M	I80	II0	90	D	Coronary thrombosis.
5	59	M	I60	I00	80	?	Cerebral haemorrhage.
6	52	M	200	80	70	?	Aortic incompetence.
9	6I	M	220	I20	I00	?	Chr. inter. nephritis.
I0	34	M	I70	90	70	?	Traumatic headache.
I3	60	F	I70	II0	90	D	(Functional hypertens. (Died of pneumonia.
I9	64	M	I70	II0	90	A	Arteriosclerosis.
2I	67	M	I70	I00	90	D	Arteriosclerosis.
30	24	M	I60	I20	90	A	No symptoms.
3I	34	F	I60	I20	90	A	Toxaemia of pregnancy.
39	3I	M	I60	90	60	D	Aortic incompetence.
47	52	F	I60	II0	90	A	Retinal detachment.
59	70	F	I60	90	70	D	Aortic incompetence.
60	24	F	I60	II0	90	A	Pregnant; ?excitement.
69	27	M	I60	I20	90	A	Mitral incompetence.
76	60	M	I60	I20	I00	A	Arteriosclerosis.
82	60	M	I80	I20	I00	D	Arteriosclerosis.
88	78	M	200	II0	I00	A	"" "" "" ";angina
I0I	56	M	I70	I20	80	D	Chr. inter. nephritis.
I07	44	F	I70	II0	90	A	Toxic goitre.

(over-

Table 22 (contd.)

Case	Age	Sex	Mx	My	Mn	Result	
110	62	M	170	100	80	?	Arteriosclerosis.
117	81	F	180	110	90	D	Pneumonia.
118	47	F	160	110	90	A	Hemiplegia.
119	70	M	190	120	100	A	Paraplegia.
121	70	M	180	110	90	A	Tabes.
123	65	M	180	120	110	D	Chr. inter. nephritis.
135	72	M	180	120	100	D	Carcinoma of rectum.
136	58	M	180	110	90	D	Epithelioma of jaw.
138	64	M	170	100	80	A	Arteriosclerosis.
139	67	M	160	80	60	D	Arteriosclerosis.
141	69	F	180	110	90	D	T.B. hip.
160	59	F	160	100	90	A	?myocarditis.
161	65	M	210	120	100	D	Cerebral syphilis.
162	62	F	190	110	90	A	Aortic incompetence.
188	79	M	170	110	90	A	?normal.

2j. RECORDS FROM A CASE OF MYOCARDIAL INFARCTION: (Fig. 19)

Alex. A., aet. 61; labourer; case of arterio-sclerosis with hard, tortuous peripheral arteries and heart showing forcible apex beat in 6th space, 1 in. outside nipple line; chief complaint was of dyspnoea and precordial pain on exertion.

On 29/10/33 his blood pressure readings were 210/140/100, and on 20/12/33 they were 210/120/100.

On 1/2/34 he suffered a coronary thrombosis and the following day his readings had dropped to 140/90/70.

During the next six weeks he made a gradual recovery and there was a corresponding rise in the level of his B.P. readings, till on 25/3/34 they were 180/140/120.

