THESIS

Presented for the degree M.D.

EDINBURGH UNIVERSITY

ON

THE CORRELATION OF VITAL CAPACITY READINGS AND CLINICAL EXAMINATION:

With special reference to Vital Capacity Observations in obese subjects.

by

J O H N B. R O L T.

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I N T R O D U C T I O N.

Since the war a large amount of work has been done on the vital capacity of the lungs both in health and in disease. In America especially the subject has had wide publicity and strong claims have been made for it as an aid to diagnosis, and prognosis in cardiac cases.

The dyspnoea so commonly associated with cases of obesity interested me. Very little seemed to be known as to the cause of this excessive breathlessness "Fatty Heart" was nearly always suggested but could after all only be suspected.

In the hope that vital capacity observations on the obese person might throw some light on the question I set out to study from the point of view of the vital capacity a series of obese patients.

While working on vital capacity in general several other problems presented themselves. I have included in this thesis a study of the cases of a large series of coal miners from whom vital capacity readings had been taken.

With a view to observing how closely the vital capacity reading corresponded to the Clinical condition of the patient I have taken daily readings from/

from a series of cases showing heart failure.

Throughout a critical attitude has been adopted. The question as to how much help the clinician can expect from vital capacity readings has always been before me.

I wish to thank PROFESSOR MURRAY LYON for his kindness in allowing me to use the cases under his care in the wards and in the Dietetic Department, and in permitting me to work in the Clinical Medicine Laboratory.

THE HISTORY of the STUDY of VITAL CAPACITY.

As far as can be ascertained the first attempt to estimate the amount of air in the lungs was made by BORELLI in 1679. TURIN working at the same subject some years later, found that the greatest volume of air that could be forced from the lungs after the deepest possible breath, (the vital capacity) was 220 cu.ins. HALES also found that the vital capacity was about 220 cu.ins. GOODWYN found it to be in the neighbourhood of 200cu.ins.

Other workers showed variations from 100-250 cu.ins. For the most part their methods were crude and the results merely of historical interest. HUMPHREY DAVY in 1799 found his own vital capacity to be 213 cu.ins. Following on his work, the most important contribution is that of HUTCHINSON. In 1846 he published his paper, "On the Capacity of the lungs, and on the Respiratory Functions with a view to establishing a precise and easy method of detecting disease by the spirometer".

It was HUTCHINSON[1] who introduced the term vital capacity, as being the greatest voluntary expiration/

expiration following on the deepest inspiration.

He invented a simple form of spirometer of a type which remains practically unmodified at the present day. With this instrument he collected data from 2200 people, drawn from all classes and of whom 60 were diseased.

HUTCHINSON also paved the way to the accurate measurement of the tidal air, the supplemental air, and the complemental air and to the calculation of the residual air.

It was HUTCHINSON who first drew attention to the fact that a man's vital capacity is constant quantity, but that this quantity is modified by four circumstances. Height, weight, age, and disease and of all measurements height bears the closest relation to vital capacity.

Following on HUTCHINSON'S paper many workers studied vital capacity in men, women and children.

In 1854 WINTRICH (2) examined the vital capacity in 3500 individuals. He found that for each centimeter in height a man exhales 22-24 ccs. of air while women exhale 16-17·5 cc. Between the ages of 6-8 years in both boys and girls, 6·5-9cc of air are exhaled, from 10-12 years 11-13cc, from

12/

12-14 years 13-15 cc. per cm. of standing height.

He stated that age had a great influence on the vital capacity. The largest readings he obtained were between the ages of 20 and 40. There was a slight decrease between 40 and 50. Between the ages of 50 and 60 he found a great variation in the vital capacities. A well developed subject of 64 might have a vital capacity as good as those whose ages ranged from 44-45. After 75 there was a marked decline, approaching that of the childhood ages.

During the present century BOHR (3) made further studies on lung volume and showed its importance "in the understanding of the disturbed physiological processes in many pathological conditions", particularly, heart disease.

During the war the vital capacity test was used as a test of physical fitness, especially in the examination of candidates for the flying corps. A definite minimum standard of vital capacity was fixed more or less arbitrarily. A number of successful pilots were examined and the lowest vital capacity (3400 cc) was laid down as a minimum. No regard was taken of the size of the man. (11)

DREYER heard of this method and it stimulated him to investigate further the subject of vital capacity/

capacity. Others in America notably PEABODY, WENTWORTH, WEST, MYERS, were working on the same subject by 1920.

SUMMARY/

SUMMARY.

Not until 1679 did anybody investigate the vital capacity of the lungs, after that the subject was studied by several scientists; but it was not until 1846 that a really scientific investigation was made. In that year HUTCHINSON published his paper on the vital capacity of man.

Since then the subject of vital capacity has been worked on both in Europe and America; but even up to the end of the war there were no definite normal standards set up, although large numbers of men women and children had been examined.

By 1920 there were numerous investigators at work chiefly in America and since then the subject has had wide publicity.

THE PHYSIOLOGY OF RESPIRATION.

From the point of view of the Physiology of respiration the important portions of the lung are the terminal Bronchioli and their ramifications. These have been described by MILLER (7) as consisting of:-

(1) Bronchioli

(2) Bronchioli respiratorii

(3) **Ductus alveolares**

(4) Atria

(5) Sacculi alveolares

(6) Alvolares pulmonis.

The Bronchioli are the terminal portions of the bronchial tree which still have smooth walls. They divide into two or more bronchioli respiratorii their walls are irregular, because of the presence of shallow alveoli. And so they show the first evidence of haemo-respiratory function.

The Bronchioli respiratorii divide into Ductuli alveolares which have on their walls a larger number of deeper alveoli. Where the ductuli open into the atria the muscle fibres of the bronchial tree terminate abruptly in a sphincter-like ring.

The Atria which are also lined with alveoli serve as mixing chambers for the air which is distributed/

distributed to the principal respiratory elements, the sacculi alveolares, with their numerous alveoli pulmonis.

MILLER has limited the term "lobule" to mean the ductus alveolaris, with its atria, sacculi alveolares and alveoli pulmonis and their blood vessels, lymph-vessels, and nerves.

The alveoli, whenever they are found are lined by a single layer of epithelium, and may be considered as the glands of the pulmonary system. The alveoli of adjacent sacculi and lobules are very intimately associated, and in these thin partitions the capillaries of the pulmonary artery ramify and anastomose.

According to MILLER'S investigations the branches of the pulmonary artery on reaching a bronchiolus divide into three terminal branches. They ramify into capillaries one around the alveoli of the bronchiolus respiratorius another round the alveoli of the atria, and the third about the alveoli of the sacculi; toward the periphery of the alveoli the capillaries unite when they form the radicals of the pulmonary veins.

The atria sacculi alveolares and alveoli pulmones are considered as a whole and called the infundibulum/

infundibulum, in these, the expansion of the lungs, the major part of the mixing of gases, and their transference to and from the blood, takes place.

KEITH (8) has shown that the infundibula are larger in the basal parts and in the subpleural regions than elsewhere. On inspiration the base expands more easily and more extensively than does the apex and the subpleural part more than the root zone.

KEITH has pointed out the importance of the sphincter like musculature of the terminal bronchioli. He thinks that the more the calibre of the bronchioli is diminished during inspiration the more will be the negative tension in the infundibula. This bronchial musculature by diminishing or increasing the access to the infundibula in various parts of the lung may regulate the distribution of the indrawn air.

By regulating the intra alveolar pressure it may influence the distribution of the blood throughout the lung and so take the place of the vaso-motor mechanism which has not been proved to exist in the lung.

According to KEITH (8) the lungs expand like the/

the opening of a Japanese fan and not like a rubber bag. The part nearest the diaphragm and costal surface expand first and is followed by the apex and then the intermediate and root zones.

As we have seen the infundibula are larger in the basal parts than in the apex, and in the subpleural areas than the root zones.

In emphysema these large infundibula are the first to become distended and it is common knowledge that emphysema starts along the margins of the lower lobe particularly towards the diaphragmatic surface.

The diaphragm moves downwards and forwards and acts as a true piston. HASSE states that the spinal part elongates the thoracic cavity from above [illegible] downwards while the sterno costal part increases the antero posterior diameter of the lower thorax.

Diagraphmatic movement is the chief factor in inspiration 1·3 cm of movement represents about 400 cc of inspired air, thus there may be no relation as to the amount the chest can expand and the amount of air it can take in.

The costal series can be divided as follows:-

(1)/

(1)	First rib	Apices
(2)	2nd - 5th ribs	Upper lobes
(3)	6th - 10th ribs	Lower lobes apart of the diaphragmatic mechanism.
(4)	Floating ribs	Functionally part of the abdominal wall.

The movement of the diaphragm is dependent to a large extent on the muscles of the abdominal wall which are its antagonists. If the abdominal muscles have good tone the lower margin of the Thorax moves towards the domes of the diaphragm. If the ribs are fixed and the abdomen relaxed the domes of the diaphragm move towards the lower aperture of the Thorax. KEITH suggests that in viceroptosis the diaphragm has no fulcrum and as a result the respiration is largely thoracic.

In 1868 **HERRING** and BREUER (9) found that on dilating the lungs of an animal promoted expiration. Aspiration from the lungs on the other hand caused inspiration.

They propounded the theory of automatic regulation of respiration they assumed that the respiratory movements comprised a respiratory mechanism in themselves regulated by the centripetal fibres of the pulmonary vagi since these excite the inspiratory/

inspiratory centres when the lungs are expanded.

From their experiments it may be inferred that the vagi consist of two sets of fibres one of which when a certain phase of expiration is reached convey afferent impulses to the respiratory centre, and thus start inspiration, the other at a certain phase stimulates expiration.

The HERRING BREUER reflex therefore forms a means whereby the rate and depth of breathing is normally controlled.

HALDANE (10) working with MAVROGORDATO showed that the degree of response of the respiratory centre to the HERRING BREUER stimuli is determined by chemical factors. A very slight increase of free Co_2 caused the respiratory centre to send out more powerful efferent impulses. And to be less sensitive to the normal HERRING-BREUER inhibition and as a result there is increased depth of breathing.

The reverse is seen after voluntary forced breathing when an excessive amount of Co_2 is washed out from the blood and tissues and temporary apnoea results.

There is a degree of inertia of the respiratory centre, otherwise speaking swallowing, singing, playing/

playing wind instruments would be interfered with, or at any rate attended by embarrassment, as it is they are compensated for by a period of hyperpnœa which gets rid of the small accumulation of carbon-dioxide.

In cases of dyspnœa where the centre is already reacting to an excessive stimulus interruptions in breathing may cause great distress.(5)

Thus I have noted that cardiac cases who show dyspnœa on slight exertion are unable to hold their breath long enough to use the small spirometer. PEABODY states that cardiac cases are able to hold their breath just as long as normal people but it is probable that he refers to cases in which compensation is established.

THE/

THE LUNG AND ITS SUBDIVISIONS.

The total lung volume is made up by 5 portions.

(1) THE RESIDUAL AIR. That which remains in the chest after the greatest possible expiration.

(2) THE RESERVE AIR. That portion of air which can be expelled after a normal expiration

(3) THE TIDAL AIR. (The breathing air of HUTCHINSON). The volume of air inspired and expired during normal respiration.

(4) THE COMPLEMENTARY AIR. The largest amount of air which can still be inspired after normal inspiration.

(5) THE VITAL CAPACITY. The sum of the reserve tidal and complementary air. That is to say the maximum amount of air which can be inspired at the end of forced expiration or conversely the maximum amount of air which can be expired at the end of the largest possible inspiration.

These are the definitions given by HUTHCINSON and are used by the majority of Physiologists. Recent workers have used the terms reserve and complementary air in a slightly different sense and some confusion has arisen.

PANUM (4) and BOHR (3) introduce a middle capacity. If one stops breathing at a point midway between normal inspiration and expiration the amount of air left in the respiratory tract is called the middle/

middle capacity.

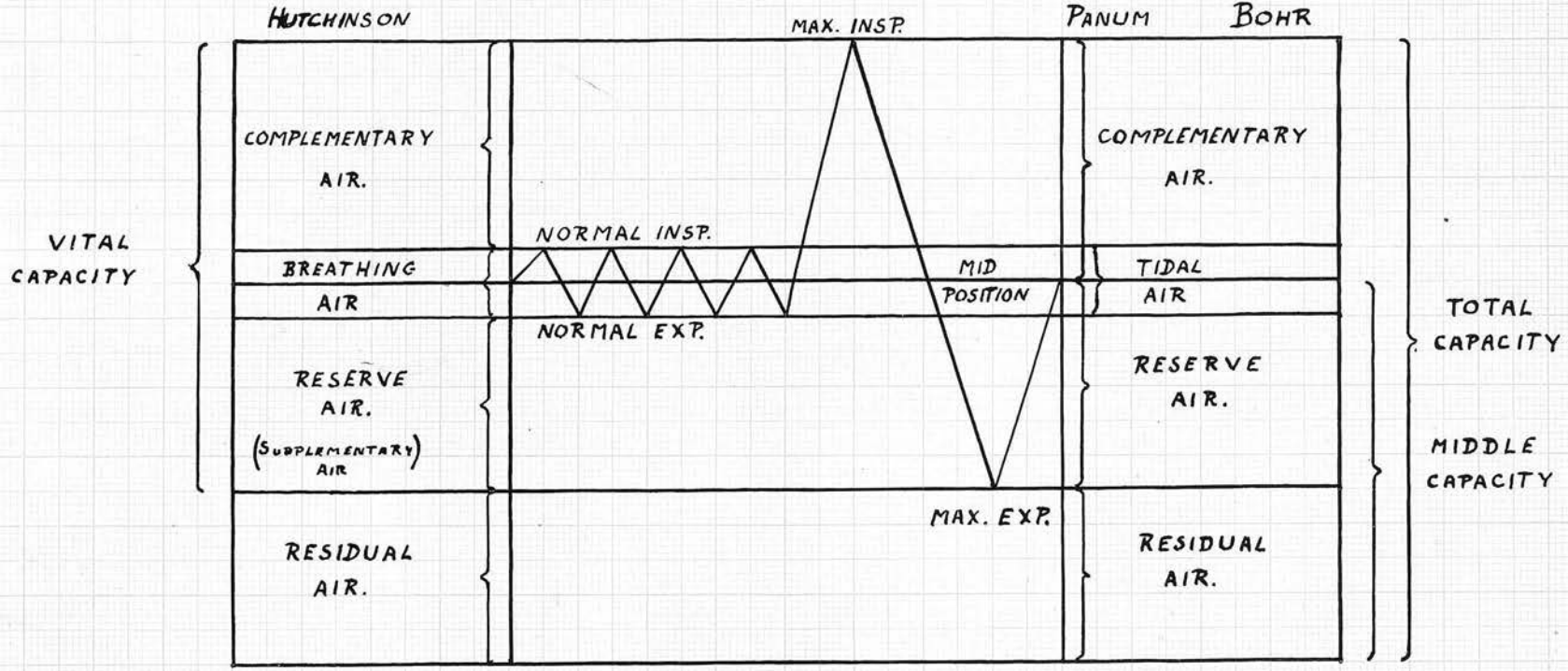
The maximum amount of air which can still be inspired is referred to as the complementary air, while all that can be expired is called the reserve air.

From the diagram it can be seen that the complementary air and reserve air of PANUM and BOHR are each greater than those of HUTCHINSON by half the tidal air.

MEAKINS and DAVIES (5) prefer the inclusion of the mid position. The average amount of tidal air varies greatly in different individuals and in a given individual at different times and under different conditions of rest and work. They think that the rather arbitary mid position is somewhat less variable.

BINGER and BROW (6) have introduced another subdivision the functional residual air, the total amount of air left in the lung after normal expiration. They found the normal expiratory position to be very constant in a given individual and they considered the functional residual air more constant quantity than the total capacity and vital capacity which to a certain extent represent "abnormal gymnastic achievements".

They/



THE TOTAL LUNG VOLUME AND ITS SUBDIVISIONS AS DEFINED BY HUTCHINSON AND BY PANUM AND BOHR.

They point out that the functional residual air can readily be ascertained without discomfort to the patient in cases such as pneumonia and pleurisy where it is undesirable to ask the patient to make forcible respiratory efforts.

So far as BINGER and BROW were able to determine the functional residual air was not correlated with any other body measurement.

THE/

THE CHOICE OF A STANDARD.

The ideal standard would be the individuals own, recorded in time of good health. In the vast majority of cases such is not available so we have to resort to a theoretical normal standard.

HUTCHINSON (1) in 1846 was the first to try and correlate vital capacity and other measurements of the body. He considered the relationship of vital capacity to height, weight, age and disease.

He found that height bore the most marked relationship to the vital capacity. "I find if I be allowed to take a man's height I can tell what quantity of air he should breathe to constitute him a healthy individual".

HUTCHINSON found that for every inch in height (from 5 ft to 6 ft) 8 cu.ins. of air at 60° are given out by a forced expiration.

Since HUTCHINSON'S work many British and American workers have investigated the subject but up to the end of the war HUTCHINSON'S height standard had not been improved on.

HUTCHINSON had given a normal standard

but/

but how close an individual had to approximate to these averages, in order to be classed as "fit" was not known.

In 1919 DRYER (12) published formulae for obtaining the normal vital capacity. He chose 16 men, & boys who were selected on account of their physical fitness and covered a wide range in height and weight etc.

A criticism which was at once brought up was how did **Professor** DRYER assess physical fitness.

From data obtained from these 16 subjects DRYER brought forward four relationships.

I. The vital capacity is a function of the weight. This can be expressed in the Formula $\frac{W^n}{V\ C} = K$, where W is the nett wt. of the body in grammes. V.C. is the vital capacity in cu. cms. and the power n is approximately 2/3 or •72 and K, is a constant. As it has already been established that $\frac{W^n}{S} = K_2$ where W = Nett wt. S = body surface and the power n = •72 it follows that the V.C. is a simple fraction of the body surface.

In other words the smaller and lighter/

lighter individual with his relatively larger surface has a greater vital capacity per unit of body weight than the larger individual.

II. The relationship between V.C. and stem length can be correctly expressed by the formula $\frac{\lambda^n}{V\ C} = K_3$ where λ = stem length in cms. V.C. = Vital Capacity in cu. cms. The power n is approximately 2 and K_3 is a constant.

The stem length is measured with the legs drawn up so that the individual rests on the ischial tuberosities. The sitting height of other observers is about 3% greater owing to the thickness of the gluteal muscles.

III. The relation between the vital capacity and the circumference of the chest can be expressed by the formula $\frac{ch^n}{VC} = K_4$ where ch = chest circumference expressed in cms. V.C.= vital capacity expressed in cu. cms. The power n is approximately 2 and K_4 is a constant.

IV. Finally $\frac{\lambda\ ch}{V\ C} = K_5$ where λ = stem length in cms. ch = chest circumference in cms V.C. the vital capacity in cu. cms. and K_5 is a constant.

CRIPPS/

CRIPPS GREENWOOD and NEWBOLD[13] examined 1000 Air Force Cadets together with other published data. They found that "the substitution of stem length for height does not improve the accuracy of the prediction and in general HUTCHINSON'S method is as good as, or better than recent modifications".

ROGERS[14] found the standing height standard quite satisfactory.

WEST[15] examined data from 129 individuals Harvard medical students and nurses from the Peter Bent Brigham Hospital, Boston. 85 of his males gave results which approximated closely with those calculated from DREYER'S weight formula.

WEST also took up a suggestion of PEABODY and WENTWORTH who had found in a few observations that there was a close correlation between the vital capacity and the surface area of the body.

WEST used the height, weight formula of DU BOIS.

He found that the vital capacity expressed in litres was 2·5 times the body surface area (sq. metres) in men and twice the body surface area in women.

MEYERS found that in women the ratio was 1·87/

1·87 litres per sq. meter of body surface.

WEST using his surface area standard found that in his comparatively homogeneous series of subjects the individual limits of variation are wide and he concludes that too much weight should not be given to a single determination unless a variation of more than 15% from the chosen standard is observed.

WEST showed that 71% of his subjects had vital capacities within 70% of normal 5·5 were below 90%. He also found that the vital capacity in c.cs. was 25 times the height in cms. in men and twenty times in women. In this case 63% were within 10% of normal.

Further work by MYERS[16] led him to produce two empirical formulae for determining the approximate normal vital capacity for men and women.

17·6 X body wt. + 900 = V.C. for women.

21·2 X body wt. +1168 = V.C. for men.

In 1280 men aged from 17-32 years 11% had vital capacities below 85% and in 1058 women aged from 17-23, 27·1 had vital capacities below 85% of the normal.

HEWLETT and JACKSON[17] examined 400 healthy/

healthy young men and on a basis of their own results with those of SCHUSTER, who examined 959 Oxford undergraduates and WEST examining Harvard students deduced the following formula:-

$$V.C. = 50\ H - 4400$$

$$V.C. = 2900\ S.A. - 1000.$$

where V.C. = vital capacity in cc^s

H. = height in cms.

S.A. = Surface area in sq. meters using the DU BOIS formula.

Using their own figures together with WEST'S they deduced another formula including both height and weight.

$$V.C. = 27\ W + 31{\cdot}4\ H - 3000.$$

W being the weight in kilogrammes.

Comparing the observed vital capacities and those deduced from this formula they found 20% were below 90% of the calculated value, 11% below 95%, 5% below 80%, and 2% below 75%.

They say that a reduction below 70% of the calculated normal is almost always pathological.

The correlation of vital capacity with height and vital capacity with weight were approximately equal while the correlation with height and weight/

weight together is slightly better than with either separately.

Since DRYER'S presented his formula in 1919 he has made an extensive study of vital capacity and came to the conclusion that vital capacity is more closely correlated with surface area than any other measurement.

LEMON and MOERSCH writing in 1924 confirm this view.

It would seem that standard tables based on surface area weight or standing height are the best.

In emaciated cases it is best to use the patients normal weight.

In obese cases it may be best to use the standing height standard.

In practice WEST'S formula is the simplest and most convenient method of estimating what the normal vital capacity of any individual should be.

SUMMARY/

S U M M A R Y.

The difficulty of deciding on some standard is one reason why measurements of vital capacity have not been used more widely.

Three methods have been found satisfactory.

(1) Standing height.

(2) Surface area.

(3) Average of chest measurement and stem length (DRYER)

WEST conluded that the most accurate standard was the use of the DU BOIS surface area with an allowance of each sq. meter of 2500 cc. for men and 200 cc. for women and children. This last figure has been modified to 1920 cc. by MYERS. These figures represent an average based on reports of WEST. LEMON & MOERSCH and MYERS.

PEABODY & WENTWORTH used the standing height standard and many observers have found this quite staisfactory for quick clinical work though the results were admittedly rather more variable.

Chest measurements and stem length are considerably less accurate and weight alone especially in fat and thin persons (a very important class) often gives results which are quite misleading/

misleading.

Of the three satisfactory methods surface area is the most reliable. Height is the simplest and is used extensively by some.

A criticism that has been levelled against the surface area standard is that, as the patient loses weight and vital capacity as a result of his illness, the loss of weight will,itself lower the calculated surface area,thus minimising or perhaps hiding the real loss of vital capacity. This is perfectly true but in practice it can be got over by using the patient's usual weight while in health.

In my series I have used the surface area standard using MYER'S tables.

In the obese cases the height standard has been employed.

SOME FACTORS WHICH INFLUENCE THE NORMAL VITAL CAPACITY.

It has been pointed out by many observers that physical training or hard manual labour goes hand in hand with a high vital capacity. Singers, players of wind instruments, or glass blowers are expected to have large vital capacities.

But now and then cases are met with large capacities and no history of having ever played games seriously, individuals with long chests and low placed diagraphms. The hyposthenic or asthenic type described by MILLS fall into this class.

In my series of normals it will be seen that many show capacities in excess of 100%. The majority of subjects are medical students or nurses and, consequently have at one time or another played games.

The class showing a normal vital capacity of well over 100% is not only a very big one but is also very important.

DREYER and others have stated that a 15% decrease/

decrease from the expected normal, probably indicates the presence of some health depressing factor.

DREYER believes that occupation has a great influence on vital capacity and he groups all people into three classes.

He believes that an individual who spends a considerable time in the open air playing games etc. may bring himself into class A regardless of his occupation.

FOSTER/

FOSTER and HSIEH (18) found that occupation influenced vital capacity to some extent although not in proportion to the degree of physical activity.

SEX. FABIUS and later SCHNEEVOGL and then WINTRICH pointed out that there was a difference in the vital capacity of men and women.

MYERS states that WINTRICH found the sex variation very slight during early years, but becomes more marked after the fifteenth year when boys show a larger capacity. About 6% according to WILSON and EDWARDS.

The differences due to sex are such that after young adult life separate normal standards are required.

AGE. HUTCHINSON stated that an individual reached his maximum vital capacity about the 30th year.

Recent observation by numerous workers indicate that the maximum vital capacity is reached at about 20 in boys and a little earlier in girls. (Stewart)

BOWEN and PLATT (19) found that up to the age of 50 years the average vital capacity was 95.8% of the normal. Then it gradually reached 50% at the age/

age of 85 years. The greatest fall in any decade being between the ages of 50 and 60 years.

OBESITY. HUTCHINSON stated that 7% overweight interferes with the vital capacity. More recent investigation has shown that only extreme obesity is capable of reducing the reading. The subject will be discussed more fully later on.

POSTURE. It has been found that a subject lying down records a smaller vital capacity than when standing or sitting.

