A STATISTICAL COMPARISON OF THE BLOOD SEDIMENTATION RATE AND WHITE BLOOD CELL COUNT IN ACUTE RESPIRATORY CONDITIONS, WITH SPECIAL REFERENCE TO LOBAR PNEUMONIA

A Thesis presented for examination for the

Degree of Doctor of Medicine of the

University of Edinburgh

by

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INTRODUCTION

The blood sedimentation rate has, of recent years, been commonly employed as an aid to clinical study. The virtue of the test has been recognised particularly as giving an indication of chronic inflammatory, and other destructive changes in cases where physical signs and examination of the blood cells provide no such guide; and it has been found especially helpful in the diagnosis and exclusion of early pulmonary tuberculosis and in the assessment of progress and cure of other inflammatory conditions, such as rheumatic fever.

The test is, unfortunately, the least specific of any in clinical pathology. Owing to this lack of specificity which has caused the wariest to be trapped into fallacious conclusions, the value of the test remains as it always will remain, self-limited.

The sedimentation rate is, however, of proven value as an accessory to other findings and frequently provides valuable confirmatory evidence.

It appears that whereas the worth of the test in its lower range of values has been appreciated, insufficient attention has been paid to the possible application of a grossly increased sedimentation rate. Moreover, no accurate simultaneous study of the sedimentation rate and white blood cell count in acute inflammatory diseases has hitherto been undertaken, although in such disorders, an approximate parallelism between these two forms of estimation has been recorded.

I have therefore made a statistical comparison of the white cell count and sedimentation rate in a series of cases of Lobar Pneumonia and, have, at the same time, investigated the influence of pyrexia on the sedimentation rate.

The volume of work on the sedimentation rate published in Britain and the United States has been truly enormous, yet this represents but a fraction of that written by Continental and especially German, workers, who have done much to elucidate the theoretical aspect of the subject. In the absence of corresponding reports in the English language, repeated reference throughout the text, to French and German authorities, has been inevitable.

In this paper, the history, technique, methods, and theory of the sedimentation rate have been described in successive sections. A survey of previous work on the subject under investigation, is followed by a statistical comparison of the sedimentation rate, white cell count and temperature in forty four cases of Lobar Pneumonia; in this section, reference is made to certain trends observed in these

factors. Allusion is then made to the sedimentation rate as an aid to diagnosis in Lobar Pneumonia and finally, to its value in other respiratory conditions. The results in individual cases and the statistical figures have been included in the Index.

I wish to express my thanks to the Physicians-in-Charge, Royal Infirmary, Edinburgh, for kindly allowing me free access to the cases in their wards; to Professor D. M. Dunlop and Dr. A. Fergus Hewat for much helpful advice; to Colonel W. F. Harvey for instruction in the technique of blood examination; and to Dr. R. G. Macfarlane, Hammersmith Hospital and Mr. W. T. Russell, the Department of Epidemiology, London School of Hygiene, for assistance in the interpretation of statistics.

ERYTHROCYTE SEDIMENTATION RATE

I. Historical Survey

In the past two decades medical literature has been inundated with a wealth of publications dealing with the erythrocyte sedimentation rate in all its aspects, and it has been computed that an article on this subject now appears in the literature of the world every third day.

This recent plethora has tended to obscure the fact that the physicians of older days were fully aware of the occurrence, if not of the implications, of a changed appearance of the blood in ill-health; indeed consideration of this change, which forms the first recorded biochemical observation, gave rise directly to the humoral theory of the causation of disease, which was the mainstay of pathology for some nineteen hundred years.

The first published reference to this subject was made by HIPPOCRATES (B.C. 460 - ? 380). The pathologists of that time conceived that health was dependent on the maintenance in the blood of the proper proportions of four constituents: cholera, or the yellow bile, the serum which separates from the dark red clot; melancholia, or the black bile, the dark coloured substance in the lowermost portion of the tube; sanguis, the upper bright red portion and phlegma or mucus, or fibrin as it would now be called. It was noticed that the phlegma was increased in the presence of disease, and so constant was this change, that the Hellenic physicians concluded that this substance was the most important cause of all diseases.

GALEN (131-201), the most prominent of the Roman physicians adopted and developed this hypothesis which was accepted without serious debate for the

following nineteen centuries, as is witnessed by the universal practice of venesection as a remedy for all ills throughout that time. Spasmodic attempts were indeed made to denounce the humoral theory, notably by PARACELSUS (1493-1541), QUENSAY (1694-1774) and HEWSON [1] (1739-1774), but these workers received scant attention from contemporaries hidebound by a traditional servitude to the Greek school of teaching.

Throughout the seventeenth, eighteenth and early nineteenth centuries, the naked-eye appearance of the blood in disease was the subject of persistent discussion by physicians throughout the world, but while some progress was made, old fallacies persisted, and new ones appeared. FAHRAEUS [2] points out that whereas the Greeks had rightly concluded that the coagulation of the blood depended on the phlegma or fibrin, the pathologists of this time decided that the clustering

of the red blood cells was the factor responsible for clotting, and that the fibrin was an alien substance.

At this early date, the altered blood state in pregnancy was recognised, and its mechanism was a fertile source of argument. PRIORRY (1794-1879) went so far as to suggest that every pregnant woman, from whose blood a fibrin clot separated, must be regarded as ill. Others considered it an indication of uterine congestion (VAN SWIETEN) or of increased vigour (HUNTER [3]).