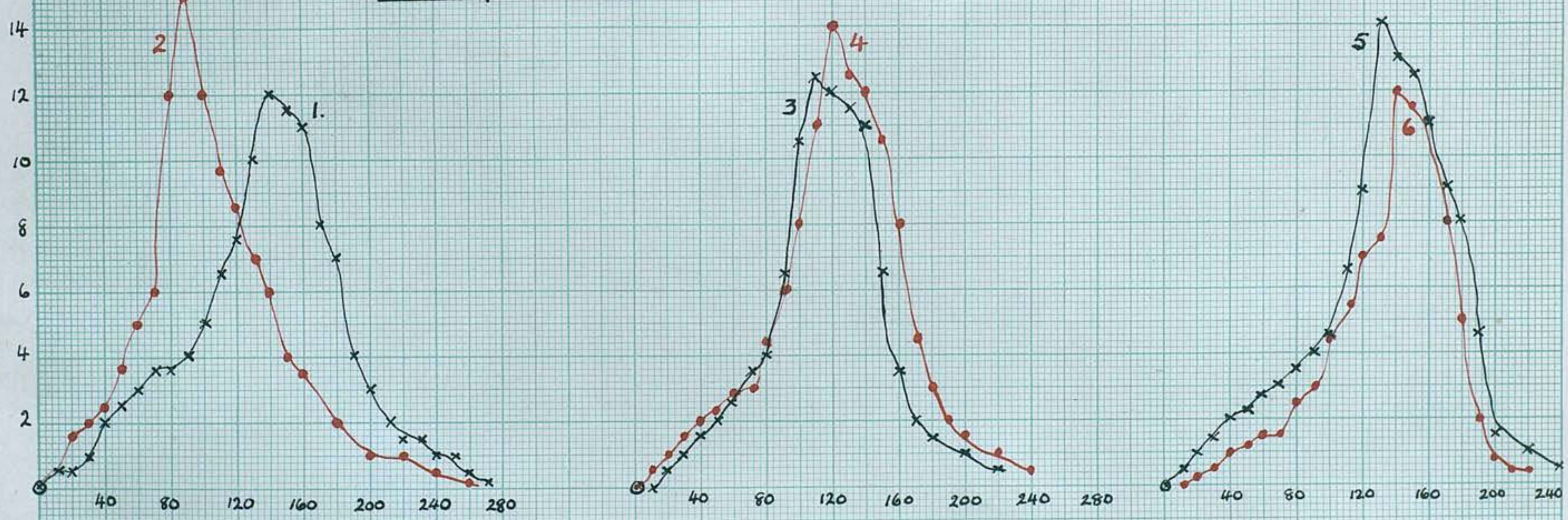
On 9/3/34 he underwent a very mild exercise test - sufficient however to cause some dyspnoea and precordial discomfort. After exercise his Mx rose only 10 mm. but his My increased by 30 - a bad prognostic sign. He died three months later of a further coronary thrombosis.

The interesting features of this case appear to be:-

1. The complete shift to the left of his curve on the day following his thrombosis,

Case 1.

Coronary thrombosis.



Curve 1 29/10/33 { Mx 210, My 140, Mn 100 }

Curve 2 2/2/34 { Mx 140, My 90, Mn 70 }

Curve 3 11/2/34 { Mx 150, My 110, Mn 90 }

Curve 4 18/2/34 { Mx 160, My 120, Mn 110 }

Curve 5 9/3/34 { Mx 180, My 130, Mn 120 }

Curve 6 25/3/34 { Mx 180, My 140, Mn 120 }

Records taken (1) 3 months prior to thrombosis; (2) the day after; (3),(4),(5),(6) during recovery.

Note sudden complete shift to left in curve 2 — presumably the effect of shock resulting from thrombosis.

Also gradual shift to right during stage of recovery, 'My' having regained original level after 7 weeks,

but 'Mx' never reaching original level owing to myocardial damage. — a bad prognostic sign.

(Patient died 3 months later, following a further infarction.)

Fig. 19.

indicating lowering of all the B.P. readings - whether as a result of the shock accompanying his thrombosis or simply as a result of impairment of cardiac action.

2. After 7 weeks Mx had not regained its former level though My was back to the original figure - probably an indication of myocardial damage.

3. His early death was a confirmation of the fact that a high My which rises after exercise is a feature of grave prognostic significance.

...

CONCLUSIONS:

Although records have been taken from a considerable number of patients it is felt that the number of cases falling into each group is so small that it would hardly be justifiable to be dogmatic in drawing any conclusions from them.

With only a few exceptions however the conclusions are merely confirmation of the results of other observers, and so though they might be of little value standing alone, they are of some significance in adding weight to results which have already been published.

Certain groups - e.g. toxaemias of pregnancy - have had to be omitted on account of lack of clinical material available, and it is felt that in order to establish a true guide to prognosis a longer follow-up would have been necessary over a larger number of cases.

The following however are the conclusions which have been arrived at in the course of the investigation:-

A. <u>Technical points in oscillometry:-</u>

1. A definite system must be adopted as regards recording the amplitude of oscillations, as this affects the evaluation of Mx, My and Mn.
2. Variation in the tightness of application of the armlet affects the oscillatory amplitude throughout, but does not in any way affect the reading obtained for My.
3. The oscillatory amplitude was found to be higher than normal:-
    a) In direct proportion with the tightness of application of the armlet.
    b) Where there was increase in the stroke-volume of the cardiac output - e.g. after exercise.

c) Where high blood pressure was acting on an elastic arterial wall, as in the early stages of arteriosclerosis.