RABINOWITCH (20) investigated the problem and found that the obtained vital capacity in the recumbent position has to be multiplied by 1·075 the product being very near the normal capacity, the patient would present, had the reading been taken standing.

CHRISTIE and BEAMS (21) report on 5·5% reduction of vital capacity when the reading is taken with the patient lying down. This figure has been generally accepted and I have used this correction in the cases I report when for any reason it was impossible to have the patient sitting or standing.

Naturally any cramped or awkward

position/

position such as bending forward owing to too short a tube leading to the spirometer will cause a low reading. Tight clothing whether it be stays or an unduly tight waistcoat or trouser top will reduce the vital capacity.

The observer must always satisfy himself that there is nothing constricting the chest and abdomen.

RACE. It has been pointed out by American writers that coloured races have a lower vital capacity than the Western (MYERS).

LACK OF CO-OPERATION AND WILL POWER.

I have found that there is quite a large class of patients who are quite unable to take the deepest possible breath and then expire to the greatest possible extent. Several cases which I started to investigate from the point of view of their vital capacity, I have had to give up owing to their inability to record reasonably regular readings. All such cases have used the spirometer every day for a week and if, at the end of that time, they have not shown a marked improvement, I have given up the unequal struggle.

Women/

Women of the hospital class are the worst offenders. Some of them seem quite incapable of taking a deep breath or of emptying the chest as far as possible. The majority are of the highly strung type who would, in any case, be incapable of doing anything to order which required a little concentration and effort.

I speak of course, of the comparatively fit patient, one can understand the dyspnoeic cardiac case not co-operating as well as she might, though my experience is that these cases really do try especially/

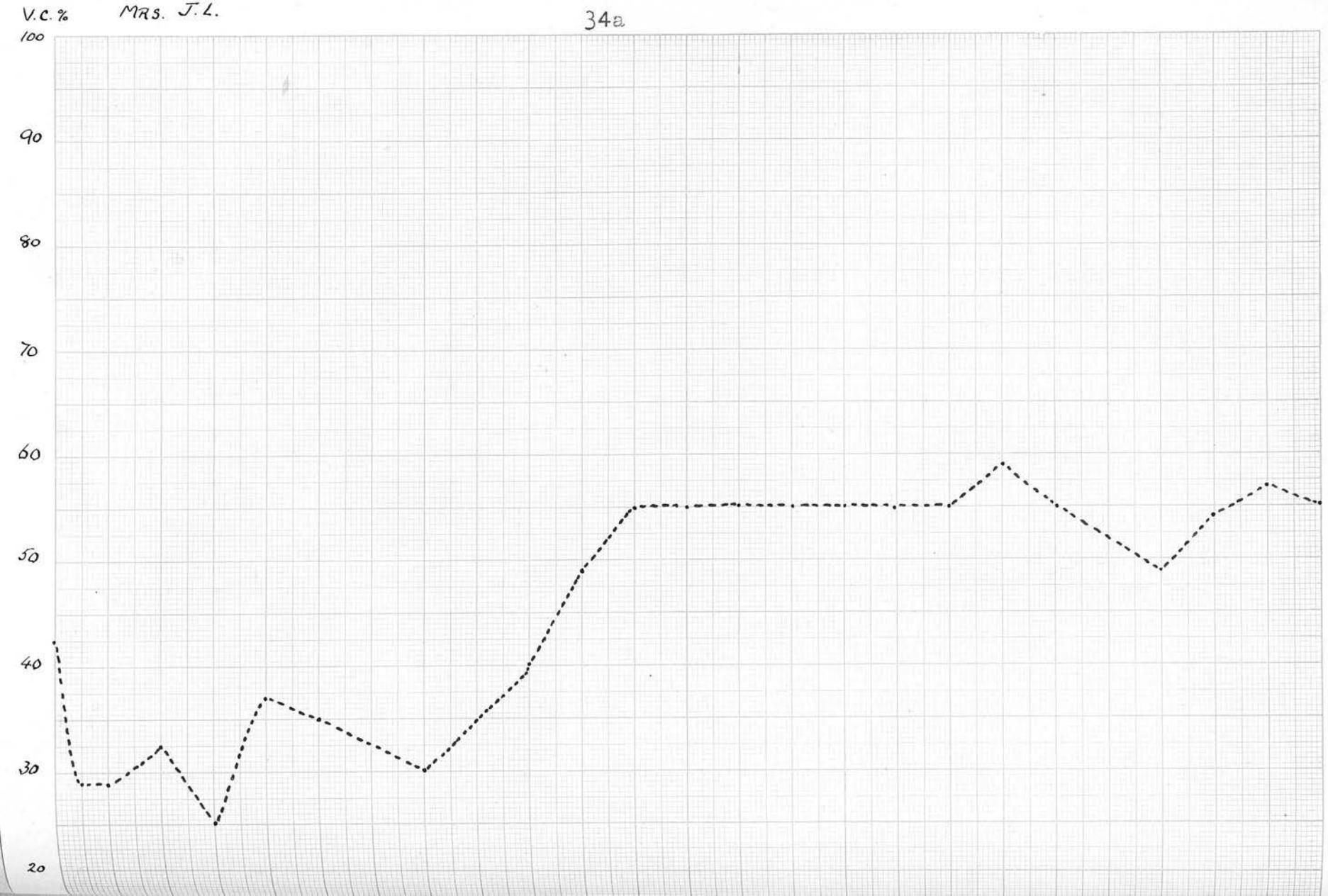
especially when they see the readings improving day by day.

I do not think I exaggerate when I say that the majority of women of the hospital type over 40 years of age, are unable to cooperate to the fullest extent of their powers. But when a long series of daily readings are taken, one is able to set up a standard for that particular patient, and provided the same observer takes each reading, the series will be comparable.

The spirit of competition has to be encouraged, almost any healthy young adult male will blow two or more hundred cc. than his previous best if a competition be started. In the case of women the results are not always so satisfactory.

Malingering is so easily detected that it calls for no special description.

SUMMARY/


V.C. %
MRS. J. L.
100
90
80
70
60
50
40
30
20

The following two examples of LACK OF COOPERATION and WILL POWER are of interest.

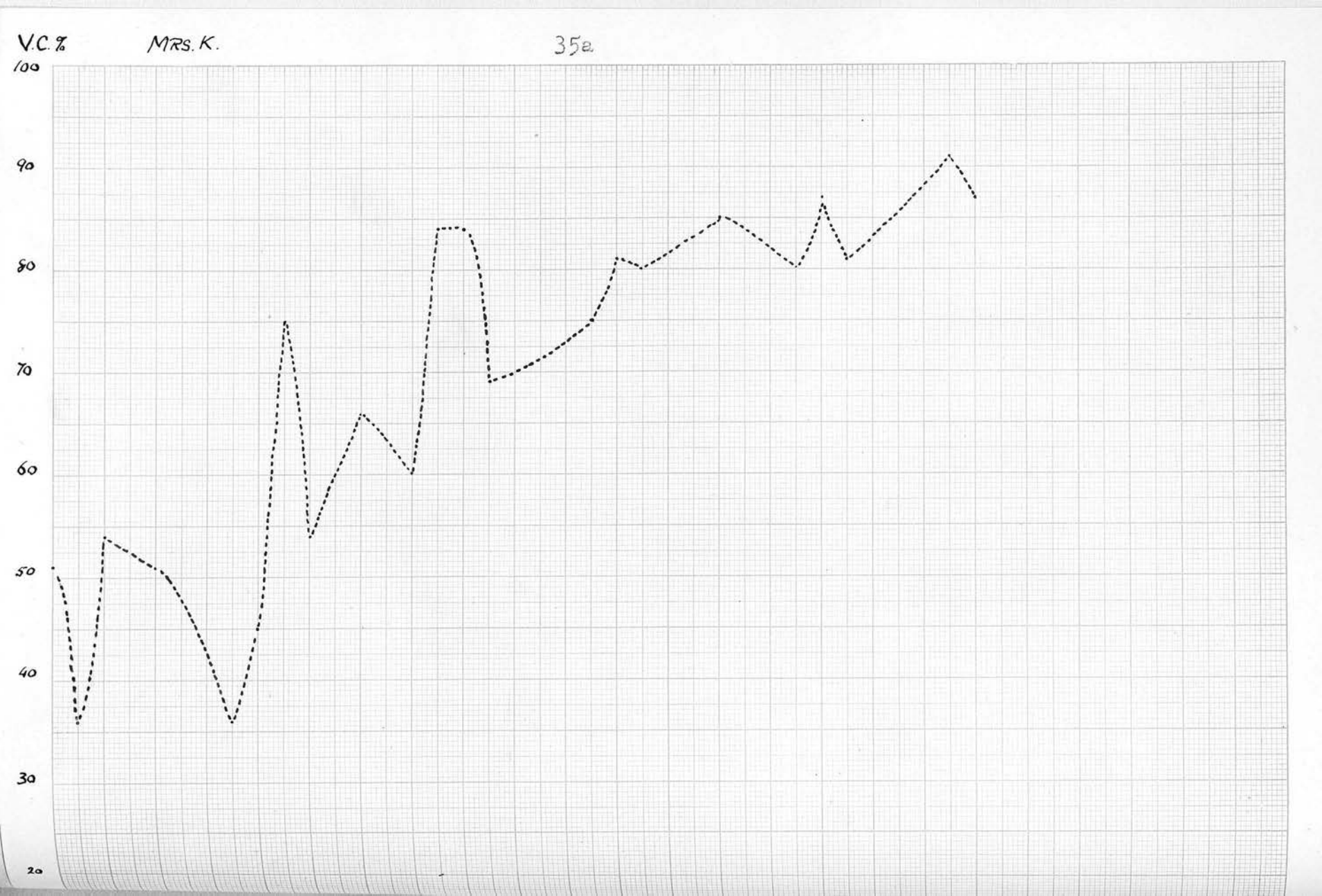
MRS J. L.

The patient was suffering from a primary Carcinoma of the left lung.

The chart shows a gradual increase in vital capacity until 15/11/29.

This increase was quite contrary to the clinical aspect of the case, and may be explained by the fact that the patient took 10 days to learn to inspire and expire to the fullest extent of her powers.

All the readings from 5/11/29 - 15/11/29 are quite valueless.


V.C.%
MRS. K.
35a
100
90
80
70
60
50
40
30
20

MRS K.

The Patient was admitted as an asthmatic, but the diagnosis was revised to that of Neurosis. Daily vital capacity readings were taken and only after 4 weeks were anything like reliable readings obtained.

S U M M A R Y.

The vital capacity of normal healthy people may be influenced by several factors.

Physical training and certain occupations may result in an over development of the vital capacity, while other occupations may decrease it.

Sex effects the vital capacity to such a degree that it is necessary to have separate standards for men and women. There is a fall in vital capacity after the age of 50. In dealing with obese persons it must be remembered that the usual correlation between weight and surface area breaks down.

A reading taken with the patient lying down is 5·5% less than if the subject were standing.

Race and nationality effect vital capacity. Some patients are quite unable to cooperate with the observer. It is essential that the observer should encourage the patient to do better on the second attempt.

DISEASES/

DISEASES WHICH INFLUENCE THE VITAL CAPACITY.

Diseases besides those of the lungs and in particular those of the heart can cause a reduction in vital capacity, old standing Pleural adhesions. It has been shown that cases having old standing pleural adhesions from one cause or another may show a decrease in vital capacity.

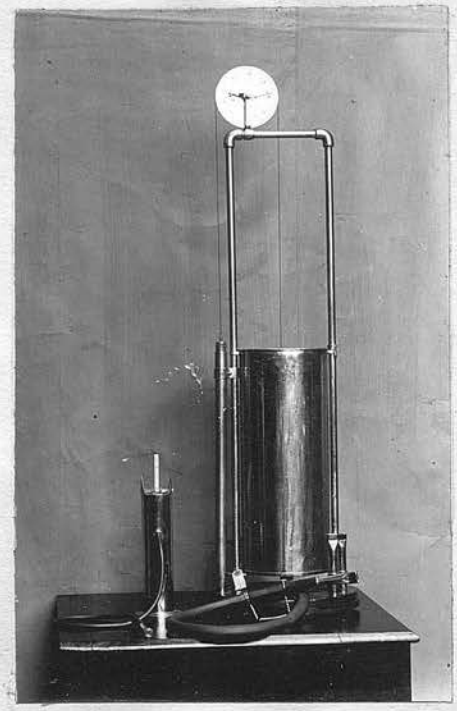
MYERS states that in 54 cases of thickened pleura as revealed by X rays, the average vital capacity was 94·1% while 26 cases of healed empyema had an average vital capacity of 91%. Old deformities of the Thorax and ossification of the costal cartilages and costo-vertebral joints reduce the vital capacity to some extent.

The chest capacity is increased by the elevation of the ribs and sternum and the descent of the diaphragm. Any condition which prevents the usual movements of the Thorax will decrease the vital capacity.

Factors of clinical significance which decrease vital capacity.

Cardiac disease, Hyperthyroidism, asthma, emphysema, bronchitis, pleuresy, pneumonia, pulmonary abcess, bronchiectasis, new growths of the lungs and mediastinum/

mediastinum. Aneurism pneumonia, and pulmonary Tuberculosis. MYERS. (22)



The photograph shows the two types of Spirometer used.

On the left is the very portable Pneumameter, on the right the Standard Sanborn Spirometer.

THE CHOICE OF A SPIROMETER.

Unquestionably the most accurate type of Spirometer are those of the wet type. The dry type is easily transported, but needs constant checking against an instrument which is known to be accurate.

The Modern Sanborn Spirometer is the one in general use, and is every way reliable. It has the great disadvantage, however, of being very bulky. It stands about 3 ft. high and contains a gallon or more of water.

Even the most enthuasiastic worker would find the instrument very cumbersome to carry from ward to ward every day. It is not of course meant for such work.

The writer looked around for a more convenient instrument, and found one made by the Electric & Manufacturing Company of London. It is called the OLIVER-PELL Pneumameter.

This Spirometer is of the water type but is only 11 ins. high. Each instrument bears a certificate of accuracy from the National Physical Laboratory.

I found it invaluable for taking normal readings and also for cases in the wards.

There/

There is no question that it is perfectly accurate. It has, however, its disadvantages.

(1) A certain amount of skill is needed in controlling the rate of blowing. This did not prove a great draw back, the vast majority of patients mastering the instructions after one or two attempts.

(2) The instrument is unreliable in cases which show extreme dyspnoea. They are unable to hold their breath long enough to record a proper reading. In any doubtful cases the reading was checked, and if necessary disregarded, against the SANBORN Spirometer.

Using the Pneumameter about 15 seconds are necessary to record a vital capacity of 5000 cc. and a practical minimum of 5 seconds to record the vital capacity of a cardiac (1500 cc.)

It is quite impossible to get a wrong reading if the following instructions are carried out.

As I believe that this is the first instrument of its type to be used in EDINBURGH I have written out the instructions in full.

In order to put water into the instrument the inner cylinder should be removed. This is done by taking the pointer between the finger and thumb and raising it until the cylinder is lifted right out.

The water should be poured into the outer cylinder to a point indicated by the ring on the air inlet/

inlet tube which is about 1¼" below the rim of the outer cylinder.

When replacing the inner cylinder the double spoke seen at its base, should be directed away from the inlet tube.

After gently pulling up the ivory scale to the zero mark, according to natural yielding (no quick force should be exercised) the user should first, by way of practice keep the rising cylinder poised in the space between the uprights by means of gentle blowing. When this can be easily managed the scale should be reset to zero, and a full expiration can be measured by keeping the cylinder posed or playing within the limits of the uprights so long as expiration lasts. At the end of expiration the cylinder will fall, the scale is automatically fixed and can then be read.

If the guiding levers go above the uprights or touch the bottom of the slots during the observation then the scale must be returned to zero, and the user begin again.

Three glass mouth pieces are provided and are placed in disinfectant or washed after use. A fresh one being provided for each patient.

TECHNIQUE/

TECHNIQUE AND CALCULATIONS.

In taking the vital capacity, the subject is instructed to take the deepest possible inspiration and exhale as much as possible into the mouth piece.

Three trials are allowed and the highest reading is recorded as the vital capacity . It is just as well to point out to the patient how much he has blown, and to encourage him to try and beat that at the next attempt. A patient blows far better if he watches the scale. A figure a little beyond his powers is mentioned as being about the average for his size, and in this way the observer can hope to obtain the patient's full cooperation which is so essential to success. Malingering is so easily detected, that it is quite safe to "give away" the figure one expects the patient to reach.

The reading is taken with the patient standing in front of the instrument which is placed on a table or shelf so that it is level with his mouth. All tight clothing is loosened.

Patients in bed should whenever possible sit up. If this is impossible the reading is taken them lying down, a correction of 5.5% being added to the result. (CHRISTIE & BEAME (21))

The standing height is taken and also the weight/

weight, suitable allowance being made for clothes. From Life Assurance Tables the percentage of the theoretical normal which the actual weight represents can be worked out.

To find the Surface Area, a graphic chart completed by BOOTHBY & SANDIFORD after the work of DU BOIS & DU BOIS was used.

As great difficulty was experienced in obtaining such a chart the reproduction in MYER'S book on vital capacity was photographed and enlarged to 12" X 10" and so accurate readings were assured.

By the aid of this chart one may obtain the body surface area of an individual when only weight and standing height are known. No mathematical calculations are necessary.

At first I worked out the % of the theoretical normal vital capacity of the patient, but as soon as I began to realize that it was unnecessary to be accurate to .5%, the tables given by MYERS were used, hence all my results are in round figures. The following examples will make the method quite clear.

MR A. AGE 26. WEIGHT 162 lbs. HEIGHT 5'8"

v.c. 4500.

Surface/

Surface area 1.85 (From BOOTHBY & SANDIFORD'S chart). From WEST'S work we know that a man should blow 2.5 litres for each sq. meter of body surface so the theoretical normal in this case would be :-

1.85 X 2.5 = 4625 cc.

The actual amount he blew was 4500 or 97% of the theoretical normal.

MRS B. AGE 30. WEIGHT 140 lbs. HEIGHT 5'9"

v.c. 3500.

Surface area = 1.75

From the fact that LEMON & MOERSCH and MYERS found that women blow 1.92 litres of air per sq. meter of body surface the theoretical normal would be :-

1.75 X 1.92 = 3325 cc.

The actual amount blown was 3500 or 104% of the theoretical normal.

In using the standing height standard the patient's height is converted to centimetres and in the case of a man WEST found that 25 cc. of air were blown, for every cm. in height and 20 cc. per centimeter in the case of women.

In the case of MR A.

HEIGHT 5'8" = 173 cms. v.c. = 173 X 25 = 4325 cc.

He blew 4500 or 104% of the theoretical normal.

In/

In the case of MRS B.

HEIGHT 5.9 = 175 cms. v.c. 175 X 20 = 3500 cc.

100% of the theoretical normal.

A G E G R O U P S.

	20 - 24	25 - 29	30 - 34	35 - 39	40 - 44	45 - 49
NO. of CASES	17	18	11	11	7	6
AVERAGE V.C. S.A. STANDARD	102%	101%	97%	97.8%	93.7%	88%
" " Ht. STANDARD	104%	105%	100%	98.3%	93%	94%
" " Wt. STANDARD	105%	106%	99.9%	10.1%	97.4%	91%

VITAL CAPACITY READINGS ON NORMAL SUBJECTS.

In order to test the various standards which I proposed to employ, a series of 70 normal subjects were examined.

The majority were medical students, porters or patients in the wards. Several nurses were included. None of them had any history of past respiratory disease. In six of the medical students, minor cardiac abnormalities were found. As these appeared to have no clinical significance, those subjects have been included.

The medical students were all taking an active part in some form of athletics. This I think accounts for the rather high readings in the younger age groups.

AGE/

C O N C L U S I O N S.

It will be seen that the height and weight standard tend to give slightly higher readings. The height standard approximates more closely to the surface area than the weight standard. The average error for allages being + 2.45 v.c. in the case of the height standard and + 3.5% v.c. for the weight standard over the S.A. standard.

The average V.C. by the surface area standard for all age groups was 96.6%.

VITAL CAPACITY READINGS FROM 70 NORMAL ADULTS.

CASE NO	AGE	WEIGHT St. Lbs.	HEIGHT Ft. Ins.	SURFACE AREA	VITAL CAPACITY	% V.C. .A. STANDARD	% V.C. Ht. STANDARD	% V.C. Wt. STANDARD	SEX
1	27	12•7	6•1	2	5600	112	121	115	M
2	27	12•11	6•2	2•05	4800	94	101	96	M
3	30	11•4	6•2	1•9	5250	114	112	110	M
4	26	11•3	5•9	1•85	4800	104	109	105	M
5	25	11	5•11	1•9	5000	105	111	112	M
6	20	11•12	6	1•95	4500	89	99	96	M
7	24	11	5•11	1•9	4900	104	108	109	M
8	24	11•7	5•9	1•87	4600	99	105	100	M
9	25	10•7	5•9	1•8	5400	119	124	123	M
10	23	11•4	6	1•9	5400	112	119	118	M
11	40	10	5•10	1•91	4300	104	97	103	M
12	24	9•2	5•4	1•6	4000	125	123	125	F
13	30	12•12	5•1	2•04	6100	120	130	124	M

14	26	11•10	5•8	1•85	4400	95	101	96	M
15	20	11•10	5•10	1•86	4000	86	890	86	M
16	21	11•6	5•11	1•9	5300	112	118	115	M
17	26	10•7	5•9	1•8	4600	102	105	106	M
18	28	8•7	5•2	1•51	3700	91	86	99	M
19	22	12	5•11	1•95	4900	99	109	105	F
20	27	10•2	5•9	1•77	4500	102	103	108	F
21	24	11•12	5•9	1•89	4100	87	94	88	F
22	26	10•7	5•9	1•8	4600	102	105	108	M
23	35	9•4	5•6	1•65	3400	109	101	106	M
24	30	10•5	6	1•85	4500	97	101	105	M
25	20	12	6•2	2	5300	106	112	113	M
26	34	10•4	5•6	1•72	3700	88	88	89	M
27	23	11•4	5•10	1•87	5200	111	116	116	M
28	43	8•5	5•3	1•51	2800	92	88	90	F

CASE NO	AGE	WEIGHT St. Lbs.	HEIGHT Ft. Ins.	SURFACE AREA	VITAL CAPACITY	% V.C. S.A. STANDARD	% V.C. Ht. STANDARD	% V.C. Wt. STANDARD	SEX
29	27	10·7	5·9	1·81	5650	122	128	127	M
30	32	9·12	5·5	1·63	3700	91	91	91	M
31	31	10·4	5·6	1·7	4000	94	96	96	M
32	47	13·	5·9	1·95	4000	83	91	82	M
33	22	8·2	5·4½	1·5	2900	97	89	102	F
34	21	7·8	5·3½	1·45	3000	103	94	109	F
35	24	9·6	5·4	1·61	3400	105	105	106	F
36	36	7·10	5·3	1·48	4000	108	100	118	M
37	24	13·2	6·1	2·05	5600	109	121	112	F
38	35	9·12	5·10	1·75	4900	112	110	121	M
39	22	10·4	5·7	1·72	3800	87	89	91	M
40		8·10	5·4	1·36	2500	84	77	85	F
41		9·	5·2	1·55	3200	107	102	103	F

42	37	10•1	5•9	1•75	4700	107	107	115	M
43	36	11•6	5•11	1•9	4600	97	102	103	M
44	29	9•11	5•7	1•7	3600	85	85	90	M
45	39	12	5•1	1•92	4600	96	107	98	M
46	25	10•3	5•11	1•8	4800	107	106	115	M
47	41	11•1	5•5	1•72	4300	99	104	98	M
48	48	10•5	5•11	1•84	3600	79	80	86	M
49	39	10•7	5•7	1•75	4100	94	97	95	M
50	43	9	5•5	1•6	3300	83	80	89	M
51	48	12•2	5•6	1•84	4100	90	98	88	M
52	39	12•2	5•8	1•87	3800	81	88	81	M
53	40	11•11	6•1	1•98	4700	95	107	102	M
54	41	9•12	5•10	1•8	4200	93	94	105	M
55	34	10•13	5•8	1•8	4300	96	100	98	M
56	29	11•11	5•10	1•92	4800	100	108	105	M

CASE NO	AGE	WEIGHT St. Lbs.	HEIGHT Ft. Ins.	SURFACE AREA	VITAL CAPACITY	% V.C. S.A. STANDARD	% V.C. Ht. STANDARD	% V.C. Wt. STANDARD	SEX
57	28	10•2	5•11	1•8	4500	100	100	108	M
58	26	11•1	5•7	1•8	4650	103	110	106	M
59	27	11•13	5•9	1•9	4100	86	94	88	M
60	33	12•9	5•10	1•95	4200	86	94	86	M
61	33	10•1	5•6	1•7	4400	101	105	106	M
62	41	9•6	5•6	1•65	3700	90	88	95	M
63	33	13	5•11	2	4400	88	98	90	M
64	38	10•13	5•7	1•77	4100	94	97	94	M
65	38	11•	5•7	1•76	3800	16	89	87	M
66	39	10•4	5•10	1•77	4100	93	92	98	M
67	33	10•4	5•8	1•75	4100	94	95	98	M
68	48	11 11	5•11	1•89	4700	100	104	105	M
69	46	11	6	1•91	4300	90	96	96	M
70	45	10•9	5•9	1•83	4100	90	94	94	M

VITAL CAPACITY READINGS FROM 140 COAL MINERS.