Throughout the eighteenth and early nineteenth centuries the use of venesection had persisted and extended, and it is recorded that it was no rare experience for wretched individuals to be relieved of as much as four litres of blood within the space of two days; but the nineteenth century witnessed a sharp reaction against the unquestioning use of this

therapeutic measure. The rebellion was led by a homeopathist, HAHNEMANN (about 1810), DIELTEN, a German doctor, and HUSS, a Swede (both about 1850).

For the next fifty years or so, the study of the blood in disease was in abeyance. This was doubtless due in part to the revulsion against blood-letting, with the consequently diminished facilities for investigation; but another and equally important factor was the displacement of humoral by cellular pathology, largely through the agency of VIRCHOW. In this immense new field, pathologists found more interesting and important subjects for research than the naked-eye examination of the blood.

REICHEL [4] comments on the advanced state of knowledge concerning the macroscopic appearances of the blood at the beginning of the last century. HUNTER [3] in 1772 had enunciated that "the inflammatory crust or

size is not a new-formed substance, but merely the coagulable lymph separated from the rest of the blood." This great advance was confirmed and developed by HERMANN NASSE [5] in a massive tome on haematology published in 1836. He showed that the buffy coat owed its origin to an increased sinking velocity of the red blood corpuscles, and that this phenomenon was connected with the peculiar clustering tendency of the red blood cells. He writes "I have found that the more the cells cluster, the quicker they settle to the bottom, and that this tendency to cluster is most notably present in buffy blood."

At that time it was appreciated that the degree of sedimentation depended on the height of the column of blood, and the time taken by the blood to coagulate. Sedimentation was said to depend on the erythrocyte content, and on the degree of erythrocyte clustering,

the process being hastened by increasing the viscosity of the blood, and retarded by lowering it. It was, moreover, hastened in the presence of increased fibrin content.

The erythrocyte sedimentation was hastened in pregnancy, infectious diseases, notably pulmonary tuberculosis, inflammatory illnesses, such as pneumonia, rheumatic fever, hepatitis, nephritis and erysipelas. The rate of sedimentation might show an increase corresponding to the spread of a disease process, and in the very gravely ill, might again return to normal.

This not inconsiderable knowledge was forgotten or at least neglected, until BIERNACKI,[6.7.] some sixty years later revolutionised the study of blood sedimentation by a series of articles published between 1893 and 1897 in which he suggested that clotting should be prevented by the addition of oxalate, that

the blood should be set up in tubes and readings taken after a half, one and twenty four hours. He indicated the importance of the test in diagnosis, and drew attention to the marked hastening of sedimentation in arthritis, pneumonia and other febrile disorders, and the normal or slowed readings in functional neuroses, emphysema and heart disease. Whilst he made numerous mistakes both in theory and in method, he must nevertheless be regarded as the progenitor of the sedimentation test as it stands to-day. Indeed, many Polish and French writers allude to "BIERNACKI'S reaction."

In view of BIERNACKI'S work, it is surprising that twenty years elapsed before interest in the reaction became general. At the end of 1917, HIRSZFELD [8] described the use of the reaction in the diagnosis of malaria, and in the following year,

FAHRAEUS [9] published his first work on the subject in which he dealt with the test mainly in connection with the diagnosis of pregnancy. Two years later, PLAUT, [10] who had begun his work unaware of FAHRAEUS' publication, reported its application in the diagnosis of mental disorders.

In 1921, FAHRAEUS [11] published a pioneer work on the history, theory and diagnostic significance of the sedimentation test. Further distinguished work has since been done by ABDERHAUER (theory), ASCHER (eye affections,) HÖBER (theory) LINZENMEIER (method; theory; gynaecology), PLAUT (psychiatry) and WESTERGREN (method; theory; internal medicine, more especially pulmonary tuberculosis.)

II Technique of Estimation

For the purpose of estimating the sedimentation rate, blood is withdrawn, mixed with an anti-coagulant substance, the mixture set up in a tube and the degree of separation of the cell content from the clear plasma in a given time noted. This is not to be regarded as a physiological action, for all anti-coagulants exercise an influence on the blood, some by diluting the serum, others by altering the nature of the cells. These substances act not only qualitatively but also quantitatively. It is therefore essential that the same anti-coagulant substance in identical proportions be employed on each occasion.

Withdrawal of Blood

REICHEL [12] has demonstrated that the sedimentation rate is identical in blood drawn from an artery, capillary or vein of the same individual. In

practice, a vein on the forearm is usually selected, although in the absence of available veins, and with adequate precautions, there is no objection to the withdrawal of arterial blood.

A tourniquet is applied, the needle immediately inserted into the vein and the tourniquet released. Blood is then withdrawn into a syringe containing the selected anti-coagulant. Isotonic citrate solution (3.8%) is usually chosen for the prevention of clotting and this should be drawn into the syringe before the blood is aspirated. SCHÖTT [13] has demonstrated the necessity of absolute cleanliness of the needle and syringe, and finds their preliminary irrigation with citrate a useful practice. Sterility of the instruments is, however, unnecessary.