4. The fact that deflation is performed in steps of 10 mm. at a time gives a margin of error of 9 mm. in all readings, but it is pointed out that blood pressure is naturally so labile that this error is of no significance and may well fall within the range of observational error.
5. The plateau curve should give rise to no difficulty as it can always be abolished by repeating the reading after tighter application of the armlet.
6. There are no points on an oscillometric curve which can be taken as the index of either systolic or diastolic pressure. For these readings reliance must be placed on the older and more generally accepted auditory method.
7. No characteristic features have been found which from a study of the oscillometric curve would enable the subject to be classed as normal or pathological, or to be suffering from cardiovascular disease or any other specific pathological condition.

It would thus appear that the oscillometric curve is unnecessary, all that requires to be noted being the three levels Mx, My and Mn.

B. Oscillometric records from patients:-

1. Normal values for My:

In healthy individuals My was found to lie within the limits of 70 - 110. (Many observers consider 120 as the upper limit of normal.)

The readings for males were in all age groups very slightly higher than those for females.

In both sexes there was a slight increase in the value of My with advancing years.

2. Site of application of the cuff:

In the recumbent position the site of application of the cuff has no appreciable effect upon the reading obtained for My.

3. Normal pregnancy:

A normal pregnancy does not affect the level of My.

4. Records from the same patient at different times:

Every individual has a characteristic oscillometric curve to which he remains true even if records are taken at wide intervals of time or under varying conditions.

5. Exercise:

a) In healthy subjects My remains unaltered after exercise.

b) In hypertensive subjects My rises after exercise - the gravity of the prognosis corresponding fairly closely to the extend of this rise.

6. Arteriosclerosis:

There is a rise in the level of My as well as of Mx, and the prognosis depends on the level of My rather than of Mx.

7. Aortic incompetence:

Where this valvular lesion is uncomplicated My remains within normal limits in spite of increase of Mx and P.P.

8. Chronic interstitial nephritis:

In this disease there is elevation of My as well as of Mx and the prognosis appears to depend on the height of My and not upon the height of Mx.

9. Right-sided heart failure:

Right-sided heart failure of any degree or valvular lesions of the right heart cause no departure from normal in the level of My.

10. Malignant disease &c.:

Neither malignant disease nor chronic diseases causing prolonged confinement to bed of themselves cause any change in My.

11. Lobar Pneumonia:

In the three cases of lobar pneumonia recorded the level of My was within normal limits throughout the illness.

12. Spinal anaesthesia:

Where the patient suffers from shock - whether as a result of the anaesthetic used or as a result of the operative procedure - there is a fall in all three levels of blood-pressure.

13. From an analysis of cases (1) with elevation of My and (2) with elevation of Mx but normal My, it was concluded that in a case of hypertension the gravity of the prognosis depends upon the elevation of My to a far greater extent than upon the elevation of Mx.

14. The condition described as "hypertension "moyenne solitaire" has not been encountered.

15. From a study of the results obtained the conclusion is arrived at that the recording of My is an essential part of every blood pressure estimation. The fact that My is within normal limits is of no more significance than the fact that Mx and Mn are normal. Any departure of My from normal

is, however, a feature of the utmost import.

It has been shown that in arteriosclerosis, chronic interstitial nephritis and in fact in any condition giving rise to "high blood pressure" the prognosis depends to a much greater extent on the level of My than of Mx.

Any elevation of My after exercise must be regarded with gravity, and it follows that the estimation of My both at rest and after exercise should be insisted upon in every Life Assurance examination, and in every case where the ultimate prognosis is to be assessed.

SUMMARY OF PART 2:-

1) A specification of the "Kynomètre" of Vaquez, Gley & Gomez is given.
2) Technique and technical problems of oscillometry are discussed.
3) From observations on 252 patients (a) a scale of normal readings for My is drawn up and (b) records from pathological cases are compared with this scale and the significance of deviations from normal discussed.
4) Conclusions which may be drawn from the records are tabulated and the importance of recording the level of My in every blood pressure estimation is emphasised.