A statement by MYERS "not enough work has been done to determine the effect of pneumonoeoniosis on the vital capacity" interested me.

I found that Dr. A. RAE GILCHRIST had examined a large group of miners who were candidates for the "Rescue Gangs" in the various mines concerned.

Dr. GILCHRIST had taken vital capacity readings on these men but the readings had never been worked out. He very kindly gave me permission to use the case records.

I have worked out the percentages in 140 cases from the point of view of the surface area height, and weight standards.

In the majority of cases two vital capacity readings had been taken at the interval of

1/

1 year. As a very careful clinical examination was carried out on each patient. I think an analysis of these readings should be of value.

All the men have worked as miners from their youth up, with the exception of some who were away during the war.

All were engaged in Bituminous coal mines, around Edinburgh and the vast majority work underground.

PNEUMOKONIOSIS or dust disease of the lung comprises all the pathological changes induced in the bronchi lungs, and pleura by the inhalation of dust particles. It is one of the occupational diseases and is practically limited to men. Usually it develops between 25 and 40 years of age.

Many forms of dust will cause pneumokoniosis both organic and inorganic. The harder and more gritty the particles, the greater is the change produced in the chest. As a rule organic forms cause bronchitic changes, while inorganic dust leads to fibrosis of the lung.

Anthracosis is the variety due to coal dust.

Siderosis or "grinders rot" is due to metalic dust.

Silicosis/

Silicosis is found in quartz workers, workers in slate quarries, and gold mines, etc.

Byssinosis is rare, and is found in cotton and felt workers. A type due to Asbestos dust has recently been described.

PATHOLOGY. The lung of the country dweller is nearly free from deposits of pigment. Town dwellers always show a certain amount of carbon which gives the lungs a dark grey mottled appearance, but no fibrosis of the lungs is produced.

In coal mines the lungs are quite black but even here little fibrosis occurs, unless the men are working in steam coal (anthracite) mines where the coal is very hard.

In Siderosis and Silicosis fine sharp particles are deposited in the lungs.

Particles of dust are conveyed to the bronchi and alveoli by inhalation. This is the generally accepted theory, but Calmette and others have suggested that the dust is swallowed and the particles reach the lungs through the mesenteric and bronchial glands. A good deal of experimental work has been done to prove this, but the results seem so contradictory, that I do not think we should gain anything by quoting them.

Even/

Even if a small amount is absorbed from the stomach, it would be a very small quantity compared to what is inhaled.

The natural protection against particles of dust reaching the lungs is very efficient. Most of the dust is detained in the nose, but in mouth breathers it will readily gain access to the trachea and bronchi. Even then the ciliated epithelium will tend to waft the coarser particles up again, and they will be discharged with the expectoration.

It has been proved, however, that repeated inhalation of dust laden air, produces irritation in the bronchi. Some of the epithelium desquimates and absorption of the particles takes place. As a further consequence of this initial bronchitis, the finer particles may reach the alveoli and passing between the epithelial cells, reach the tissue spaces or they may be taken up by the special "dust cells".

Particles of dust become deposited in the connective tissue and chronic peribronchial and perialveolar fibrosis develops.

The bronchial glands become enlarged and pigmented by particles carried by the Lymphatics.

Emphysema, pleural adhesion and bronchiectasis develop.

The question of predisposition to tuberculosis has been much discussed. It has been shown that coal miners are remarkably free. In fact they suffer less from the disease than any other male community in this country.

Silica on the other hand definitely seems to predispose to Tuberculosis, gold miners are especially liable.

HALDANE & MAVROGORDATO have shown that coal particles are obsorbed by the "dust cells" whose movements are thereby stimulated with a result that they appear in the expectoration. "Black spit" is therefore a healthy sign.

Silica is also taken up by these cells but they are not stimulated and consequently remain in situ.

POST MORTEM. the lungs are firm and pigmented, the pleura is generally adherent, especially the bases. On section the lungs are firm and often gritty fibroid changes are especially marked in Silicosis. The bronchi are inflamed and sometimes dilated. Some degree of emphysema may be present.

Microscopically in silicosis the alveolar walls are thickened and the connective tissue increased/

increased, "Dust cells" may be seen in the connective tissue or in the alveoli, and particles of silica are found widely deposited in the cells of the connective tissue. R.A. YOUNG (45) and G.E. BEAUMONT. WILLIS (46) has done some experimental work with soft coal dust on animals. He finds that after 1 year's exposure, there is some lymphoid tissue proliferation in response to the dust but no development of fibrous tissue.

SUMMARY/

S U M M A R Y.

It would seem that anthracosis cause very little if any harm to the lungs. The work of HALDANE & MAVROGORDATO and WILLIS tends to confirm clincal experience on this point.

Silicosis on the other hand is a serious disease, causing gross damage to the lungs.

From this we should expect to find very little, if any change, in vital capacity readings from bituminous coal miners who presumably suffer from some degree of anthracosis.

VITAL CAPACITY READINGS FROM 140 COAL MINERS.

A G E G R O U P S.

AGE	20 - 24	25 - 29	30 - 34	35 - 39	40 - 45	45 - 49	
NO. of CASES	7 cases	34 cases	30 cases	28 cases	30 cases	11 cases	

The average vital capacity for all ages was found to be 92% which is very close to MYERS 91.4%.

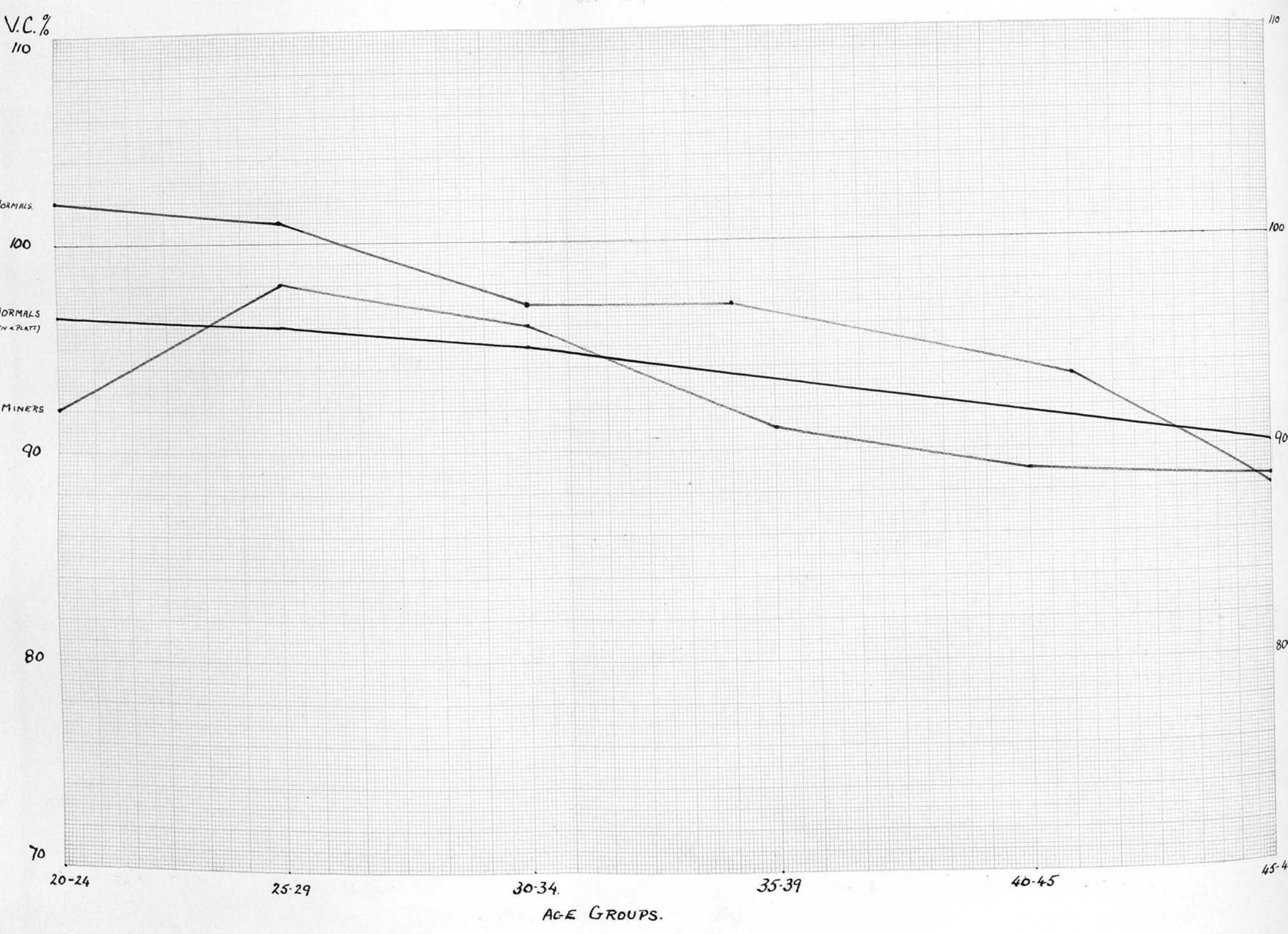
On searching through the papers on vital capacity the only work I could find was by MYERS (4) who examined 80 Bituminous coal miners in 1924.

In this series no clinical examination was made. MYERS found that the average vital capacity for all ages was 91.4%. There was no decrease in capacity up to the age of 46 years, the lowering of the capacities after that could quite well be explained by advancing age.

Only after 24 years work in the mines was any decrease in the readings noted and here again the age factor would quite well explain the reduction.

I have divided up the 140 miners into age groups as follows:-

TABLE/


V.C.%
110
ORMALS.
100
ORMALS
N & PLATT)
MINERS
90
80
70
110
100
90
80
20-24
25-29
30-34.
35-39
40-45
45-4
AGE GROUPS.

The three curves in the graph represent.

(1) My series of 70 normal people (Red)

(2) The series of 140 miners. (Green)

(3) BOWEN & PLATT'S curve for 100 normal people. (Black)

The ordinate represents percentage vital capacity taken by the surface area standard.

The Abeissa is divided into the different age groups.

The low reading in the 20-24 age group in the green curve, may be due to the fact that the average is taken on only 7 cases.

I have put in BOWEN & PLATT'S curve which was the result of an analysis of 100 normal people, between the ages of 20 and 50.

The miners 'curve is nowhere more than 2.75% v.c. below BOWEN & PLATT'S average and I think it is quite fair to conclude from the series that ANTHRACOSIS CAUSES NO DECREASE IN VITAL CAPACITY other than that which can be explained by advancing age.

In 41 cases some abnormality was found in the heart, not necessarily of course, of clinical significance, I have, however, separated this group.

REDUPLICATED 1st MITRAL.

10 cases Highest V.C. 102% Lowest 76% Average 90%

ACCENTUATED 1st MITRAL.

2 cases Highest V.C. 86% Lowest 86% Average 86%

ACCENTUATED 2nd MITRAL.

1 case V.C. 87%

SYSTOLIC IN MITRAL AREA.

16 cases Highest V.C. 104% Lowest 70% Average 91%.

ACCENTUATED 2nd AORTIC.

3 cases Highest V.C. 106% Lowest 89% Average 95%

SYSTOLIC MURMUR AT THE BASE.

4 cases Highest V.C. 110% Lowest 84% Average 99%

ENLARGED HEART 6th sp.

3 cases Highest V.C. 105% Lowest 87% Average 95%

EXTRA SYSTOLES.

1 case V.C. 95%

RAPID HEART.

1 case V.C. 92%

These figures for what they are worth all bear out the fact that a low reading is only obtained when there is some degree of decompensation.

In/

In all cases the vital capacity was worked out in relation to three standards. Surface area, Height, and weight.

If in the series we compare the height standard to the surface area standard we find that:-

87.1% of the cases were above the S.A. standard by an average of 4.3% v.c.

6.45% of the cases were equal with the surface area standard

6.54% of the cases were below the S.A. standard by an average of 2%

In comparing the weight standard and the Surface Area Standard:-

80.5% of the cases were above the S.A. standard by an average of 4%.

8.6% of the cases were equal with the S.A. standard.

10.9% of the cases were below the S.A. standard by an average of 2%.

All the pioneers in their work on vital capacity are unanimous in the opinion that the surface area standard is the most accurate.

WEST and others have found that for quick clinical work the Height Standard is quite satisfactory.

HEWLETT & JACKSON examining college students came to the conclusion that a combination of the height and weight standard (i.e. the Surface area standard) is little better than either separately.

It is interesting, therefore, to look at the above results taken in a series of absolutely "fit" men.

We see that the Height standard gives in general a slightly higher reading than the Surface area standard by 4.3% in 87.1% of the cases.

The weight standard gives a 4% increase in 80.5%. For all practical purposes the two standards would appear to be equal, if anything the weight standard is slightly more accurate.

We must not forget, however, that these men were picked for special work, and all were fine examples of normal healthy manhood. In very few cases were their weights very far from the normal, for their height and age as shown by life insurance tables. While the weight standard is quite satisfactory in normal people, I will show in a study of a series of obesities, that the weight standard is breaks down completely, while the height standard is still satisfactory.

Of the 140 miners 87 had second V.C. readings taken 1 year after the first examination.

In no case was their any marked change on physical examination, yet we find variations in the readings of these 87 cases:-

42 cases (48.3%) showed a 5% increase or decrease over their previous reading. Of these 42 cases 22 showed an increase and 20 a decrease.

13.8% showed a 5% - 10% increase from the first reading.

12.6% showed a 5% - 10% decrease - - - - - - - -

5.75% showed 11% - 13% increase - - - - - - - -

8.05% showed 11% - 20% decrease.

11.5% gave a similar reading to the first, on the second examination.

If we look at these figures it would seem that the law of chance had taken a hand. They are to all intents and purposes, mirror images of each other.

It would seem, therefore, that the factor of experimental error is a large one and I would emphasise again the necessity of the observer encouraging the subject to give his whole hearted co-operation when a reading is being taken.

In the second examination of these men an exercise tolerance test was used.

For this test the men wore their mine rescue apparatus, which weighed about 20 lbs. They wore respirators and were supplied with oxygen during the test.

With/

With this apparatus fitted and the oxygen flowing, each man had to walk at four miles per hour for twenty minutes. At the end of this period the pulse rate over ¼ minute was taken every minute, until 5 readings had been obtained.

I have included these readings in my records, but it is, of course, very difficult to correlate them. Not until a further large series has been done will it be possible to assess their value.

After all the men had been examined for the second time, five were failed .

Below is a short summary of their cases.

JAMES D. aet.37 vital capacity 97%.

Auscultation revealed a systolic murmur which was heard all over the heart. The man was of a nervous and excitable type and not fitted for mine rescue work. Exercise Tolerance Test 33.30.30. 29.30.31.

GEORGE McL. aet 26 vital capacity 104%.

The apex beat was thrusting and there was a systolic murmur in the mitral area. Exercise Tolerance Test 20.19.19.18.19.

This man had marked miners' nystagmus and was on this account excluded.

JAMES M. aet 46 vital capacity 70%.

The/

The 1st sound was not pure and the patient was overweight. Exercise Tolerance Test 21.17. 18.18.21.

WILLIAM D. aet.36 vital capacity 90%.

The apex beat was in the 6th space. The sounds were thudding B.P. $\frac{154}{90}$ Exercise Tolerance Test 22.20.19.18.18½.

W. CUMMING aet.37. vital capacity 90%. The heart was rapid. Exercise Tolerance Test. 39.33.30.29.29½ 29.

DREYER states that a patient with a vital capacity of 85% and under, would be practically certain "to be suffering from some health depressing condition".

In examining the 140 miners, I find that 28 cases had vital capacities of 85% and under, and yet after a searching clinical examination they are passed as fit to undertake arduous work in mine rescue gangs.

Once again we are faced with the fact that vital capacity is in no sense an indication of physical fitness, and unless it is supported by clinical findings, a low reading is of no account.

VITAL CAPACITY READINGS ON 140 COAL MINERS.

CASE NO	AGE	HEIGHT Ft. Ins.	WEIGHT St. Lbs.	VITAL CAPACITY	SURFACE AREA	V.C.% S.A. STANDARD	V.C.% Ht. STANDARD	V.C.% Wt. STANDARD	V.C.2nd Exam V.C.	V.C.% S.A. STANDARD %	EXCERCISE	TOLERANCE	TEST		
1	29	5·10¾	10·11	5000	1·85	108	112	112	5100	110	25	23	23	24	24
2	43	5·9¾	11·4½	3800	1·86	82	87	85	3200	68	19	16	17	17	17
3	40	5·6	10·3½	3450	1·72	80	81	83	3500	81	23	22	22	21	22
4	29	5·9¾	11·8	4100	1·9	86	94	90	4300	91	26	21	21	24	21
5	27	5·9½	10·10	4400	1·81	98	100	96	4700	104	23	19	19	19	19
6	26	5·8¼	9·12	4000	1·72	94	93	91	4000	94	23	20	18	18	
7	30	5·5	10·5	3500	1·7	82	85	82	3500	82	29	26	25	25½	25
8	28	5·8¾	10·11	3700	1·82	82	86	85	3900	86	30	22	21	22	23
9	28	5·8½	11·4	4400	1·84	95	102	98	4500	96	31	27	25	26	26½
10	33	6·1¾	10·12	5000	1·9	107	108	114	4500	96	23	18	17	16½	17
11	35	5·10	11·4	5000	1·87	107	112	114	4800	103	22	18½	17	18	18
12	22	5·6	10·6	4200	1·79	93	100	98	4200	93	20	16½	16	17	17
13	18	5·10	11·	4100	1·86	89	92	94	4200	91	28	23	21	21	20

14	25	5·11½	10·9	4800	1·85	104	106	112	4600	99	33	29	29	28	27½
15	27	5·10¾	11·10	5300	1·91	111	119	115	5250	110	31	26	26	25½	26
16	31	5·10	10·8½	4000	1·83	88	90	94	3800	83	19	17	17	16½	17
17	29	5·7	10·9	5000	1·76	113	118	117	4600	104	35	31	30	30	30
18	25	5·9	10·11	4500	1·83	98	103	103	4300	95	32	28	28½	29	29
19	29	5·9	10·3	4000	1·78	90	91	96	4100	92	34	27½	27	26	27
20	43	5·5¾	9·2	3300	1·63	81	80	86	3300	81	25	23	21	21	22
21	45	5·9	10 11	4100	1·83	90	94	94	3400	70	21	17	18	18	21
22	25	5·8¼	10·3	4400	1·75	101	102	106	4200	102	23	17	18	17	17
23	19	5·11½	12·4	5050	1·97	103	111	105	5000	102	25	23	22	23	22
24	42	5·5½	9·12	2950	1·69	69	70	73							
25	38	5·11½	13	4000	2·	80	89	80	4400	88	31	28	27½	25½	26
26	35	5·7¼	10·3	3600	1·73	83	85	86	4000	94	29	25	25	24	24
27	40	5·4	8·3	3000	1·54	78	74	86	3200	84	29	24	22	21	21
28	40	5·8	11·5	3200	1·83	70	74	72							
29	37	5·8½	11	4100	1·82	90	95	96	4400	97	19	16	15	14½	15

CASE NO	AGE	HEIGHT Feet. Inches	WEIGHT	VITAL CAPACITY	SURFACE AREA	V.C.% S.A. STANDARD.	V.C.% Ht. STANDARD	V.C.% Wt. STANDARD	V.C.2nd Exam V.C.% S.A. STANDARD V.C. %		EXCERCISE	TOLERANCE	TEST			
			St. Lbs.													
30	33	5·9½	10·6	4200	1·80	93	96	98	4300	96	21	18	18	19	18	
31	25	5·10½	10·11	4400	1·85	96	99	100	4000	87	21	17½	16	17	17	
32	40	5·6½	11·7	3650	1·81	79	86	80	3500	77						
33	48	5·6¾	9·12	3900	1·7	92	93	96	4000	94	21	18	18	19	18	
34	42	5·3¾	9·5	3400	1·6	85	85	86								
35	31	5·6½	10·3½	3800	1·72	88	91	91	3860	88	24	23	22¼	23	23	
36	30	5·2½	10·1	3500	1·65	85	89	84	3300	81	27	23	22	20	20	
37	30	5·9¼	10·5	4300	1·8	96	98	101	4600	102	22	19	20	20		
38	31	5·9	11·6	4500	1·87	97	103	98	4000	86	24	21	21	21		
39	38	6	11·6	4100	1·92	86	90	90	4150	87	25	23½	21	21		
40	37	5·8½	10·4	3600	1·76	82	83	86	3700	84	23	18	18	17	17	
41	42	5·10	11·11	3800	1·91	80	86	81	3700	78	35	33	33	31	31	30
42	35	5·8½	10·	3800	1·75	87	88	91	3700	85	32	26	26	26	26	

43	34	5•6¾	10	4500	1•71	105	107	108	4800	112	32	29	28	28	29	
44	29	5•9	11•3	4325	1•85	94	98	96	4000	87	34	31	30	28	29	
45	33	5•5	11•10	4500	1•8	100	109	98	4600	102	24	20	19	20	20	
46	32	5•9	12•9	4450	1•95	91	100	91	4300	88	28	22	22	22½	22	
47	30	5•1½	10•1	3900	1•62	97	101	94	4000	99	24	22	22	21½	21	
48	23	6•½	11•3	4300	1•91	88	94	96								
49	29	5•7¾	10•9	4150	1•78	93	97	90	4100	92	22	18	19	18½	18	
50	33	5•4¾	10•11	3880	1•73	89	95	82	4000	93	21	18	17	18	17	17
51	24	5•5½	9•11	4400	1•66	106	107	109	4300	104	25	22	23	22	22	x
52	25	5•11	11•11	4900	1•92	102	109	105	4700	99	17	14½	14	14	15	
53	30	5•8	10•11	3850	1•8	86	88	88	4000	89	24	19	20	20½	21	21
54	29	5•8	9•10	4450	1•71	103	102	110	4350	101	16	14½	13½	13	13½	
55	31	5•6	10•11	3600	1•76	82	86	82	5700	84	19½	17	17	17	17	
56	25	5•8¾	10•10	3800	1•8	87	88	87	3800	87	19	16	16	16	16	
57	30	5•6¾	10•2	4500	1•72	105	107	108								
58	38	5•6	9•3	3900	1•64	95	93	102	4100	100	24	16	16	16	16	

CASE NO	AGE	HEIGHT Ft. Ins.	WEIGHT St. Lbs.	VITAL CAPACITY	SURFACE AREA	V.C.% S.A. STANDARD	V.C.% Ht. STANDARD	V.C.% Wt. STANDARD	V.C. 2nd Exam V.C.	% S.A. STANDARD	EXERCISE	TOLERANCE	TEST			
59	29	5·9½	10·3	4600	1·77	104	105	110								
60	25	5·7	9·9	4400	1·69	104	104	109								
61	41	5·11	10·3	4100	1·81	91	91	98	4700	103	26	21	21	21	20	
62	40	5·7¼	10·3	4300	1·74	98	101	103	4300	98	22	19	19	19½	19	
63	34	5·5	10·3	4000	1·69	97	97	96								
64	43	5·9	12	3700	1·9	78	84	79	3700	78	26	24	22	22	21	
65	40	5·6	8·9	3100	1·6	78	74	83	3300	83	27	23	23	24	22	
66	36	5·8	10·1	3950	1·74	90	92	94	3900	92	39	33	30	29	29½	29
67	29	5·11	11·8	4900	1·92	104	109	107	5000	106	26½	22	22	21	22½	
68	48	5·8¾	10·3	3500	1·76	80	81	84								xA
69	47	6	11·3	4300	1·91	90	96	96	4100	84	21	16½	17	17	17	x
70	46	5·8	9·13	3600	1·72	84	83	89			33	29	27	27	26	0
71	27	5·10	11	4400	1·86	94	99	100	4300	92	21	17	17	16	16½	

72	26	5•9	10•9	5000	1•81	110	114	117	5000	110	28	22	23	22	22½
73	43	5•6	9•10	3800	1•69	90	91	94	3700	88	26	25½	25	25	2624
74	28	5•10	13•8	4600	2•01	92	103	92	4780	95	25	24	22	23	23
75	40	6	13•1	5000	2•01	100	109	100	4400	87	17	14	14	15	16
76	32	5•9	11•1	5300	1•84	115	121	118							
77	24	5•7	10•2	4100	1•73	95	97	98	4000	92	22	20	19	20	19½
78	26	6	12•8	3700	1•97	75	81	76	3800	77	26	23	22	21½	22
79	31	5•6	10•9	4000	1•75	94	95	94	3500	80	24	21	21	21	21
80	35	5•6	10•6	3200	1•73	74	76	75	3200	74	30	24	24	22½	23
81	25	5•9	10•12	4600	1•83	100	105	105	4100	90	27	23	23	23	22½
82	41	5•6¾	9•5	4450	1•66	107	105	112	4000	96	22½	19	21	20	20
83	32	6	11	4300	1•9	91	94	98							
84	27	5•6¾	9•12	4050	1•71	94	96	99	3500	81	28	25	25	24	25
85	38	5•10¾	10•4	4200	1•8	93	97	101							
86	32	5•10½	10•11	4000	1•85	87	90	91	4000	87	18	16	16	16	16½