After aspiration of the blood, which should not be performed so quickly as to permit the admixture

of air bubbles (thus preventing accurate volumetric estimation), a small amount of air is aspirated, and the mixture gently shaken, before being set up in the selected glass tube.

Where the capillary method is employed, smaller quantities of blood (·1-·2 cc) are sufficient, and puncture of the ear or thumb is an adequate method.

Anti-Coagulants

For the study of blood sedimentation, the blood must be rendered unclottable, and to this end, three main methods are available :-

(1) Smoothing of the tube wall (paraffin, oil, etc.).

This method is not now practiced.

(2) The addition of an anti-coagulant substance :

a. in solid form (heparin, oxalate, etc.)

b. in isotonic solution.

Those in the first category (a) are excellent anti-coagulants, but have the disadvantage of altering the osmotic tension of the blood, and therefore change the agglutinability of the erythrocytes. Moreover, their action varies with the amount used, and they cannot be readily estimated quantitatively.

Those anti-coagulants in the latter class (b), have the drawback of changing the concentration of the plasma, but they do not affect the erythrocytes, as do the solid forms, and they are easily measured quantitatively.

There is still some difference of opinion concerning the anti-coagulant of choice, but most workers now hold that a solution is best, and in this class, isotonic citrate (3.8%) is most commonly employed. Writers are generally agreed that the optimum dilution is one of blood four to citrate one,

but WESTERGREN and others [14] have found that small differences in the concentration of the solution do not affect the results, and LINZENMEIER [15] uses a 5% solution. MEIER [16] reports that the variation between the readings obtained with the use of this solution and the more commonly accepted 3.8%, is negligible.

Storage of the citrate and blood.

A sterile solution of citrate should be employed and kept in an air-tight container, as growths of fungi otherwise change its nature.

Some authorities hold that citrated blood may be kept at room temperature for up to twelve hours prior to estimation (LEFFKOWITZ,[17] KOVACS,[18] ROURKE and PLASS.[19]) WESTERGREN,[20] in a large series of controlled observations, found that a delay of four or five hours before estimation had no effect on the

result, that after six to eight hours, a slowing of the sedimentation occasionally occurred, and that this became pronounced after twenty four hours' delay.

Temperature.

Room temperature should be obtained for the test. With lowering of the temperature, sedimentation is retarded, and with raised temperatures it is accelerated (ROURKE and PLASS,[19] RIMINI [21] and others).

The effect of diameter of the tubes.

The diameter of the tube chosen for the test exercises some influence on the sedimentation rate. In an exhaustive literature on this subject, a striking diversity of opinion is found. Some have pronounced that the sedimentation rate is slowed in a narrow tube (WESTERGREN, WIEMER [22]) and others, the reverse (HORVAT,[23] DUCCESCHI,[24] YAMAMOTO,[25] FEUERSTEIN.[26]) The opinion now held is that in narrow tubes (under 1 mm.

diameter) sedimentation is at first slower, but later more rapid than in tubes of wider bore, and that tubes over 1·4 mm. in diameter produce uniform results which obviate these discrepancies.

Height of the blood column

The height of the column of blood affects the sedimentation reading, and this is easily appreciated in view of the work of ROTHE,[27] who suggests that if the sedimentation rate be registered in the form of a curve, three stages can be distinguished :-

1. Slow - The single red blood corpuscles settle and collect.

2. Quick - Sedimentation of aggregations of erythrocytes at an increasing speed, which soon reaches a constant.

3. Gradual slowing, through packing of the aggregations, the on-set of this stage, varying with the height of the column and the erythrocyte volume.

This last mentioned braking effect ("Bremsung") is said first to take effect when the top of the

erythrocyte column has fallen to the second third of the total column (REICHEL,[4] STRÖM[28].)

REICHEL[4] has estimated the height of blood-columns necessary to obviate this "Bremsung" on the basis :-

Slowed and normal sedimentation	(0 - 12 mm)	50 mm. column
Moderate acceleration	(up to 30 mm)	-100 mm. "
Marked "	(up to 60 mm)	-200 mm. "
Very marked "	(over 60 mm)	Over 200 mm "

It will be noted that according to this table, the column in the WESTERGREN tube (200 mm.) may be sometimes inadequate.

Clearly the degree of slowing through cell-packing will vary with the cell volume; and this has given rise to numerous attempts to correct the results according to the red cell count (WESTERGREN,[29] PFAFF,[30] WELLNER,[31] STRÖM[28] and others), but PFAFF has demonstrated the uncertainty of such methods.

Times of readings

WESTERGREN recommends that readings be taken after one and two hours, but most authorities are now agreed that the influence of cell-packing is so great as to render all but the first-hour reading valueless.

It has been suggested that a twenty-four hour reading is of value as giving, in conjunction with the one-hour result, a rough estimation of haemoglobin content, and of the white cell count. The white cells settle in some cases at the top of the column of sedimented red cells, and when this visible heaping of white cells extends more than 1% of the length of the total column, the white cell count is said to be raised. The author has observed this phenomenon infrequently, and only when the sedimentation rate was markedly increased. Many specimens with a pronounced leucocyte increase failed altogether

to exhibit such a phenomenon.

Summary of technical factors.