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BIBLIOGRAPHY

1. HALL, Marshall. Ed. Med. Jl. 1832: 37: 164.

2. MAREY, E.J. Trav. du Lab. 1876: 2: 309.

3. MAREY, E.J. " " " 1880: 4: 253.

4. MAREY, E.J. "La circulation du Sang à l'état "physiologique et dans les malades". Paris 1881; p.169.

5. MAREY, E.J. "Physiologie médicale de la Cir-"culation du Sang" Paris 1863; p.143.

6. POTAIN. Arch. de Physiol. 1890; Ser. 5; Vol.2; p.681.

7. POTAIN. Arch. de Physiol. 1889; Ser. 5; Vol.1; p.556.

8. GLEY, P. & GOMEZ, D.M. Jl. de Phys. et de Path. Gén. 1931; 29; 38.

9. ROY, C.S. & ADAMI, J.G. Practitioner; 1890; 45; 27.

10. HILL, L. & BARNARD, H. Journ. of Physiology; 1898; 23; 67.

11. ERLANGER, J. John Hopkins Hosp. Report; 1904; 12; 53.

12. ERLANGER, J. & HOOKER, D.R. John Hopkins Hosp. Report; 1904; 12; 145.

13. PACHON, V. Compt. Rend. Soc. de Biol. 1909; 66; 776.

14. RUSSELL, W. "The Sphygmometer". Baillière, Tindaell & Cox; 1921.

15. RUSSELL, W. B.M.J. 1908; 2; 1076.

16. HILL, L. & FLACK, M. B.M.J. 1909; 1; 272.

17. PACHON, V. Compt. Rend. Soc. de Biol. 1921; 84; 868.

18. PACHON, V. &. FABRE, R. Compt. Rend. Soc. de Biol. 1921; 84; 871.

19. PACHON, V. & FABRE, R. Compt. Rend. Soc. de Biol. 1923; 89; 951.

20. VAQUEZ, H., GLEY, P. & GOMEZ, D.M. Presse Médicale. 1931; 39; 281.

21. VAQUEZ, H. & LAJOIE, R.J. Libman Ann. Vol. 1932. Internat. Press. N.Y.

22. GOMEZ, D.M. & LAJOIE, R.J. Presse Médicale; 1931; 39; 586.

23. VAQUEZ, H., GOMEZ, D.M. & LAJOIE, R.J. Presse Méd. No. 84; 21/10/31.

24. VAQUEZ, H., KISTHINIOS, N. & PAPAIOANNOU, M. Presse Méd. 1931; 39; 585.

25. GOMEZ, D.M. Presse Médicale. 1931; 39; 1768.

26. VAQUEZ, H. & KISTHINIOS, N. Bull. de l'Acad. de Méd. 24/3/31. Vol. CV; No.12.

27. VAQUEZ, H. & GOMEZ, D.M. Bull. de L'Acad. de Méd. 17/2/31. Vol. CV; No.7.

28. GLEY, P. & GOMEZ, D.M. Presse Médicale. 1931; 39; 284.

29. VAQUEZ, H., GLEY, P. & GOMEZ, D.M. Presse Méd. 1931; 39; 1802.

30. VAQUEZ, H. & GOMEZ, D.M. Presse Méd. 1931; 39; 1789.

31. VAQUEZ, H., GLEY, P. & GOMEZ, D.M. Presse Méd. 1931; 39; 1309.

32. TETELBAUM, A.G., KRINSKY, M.I. & ROMANOWA, O.F. Presse Méd. 1933; 2; 2111.

33. PACHON, V. & FABRE, R. Compt. Rend. Soc. de Biol. 1924; Vol. CX; 210.

34. PACHON, V. Compt. Rend. Soc. de Biol. 1909; Vol. 66; 733.

35. GLEY, P. & GOMEZ, D.M. Paris Médicale; 1931; 2; 31.

36. LAJOIE, R.J. Canadian Med. Journ. 28; 3; 276.

37. MILLER, H.R. & CHESTER, R.W. American Heart Jl. 1933; 8; 388.

38. LIAN, C. & ZAMFIR, C. Presse Médicale; 1933; 9; 169.

39. GALLAVARDIN, L. Presse Médicale. 1922; 72; 776.

40. MacWILLIAM, J.A. & MELVIN, G.S. B.M.J. 1914; 1; 693.

41. MacWILLIAM, J.A. Physiological Reviews. 1925; 5; 303.

42. MELVIN, G.S. & MURRAY, J. Quart. Jl. of Med. 1914; 7; 419.

43. MacWILLIAM, J.A. B.M.J. 1927; 1; 125.

44. ELDAHL, A. "Investigations into the oscilla-"tory method &c." Thesis, Copenhagen; 1933.

45. THOMPSON, R.J.C. & TODD, R.E. Lancet. 1922; 2; 503.

46. BLAVIER, L. Rév. Belge des Sciences Méd. 1932; 4; 559.