CASE NO	AGE	HEIGHT Ft. Ins.	WEIGHT St.Lbs.	VITAL CAPACITY	SURFACE AREA	V.C.% S.A. STANDARD	V.C.% Ht. STANDARD	V.C.% Wt. STANDARD	V.C.2nd Exam V.C.	V.C.% S.A. STANDARD %	EXERCISE	TOLERANCE	TEST			
87	29	5·8½	11·2	4500	1·82	99	104	101								
88	33	5·10	12·2	4700	1·92	98	106	100	4600	96	27	24	24	24	22	
89	30	5·10¾	10·9	4450	1·84	97	100	103	4300	94	25	22	22	22	21	
90	43	5·7	10·13	4900	1·8	109	115	112	4300	96						
91	37	5·8	11·3	3100	1·83	70	72	69	3800	83	31	27	26	26	26	
92	35	6·¾	11·10	5600	1·95	115	122	121	5350	112	22	19	19	19	17	
93	36	5·6	10·6	3700	1·74	86	87	87	3300	76	28	24	23	24	23	23
94	29	5·10	11·8	5000	1·9	105	111	109	4650	98	25	21	22	22	21	21
95	31	5·9	12·4	4700	1·91	99	107	100								
96	40	5·7	12·10	3900	1·9	82	92	80	3600	77	28	25	25	24	24	
97	44	5·4¾	10	4200	1·67	101	103	101	4000	96	22	19	18	19	18	19
98	29	5·8¼	10·3	3900	1·76	90	90	94	4000	91	27	23	22	22½	23	
99	29	5·7	10·8	3950	1·76	91	93	92								

100	39	5•5	10•4	3200	1•7	75	78	77	3500	82	25	23	23	24		
101	42	5•11	12•4	4700	1•96	96	104	98	5200	106	23	21	20	19½	20	
102	27	5•10½	12	4650	1•94	98	104	99	4800	102	25	23½	23½	24	24	
103	43	5•8	12•6	3800	1•91	79	88	80								
104	46	5•11	11•3	4700	1•59	100	104	105	5300	113	23	19	20	20		
105	41	5 11	10•11	4600	1•85	99	102	108	5300	112	24	22	22	22		
106	30	5•7	11•1	4800	1•78	109	113	110			40	36	32	31	30	
107	39	5•11	12•1	4600	1•92	96	102	98			33	28	26	25	25	
108	42	5•11	11•10	4500	1•9	95	100	98			14	13	15	15		
109	35	5•9	12•7	3600	1•91	76	82	75			30	22	24	23½	24	
110	37	5•8	9•12	4200	1•72	99	97	104			33	30	30	29	30	31
111	26	5•8	11•3	4700	1•8	104	109	106			20	19	19	18	19	
112	36	5•8	11•6	3900	1•82	87	90	87			22	20	19	18	18½	
113	37	5•8	10	3900	1•72	92	90	94			39	33	30	29	29½	29
114	43	5•9	10•10	4100	1•8	91	94	96			32	28	24	23	24	
115	31	5•10	11•7	3600	1•86	80%	81	80			30	23	22	22	21½	

CASE NO	AGE	HEIGHT Ft. Ins.	WEIGHT St. Lbs.	VITAL CAPACITY	SURFACE AREA	V.C.% S.A. STANDARD	V.C.% Ht. STANDARD	V.C.% Wt. STANDARD	V.C.2nd Exam V.C. % S.A. STANDARD	EXERCISE	TOLERANCE	TEST			
116	37	5·8	10·8	4100	1·77	92	95	97		29	23	22	21		
117	38	5·8	12·6	4300	1·86	92	100	90		25	20	20	21	21	
118	33	5·6	10·2	3900	1·7	92	93	94		27	22	22	23	22	21
119	31	5·10	12·10	4500	1·95	92	101	94		24	19	18	19	18	
120	30	5·10	12·1	4850	1·9	102	109	102		23	20	19	19	19	
121	25	5·10	10·8	4300	1·8	96	97	101		17	14½	14	14	15	
122	38	5·11	13·6	4400	2·01	87	98	88		26	22	22	22	23	
123	35	5·10	11·8	4800	1·85	104	106	105		22	18½	17	18	18	
124	40	5·8	10	3500	1·72	81	81	86		36	32	32	30	29	
125	25	6	10·5	5100	1·85	110	111	124		31	24	25	24	24	23
126	28	5·8	10·7	4350	1·77	98	101	103		39	35	33	32	31	32
127	35	6	10·12	4780	1·86	103	105	107		27	24	23	24	24	23

128	41	5·9	11·4	4680	1·85	100	107	104		36	31	30	30	30
129	32	5·9	12·8	4700	1·94	98	107	94		29	21	21	22	20
130	44	5·8	11·6	3900	1·83	85	90	87		24	22	20	21	21
131	37	5·9	10·1	4700	1·75	107	107	115						
132	40	5·8	11	4150	1·8	92	96	96		19	14	14	13	
133	30	5·2	10	3300	1·61	82	84	81		27	23	22	20	20
134	45	5·8	12	3750	1·85	81	87	80		26	21	20	15	
135	42	5·7	10	3500	1·7	82	82	86		21	16	16	16	16
136	32	5·10	12·2	4800	1·92	100	108	102		28	24½	25	24	24
137	33	5·10	10·8	4300	1·8	96	97	101		26	22	22	22	23
138	34	5·6	10	4400	1·7	103	105	109		23½	20	20	21	20
139	34	5·6	9·9	3900	1·67	93	93	96		22	19	18	17	18
140	47	5·9	10·8	4400	1·8	98	100	103		35	31½	30	30	31

C A R D I A C F A I L U R E.

As I have examined from the point of view of their vital capacity a series of cases showing cardiac failure, it will be necessary to consider the various factors involved.

Failure in the function of contractibility is the essential factor in cardiac failure. Signs of failure will only appear when the contraction of the heart is not sufficiently strong to maintain an adequate circulation.

The force of cardiac contraction depends on the degree of stretching of the individual muscle fibres at the beginning of systole. (STARLING'S LAW). This depends on the volume of blood in the heart at the end of diastole. The force of contraction is, therefore, regulated by the volume of blood received from the venous system during diastole. The greater the volume of blood in the heart, the greater will be the contraction up to the maximum.

If the point of optum stretch is passed the contraction begins to weaken. This pathological dilatation may result in permanent stretching of the muscle fibres and the chambers be permanently enlarged/

enlarged.

HENDERSON & PRICE (35) have shown that there is a mechanism whereby each ventricle expels the same quantity of blood during systole. Such a mechanism is obviously necessary, otherwise the blood would accumlate in one or other of the circulations depending on which ventricle was contracting the weaker.

They found that when the Right Ventricle is pumping more blood than the left, the rise in pressure in the pulmonary veins stimulates the left to contract more forcibly, and the output of the two sides is kept constant.

To a large extent the force of the Left Ventricle is controlled by the right, when the heart suddenly fails, the blood tends to accumulate in the systemic veins.

In recent years many investigators have tried to find out what happens to the circulatory rate when the heart fails.

The circulatory rate may be defined as the time taken for the blood to move from point to point in the body. It is of course not a constant factor/

factor since owing to the widening of the total stream bed the velocity decreases from the aorta to the periphery. If, however, corresponding points are taken in different individuals the results will be comparable.

Several methods have been devised but that of BLUMGART & YENS (36) and WEISS (37) seems to have given the most valuable results. As the question of pulmonary congestion is so important in the etiology of the decrease in vital capacity, in cardiac failure, I will quote their work in some detail.

A solution of sodium chloride which has been exposed to radium emanations is injected into the median basilic vein of one arm. The arm is kept at the same level as the Right Auricle. At a corresponding position on the other arm a radium detector is placed. The detector shows the presence of radium emanations as soon as they reach the arteries at the bend of the elbow.

A series of lead screens suitably arranged prevent the emanations reaching the detector except by means of the blood stream. The time taken for the emanations to travel from arm to arm is measured by a stop watch and expressed in seconds.

In/

In a number of normal subjects the arm to arm times, was found to vary between fifteen and twenty seconds. Normal variations in the blood pressure made no difference in the velocity. Acceleration of the heart to above 100° produced only a slight rise. Low ventricular rates did not retard it.

A series of cases showing cardiac lesions were examined and in cardiac failure alone were the times greatly increased.

They were normal in groups of cases showing valvular lesions where cardiac failure was absent.

In auricular fibrillation the velocity was always diminished. In cases of extensive heart disease where fibrillation was added, the rate was very slow. In high blood pressure the rate was noted to be either slow or normal, it was never rapid.

In the cases showing cardiac failure the time taken was from fifty to seventy seconds as opposed to the normal of fifteen or twenty seconds, and the length of time taken was directly proportional to the amount of failure as shown by dyspnoea and oedema.

A/

A modifaction of their method enabled them to estimate the time taken by the blood stream to pass through the pulmonary circuit.

The detector is placed over the skin corr-esponding to the position of the Right Auricle. By this means they obtained the time taken for the blood to pass through the veins from the ante cubital fossa to the Right Auricle. This they called the "arm to heart time" and it was a register of the velocity of the blood in the veins. The time spent by the blood in the pulmonary circulation was estimated by the following calculation.

In travelling from the Right Auricle to the arteries of the forearm, the blood has to pass through the Right side of the heart then through the lungs to the left side of the heart and from the left ventricle to the arteries. They had previously calculated that the time spent in the heart was one second and estimated that the velocity in the arteries was double that in the veins. In normal subjects the average arm to heart time was seven seconds.

Therefore it follows that the time in the arteries was three and a half seconds. As we have seen the total arm to arm time varied from fifteen to twenty/

twenty seconds. Taking the average to be eighteen seconds and subtracting from this the arm to heart time seven seconds, and the three and a half seconds for the time in the arteries, and one second for that in the heart, the average pulmonary circulation time is six and a half seconds.

It was found that an increase in the ventricular rate showed a slight but definite increase in the rate of the pulmonary circulation.

Variations in systemic pressure did not effect it. Patients with emphysema were found to have normal velocities. In aortic regurgitation (syphilitic) the pulmonary times increased although the arm to heart times might be normal.

They suggest that in this condition it is the left ventricle that is failing, so the pulmonary circulation is slowed, although the venous return to the right side of the heart is still efficiently dealt with.

This statement is important since it implies that an inefficient left ventricle is associated with pulmonary congestion. The left ventricle fails to deal with the blood coming to it so the pressure is increased behind it.

They/

They also examined cases of myocardial degeneration with normal rhythm. Even when there had been no history of failure the pulmonary circulation times were found to be increased up to 50%, but the arm to heart times were normal.

Where there was a history of failure the pulmonary circulation times were doubled, and the arm to arm times increased by 80%.

In the cases where heart failure was actually present at the time of investigation both the pulmonary and arm to heart times were increased 400%, so the blood stream was only travelling at a quarter the normal speed.

From their work it is seen that in conditions in which the chief strain falls on the left ventricle, the pulmonary times exceed the arm to heart times, until the stage of cardiac failure is reached.

SUMMARY/

S U M M A R Y.

From the work of BLUMHART, YENS & WEISS, we see that in cardiac failure the circulation rate is slowed. This slowing is present both in the pulmonary and systemic circulations. The speed of circulation is unaltered in other conditions causing dyspnœa such as emphysema.

In those conditions where the left ventricle is likely to fail first, the stream is slowed more in the pulmonary than in the systemic circulation.

The primary factor in cardiac failure is the heart muscle, only when it ceases to perform its work efficiently do the signs of cardiac failure become apparant.

Heart failure is usually more or less sudden either some exertion is attempted beyond the power of the heart or an abnormal rhythm such as fibrillation is set up. The heart suddenly becomes inefficient and fails to deal with the volume of blood being returned to it.

The muscle fibres are stretched beyond the optimum point. The heart dilates and the pressure behind it rises.

On account of HENDERSON'S Law of the Coordination of cardiac contraction the venous system will be engorged and the liver enlarged.

When one or other side of the heart is primarily affected it will be the circulation immediately behind that side which will first show signs of engorgement.

In left heart failure as in cases of aortic regurgitation or of increased peripheral resistance the pulmonary circulation is slowed while the

systemic/

systemic remains normal. Congestion of the lung bases. Acute pulmonary oedema will occur in left heart failure depending on suddeness of the onset.

In Mitral Stenosis there is an obstruction to the pulmonary outlet, the pulmonary pressure is raised and the flow retarded. The pulmonary congestion in mitral stenosis is not connected with left heart failure.

Right heart failure owing to HENDERSON'S Law of Coordination of contraction in any sudden failure the blood will accumulate behind the right side. A primary right sided failure is seen in pulmonary embolism, and a chronic lung condition such as emphysema. The venous system will be engorged, as shown by an enlarged liver or pulsating distended external jugulars.

CAUSES OF DYSPNŒA.

Dyspnœa is the most constant and early sign of cardiac failure. At first it may only be present on exertion, but later as the condition progresses, it becomes constant until the patient is forced to adopt the upright position when orthopnœa is said to be present.

Slowing of the blood flow is the principal cause/

cause of cardiac dyspnoea. It follows that in any given time less blood is oxygenated and when the flow to the tissues is decreased their supply of oxygen will be diminished. The respiratory centre along with the rest of the body receiving a dim-inished supply of oxygen.

Several factors in the quality of the blood effect the respiratory centre.

Reduction in oxygen pressure, an increase in carbon dioxide pressure or an alteration in the hydrogen ion concentration towards the acid side will cause dyspnoea. Any alteration in the carbon dioxide pressure will disturb the acid base balance so the last two factors are closely related.

FRASER (38) examined these factors in cases which had heart failure and dyspnoea. He found that in general the oxygen saturation was normal. Cardiac dyspnoea is therefore not due to deficiency in oxygen saturation of the blood.

He determined the carbon dioxide saturation, and found that the pressures were normal except in cases which had pulmonary lesions. Only in patients who were near death did he find any change in the hydrogen ion concentration.

He/

He concludes that the increased respiratory activity seen in all cases of cardiac failure must be independent of the quality of the blood supplied to the respiratory centre. The oxygen saturation is normal. The carbon dioxide pressure is normal or more often below the usual level in cases where there are no marked pulmonary complications. The hydrogen ion concentration is nearly always normal.

EAST & BAIN (39) suggest that although the quality of the blood is normal the diminished quantity leads to a smaller supply of oxygen to the respiratory centre, and the tissues each minute. They think it probable that "this lowered supply of oxygen is the constant stimulus to the respiratory centre in cardiac dyspnoea". Where there are pulmonary lesions in addition carbon dioxide retention will act as another stimulus to the centre. In terminal stages of cardiac vascular disease acid products may be retained probably through a concomitant renal failure. They will act in the same direction through an alteration in the hydrogen ion concentration.

We have seen that the Dyspnoea of cardiac cases is anoxaemic in origin and not due to any/

any variation in the PH of the blood.

"In those forms of renal disease which are so frequently associated with disease of the heart, there is a defective elimination of acids and a consequent diminution in the alkali reserve. The incidence of the dyspnoea is often paroxysmal and nocturnal and is not related to exertion. Thus the dyspnoea of acidosis can be differentiated from the more common dyspnoea of cardiac origin." (COWAN (27) & RITCHIE)

ORTHAPNOEA is due probably to the increased resistance thrown on the diaphragm when lying down. In the upright position, the weight of the liver and other abdominal organs tend to assist the descent of the diaphragm. X rays taken of recumbent cases tend to favour this view, in that the position of the diaphragm appears to be raised. (HALDANE (28)).

THE/

THE QUANTITATIVE RELATION OF VITAL CAPACITY TO DYSPNOEA IN CARDIAC FAILURE.

PEABODY & WENTWORTH (23) and many other workers in Europe and America have shown that there is a decrease in vital capacity in cases of cardiac disease with dyspnoea. They found that the decrease was not due to mechanical factors such as pleural exudate.

In comparing cases with pleural effusion to those cardiac cases with pleural transudate, it was seen that the vital capacity in the cardiac cases was much less, although the amount of collapsed lung might be smaller.

The more severe the cardiac condition the greater was the diminution in vital capacity, and the degree of dyspnoea increased as the vital capacity became less.

They suggested that this decrease in vital capacity might be due to some disturbance of the HERRING & BREUER Reflex, but concluded that this could not be the case, as they found that cardiac cases could hold their breath as long as or nearly so as normal people.

Other/

Other observers were unable to confirm this statement MEAKINS & DAVIES found there was a decrease in the time during which the breath could be held in cardiac cases where there were symptons on exertion. Cases with no dyspnoea or where there was an increase in the bicarbonate reserve as is often found in association with emphysema were able to hold their breath mearly as long as normal people.

As I have remarked elsewhere I found that the small spirometer was useless in cardiac cases, who showed very marked dyspnoea as they were unable to hold their breath long enough to record a reading.

In considering the question of a change in the HERRING & BREUER Reflex, several experiments are of interest.

DURIN (42) produced multiple pulmonary emboli in goats, by introducing a starch solution into the venous blood stream thereby producing a pronounced degree of rapid shallow breathing. The breathing was restored to normal by section of the vagi.

BRINGER BROW & BRANCH (43) carried out a further series of the same type of experiment.

MEAKINS & DAVIES after having care-

fully/

carefully considered the subject state that "Small vascular and alveolar lesions may set up a restriction of the HERRING BREUER Reflex". In a number of these cases the dyspnoea set up was very similar to that found in cardiac cases.

In cases of irritable heart or "effort syndrome" there may be extreme dyspnoea on exertion and yet no reduction in vital capacity.

WINTRICH & NATHANSON (29) point out that in a series of patients who appeared to have marked symptoms of cardiac disease, had normal vital capacities and clinical examination proved the absence of disease.

ADAMS & STURGIS (30) found that in 100 cases of "effort Syndrome" the vital capacity was within normal limits.

MEAKINS & DAVIES (5) have proved that lowering the carbon dioxide content or oxyhaemoglobin saturation of the blood, did not decrease the vital capacity.

After breathing an atmosphere containing only 5% oxygen until cyanosis and hyperpnoea were marked, the vital capacity reading was found to be normal.

It may be said then that lowering of the/

the carbon dioxide content or the oxygen partial pressure in the alveolar air does not reduce the vital capacity in the normal person.

DRINKER PEABODY (24) & BLUMGART carried out some experiments in animals to demonstrate that pulmonary congestion and oedema of the lungs, reduces the vital capacity by mechanical interference with the intra alveolar space.

The pulmonary veins of a dog were intermittently compressed as they entered the left auricle. It was found that as a result of this procedure the volume of air per minute entering the lungs by mechanical propulsion was greatly diminished. They pointed out that in such lesions as mitral stenosis, there might be a very acute increase in pulmonary blood pressure, with resulting pulmonary oedema and claimed that their experiments were comparable.

Many years ago VON BASCH suggested that congestion of the pulmonary blood vessels might reduce the elasticity of the lung.

LIEBECK suggests that the primary cause of dyspnoea is due to engorgement of the capillaries of the lung alveoli, which in turn cause a distension of the alveolar walls, and eventually an increase in fibrous tissue. Pathological examination of/

of the lung in passive congestion bears this out. He thinks therefore, that the dyspnoea is due primarily to alveolar insufficiency. He points out that this insufficiency may vary in different persons with the same amount of alveolar tension and the same amount of alveolar insufficiency, just as an increase in arterial tension does not effect all hearts in the same way.

MEAKINS & DAVIES are of the opinion that the reduction in vital capacity in heart disease is due to some structural change in the lungs. JONES (26) favours this view owing to the slow alteration in readings which he got with changes in compensation, and from the fact that the diminution in vital capacity was greatest in cases of mitral stenosis when associated with auricular fibrillation, a lesion which is extremely likely to produce permanent lung changes.

THE/

THE CAUSE OF THE REDUCTION OF THE VITAL CAPACITY IN CARDIAC FAILURE.

SUMMARY.

When we consider all the facts at our disposal it would appear that the decrease in the vital capacity of the lungs in cases of organic heart disease, is probably due to a decrease in the capacity of the alveolar spaces, owing to the encroachment of the dilated capillaries coupled with a general loss of elasticity through vascular engorgement.

We have seen that the venous flow through the lung is slowed, when this slowing reaches a certain point leakage occurs, recognisable as pulmonary oedema.

Experimentally produced pulmonary congestion caused a definite decrease in the amount of air entering the lung per minute. Although this experiment was not strictly comparable to vital capacity measurements, DRINKER PEABODY & BLUMGART'S results are very suggestive that some such mechanism is operative in certain cardiac conditions.

The suggestion of VON BASCH that vascular congestion "rendered the lung inelastic through/

through vascular turgesence" is to my mind very important.

It is difficult to imagine a simple congestion causing a large reduction in vital capacity, unless it were of a very extreme degree. But add the fact that there is also a loss of elasticity one can at once realise that the vital capacity would be greatly reduced.

Long continued congestion may cause a fibrosis and that in its turn help to reduce the capacity in those cases of chronic heart failure.

When the stage of marked exudation is reached and we recognise a clinical pulmonary oedema the mere mechanical blocking of the alveoli will without a doubt reduce the vital capacity. The difficulty is to explain the reduction in the early case, and for myself, I favour the theory of congestion causing loss of elasticity.

I am not able to agree with JONES when he says that the vital capacity changes only slowly with changes in compensation. Several of may cases show an immediate rise or fall in vital capacity as compensation changes for better or for worse. This is especially striking in cases of auricular fibrillation/

fibrillation. This would favour my view that the primary cause of a decrease in vital capacity in cardiac cases is congestion, and loss of elasticity of the lung. It would hardly be possible for a structural change to take place in a few hours and to disappear again after the administration of a drug such as digitalis which restores compensation in a few days.

I have no doubt that in old standing cardiac cases, who show varying degrees of dyspnoea on exertion, that there is some structural change such as fibrosis suggested by LEIBICK and certainly a period of rest and medication in these cases only produces a gradual rise in capacity extending over weeks or months.

A/

A REVIEW OF THE MORE IMPORTANT WORK DONE WITH RELATION TO VITAL CAPACITY AND HEART DISEASE.

BRINGER investigated heart disease from the point of view of lung volume. He found a diminution in the reserve and complementary air and therefore of the vital capacity at the time when oedema was most marked and when the difference between radial pulse rate was greatest.

His results may be summarised as follows:-

(1) In patients with heart disease the vital capacity forms a portion of the lung volume relatively smaller than in normal individuals and the mid capacity and residual air form relatively larger portions.

(2) When the absolute lung volumes determined from patients are compared with those calculated from normal individuals with the same surface area and chest measurement the following differences are found.

  (a) The vital capacities are always smaller.
  (b) The residual air is always larger.

(3) When decompensation occurs the absolute lung volume changes as follows.

(a)/

(a) Vital capacity mid capacity and total capacity decrease in volume.
(b) The residual air may either decrease or increase according to the severity of the state of decomposition.

ZISKIN (32) agree with PEABODY & WENTWORTH when they say that the vital capacity tends to be lower in Mitral disease than in Aortic but that in general the severity of the lesion rather than the type determines the decrease in capacity. ZISKIN states further that there is no definite relationship between the vital capacity and cardiac efficiency in ambulant cases who have no dyspnoea.

It has been the aim of investigators for many years to devise some simple test to estimate the functional capacity of the heart.

The simplest in use consists of taking the pulse rate before a fixed amount of exercise, after exercise has ended and after an interval of a few minutes.

The Ministry of Pensions use an exercise tolerance test in assessing cardiac disability.

A test such as this, however, seems more a measure of the excitability of the sino-auricular node than an indication of the capacity of the heart and depends on many other factors quite independent of cardiac reserve power.

WALLACE JONES/

WALLACE JONES (26) concludes after investigating a series of cardiac cases that:-

(1) The vital capacity is a valuable numerical expression of the amount of dyspnoea in organic cardiac cases.

(2) It is a useful indication as to the general progress of treatment though the changes in reading follow slowly the varying degrees of compensation.

(3) The vital capacity is most diminished in cases of mitral stenosis especially when associated with auricular fibrillation.

(4) Vital capacity readings are of little value in early diagnosis of cardiac conditions owing to the variability of normal standards.

MAURICE CAMPBELL (40) working in the cardiographic department of Guy's Hospital has used vital capacity readings as a guide to prognosis and treatment. He has followed the changes in vital capacity in some Rheumatic cases for a period of three years, and found readings helped greatly, in prognosis and in deciding on the patients capacity for work. Should the vital capacity in an adult remain stationary for months or years, it is good evidence that progress is satisfactory.

When/

When the vital capacity was above 70% he found that the patient was generally able to earn his own living, and was hardly short of breath on exertion.

When, however, it was below 40% the patient was breathless on walking, or even at rest, and was rarely able to do anything. Unless the breathlessness was due to some remediable cause the prognosis was bad.

CAMPBELL found that of all lesions auricular fibrillation produced the lowest readings.

The work of LIBRICH[29] & NATHANSON and ADAMS and STURGES[30] is important in that it proves that a vital capacity reading is of great value in distinguishing true from false dyspnoea.

The belief that vital capacity is a good indication of physical fitness, has of course been exploded.

PEABODY & STURGES[31] took the vital capacity readings from 12 cases suffering from anaema. All gave a history of weakness and some in an extreme form. Their average vital capacity was 88.7% such a figure would have no significance in organic heart disease.

They found that even in extreme weakness in/

in cases recovering from enteric fever that the vital capacity was rarely below 75% and in those cases which did give low readings, the reduction could be explained on other bases, such as development of pleurisy or pneumonia. It is interesting to read of one case they report.

"Test made on 36th day of Typhoid Fever, second day of normal temperature. Could not register grip testing apparatus with either hand, had a vital capacity of 86% of the normal".