The factors in technique affecting the rate of sedimentation may be summarised as follows :-

Hastening	Slowing
1. Temp: above room Temp.	1. Dilution of blood with anti-coagulant salts (Sod.citrat., etc.)
2. Tube off vertical	2. Temp: considerably under room temperature.
3. Moistening of the tube above the blood column.	3. Temp: above 50° C. (precipitation of fibrinogen).
	4. Packing of the R.B.C. in the later stages of sedimentation.
	5. Too narrow tube (under 1 m.m.)
	6. Delay in use of specimen (over four hours) especially in raised temperature.

III

Method of Estimation

It is said that more than one hundred methods of testing the sedimentation rate of the blood have been evolved, and that most of these are in use (REICHEL.[4]) Comparison of results has hitherto been difficult, owing to a multiplicity of standards, but fortunately there is some promise that this confusion will shortly be remedied.

Mention will here be made only of methods in common use or of special interest. Amongst these are :-

a. WESTERGREN'S

This was used by the writer in the present work, and all readings quoted in later sections are one-hour results following WESTERGREN'S method. This method was selected because it reputedly entails fewer

technical difficulties than others (notably capillary) and because its wide quotation in the literature offers the maximum opportunity for comparison with other work.

WESTERGREN'S[32] apparatus, which is a modification of that described by FAHRAEUS,[9] consists of a tube 300 mm. long with a uniform internal diameter of 2·5 mm. It is filled by suction with citrated blood, up to the level of a mark at 200 mm., and placed vertically in a stand, where it is held above by a spring arm, which exercises firm downward pressure; the base of the tube is fitted into a circle of rubber, hollowed out to contain it.

The blood for examination is obtained by the method already described: ·4 cc. sodium citrate (3·8%) is previously drawn into the syringe, and blood is aspirated to the 2 cc. mark, these proportions giving the desired dilution of one in five. The writer's

practice after withdrawal of the syringe was to aspirate a little air, shake the syringe gently, and after removal of its cap, to draw the blood into the tube directly from the syringe. The interval between venesection and this step was in no case more than four minutes.

In some instances a small collection of air bubbles rose to the top of the blood column, but controlled observations failed to indicate that these had a perceptible effect on the readings.

To ensure uniform results, the apparatus was cleaned by the same method on each occasion. Liquor potassae, water, methylated spirit and ether were used in succession, and in the case of the tubes, air was blown through before they were set beside warm pipes for twenty-four hours. A similar procedure was observed in cleaning the syringe, but as this was usually required for further tests on the same day, a

rapid method of drying was necessary, and for this purpose, repeated pulling-through of gauze swabs proved satisfactory.

Though the investigation of the reaction was being carried out for the most part on seriously ill patients, the operation caused little or no disturbance to the subjects, however ill, and in only one case, that of a highly-strung girl of fifteen, were anxiety and resentment exhibited.

Where intravenous injections were being given therapeutically, the synchronous withdrawal of blood for the test was found to be a useful practice. This procedure requires the co-operation of two people, but has obvious advantages in that disturbance of the patient is minimal and unnecessary devastation of a vein by two punctures is avoided.

First-hour results up to 10 m.m. are now

accepted as normal and figures between 10 m.m. and 12 m.m. as being on the border-line (WYSS [33] et alt.) This standard is somewhat higher than that originally proposed by WESTERGREN.[32] The writer at first took additional readings after two hours and twenty four hours, but these were found to give no additional information. At the Mayo Clinic, where WESTERGREN'S method is favoured, reliance is placed solely on first-hour results (BANNICK, GREGG and GUERNSEY. [34])

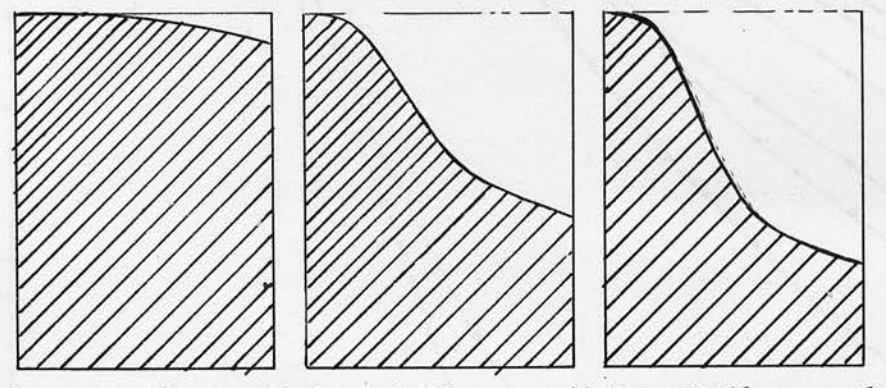
b. Other Methods.

1. Photographic reading of the rate of sedimentation

This method was described by STAMMREICH in Germany, was introduced into the U.S.A. by SULKOWITCH[35] and more recently into this country by LEE.[36]

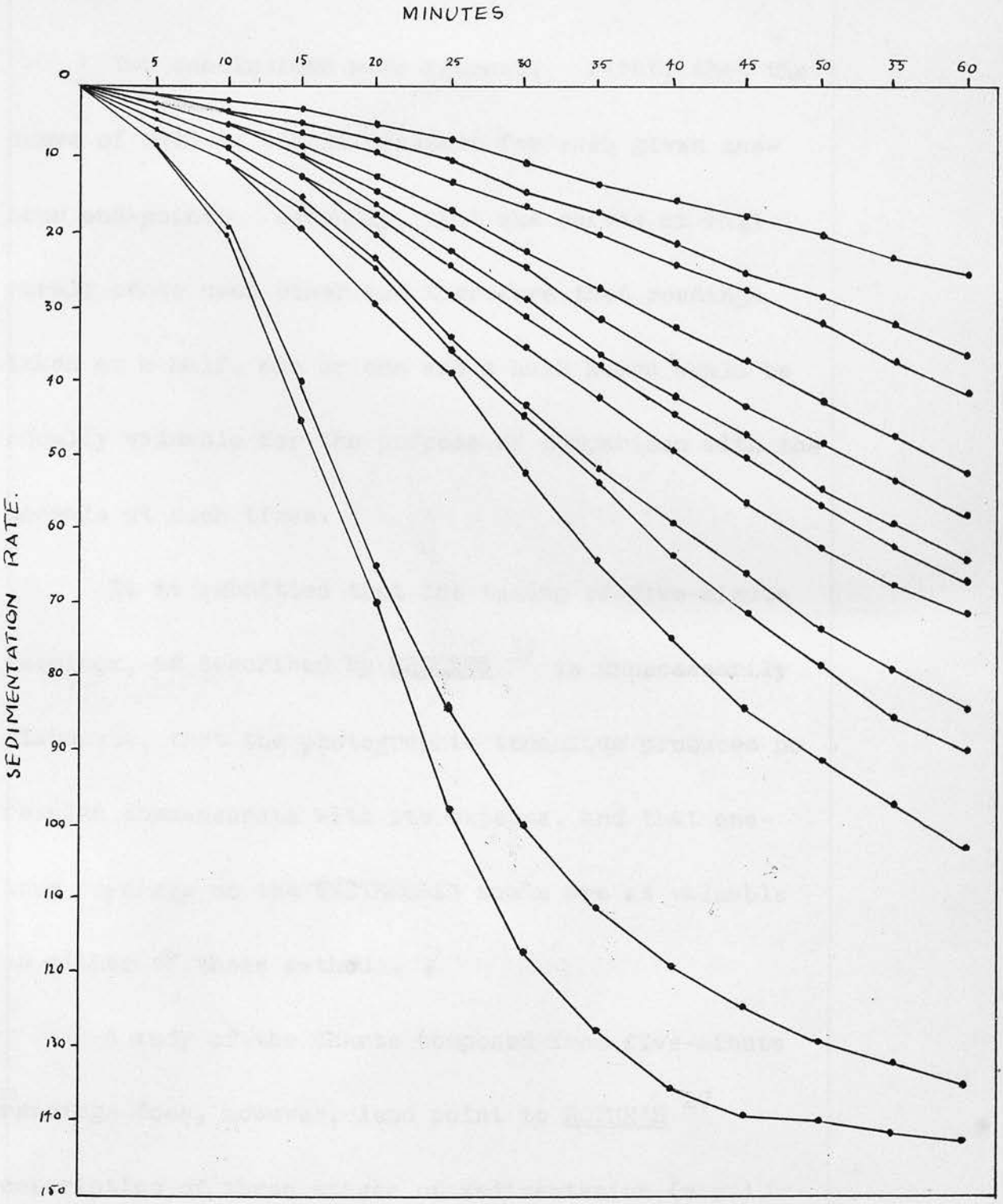
Its adherents assert that this method gives information unobtainable by other forms of estimation. Thus, LEE, studying the top of the blood-cell column on a continuous photographic record, associates its

fall in a straight diagonal line with a quiescent lesion, in a diagonal curve with a moderately active lesion, and in a perpendicular curve with pronounced activity. The three types of fall described may be diagramatically represented thus :-



THREE TYPES OF SEDIMENTATION RECORDED PHOTOGRAPHICALLY by LEE.

The writer had not the advantage of a photographic apparatus, but by taking readings every five minutes and charting the results, fairly faithful reproductions of the curves of fall were made. In some cases the readings were extended to ninety minutes.



EXAMPLES OF THE RATE OF SEDIMENTATION RECORDED EVERY 5 MINS IN A SERIES OF CASES OF LOBAR PNEUMONIA.

Two conclusions were reached; first, that the curve of fall is almost constant for each given one-hour end-point; secondly, that the curves of fall rarely cross each other and therefore that readings taken at a half, one or one and a half hours would be equally valuable for the purpose of comparison with the normals at such times.

It is submitted that the taking of five-minute readings, as described by BROOKES [37] is unnecessarily elaborate, that the photographic technique produces no results commensurate with its expense, and that one-hour readings on the WESTERGREN scale are as valuable as either of these methods.

A study of the charts composed from five-minute readings does, however, lend point to ROTHE'S [27] description of three stages of sedimentation (v.s.). Where the straight diagonal line of fall is observed

the influence of cell-packing is not apparent, and the second stage prevails throughout, but in cases where a sharp fall is followed by a flattening of the curve, the third stage of cell-packing is superimposed. Plainly, therefore, the type of curve will vary with the height of the column of blood, for the higher the column, the later the onset of slowing by cell-packing. With the high column recommended by WESTERGREEN, the influence of cell-packing within one hour is seen only with very pronounced acceleration of sedimentation.