Obviously in these cases the vital capacity does not indicate the true physical fitness of the patients. PEABODY & STURGES also cleared up the point as to whether the taking of vital capacity readings did not cause a low reading to be recorded through fatigue of the patients. They took vital capacity readings on cardiac and pernicious anaemia cases every 15 seconds for 10 minutes. At the end of the experiment the vital capacity readings were as great as at the beginning.

THE/

THE VITAL CAPACITY in CASES of CARDIAC FAILURE.

While looking through some articles on vital capacity in heart disease, I was struck by the comparatively few readings which were taken during the weeks or months that the patient had been ill. The majority seemed satisfied with a reading every two to four weeks.

I thought it would be interesting to see how the vital capacity varied from day to day in cardiac cases. Six cases had I found, had daily readings taken, while in the Royal Infirmary. Looking at the charts gave one little indication as to how the vital capacity varied. I worked these readings out on the Surface area standard and plotted the % result as a graph.

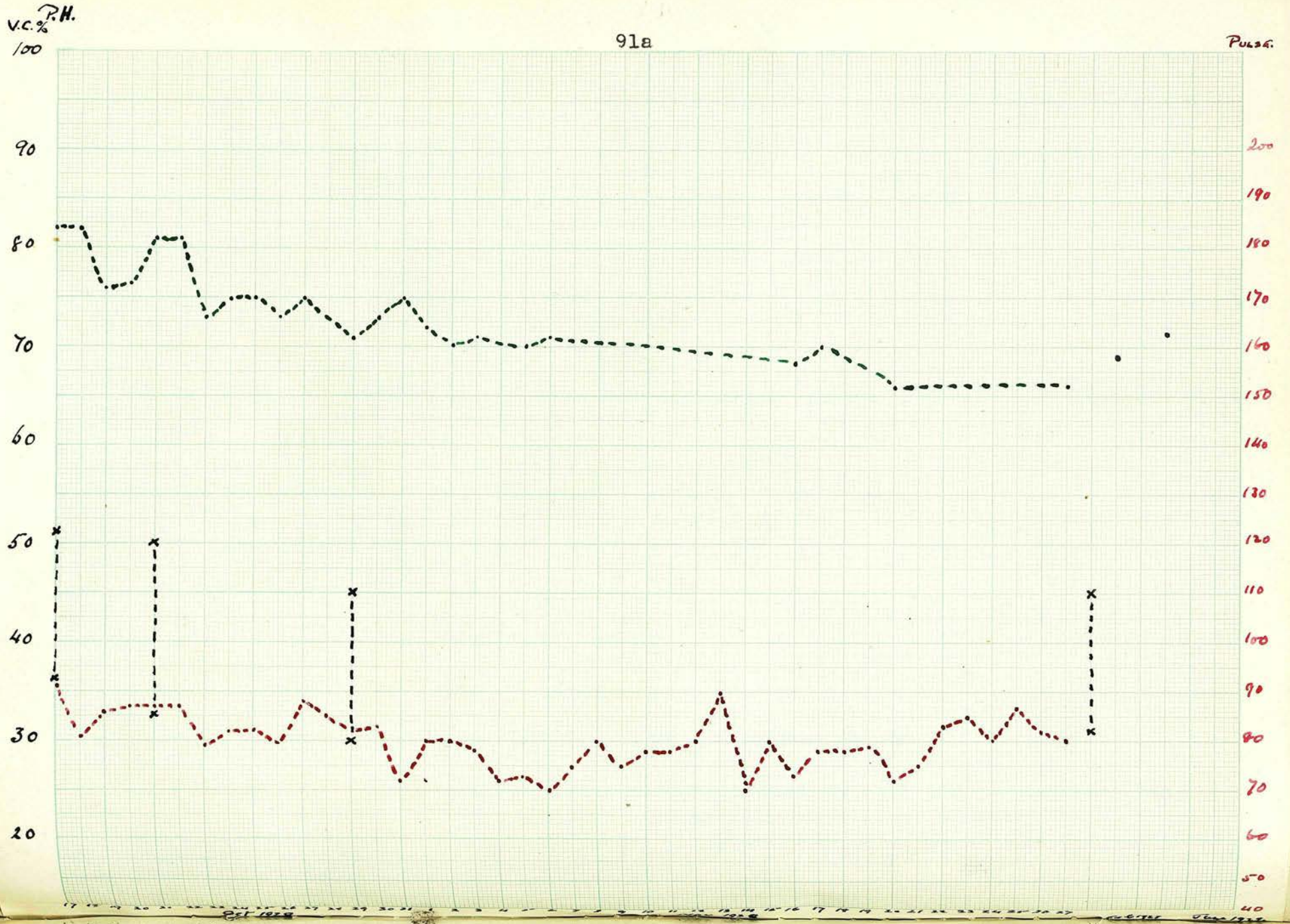
The results were so striking that I carried out a series of daily readings on cardiac cases while in hospital. In order to appreciate the daily changes, graphic representation seemed essential.

In all the charts the ordinate represents the % vital capacity calculated on the surface area standard.

The vital capacity is represented by a dotted/

dotted green line The Abcissa represents the days.

The Right hand ordinate, unless otherwise stated, gives the pulse rate represented by a dotted red line, Drugs and the dates they were given are also included.


V.C. % P.H.
100
90
80
70
60
50
40
30
20
PULSE.
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
40

A SERIES of CARDIAC CASES FROM WHOM DAY to DAY VITAL CAPACITY READINGS HAVE BEEN TAKEN.

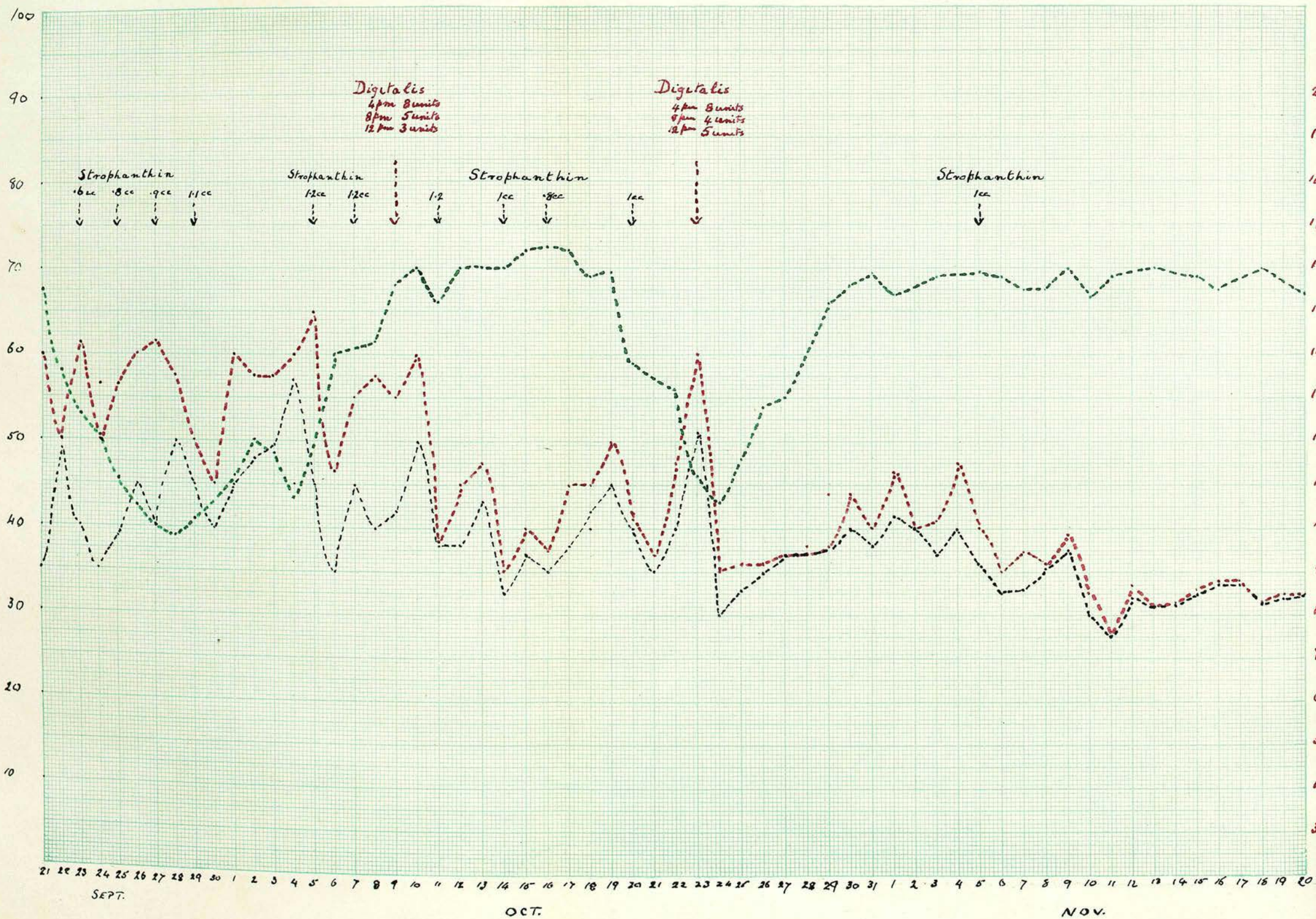
CASE I.

PETER H. aet. 50

For 7 years the patient has had constant pain over the heart. X ray examination revealed what was considered to be an Aneurism of the Heart Wall.

From the chart the vital capacity (green) is seen to be falling gradually. This fall was comparable with the patient's clinical condition. He made no progress and at the end of his stay in hospital said he "felt more tired than ever".

F.K.


V.C%
PULSE.
100
90
80
70
60
50
40
30
20
10
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
40
30
Digitalis
4 pm 8 units
8 pm 5 units
12 pm 3 units
Digitalis
4 pm 8 units
8 pm 4 units
12 pm 5 units
Strophanthin
.6 cc .8 cc .9 cc 1.1 cc
Strophanthin
1.2 cc 1.2 cc 1.2
Strophanthin
1 cc .8 cc 1 cc
Strophanthin
1 cc
21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
SEPT.
OCT.
NOV.

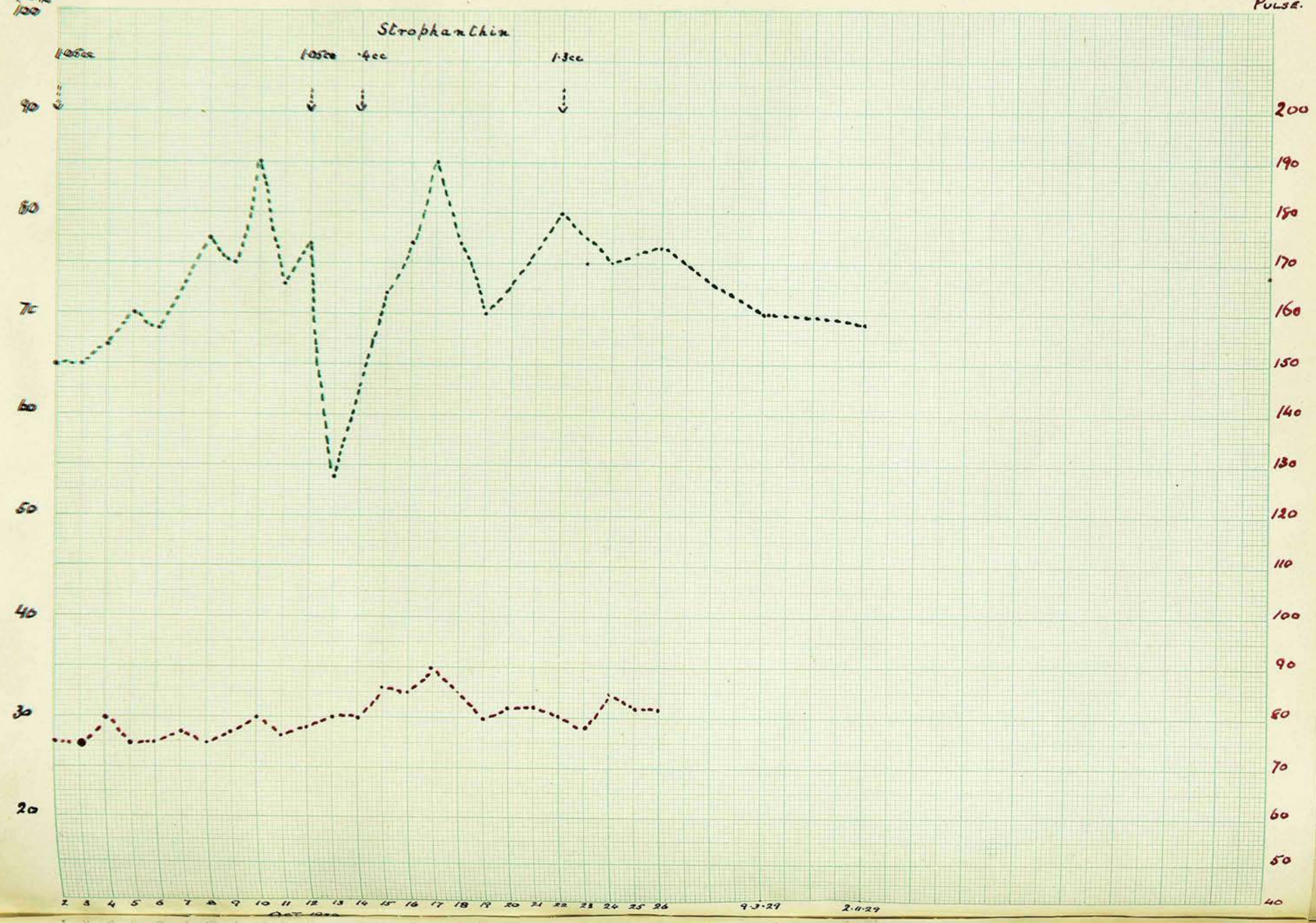
CASE II

FLORENCE K. aet. 24 Admitted 1/8/28.

Patient was in hospital 1 year ago with advanced mitral stenosis following on Rheumatic Fever. Since then she has been able to walk about quite comfortably. During the last 3 weeks she became very breathless. The auricles were found to be fibrillating, there were crepitations at both bases. Heart failure was advanced. Strophanthin was given, but there was no great improvement shown until a massive dose of Digitalis was administered. The chest then cleared rapidly.

On 20/10/28 the patient did not feel so well, the vital capacity (green) was falling, and the heart rate (red) began to rise again, so a further massive dose of Digitalis was given. The result was very satisfactory, the effect was prolonged and the patient was discharged much improved.

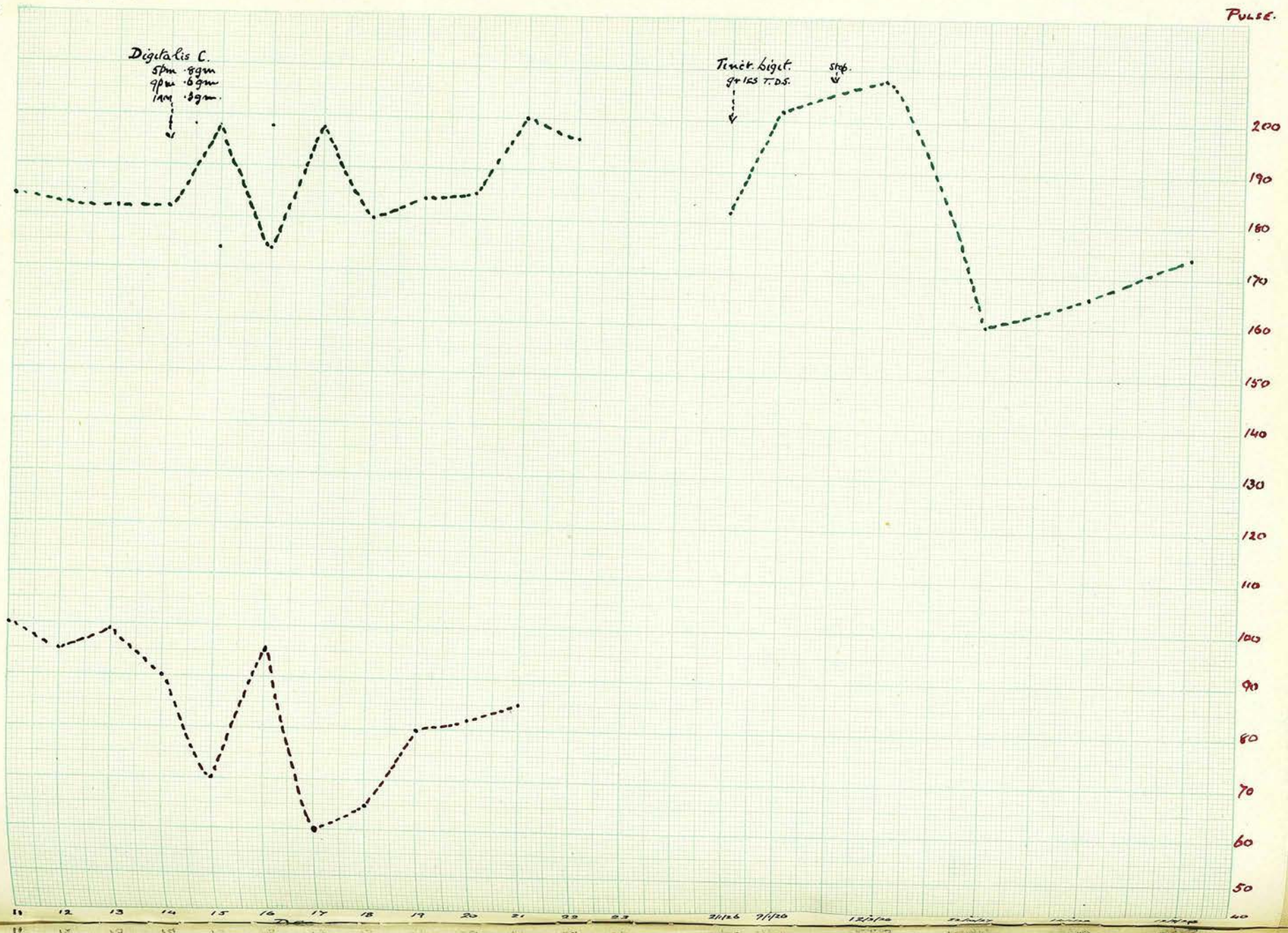
Through her stay in hospital the vital capacity readings went parallel with the patient's clinical condition. From the chart it can be seen that a rise in heart rate corresponds with a fall in vital capacity. The steady rise in vital capacity after the second dose of digitalis is striking.


W.B.
Vc %
100
90
80
70
60
50
40
30
20
PULSE.
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
40
Strophanthin
1·05cc
1·05cc
·4cc
1·3cc
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26
OCT 1929
9·3·29
2·11·29

W.B.

V.C.%

PULSE.


Digitalis C.
5pm ·8gm
9pm ·6gm
1am ·3gm.
Tinct. digit.
gr 1ss T.D.S.
stop.
100
90
80
70
60
50
40
30
20
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
40
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12
13
14
15
16
17
18
19
20
21
22
23
21/12/26
7/1/20
13/2/26

CASE III.

WILLIAM B. aet. 21 admitted 28/11/25.

The patient gave a history of three years shortness of breath, which was found to be due to a Mitral Incompetence and Stenosis.

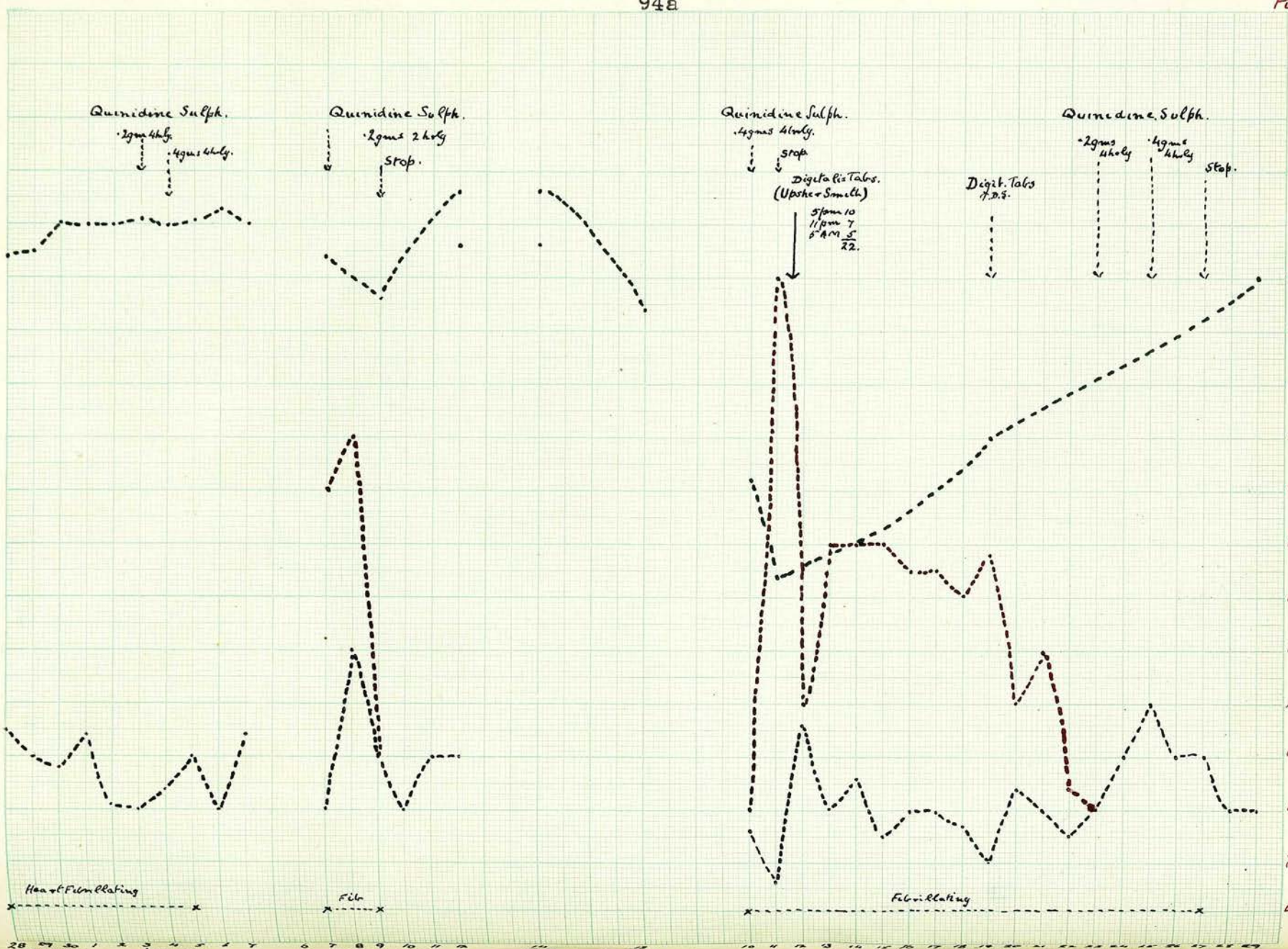
Rest and Digitalis improved his condition and the vital capacity rose from 82% - 90%.

On 2/1/26, the patient reported and was given digitalis and a week later he felt so much better that he returned to work, his vital capacity being now 91%. He was still keeping well on 12/3/26. In October 1926 he returned to hospital having had a brisk Haemoptysis. His vital capacity remained between 70% and 78% from 22/10/27 - 12/2/28.

He was again readmitted on 14/9/28 with haemoptysis. He improved until 10/10/28, when he started to get breathless again, and the chart showed the vital capacity falling to 54%, on 13/10/28. Further injections of strophanthin caused a slight improvement in the vital capacity which, however, gradually began to fall again.

V.C. % J. W.

PULSE.


100
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80
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60
50
40
30
20
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
Quinidine Sulph.
·2gms 4hrly.
·4gms 4hrly.
Quinidine Sulph.
·2gms 2 hrly
Stop.
Quinidine Sulph.
·4gms 4hrly.
Stop.
Digitalis Tabs.
(Upsher Smith)
5 pm 10
11 pm 7
5 AM 5
22.
Digit. Tabs
t.d.s.
Quinedine. Sulph.
·2gms 4hrly
·4gms 4hrly
Stop.
Heart Fibrillating
Fib
Fibrillating

CASE IV.

JAMES W. aet. 39 Admitted 7/3/28.

A history of previous admissions for auricular fibrillation was obtained. Up to now quinidine had always restored the rhythm.

From the chart it can be seen that quinidine has again given good results. The vital capacity (green) is seen to rise as soon as normal rhythm is established.

Red line = Apex beat rate

Black line = Pulse rate.

On 10/1/29, patient was admitted again with acute heart failure, quinidine failed to control the heart rate so a massive dose of Digitalis was given followed by quinidine again. The vital capacity rises as the heart rate falls.

V.C.% A.T.