2. Micro-pipettes

Amonst others, BROOKES [38] has evolved a micro-pipette for which he claims accurate results. He recommends the use of heparin or crystalline calcium oxalate as anti-coagulant.

In this country, HARVEY and HAMILTON[39] have reported the use of a capillary tube of 1·1 m.m. diameter, and made simultaneous studies of blood volume.

On the continent, STEIGER [40] and others recommend the use of a small pipette, not unlike a white blood cell chamber, for which easy and rapid estimation at the bedside without disturbance of the patient is claimed.

3. Rapid Methods

Various attempts have been made to obtain a quick reading of the sedimentation rate: these have included the addition of gum arabic to increase the viscosity of the plasma (KAUFMANN [41]); tilting of the tube (BALACHOWSKI [42]); and increasing the temperature (PRISELKOV [43]). No advantage has been found in these methods which are rarely used.

4. New Modified Test.

SERENY [44] has recently elaborated an interesting modification of the usual test of sedimentation rate. He has attempted to obviate the fallacies inherent in the test and arising through the red blood corpuscles,

by taking cell-free serum, and estimating the time taken by added sodium hydroxide to settle. The technique is complicated and the results so far, inconclusive, but this work, which is still in the experimental stage, is mentioned as possibly indicating the line of future advances in the accuracy of the test.

Comparison of Methods

BEAUMONT and MAYCOCK [45] report that most recognised methods including the micro-method and those of WESTERGREN and ZECKWAR and GOODELL [46] give parallel and comparable results.

IV. Summary of Theoretical Knowledge.

The mechanism of erythrocyte sedimentation has been the subject of prolonged enquiry by many workers, who have sought to offer a full and satisfactory explanation of the phenomenon, but while the roles of certain factors are now clearly defined, those of others are less surely established, and it may even be that some have entirely eluded detection.

Writers have seldom been able to investigate at one time all the relevant influences, and a comprehensive understanding of the mechanism, so far as it is known, is to be obtained only by the study of numerous individual reports.

The blood consists mainly of two parts, one, the cellular portion, the other, the plasma in which it is suspended. This suspension of the cells in plasma is not a vital activity, nor is cellular

sedimentation solely a post-mortem phenomenon. Investigations of the blood in vivo have shown that the corpuscles are carried along in the blood stream, much as an object of greater density than water might be floated down a river, and that slowing or arrest of the circulation results in the corpuscles settling by gravity (FAHRAEUS [9]).

Some workers, appreciating that erythrocyte sedimentation is essentially the fall of particles in a fluid medium of lesser density, have attempted to correlate the process with STOKES' Law, which relates to such conditions. This is :-

$$V = \frac{2}{9} g \quad \frac{s-s_1}{v} r^2$$

where: V = velocity of fall

g = a gravitation constant

s = specific gravity of particle

s_1 = specific gravity of fluid

v = absolute viscosity of fluid

r = radius of particle

This formula, however, is correct only when no interaction occurs between the particles in suspension, and is inapplicable to erythrocyte sedimentation on these grounds:

1. The red blood corpuscles are loaded with negative charge, , and are thus mutually repellent.

2. On the other hand, the R.B.C. tend to form large irregular aggregations.

3. Contact between cells is known to be an important factor in inhibiting sedimentation, and to account largely for the abnormal slowing in polycythaemia and the reverse (ceteris paribus) in anaemia.

According to STOKES' Law, increase in plasma viscosity would result in slowing of sedimentation: in practice, the reverse is found to be the case, for most substances which raise the viscosity,

at the same time increase the aggregation of red cells. Thus, the addition of gelatine or gum arabic hastens sedimentation, as NASSE [5] noted a century ago.

It has been found that whereas increased plasma viscosity hastens the rate of sedimentation, increased whole-blood viscosity retards it, and the degree of whole-blood viscosity varies directly with the erythrocyte count.

STOKES' formula indicates further that the greater the radius of the bodies in suspension, the quicker is the rate of sedimentation. To some extent this is true in the case of blood, for sedimentation tends to be hastened in macrocytic and slowed in microcytic states. (BÜRKER,[47] BEHRENS [48]). The influence of cell aggregation, however, far outweighs, and usually masks the effect of individual cell-radius.

During sedimentation, the red blood cells have been shown to aggregate into clumps, the size of which varies directly with the rate of sedimentation. Having attained certain proportions, they cease to enlarge further and the time taken to reach such proportions also varies according to the sedimentation rate (S.R.). FAHRAEUS [9] has estimated that if no cell-aggregation occurred, the normal one-hour result would be ·2 m.m.; that in blood with an S.R. of 1 m.m. the aggregations contain about 11 R.B.C. and that in blood, with pronounced acceleration of sedimentation, each aggregation may consist of about 58,000 cells. If the formation of aggregations be artificially inhibited, sedimentation is retarded (Sedimentation, in isotonic saline - BLOCH and OELSNER [49]: in HAYEMS' solution - BURKER,[47] BEHRENS;[48] in lymph - RUSSEL and BOYD.[50])

Electrical Charge.

The red blood corpuscles carry a negative electrical charge (HÖBER [51]). HÖBER and others, (FAHRAEUS, LINZENMEIER, MOND) have indicated that the property of promoting sedimentation possessed by the plasma proteins varies according to their individual capacity to discharge this negative load. This capacity is possessed in most marked degree by fibrinogen, less by globulin, and least by albumen.

Diminution of mutual repulsion and suspension stability of the red blood cells follows loss of this electrical charge. This explains the frequent association of increased S.R. with raised fibrinogen and globulin fractions.

The discharge theory has not been universally accepted. FAHRAEUS himself has remarked on the uncertainty of measurements of negative potential, and

SCHLECHTER and BLÜHBAUM [52] could find no connection between the rate of sedimentation and the degree of erythrocyte negative potential.

Plasma Proteins.

Divergent opinions have been expressed concerning the role of plasma proteins in the production of blood sedimentation. Most workers, [11, 53, 54] however, are now agreed that they play a predominant part, although a few have failed to confirm the connection (ALDRED-BROWN and MUNRO: [55] LUCIA and co-workers [56, 57, 58, 59]).

FAHRAEUS [2, 11] observed that where the fibrinogen was increased, the S.R. was hastened, and that this applied to a less marked extent in the case of globulin, whereas increased albumen retarded the S.R.

WESTERGREN [14, 60] concluded that a direct relationship existed between the plasma proteins and the S.R.

ZARDAY and FARCAS [61] modified normal blood by the addition of fibrinogen and globulin, and found that the S.R. was increased according to the amounts added: the addition of albumin inhibited sedimentation. COBURN and KAPP [62] obtained similar results. On the other hand, LUCIA and his associates who reproduced these experimental conditions, found that the resultant aberration of S.R. from normal was negligible.

WESTERGREN, THEORELL and WIDSTRÖM [14] have undertaken estimations of the S.R. by calculation from the plasma protein content. Their formula is :

S.R.= 140·4 Fibrinogen% + 6·22 Globulin% - 6·09 Albumin% - 24·5

BENDIEN, NEUBERG and SNAPPER [63, 64] are responsible for a similar but more complicated formula, in which allowance is made for the erythrocyte and haemoglobin contents:

$$S.R. = \frac{45}{\text{cell volume}} \times C.I. \left(12 \ (Fib.\% - 3{\cdot}5) + 2{\cdot}5 \ (Glob.\% - 22)\right)$$

BROOM [65] found that neither of these formulae gave an exact indication of the S.R., and attributed this failure to factors other than plasma proteins affecting sedimentation.

These formulae serve, however, to illustrate that the S.R. can be increased by a rise either of fibrinogen or of globulin or of both together. This is the explanation of the conflicting results reported by workers who have studied variations in the ratios of the individual proteins.

It is now generally believed that in acute illnesses the increase is mainly in fibrinogen,[74] while in chronic maladies, globulin is the fraction most notably raised.[53] STARLINGER and WINANDS [66] have made a comprehensive study of changes of the plasma proteins in various diseases.

The mode of action of the plasma proteins on the S.R. is uncertain. The erythrocytes are maintained in suspension by differences in the surface tension between cells and plasma, by mutual repulsion owing to similar electrical charges, and by the hydrophilic property possessed by the cell capsules. The plasma proteins are thought to affect all three qualities by changing the surface tension, diminishing the negative electrical charges of the R.B.C. and acting as dehydrating agents. Some writers have laid stress on the changes in electrical charge (v.s.), while others, (MONAGHAN and WHITE [67]) have established a good case for dehydration as the important factor, but it is possible that the proteins capacity for promoting sedimentation arises from all three properties.

Other Factors in the Blood

Cholesterol. The evidence of the effect

of the blood lipoids on sedimentation is inconclusive. Some [14. 72] hold that they exercise no influence, but others (RIX,[68] THEORELL) are less certain. RIX, giving rabbits a high lipoid diet, recorded a hastened S.R. in association with raised blood cholesterol. THEORELL suggests that the raised S.R. observed in some diseases may be caused only partly by an increase of fibrinogen and globulin. He believes that these proteins by taking cholesterol into combination, leave a lowered free cholesterol content, which may help to promote sedimentation.

Sugar. Sugar has no effect when present within physiological limits.[69]

Bile Salts These tend to diminish sedimentation.

N.P.N. N.P.N. in increased quantities hastens sedimentation. [71.]

Variations in the Oxygen content of the R.B.C.

Reports of the effects of such variations are conflicting.[2. 4. 19.]

Electrolytes The influence of electrolytes is variable, but in general, whatever their ionisation, they tend to delay sedimentation. [54. 69. 73.]

Haemolysis. Haemolysis is reported as having no effect on sedimentation.[70]

Erythrocyte content: Anaemia: Polycythaemia

The importance of the corpuscular content in sedimentation has already been noted, and mention has been made of its action in determining whole-blood viscosity and the degree of cell-contact.

MASSE[5] who was evidently an astute observer, recorded over a hundred years ago, that the addition of serum to defibrinated blood increased the S.R. according to the degree of anaemia produced. Since then

ABERHALDEN,[75] FAHRAEUS,[76] WESTERGREN[14] and many others have investigated the influence of the red blood corpuscles on sedimentation.

The usual experience of most forms of anaemia is that sedimentation is hastened, and conversely that in polycythaemia it is delayed:[53] but equally important as the cell content, is the solution in which it is suspended, for dilution of blood with isotonic citrate or saline inhibits sedimentation.