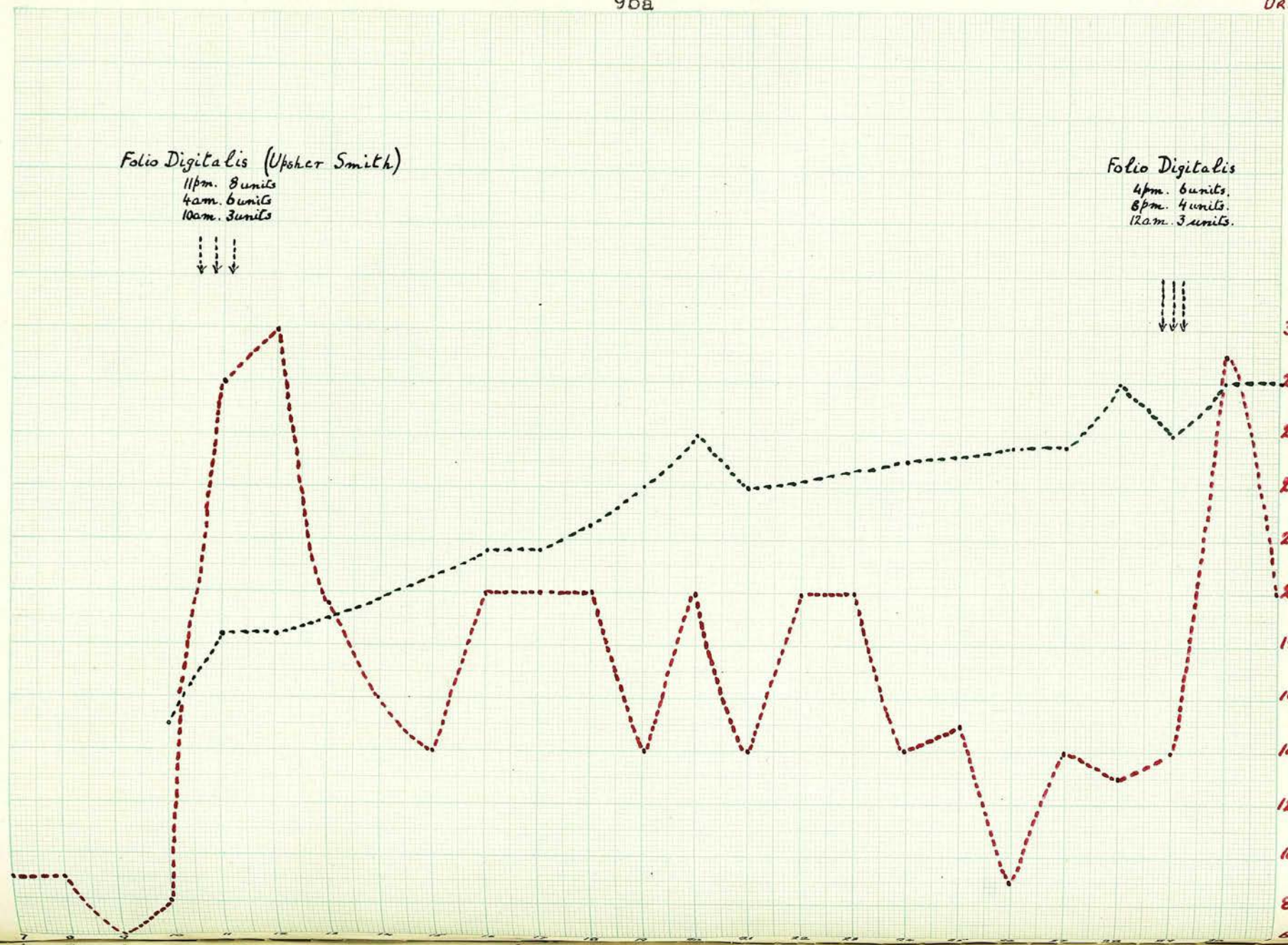
URINE c.c.s.

100
90
80
70
60
50
40
30
20

3000
2800
2600
2400
2200
2000
1800
1600
1400
1200
1000
800
600

Folio Digitalis (Upsher Smith)
11pm. 8 units
4am. 6 units
10am. 3 units

Folio Digitalis
4pm. 6 units.
8pm. 4 units.
12a.m. 3 units.



CASE V.

ANDREW T. aet. 50

The patient was admitted with a history of shortness of breath for 1 year. Breathlessness was getting progressively worse.

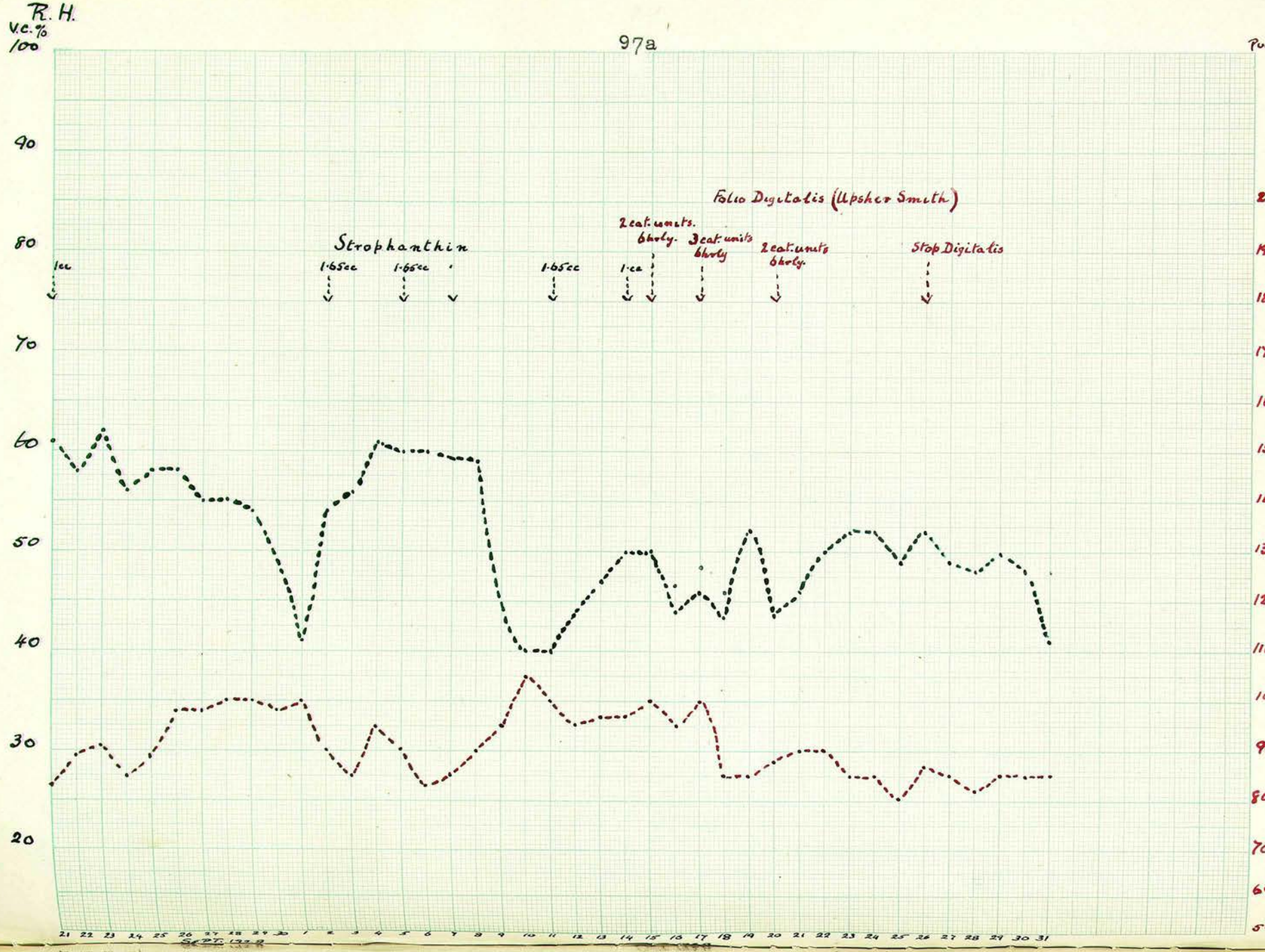
Cyanosis and orthopnoea were marked. The legs were oedematous, and there was also some oedema of the lung bases.

The heart was enlarged and a rough mitral systolic murmur was present.

A diagnosis of coronary Sclerosis was made. On admission the patient was very gravely ill. A massive dose of digitalis caused great improvement in the general condition, and the amount of urine (Red line) increased enormously.

By 16/11/28 the amount of oedema was greatly lessened, and there were only a few crepitations at the lung bases. A second dose of Digitalis was given with another increase in the output of urine.

As the output of urine increased and the condition of the myocardium improved, the vital capacity (green) is seen to rise steadily.


R. H.
V.C. %
Pulse.
100
90
80
70
60
50
40
30
20
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
Folio Digitalis (Upsher Smith)
2 cat. units 6 hrly.
3 cat. units 6 hrly
2 cat. units 6 hrly.
Stop Digitalis
Strophanthin
1cc
1·65cc
1·65cc
1·65cc
1·cc
21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31
SEPT 1928

CASE VI.

ROBERT H. aet. 40

The patient gave a long history of increasing breathlessness, Aortic Stenosis, and Incompetence were found.

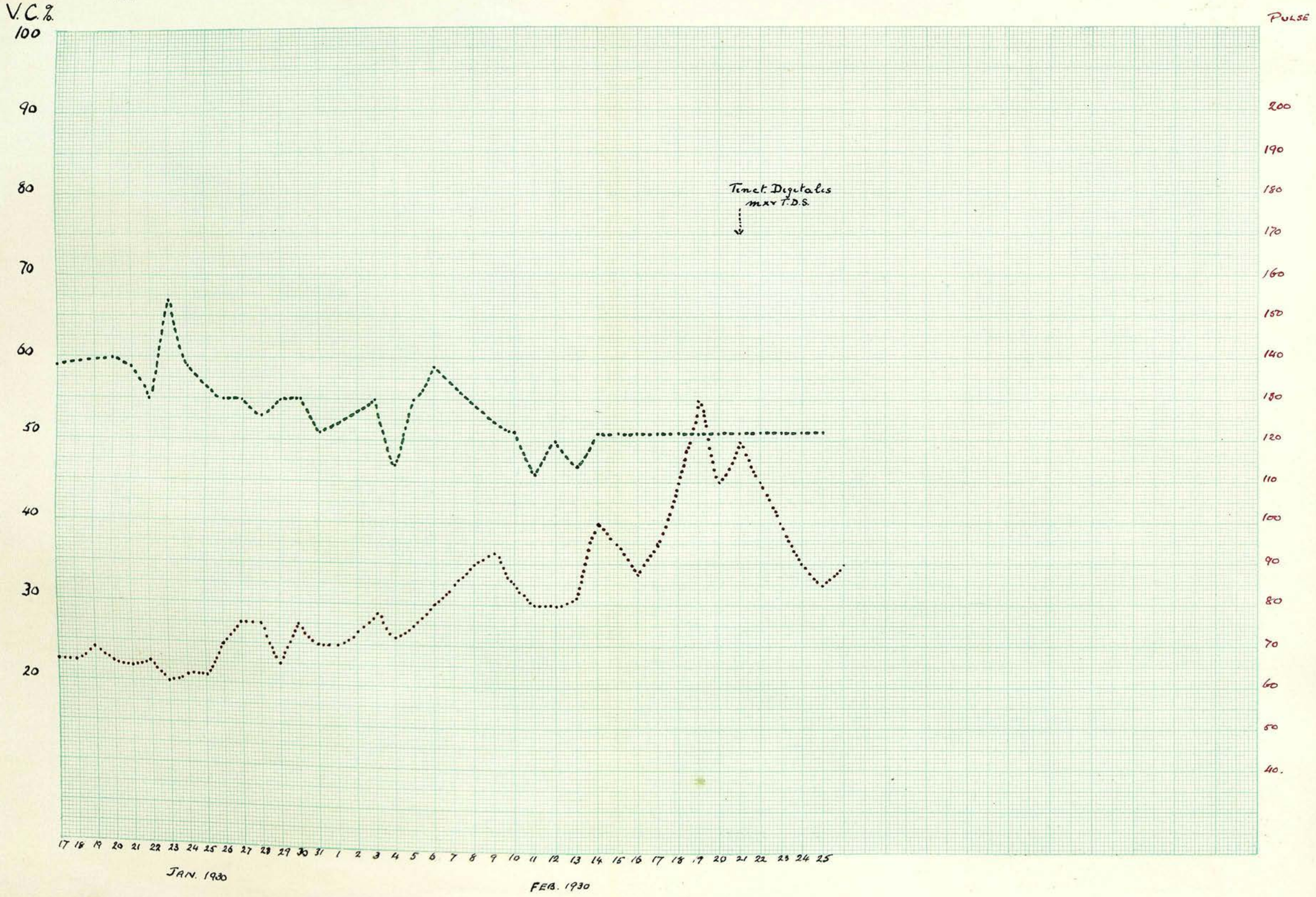
Numerous crepitations were present at both lung bases on admission. On 2/10/28 the bases were clearer and there is a rise in vital capacity (green) which continues till 5/10/28 when all signs of pulmonary oedema had gone.

On 11/10/29 Cheyne Stokes breathing commenced, and there is a corresponding drop in vital capacity.

On 16th a small Right sided pulmonary infarct was discovered, and the vital capacity remained at a low level.

The patient died suddenly on 2/11/28.

MRS. K.


V.C.%
100
90
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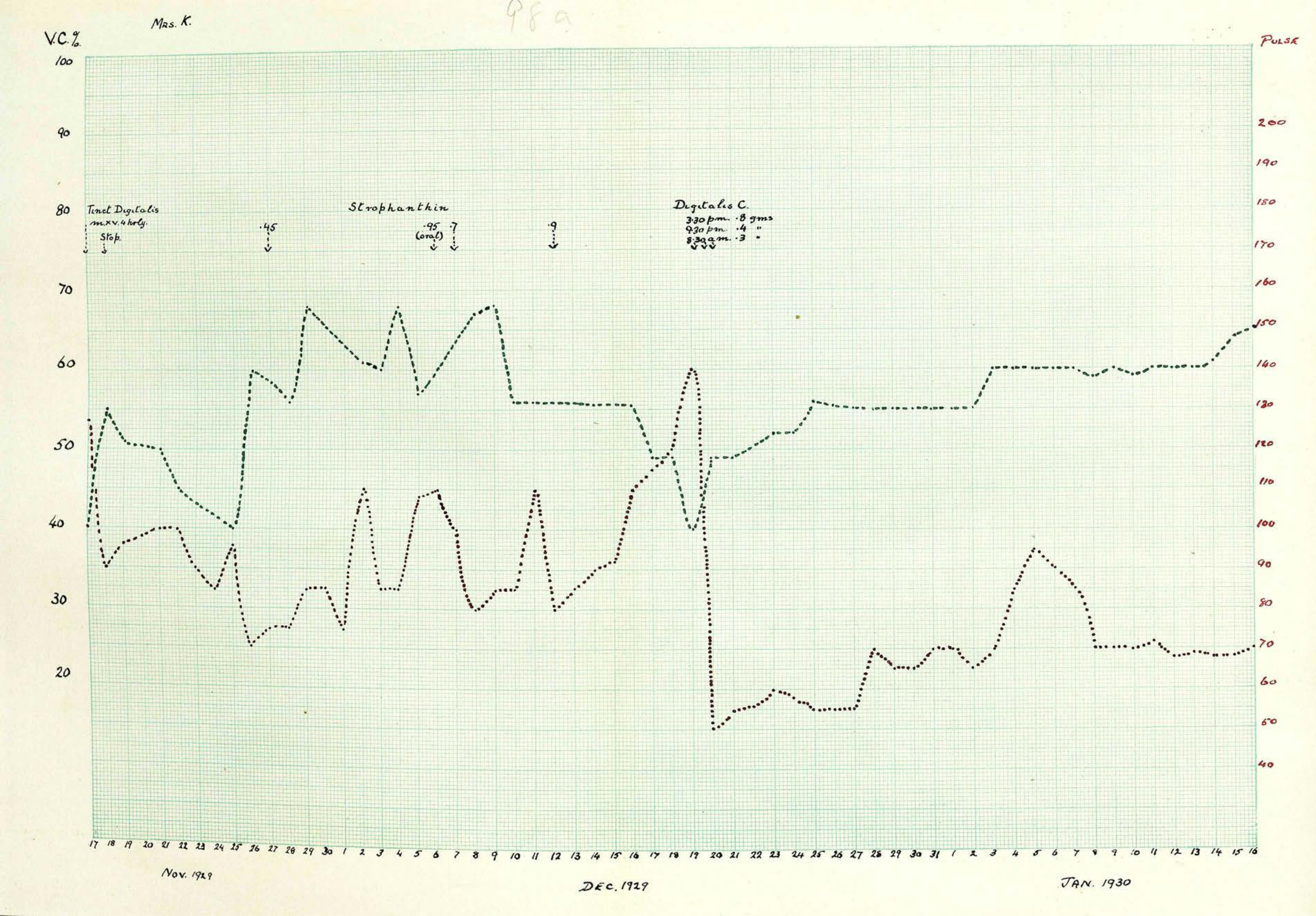
30
20
PULSE
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
40.
Tinct. Digitalis
m xv T.D.S.
17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25
JAN. 1930
FEB. 1930


Mrs. K.
989
V.C. %
PULSE
100
90
80
70
60
50
40
30
20
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
40
Tinct Digitalis
m.xv. 4 hrly.
Stop.
.45
Strophanthin
.95
(oral)
.7
.9
Digitalis C.
3.30 p.m. .8 gms
9.30 p.m. .4 "
8.30 a.m. .3 "
17 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
Nov. 1929
DEC. 1929
JAN. 1930

CASE VII

MRS. K.aet. 43

The patient complained of shortness of breath for the last 6 months, and gave a history of Rheumatic Fever in childhood.

Course of the Illness. The patient was admitted with heart failure. The heart was fibrillating. Rest and Digitalis caused improvement in the general condition of the patient, and from the chart we see that the vital capacity (green) has risen. The heart rate, however, did not settle. On 15/12/29 it is seen to be rising.. On 19th the patient was very breathless, and cyanosed. The vital capacity fell sharply. A massive dose of digitalis brought about an immediate fall in pulse rate, and a rise in vital capacity.

During the last 5 weeks of her stay in hospital, the patient was up and able to walk about the ward. During the last two weeks, the heart rate rose again. Although the rise was considerable the patient felt in her usual health, and there was no drop in vital capacity.

During this rise of pulse rate there was no decompensation and consequently no fall in vital capacity./

capacity.

Throughout the patients stay in hospital the vital capacity readings followed very closely on the patient's clinical condition.

V.C.% Mrs. L.

Puls.

100
90
80
70
60
50
40
30
20

200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50

CASE VIII.

MRS L.

The chart is taken from the case of a woman aet. 59, who suffered from a slow Fibrillation. She gave a history of being breathless for two years. The Heart was not enlarged. Both sounds were closed. From the chart it can be seen that there was a pulse deficit of about 10-14 beats (Apex beat Red. Pulse Black) With Rest and Digitalis the pulse rate came down about 15 beats and there was a corresponding improvement in the patient's Breathlessness.

The vital capacity (green) fell slightly in the first three days, while the pulse rate rose. On 5th day the vital capacity began to rise and continued to do so until the patient's discharge, when it had reached 50%.

R.W.

101a


V.C.%
PULSE.
100
90
80
70
60
50
40
30
20
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10
Dec. 1929
Jan. 1930

CASE IX.

ROBERT W. aet. 59

This man was admitted with Acute heart Failure afterwards found to be due to Right Bundle Branch Block. He gave a history of increasing breathlessness over a period of 10 days.

On admission he was orthopnoeic and at times delirious.

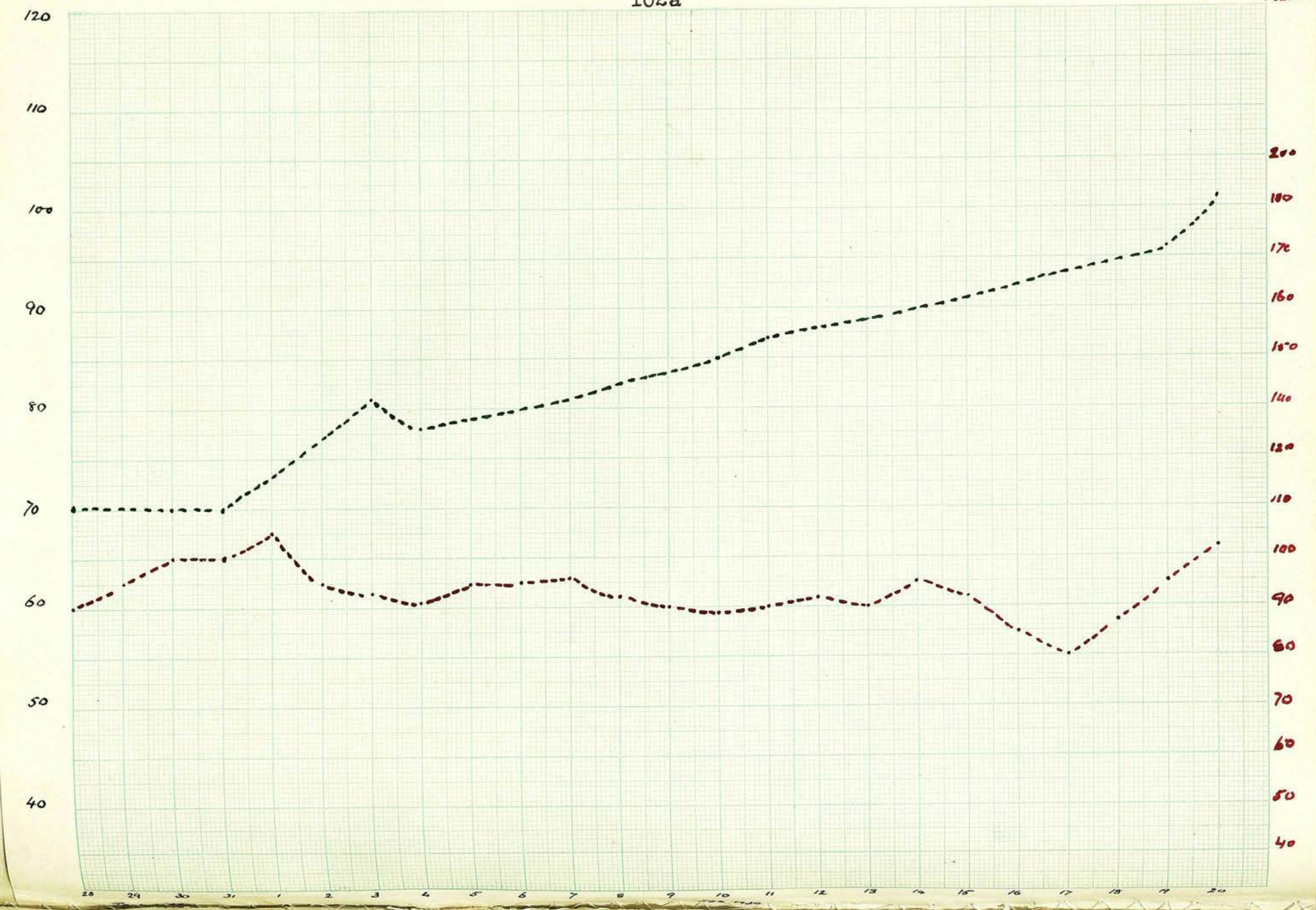
From the graph the pulse rate (red) is seen to have fallen from 90 - 52.

The vital capacity (green) rises from 22% to 75%.

V.C.% R.P.

102a

PULSE



CASE X.

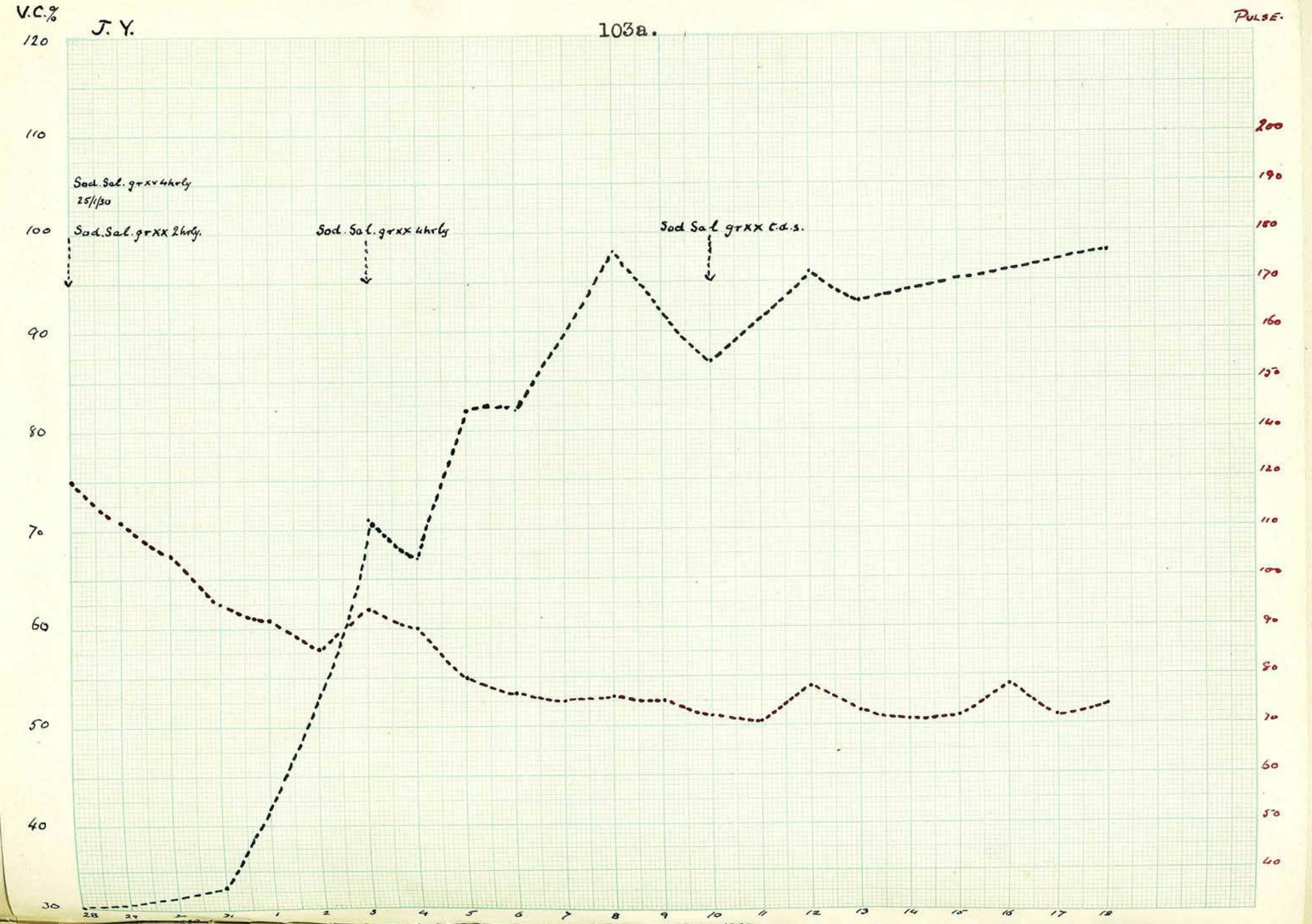
R.P. aet. 22

He was admitted with a history of Rheumatic pains over the last month. The heart was not enlarged, but there was a faint mitral systolic. Patient was very breathless on exertion.

Rest in bed improved his breathlessness to a marked extent, but the stiffness and pain in the joints persisted.

The chart shows the rise in vital capacity from 70 to 101%, which was in keeping with his improved cardiac condition.

J. Y.


V.C.%
PULSE.
120
110
100
90
80
70
60
50
40
30
200
190
180
170
160
150
140
120
110
100
90
80
70
60
50
40
Sod. Sal. grxv 4hrly
25/1/30
Sod. Sal. grXX 2hrly.
Sod. Sal. grXX 4hrly
Sod Sal grXX t.d.s.
28
29
31
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18

CASE XI.

J.Y. aet. 19

He was admitted 28/1/30 with Rheumatic Fever 1st attack. There was a localised area of pericardial friction round the apex beat. By 5th February he had improved greatly, and the rub was only heard with difficulty. Precardial pain which was never marked had disappeared. The patient made an uninterrupted recovery.

The rise in vital capacity (green) is abrupt as soon as the Sod. Sal took effect, and the pericarditis cleared.

CASE XII.

JAMES S. aet. 45

This case seemed of sufficient interest to be included. He was admitted with a history strongly suggestive of myocardial infarct.

On admission his vital capacity was 15%, two days later it had improved to 27%, and the patient seemed much more comfortable.

He died suddenly that night and post mortem showed an infarct of the posterior wall of the Right Ventricle about the size of half a crown.

CASE XIII.

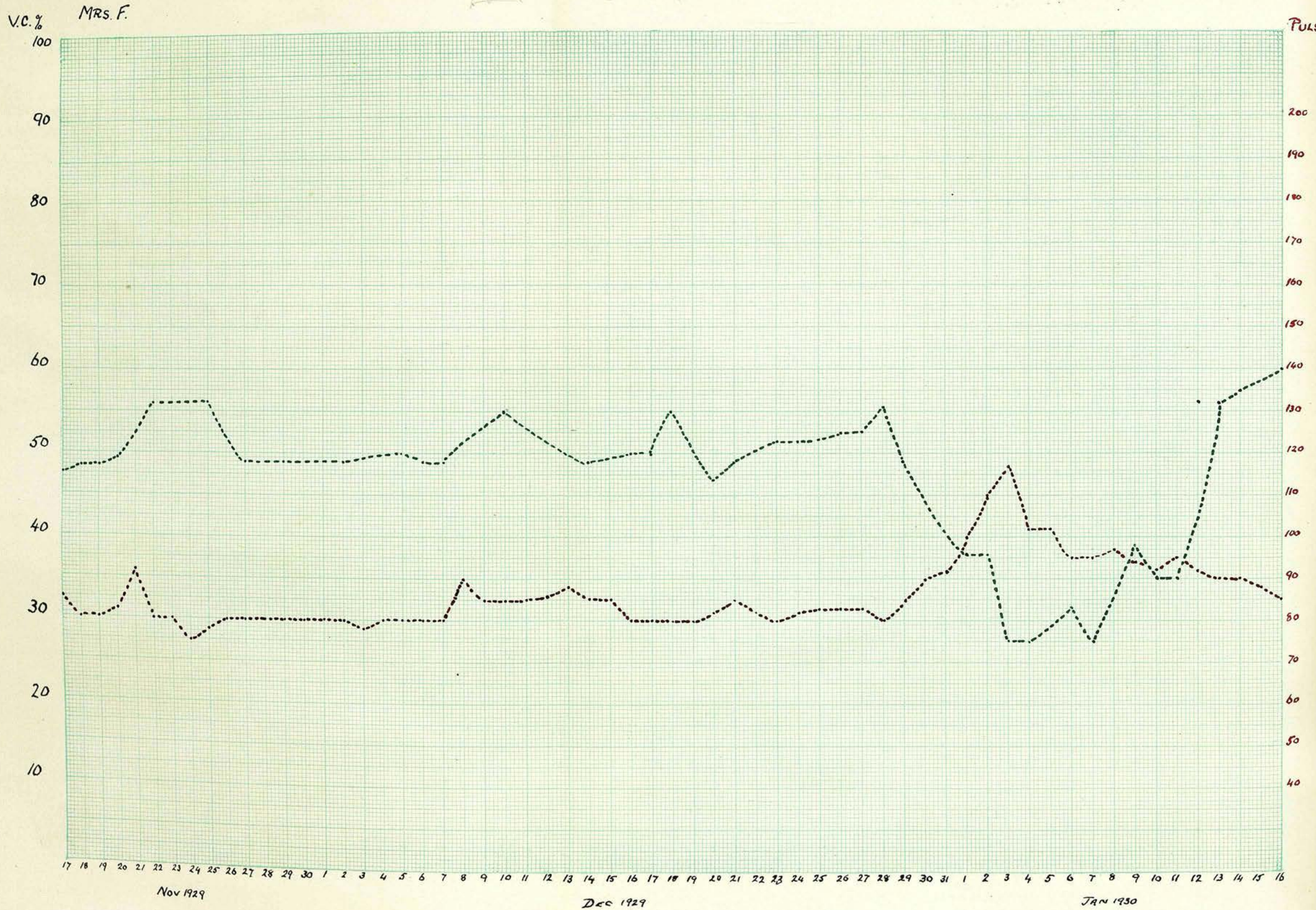
WILLIAM P. aet. 9

This little boy had complete dextrocardia. There was evidence of pulmonary stenosis, and patent intra ventricular Septum.

He was just able to walk 30 yards before having to stop and rest.

With this very marked cardiac disability, he still had a vital capacity of 71%.

~~98a~~ 105a


V.C.%
MRS. F.
PULSE.
100
90
80
70
60
50
40
30
20
10
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
40
17 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
Nov 1929
Dec 1929
Jan 1930

CASE 14. MRS F. aet. 44.

The patient was admitted with acute nephritis a Mitral incompetence was present following on Rheumatic fever in childhood.

No symptoms referable to the circulatory system were complained of.

The graph shows that the vital capacity (green) remains at about 50% until 28/12/29 when the patient developed an acute Bronchitis as well as an exacerbation of the kidney condition. There is an immediate fall in vital capacity and a rise in pulse rate. As the temperature fell and the chest cleared of râles and the vital capacity rose again.

CONCLUSIONS TO BE ARRIVED AT AFTER TAKING DAILY VITAL CAPACITY READINGS ON A SERIES OF CASES SHOWING CARDIAC FAILURE.

I. Changes in vital capacity follow very closely on the clinical condition of the patient.

II. Owing to the uncertainty as to whether on a given day the patient is co-operating properly, a sudden unexpectedly low reading should be regarded with suspicion unless a reason is found on clinical examination.

SOME ASPECTS OF OBESITY.

The association of dyspnoea and obesity led me to study the vital capacity in obese persons in the hope that it might be possible to throw some light on the cause of breathlessness.

Clinical examination of the heart in the obese is very difficult, the excessive amount of fat interferes with the examination of the size of the heart and the position and condition of the apex beat. The thick chest wall makes it difficult to estimate the quality of the first sound in the mitral area. The sound is fainter than usual and consequently it is difficult to distinguish a distinction of intensity of the first sound from weakening associated with tic-tac rhythm, for the even spacing of the sounds is not altered in the latter condition.

Breathlessness and obesity so frequently go hand in hand that one is often tempted to diagnose "fatty heart".

Three main types of so-called "fatty heart" are generally described:-

(1) Fatty loading along the blood vessels.

(2) Fatty infiltration.

(3) Fatty degeneration.

Clinically/

Clinically it is impossible to differentiate them. Ventricular Hypertrophy is common in obese people and is probably the result of extra strain. The enlarged heart has no corresponding increase in its own blood supply and as in other hypertrophied organs premature senility may result. There is not sufficient evidence to suggest that in the early case of obesity the heart is necessarily weak.

Of the secondary changes which are likely to occur, coronary sclerosis is regarded by some as the most common. High blood pressure and obesity are frequently met with. The excessive accumulation of fat in the pericardium may, in itself, lead to atrophic changes in the heart muscle and consequently to inefficiency of the latter.

EAST AND BAIN (39) are doubtful whether a fatty (i.e. obese) heart exists as a pathological entity.

The activities of the obese are necessarily curtailed and it may be that part at any rate of the dyspnoea produced on exertion depends on their being in "bad training". They also have a heavy load of fat to carry, so a vicious circle limiting exertion is set up. The fatter the patient is the less he can do, he takes less and less exercise the more his fat increases.

In/

In young and especially muscular persons it may require a long time for secondary changes to be brought about during which time the obese person may enjoy good health as far as his cardio-respiratory apparatus is concerned.

DU BRAY (46) believes that the effect of any pathological change in the heart in the obese subject is intensified by the presence of a high diaphragm which is a common finding in the obese type of body build.

EAST AND BAIN have observed that if there be no heart disease, exertion produces marked quickening of the respiratory rate rather than prolonged acceleration of the heart rate.

In studying the vital capacity of obese patients any reduction recorded must in all probability be due to one or other or both of the following causes:-

(1) Limitation of the free movement of the chest or diaphragm.

(2) Reduction in the size of the alveoli owing to vascular congestion secondary to a weak or embarrassed heart muscle.

When a normal individual over 21 years of age starts to put on weight the increase is brought about/

about to a large extent by subcutaneous and visceral fat deposits. The lung receives none of the additional weight and consequently one would expect the vital capacity to remain unchanged, when the body weight increases.

THE/

THE CHOICE OF A VITAL CAPACITY STANDARD IN CASES OF OBESITY.

BOWEN & PLATT (19) investigating 32 overweight and obese subjects found the height standard the most satisfactory. If the Surface area standard were used they found it necessary to add 20% to the observed vital capacity.

WARREN BELL (52) has studied the effect of pregnancy on vital capacity. He found, that mechanical factors play a very small part. If any diminution in vital capacity was found in a pregnant woman, it was present also after delivery and was recovered from during convalescence. In his opinion results based on weight standards are illogical during pregnancy.

MYERS (33) has shown that in the normal growing person, the vital capacity tends to follow the increase in height, rather than the increase in weight.

He suggests that this is probably due to the fact that lung growth accompanies the growth of the body in height.

In dealing with the obese subject the lungs have/

have not grown as the weight increased, so the usual correlation of vital capacity weight and surface area is lost.

It would seem that the height standard should be used. I can see no contraindication to using the surface area standard obtaining the surface area from the patient's correct weight obtained from Insurance tables.

In the series of obese subjects I have examined, the vital capacity has been worked out using

(1) The Surface Area standard, and the patient's actual weight.

(2) The Surface Area Standard and the patient's correct weight.

(3) The Height Standard.

CHRISTIE & BEAMS found that in the recumbent position the vital capacity of an obese patient was reduced by 15%, as opposed to a 5.5% reduction in the normal person. This correction has been made where necessary

THE VITAL CAPACITY IN OBESE AND OVERWEIGHT SUBJECTS.

Between November 1929 and March 1930, 50 obese and overweight females were examined from the point of view of their vital capacity, in the hope of throwing some light on the cause of the dyspnoea so commonly met with in obese subjects.

As I have already stated great difficulty was experienced in getting reliable readings from women. Unfortunately, it was impossible to get sufficient material to use male patients.

In all 64 obese and overweight women were examined. But I have had to discard 14 as they were incapable of recording accurate readings.

All the cases had vital capacity readings taken on two or more occasions.

On examining the figures of the completed series I found that in many cases the first few readings were inaccurate, or that the patient at subsequent examinations was unable to read her previous figure, although no cause could be found.

I quote the cases of 6 obese persons who lost weight and recorded a considerable rise in vital capacity/

capacity. I am reasonably certain that these patients were giving their best, and on each, from 6-20 separate readings on different days were taken, and all were comparable.

Of the 50 cases, 32 lost 5 or more lbs. in weight, but the majority of the readings in their series were palpably inaccurate and I am unable to include them.

This failure to obtain a large number of reliable readings is disappointing. I believe, however, the readings of the 50 cases I quote to be accurate. From over 350 separate readings, I have selected the height reading for each patient irrespective of a loss or gain in weight.

In view of this, I feel justified in drawing conclusions from this group.

A COMPARISON BETWEEN THE RESULTS GIVEN BY THE VARIOUS STANDARDS USED IN A SERIES OF 40 CASES OF OBESITY.

I have considered all cases 25% of overweight and up as being obese. This I admit is quite an arbitrary standard.

A detailed comparison between the three standards I have employed has been made the Height standard and capacity.

The Height Standard gave a higher reading than the Surface area standard using the patients actual weight in every case. From the table we see that the difference increases with the degree of obesity.

BOWEN and PLATT examining 32 obese and overweight people state that if the surface area standard be used 20% should be added to the result. From this table I conclude that their figure of 20% is too high. I SUGGEST THAT 13% BE THE MAXIMUM FIGURE TO BE ADDED.

NUMBER OF CASES	% OVERWEIGHT	Ht. STANDARD HIGHER THAN THE S.A. STANDARD by:-
18	25% - 49%	8·05% V.C.
14	50% - 74%	11·1% V.C.
8	75% - 91%	12·5% V.C.

TABLE showing that the Height Standard gives a higher reading than the Surface Area Standard in cases of obesity. The difference between the two Standards is seen to increase with the rise in weight.

If we compare the Height Standard and the Surface area Standard when the patients correct weight is used (I have called this for convenience the Normal Surface area Standard N.S.A.) we find:-

In 6 cases 25·49% overweight. The N.S.A. Standard was higher than the Height Standard by 2·1%

In 11 cases 25·49% overweight. The Height Standard was higher than the N.S.A. Standard by 2·2%

In one case the two Standards corresponded.

In 4 cases 50%-74% overweight. The N.S.A. Standard was higher than the Height Standard by 3·7%

In 8 cases 50%-74% overweight. The Height Standard was higher than the N.S.A. Standard by 1·5%

In two cases the two Standards corresponded.

In 3 cases 75%-91% overweight. The N.S.A. Standard was higher than the Height Standard by 1·3%

In 3 cases 75%-91% overweight. The Height Standard was higher than the N.S.A. Standard by 4%

In two cases the Standards corresponded.

I think this analysis prooves my contention that in dealing with cases of obesity it is permissable to use The Surface area Standard where the Surface area is obtained form the patients height and his CORRECT weight.

% Overweight	25%-49%	50%-69%	70%-90%
Number of Cases	18	11	11
Area of the Surface average Vital Capacity Height Standerd.	81%	68.3%	63%

From this table we see that there is a reduction in Vital capacity as the weight increases. The average vital capacity for all the obese Cases is 70.7%.

THE EFFECT OF A REDUCTION IN WEIGHT ON THE VITAL CAPACITY.

Five patients were found to be able to record consistent readings and each of these lost 20 lbs. or more during the time they were under observation.

In the following cases the Height standard is used. The numbers refer to the Main Table.

CASE 4.

MRS. S. aet. 45 obese for 11 years.

	WEIGHT	OVERWEIGHT	VITAL CAPACITY
19/11/29	190 Lbs	32%	56%
26/2/30	164 Lbs	12%	73%

Her weight was reduced by 26 lbs. and her vital capacity rose 17%.

CASE 6.

MRS. J.B. aet. 49 obese for 20 years.

	WEIGHT	OVERWEIGHT	VITAL CAPACITY
19/11/29	260 Lbs	75%	72%
19/2/30	238 Lbs	51%	81%

Her weight was reduced 22 lbs. and her vital capacity rose 11%.

CASE 10./

CASE 10.

MRS. M. aet. 41 obese for 20 years.

	WEIGHT	OVERWEIGHT	VITAL CAPACITY
22/11/29	225 lbs.	61%	86%
24/1/30	203 lbs.	52%	89%
19/2/30	180 lbs.	40%	93%
2/3/30	166 lbs.	37%	105%

The patient lost 59 lbs. and her V.C. increased by 19%.

CASE 25.

MRS.M. aet. 57 obese for 8 years.

	WEIGHT	OVERWEIGHT	VITAL CAPACITY
4/12/29	228 lbs.	39%	72%
5/3/30	201 lbs.	24%	96%

The patient lost 27 lbs. and her V.C. increased by 24%.

CASE 31.

MISS M. aet. 36 years obese for 5 years.

	WEIGHT	OVERWEIGHT	VITAL CAPACITY
24/1/30	210 lbs.	52%	58%
10/2/30	196 lbs.	45%	65%
3/3/30	187 lbs.	39%	65%

The total loss in weight in this case is 23 lbs. and a rise of 7% is recorded in vital capacity.

CASE NO.	% LOSS IN WEIGHT	% RISE IN V.C.
10	24%	19%
4	20%	17%
6	16%	11%
25	15%	24%
31	13%	7%

Table showing how a reduction in weight produces a rise in vital capacity.

S U M M A R Y.

After examining these 5 cases it would seem that a decrease in weight in the obese subject is followed by a rise in vital capacity.

The table shows if we except case 25, the rise in capacity is in accordance with the amount of weight lost.

In those cases who were overweight, I found no constant fluctuation in the vital capacity as weight was gained or lost.

THE VITAL CAPACITY OF 50 OVERWEIGHT AND OBESE FEMALES.

CASE NO	AGE	WEIGHT Lbs.	HEIGHT feet Inches	VITAL CAPACITY C.C.	SURFACE AREA	% V.C. of S.A. STANDARD	% V.C. HEIGHT STANDARD	NORMAL WEIGHT Lbs.	NORMAL SURFACE AREA	% V.C.	% OVERWEIGHT
1	40	204	4•10	2200	1•87	61	75	123	1•47	76	71
2	29	168	5•2	2900	1•84	83	92	137	1•61	91	26
3	65	212	5•1	1300	1•95	35	42	135	1•59	41	61
4	45	190	5•5	1850	1•94	51	56	147	1•72	54	32
5	48	164	5	1600	1•72	48	52	134	1•56	51	25
6	49	260	5•6	2400	2•3	54	72	152	1•77	68	75
7	60	210	5•4	1850	1•99	50	56	144	1•69	54	48
8	49	198	4•10	2200	1•85	62	75	128	1•5	73	59
9	41	232	5•3	1600	2•1	40	50	139	1•65	49	74
10	41	225	5•2	2700	2	70	86	133	1•6	85	66
11	36	195	5•1	3050	1•87	88	98	126	1•54	99	63
12	52	154	4•8	1400	1•6	46	49	125	1•44	49	26
13	37	218	4•11	2200	1•93	59	73	122	1•48	75	83

14	42	236	5•2	2000	2•06	52	63	133	1•6	63	82
15	41	206	4•11	1000	1•9	27	33	126	1•5	33	68
16	65	160	5•4	1000	1•76	30	31	144	1•7	30	15
17	52	163	5•6	1700	1•88	48	51	152	1•77	49	10
18	69	153	5•3	2000	1•73	60	62	141	1•65	61	11
19	35	232	5•4	1400	2•1	35	43	130	1•61	44	83
20	44	211	5•3	3000	1•96	80	94	138	1•64	92	57
21	65	158	4•10	1500	1•64	48	51	129	1•5	50	23
22	49	251	5	1900	2•04	48	62	132	1•55	62	91
23	44	184	5•7	2450	1•82	69	72	153	1•8	68	19
24	50	156	5•1	1900	1•69	58	61	135	1•59	60	14
25	57	228	5•8	2500	2•14	61	72	163	1•86	67	39
26	50	220	5•5	2900	2•04	74	88	148	1•73	84	47
27	50	180	5•4	2200	1•85	62	68	144	1•67	69	27
28	40	174	5•1	2200	1•76	66	71	129	1•52	76	23
29	44	212	5•2	2450	2•	64	77	135	1•58	81	58

CASE NO	AGE	WEIGHT Lbs.	HEIGHT feet, Inches	VITAL CAPACITY C.C.	SURFACE AREA	% V.C. of S.A. STANDARD	% V.C. HEIGHT STANDARD	NORMAL WEIGHT Lbs.	NORMAL SURFACE AREA	% V.C.	% OVERWEIGHT
30	36	175	5•	1700	1•75	51	56	124	1•49	59	41
31	36	210	5•4	1900	2•	50	58	138	1•61	62	52
32	14	175	5•5	3100	1•86	70	94	122	1•5	108	43
33	41	185	8•1	2100	1•83	60	68	130	1•54	70	42
34	14	160	5•2	2600	1•72	80	83	112	1•47	93	42
35	58	193	5•2	2200	1•87	61	70	138	1•62	71	40
36	61	215	5•1	2200	1•95	59	71	135	1•59	76	60
37	59	253	5•2	1800	2•11	47	56	135	1•61	57	90
38	56	224	5•7	2400	2•11	60	71	158	1•81	69	42
39	28	223	5•1	2400	1•98	63	77	121	1•51	83	85
40	48	176	4•10	1800	1•71	55	61	128	1•5	63	39
41	60	174	5•3	2000	1•82	57	62	141	1•65	63	24
42	30	220	5•9	2500	2•18	47	57	159	1•81	56	50

43	55	215	5•6	2700	2•02	70	81	153	1•78	79	39
44	28	204	4•11	2000	1•85	56	67	117	1•46	72	74
45	29	236	5•5	1800	2•11	45	55	133	1•65	57	76
46	55	226	5•7	2100	2•11	52	62	158	1•8	61	42
47	26	168	5•4	2500	1•8	72	77	128	1•6	81	29
48	36	192	5•4	2200	1•9	88	98	135	1•65	101	47
49	41	236	5•5	2000	2•11	50	61	143	1•7	61	64
50	41	162	5•2	3300	1•85	93	105	133	1•58	107	40

DISCUSSION AS TO THE CAUSE OF THE REDUCTION OF VITAL CAPACITY IN OBESITY.

We have seen that the average vital capacity in the 40 obese subjects was 70.7%. Even when due regard is given to the effect of advancing age, this figure is low.

I have proved that, as the weight increases, the vital capacity tends to fall, and to rise again when the weight is reduced.

This suggests very strongly that some mechanical factor is at work causing a reduction of the vital capacity. It is conceivable that large fat deposits coupled perhaps with abdominal distension (a common finding in obese subjects) hinder the movements of the diaphragm. Yet, on the other hand it has been shown that pregnancy does not reduce the vital capacity to any great extent, and what slight reduction there may be, is also found after the birth of the child, and is recovered from during convalescense.

It is a well known fact that in patients with visceroptosis respiration is largely thoracic and KEITH has suggested that this is due to poor abdominal musculature.

The/

The muscles of the abdominal wall provide a full run for the movements of the diaphragm. If the abdominal muscles are weak diaphragmatic movement is impaired.

It is conceivable that in obese people the abdominal muscles are weak (their tendency to develop Herniae would bear this out) By limiting the movement of the diaphragm the vital capacity would be decreased.

In the recumbent position the normal person's vital capacity is reduced by 5.5%. In the obese person, however, it is reduced by 15%.

The reduction in vital capacity when lying down, is probably due to the fact that the diaphragm has to push down the liver and other abdominal organs. In the healthy normal person, this is fairly efficiently done, but in the obese person with, we have assumed, weak abdominal muscles, the diaphragm is unable to push down the viscera and consequently in the recumbent position the vital capacity is greatly reduced.

A reduction in weight in the obese subject results in increased activity. The general condition of all the muscles including those of the abdominal wall improve, and the vital capacity rises again.

Of/

Of the other possible causes of the reduction in vital capacity noted in obese subjects, factors which embarass the working of the heart, must be considered. Increase in the amount of fat in the pericardium, or the definite cardiac changes already described as being met with in obesity, might presumably reduce the vital capacity.

CONCLUSIONS/

CONCLUSIONS THAT MAY BE ARRIVED AT AFTER STUDYING THE VITAL CAPACITY IN OBESE SUBJECTS.

(i) In working with obese subjects the Height standard, or the Surface area standard using the patient's correct weight, are equally satisfactory.

(ii) If the Surface area standard using the patient's observed weight be used, I suggest BOWEN & PLATT'S correction of 20% be reduced to 13%.

(iii) As has been found before, great difficulty was experienced in obtaining reliable readings from female patients.

(iv) Obese subjects tend to record a low vital capacity 70.7% being the average vital capacity of 40 obese patients.

(v) A reduction of weight in the obese causes a corresponding rise in vital capacity.

(vi) I suggest impaired movement of the diaphragm as the chief cause in the reduction of vital capacity observed.

(vii) Breathlessness in the obese may in part at any rate/

rate be due to the very fact that the vital capacity is reduced.

(viii) Vital capacity readings afford no help in diagnosing myocardial changes in the individual obese Subject. As a class the vital capacity falls as the weight increases. I can find no definite fall in vital capacity as the age of the patients increase, nor does the length of time the patient has been obese, affect the vital capacity prediction.

(iv) No consistent changes were observed in the vital capacity of the 10 overweight persons when their weight was reduced.

A FURTHER ATTEMPT TO INVESTIGATE THE PROBLEM OF THE "FATTY HEART".

CORRELATION OF VITAL CAPACITY READINGS & ELECTRO CARDIOGRAPHIC RECORDS in 32 OBESE SUBJECTS

I have had to admit failure to throw any light on the condition of the myocardium in the individual obese subject by using vital capacity readings. While as a class the obese people have a low vital capacity, and their capacity tends to fall with increasing weight individuals show wide variations.

It occurred to me that, the electro cardiogram might give help. A search through the literature failed to reveal any work on the findings to be expected in obese patients.

An obese patient in whom, clinically one would suspect myocardial changes, and whose vital capacity was low, was chosen as the first subject. Definite changes in the cardiogram were observed.

The next patient though obese felt well and was not unduly breathless. Her vital capacity was high. A normal electro cardiogram was returned.

This encouraging start led me to take a series of electro cardiograms. In all 32 of the obese patients/

patients were studied from the point of their vital capacity and Electro cardiogram.

VITAL CAPACITY AND ELECTRO CARDIOGRAPH FINDINGS IN 32 OBESE SUBJECTS.

INDEX OF TERMS TO BE EMPLOYED IN ELECTRO CARDIOGRAPHIC DIAGNOSIS.

1. R.S.S. = Regular Sinus Rhythm.

2. S.T. = Sinus tachycardia (not paroxysmal 100 per minute and over)

3. P.C. = Premature Contractions

   (a) V.P.C.R. = Right Ventricular premature contraction.

4. "P" Wave P.

   (a) Pneg = Inverted P.

   (b) Pwd = Wide P.

5. Q.R.S. = Ventricular Complex.

   (a) Q.R.S.^{sm} = small decreased potential (.5 cms. or less)

   (b) Q.R.S.^{vy} = Q.R.S. varying in shape.

   (c) V.P.L. = left ventricular preponderence.

   (d) Q.R.S.^{sp} = splitting, notching, or distortion of the main deflection.

   Q.R.S.$^{sp\ 23}$ = Q.R.S. split in lead II and III

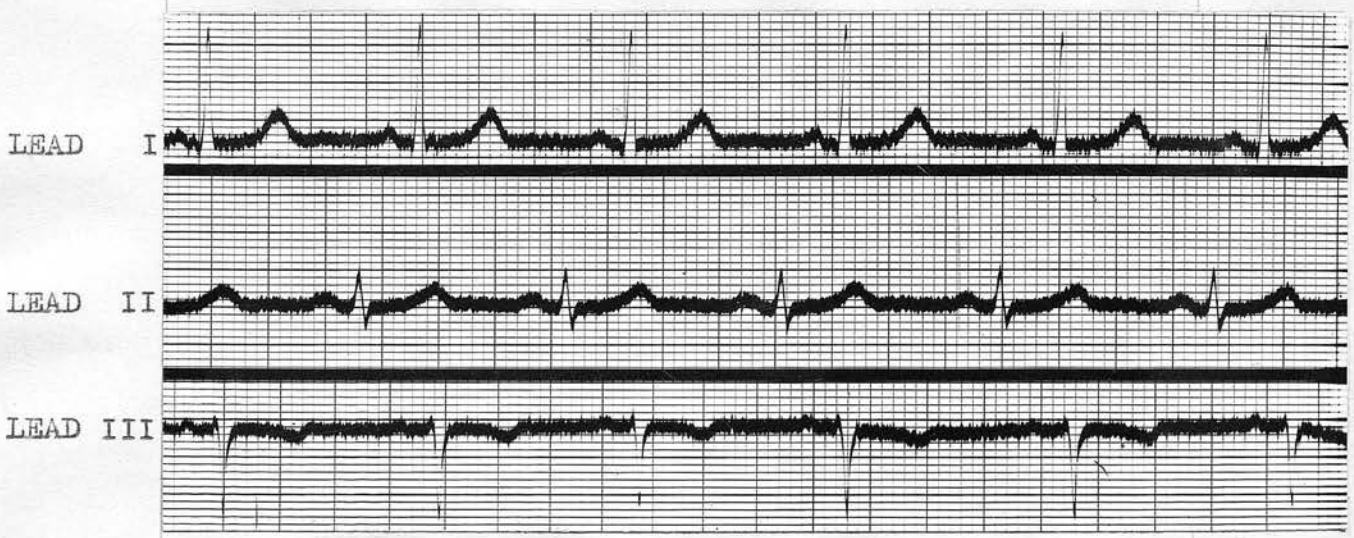
   (e) Q Prominent Q waves.

6. The T wave

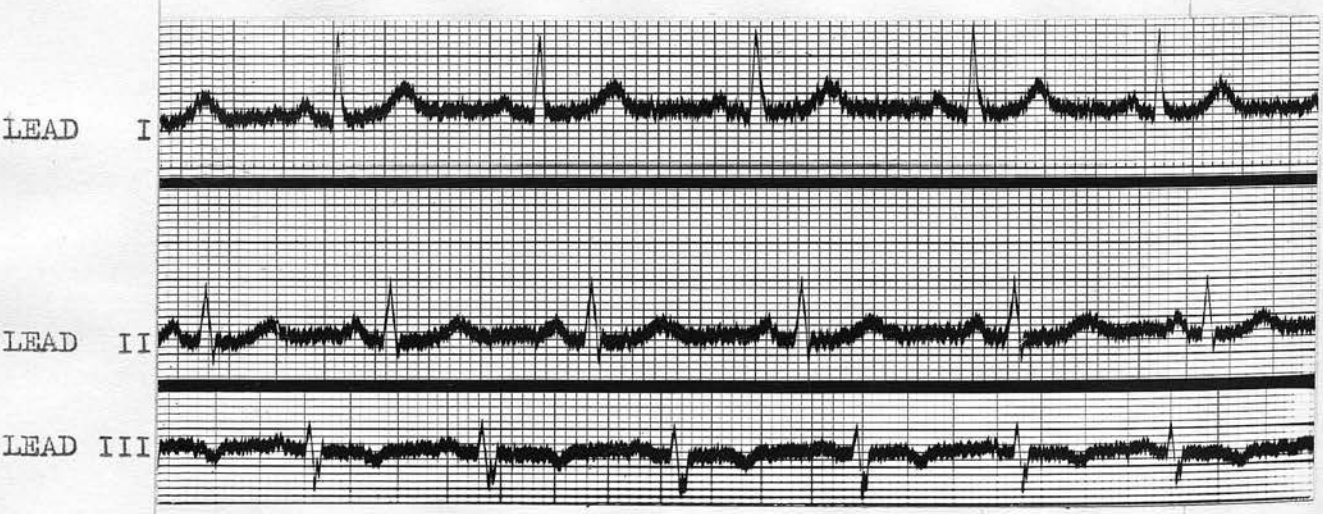
   (a) +++ All positive

   (b) ++- Negative in lead III.

TYPICAL ELECTRO CARDIOGRAMS FOUND DURING THE EXAMINATION OF OBESE PATIENTS.



I. The cardiogram shows left ventricular preponderence and inverted T. in Lead 3. 1cm = 1mv. Time Marker 1/5th & 1/25th sec.



II. Cardiogram showing left venticular preponderence a splitting of Q.R.S. and inversion of T in Lead III 1cm = 1mv. Time Marker 1/5th & 1/25th sec.

CHANGES OBSERVED IN THE ELECTRO CARDIOGRAPHING OF 32 OBESE SUBJECTS.

After examining the 32 electro cardiograms two main types can be defined:-

I. Left ventricular preponderence and an inverted T wave in lead III.

II. A split in the Q.R.S. combined with left ventricular preponderence and inverted T in lead III.

Variations of these deviations which are generally accepted as coming within the bounds of normality were also found.

Although, V.P.L. is often found in normal people, the fact that it appears in 22 out of 32 (70%) obese subjects is worthy of note.

In 18 cases (56%) the V.P.L. was associated with a negative T in lead III in 2 cases T was absent. In 16 cases (50%) Q.R.S. was split.

$Q.R.S._{sp_2}$	2 cases
$Q.R.S._{sp_3}$	8 cases
$Q.R.S._{sp_{12}}$	1 case
$Q.R.S._{sp_{123}}$	3 cases
$Q.R.S._{sp_{23}}$	2 cases

In 13 cases (40%) a split Q.R.S. is associated with V.P.L.

A negative or absent T wave occurs in 24 cases (75%).

TYPE OF ELECTRO CARDIOGRAPHIC ABNORMALITY.	OBESE SUBJECTS	NORMAL SUBJECTS
V P L.	70%	46·6% (Heard & Hein)
V P L ++-	56%	39·8% (Willins)
T neg or absent in head III.	75%	25% (Wiggers)
Q R S split	50%	no data discovered.

TABLE showing the Main Types of abnormality found in electro cardiograms of obese subjects compared with the frequency of their occurrance in normal people.

Other changes in the electro cardiogram were observed:-

$Q.R.S.vy_3$	4 cases
$Q.R.S._{sm.3}$	5 cases
$Pneg_3$	2 cases
P_{sm3}	2 cases
P_{wd2}	1 case

In only 4 cases (12.5%) out of 32 is the electro cardiogram normal and in two of these Q.R.S. is definitely diminished in size in lead III.

The electro cardiographic findings seem to have no relation to the observed vital capacity of the patients.

Such factors as the % overweight, the age, the time the patient has been obese, or the degree of breathlessness, do not in this series enable one to predict the type of abnormality to be expected in the cardiogram, or indeed whether any abnormality will be found at all.

32 CASES FROM WHOM ELECTRO CARDIOGRAPHIC RECORDS WERE TAKEN.

MISS M. aet. 41, 20 years obese, slightly breathless.
24/1/30 55% overweight v.c. 89%.
Electro cardiogram R.S.R. Q.R.S. $_{sm3}$ +++
normal.

MRS B. aet 49, 20 years obese, very breathless.
19/2/30 51% overweight, V.C. 81%.
Electro cardiogram S.T. V.P.L. Q.R.S. $_{sp3}$
+ + 0.

MRS E. aet. 60, 7 years obese, slightly breathless.
5/3/30, 43% overweight v.c. 58%.
Electro cardiogram. R.S.R. V.P.L. + + -

MRS D. aet. 49, 15 years obese, very breathless.
5/3/30, 58% overweight, v.c. 71%
Electro cardiogram R.S.R. V.P.L. Q.R.S. $_{sp3}$
+ + +

MRS H. aet. 42, always obese, breathless.
5/2/30, 70% overweight, v.c. 87%.
Electro cardiogram 11/12/29, R.S.R. Q.R.S. $_{sp\ 3}$
C.T.$_3$ + + - 5/2/30, R.S.R. Q.R.S. $_{sp3}$
V.P.L. + + -

MRS Y./

MRS Y. aet. 41, 15 years obese, very breathless.
27/11/29, 68% overweight, v.c. 33%.
Electro cardiogram R.S.R. P_{neg_3} Q.R.S. sp 3
+ + 0

MISS L. aet. 30, always obese, breathless
29/1/30, 48% overweight, v.c. 79%.
Electro cardiogram 4/12/29, R.S.R. Q.R.S. sm 3
Q.R.S. vy_3 + + - 29/1/30, R.S.R.
Q.R.S. sp_3 Q.R.S. vy_3 + + -

MRS M. aet. 35, 10 years obese, breathless.
27/11/29, 83% overweight, v.c. 43%.
Electro cardiogram R.S.R. P_{sm_3} Q.R.S. sp_{123}
+ + +

MISS W. aet. 49, 10 years obese, very breathless.
4/12/29, 91% overweight, v.c. 62%.
Electro cardiogram R.S.R. V.P.L. Q.R.S. sp 3
+ + -

MRS M. aet. 57, 8 years obese, very breathless.
5/3/30, 24% overweight, v.c. 96%
Electro cardiogram R.S.R. P_{wd_2} V.P.L. Q.R.S. sp 12
+ + -

MRS E. aet. 50, 16 years obese, not breathless.
4/12/29, 47% overweight, v.c.88%
Electro cardiogram R.S.R. V.P.L. Q.R.S. sp 23
+ + -

MRS M./

MRS M. aet 41, 10 years obese, very breathless.
5/3/30, 64% overweight, v.c. 61%
Electro cardiogram NORMAL.

MRS L. aet. 40, 1 year obese, slightly breathless,
5/3/30, 38% overweight, v.c. 65%.
Electro Cardiogram R.S.R. + + -

MRS N. aet 44, 4 years obese, breathless.
11/12/29, 58% overweight, v.c. 77%.
Electro cardiogram R.S.R. V.P.L. + + -

MRS W. aet. 36, 5 years obese, very breathless.
11/12/29, 41% overweight, v. c. 56%
Electro cardiogram R.S.R. Pvy_3 Q.R.S.vy_3
V.P.L. + + -

MISS M. aet 36, 5 years obese, very breathless.
24/1/30, 52% overweight, v. c. 58%
Electro cardiogram R.S.R. $Pneg_3$ V.P.L + + -

MRS D. aet. 41, 4 years obese, very breathless.
29/1/30, 42% overweight, v.c. 68%.
Electro cardiogram R.S.R. V.P.L. Q.R.ssm_3
Q.R.S.vy_3 + + -

MRS C. aet. 58, 10 years obese, breathless.
5/2/30, 40% overweight v.c. 70%
Electro cardiogram S.T. Q.R.S.vy_3 Q_3 + + -

MRS W./

MRS W. aet. 61, 35 years obese, slightly breathless.
5/2/30, 60% overweight, v.c. 71%.
Electro cardiogram R.S.R. V.P.L + + -

MRS S. 20 years obese, very breathless.
5/2/30, 90% overweight, v.c. 56%.
Electro cardiogram R.S.R. V.P.L. + + -

MRS M.S. aet 56, 25 years obese, very breathless
12/2/30, 42% overweight, v.c. 71%.
Electro cardiogram NORMAL.

MRS R. aet. 28, 7 years obese, very breathless.
5/3/30, 77% overweight, v.c. 87%.
Electro cardiogram S.T. V.P.L. $Q.R.S.sp_3$
+ + +

MRS I.B. aet 60, 5 years obese, very breathless.
26/2/30, 24% overweight, v.c. 62%.
Electro cardiogram R.S.R. V.P.L. Q.R.S.sp
123
+ + -

H. aet. 30, 1 year obese, very breathless.
3/3/30, 56% overweight, v.c. 57%.
Electro cardiogram $Q.R.S.sm_3$ NORMAL.

MRS K. aet. 55, 15 years obese, very breathless
5/3/30, 39% overweight, v.c. 81%.
Electro cardiogram R.S.R. V.P.L. + + 0

MRS B. aet. 28, 3 years obese, very breathless.
5/

5/3/30, 77% overweight, v.c. 87%.

Electro cardiogram R.S.R. V.P.L. Q.R.S.vy

+ + -

MRS H. aet. 29, 3 years obese, slightly breathless.

5/3/30, 76% overweight, v.c. 55%.

Electro cardiogram S.T. V.P.C.R. $Q.R.S.sp_3$

+ + -

MRS K. aet. 26, 2 years obese, very breathless

5/3/30, 29% overweight, v.c. 77%

Electro cardiogram S.T. Psm_3 $Q.R.S.sp_2$

V.P.L. + + -

MRS D. aet. 36, 6 years obese, very breathless.

5/3/30, 47% overweight, v.c. 98%.

Electro cardiogram S.T. V.P.L. $Q.R.S.sm_3$

$Q.R.S.sp_3$ + + -

MRS M. aet. 49, 12 years obese, breathless

5/2/30, 33% overweight, v.c. 73%.

Electro cardiogram R.S.R. V.P.L. $Q.R.S.sm_3$

+ + -

MRS B. aet. 60 5 years obese.

5/3/30, 33% overweight, v.c. 62%.

Electro cardiogram R.S.R. V.P.L. $Q.R.S.sp_{2.3}$

$Q.R.S.sm_3$ + + -

MRS McK. aet. 44, 10 years obese.

5/3/30, 49% overweight, v.c. 78%

Electro cardiogram R.S.R. Psm_3 V.P.L.

$Q.R.S.sp_2$ + + -

DISCUSSION ON THE ELECTRO CARDIOGRAPHIC FINDINGS IN A SERIES OF 32 OBESE SUBJECTS.

We have seen that in the main the changes observed all occur in lead III. The chief change seems to be a left ventricular preponderence (in 70% of the cases) split Q.R.S.(50%) and an absent or inverted T in lead III which is shown in 75% of the cases.

In lead III the directions of the curves Q.R.S. are opposite to lead I. The left ventricle tries to cause a downward deflection and the Right an upward. Normally lead III is more favourable to the right ventricle and the deflection is usually upwards.

The obese subject has a greatly increased body weight to carry and it is possible that hypertrophy of the left ventricle results. The preponderence might be determined by hypertension which is such a common finding is cases of obesity.

MARKEL AND PARDIE (49) examined post mortem two cases who during life showed no electro cardiographic evidence of myocardial disease but who had marked left axis deviation. In each case microscopic examination revealed the muscle fibres to be/

be fairly normal with no increase in connective tissue In both cases the coronaries were slightly thickened, but there was no narrowing or occlusion of the vessels although each of these cases had coronary changes, there was no abnormality of the myocardium on microscopic examination. In neither case was the heart enlarged in spite of the marked left axis deviation of Q.R.S. in the electro cardiogram.

HERMAN & WILSON (50) also commented on this inconsistency.

MASTER & PARDIE (51) examining a series of similar cases, came to the conclusion that the finding of a normal ventricular complex, was an indication that the ventricular muscle was normal or affected in only a slight degree. The two cases quoted by MARKEL & PARDEE (49) would seem to support this view. From their work it would seem that it is possible to have a left sided preponderence without ventricular hypertrophy.

In the obese subject, fat is laid down along the blood vessels on the heart wall. It may be that the presence of the fat may determine the preponderence. It would seem unlikely that this should be so, but the possibility must be taken into consideration.

Normally/

Normally there is a certain mass relation between each ventricle. In cases showing hypertrophy usually there is an increase in weight of both ventricles, unilateral hypertrophy as a manifestation of disease at any rate being rare. LEWIS.(60) Even though there be bilateral hypertrophy one ventricle is usually involved to a greater extent than the other, and the normal weight ratio between the ventricles is lost.

The pathologists tell us that both in the normal and the obese heart the Right ventricle seems to have the heaviest load of fat.

During starvation the fat in the sub-pericardial tissues is the last fatty deposit in the body to waste.

Fatty infiltration is found to start most commonly in the Right ventricle.

These facts would apparently tell against the argument that the fat can take a part in determining the direction of the ventricular complex, for if it can affect the cardiogram, we should expect a Right sided preponderence instead of the left, I have observed, it may be then that it is the hypertrophy of the Left ventricle due to the demand for increased work, or owing to a rise in Blood pressure which determines after all the left axis deviation.

It/

It has been shown that a change in the axis of the heart may cause a preponderence of one or other ventricle. WIGGERS (59) has established a rule "that progressive diminution in the amplitude of R leading finally to inversion in lead I or III is accompanied by a proportional diminution or even inversion of T in the same lead when occasioned by changes in the anatomic axis of the heart".

DU BRAY (46) has pointed out that the position of the diaphragm is often high in the obese subject, so it would seem that a change in the axis in the heart might be present in these cases, and some of the abnormalities I have described may be due to this factor.

It would, I should imagine, be possible to clear this point up with the aid of X ray examination to determine whether or not there is a change in axis. Then, if the patient had another electrocardiograph taken after a large reduction in weight, a normal second cardiogram and a normal axis as determined from another X ray examination, would be almost conclusive evidence. To be quite certain, however, it would seem that proof would have to be obtained, that the fatty loading takes no part in the formation of the ventricular complex.

This theory of change in axis of the heart seems/

seems to me quite a possible explanation of the preponderence noted, but against it is the fact, 5 patients between 64% and 83% overweight, do not show any preponderence.

The splitting of Q.R.S. observed in 50% of the obese patients is described by LEWIS as "not uncommon", in normal subjects, but I have failed to obtain further information on this point.

WIGGERS states that a negative or absent T in lead III is found in 25% of normal people. In 1106 electro cardiograms of normal subjects HEARD & HEIN found a negative T in lead III in 5.6%.

Only a slight deviation from the normal is needed to determine a negative T in lead III in that the Right upper zone remains electro negative. (WILLIUS).

HEPBURN & GRAHAM (47) investigated the electro cardiographic findings in 123 diabetics. Presumably some of these patients must have been obese. They, however, make no mention of the findings I have described, and they discard as being normal those cases which showed a negative T in lead III, and a left sided preponderence.

SUMMARY/

S U M M A R Y.

A thorough search through the work on Electro cardiograms has failed to reveal any mention of such changes as I have observed in relation to obesity, and as far as I am aware, this is the first time that these changes have been described.

There seems to be no correlation between the electro cardiographic findings and the vital capacity, the age, the % increase in weight, or the number of years the patient has been obese.

A change in the axis of the heart if such be present may afford a solution.

A larger series would, however, have to be examined and electro cardiograms taken as the weight changed, such a procedure coupled with X-ray and if possible post mortem examination, would probably throw some light on the problem.

The fact that 87.5 of my obese cases showed some electro cardiographic abnormality is in itself justification for further work.

GENERAL.

SUMMARY & CONCLUSIONS.

GENERAL

S U M M A R Y.

OF THE WORK DONE

A survey has been made of the more important literature on the Subject of the Vital Capacity of the Lungs. It has been shown that while there is a considerable body of opinion which takes the view that the vital capacity reading is an important clinical adjuvant in diseases of the heart and lungs, there is yet a minority of opinion who are inclined to be sceptical of its value as a prognostic and diagnostic sign. The possible factors at work in bringing about a low vital capacity reading in such cases has been discussed.

The vital capacity of seventy persons who could be pronounced healthy, as far as it is possible to make such a pronouncement, have been examined in order to establish a control series of readings.

The vital capacity records of 140 bituminous coal miners who had been subjected to a searching clincal examination by Dr GILCHRIST have been investigated with a view to observing the effect of anthracosis on the vital capacity.

Eleven/

Eleven cases of cardiac failure have been carefully studied. Daily readings of their vital capacity, extending over weeks or months, have been taken and an attempt has been made to correlate such observations with the clinical condition of the patients.

A series of 50 obese subjects have been investigated with a view to determining whether the vital capacity was reduced in such cases, and, if so whether a corresponding increase occurred as the body weight fell.

In addition this series was carefully examined, in order to find out whether vital capacity observation, in such cases, might aid in the diagnosis of the so called fatty heart. And on 32 of these obese patients electrocardiograms were taken with a view to correlating any abnormal appearances with the vital capacity readings.

GENERAL

C O N C L U S I O N S.

After taking many hundreds of observations of vital lung capacity, the following conclusions are arrived at:-

(1) Individual variations present one of the greatest drawbacks to the value of vital capacity readings.

It is necessary to know the individuals own standard while in health, before anything like complete reliance can be placed on an observation taken from a patient suffering from disease of the heart or lungs.

(2) The normal standards at present in use tend to be unreliable as a very large class of people do not conform to the so called normal body build.

(3) Standards based on surface area are probably the most accurate, but for quick clinical work the Height Standard is satisfactory.

(4) Lack of co-operation on the part of the patient is the chief factor militating against reliable readings.

(5)/

(5) Vital capacity readings must in no sense be used as a short cut to diagnosis. An observation not in accordance with clinical findings should be looked on with suspicion.

(6) Anthracosis appears to have no effect on the vital capacity.

(7) Vital capacity readings give no indication of physical fitness except in so far as the heart and lungs are concerned.

(8) In acute heart failure the vital capacity follows very closely on the daily changes in the patient's clinical condition but in general it may be said that no undue weight should be attached to a sudden unexpectedly low reading unless it can be confirmed by clinical findings.

(9) As a class obese subjects have low vital capacities but considerable individual variation is met with.

In the obese person the vital capacity increases as the weight is reduced.

(10) In working with obese patients the Height Standard/

Standard, or the Surface area standard using the patients correct weight, may be used.

If the Surface area standard and the patient's observed weight be employed it is suggested that 13% be added to the observed vital capacity instead of 20% advocated by BOWEN & PLATT.

(11) It would appear that more than one factor is at work in causing a reduction of vital capacity in the obese subject.

Vital capacity readings are of questionable value in diagnosing myocardial changes in the obese.

(12) Definite Electro cardiographic changes were observed in obese subjects. A satisfactory explanation has not been found but these changes seem to be of sufficient importance to warrant further investigation.

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