Manifold attempts have been made to obviate the fallacies caused by variations in erythrocyte content. BÖNNIGER and HERRMANN[77, 78] corrected their estimations with the aid of the haematocrit and diluted the blood to a certain volume. GRAM[79, 80] and later WALTON, adopted the same principle, but concentrated or diluted blood to five million R.B.C. per cubic m.m. Latterly, standard curves have

been published[108], which permit the ready correction of the result, according to the R.B.C. count and haemoglobin percentage, without resort to changing of the blood concentration.

Such methods are now widely used, but evidennce is available that they are not entirely satisfactory:

1. pipetting-off of citrated plasma in certain severe anaemias to raise the haemoglobin to 100% frequently results in a one-hour reading of nil (REICHEL and VAN DE STADT - unpublished work.)

2. In many cases of profound anaemia, the S.R. is normal, but this normal figure cannot be reproduced by dilution of blood to simulate the anaemia.

3. A study of the S.R. in various illnesses associated with anaemias has shown that an uncorrected result usually corresponds more closely to the state of the illness, than does a corrected figure. (GRAM, WESTERGREN,

THEORELL and WIDSTRÖM, WALTON, LEBEL and LOTTRUP.)

4. In the course of an illness associated with varying degrees of anaemia, the S.R. does not vary at all constantly with the blood changes.

LEBEL and LOTTRUP [81, 82] report such contradictory results. In a case of pneumonia with Hb.70% and S.R. 100 m.m. for which the corrected value was 55 m.m., the figures during resolution were Hb. 102% S.R. 85 m.m. and the corrected figure 90 m.m. Thus the corrected result rose paradoxically with resolution.

5. With experimental change of the blood concentration in vivo, the S.R. does not vary in relation to the R.B.C. count and haemoglobin percentage.

It would therefore appear that results are not determined, so far as the corpuscular content is concerned, solely by the number of erythrocytes and percentage of haemoglobin: additional factors may include changes

in the agglutinability of the red cells (perhaps conditioned partly by anisocytosis) variations in their specific gravity and form, and alteration of the blood viscosity.

The fact that the S.R. in severe anaemia is relatively slower than in blood artificially diluted with autogenous plasma to the same point, suggests the existence in anaemias of a retarding factor, whose influence it is difficult exactly to estimate.

WESTERGREN, THEORELL and WIDSTRÖM concluded that calculation of the S.R. from plasma protein, cholesterol and phosphate estimations gave more reliable results than ordinary readings corrected for anaemia. On the other hand, BENDIEN, NEUBERG and SNAPPER, who also used these biochemical estimations as a basis, recommended that correction be made according to the blood state.

LEBEL and LOTTRUP have shown that in disease the gross results form a more reliable guide to the clinical state than do those which are corrected, and workers of wide experience (WESTERGREN, KATZ, LEFFKOWITZ et alt.) have given up correction for anaemia, whether by haematocrit, R.B.C. or haemoglobin estimations.

Those who deny that the effects can be measured with accuracy, admit nevertheless the importance of anaemia in hastening sedimentation and of polycythaemia in retarding it. REICHEL[4] observes the following principles :-

1. In anaemic blood with more than four million erythrocytes and 70% haemoglobin, any distinct acceleration of sedimentation is not caused by the anaemia.

2. Very marked hastening of the S.R. in anaemic conditions, with erythrocytes over three million or haemoglobin above 50% inveriably indicates an influence in addition to the anaemia, promoting sedimentation.

3. Increase or diminution of a hastened S.R. in illnesses associated with severe anaemias, can be caused by corresponding changes in the degree of anaemia, as well as by the actual disease process; the gross figures are therefore not to be accepted without knowledge of the blood state. At the same time, a slowing of the S.R. invariably indicates an improvement of the general condition either by relief of anaemia, or return of plasma proteins towards normal proportions.

4. An R.B.C. count over six million or haemoglobin over 105% can inhibit sedimentation, and in such circumstances the S.R. in disease, which would otherwise be increased, may be normal.

In this country haematologists have found that correction gives reliable results (DAVIDSON, VAUGHAN) and cardiologists correct the estimations in assessing heart failure (PAUL WOOD.)

The writer has corrected none of his results, partly because the argument against correction appears to be at least as strong as that for it, but also because the restrictions of time prohibited the daily estimations of R.B.C. and haemoglobin, which would have been necessary in the acute conditions forming the subject of the present observations.

Two sets of blood showed an interesting phenomenon. In each case there was marked increase of sedimentation (c. 118 and 126 m.m.); the blood counts were respectively R.B.C. 3,800,000,Hb 54% and R.B.C. 3,970,000, Hb 69%. During sedimentation, the

supernatant serum was seen to be coloured uniformly pink, with here and there, small dark aggregations, just visible to the naked eye, and presumably composed of R.B.C. The line of junction between the serum and the erythrocyte column was hazy. These changes were at first attributed to haemolysis from faulty technique, but repeated observations produced similar results, while simultaneous tests on other bloods with the same apparatus and citrate solution proceeded normally. It was concluded that the phenomenon was due to the formation of erythrocyte-aggregations of unequal sizes with dissimilar rates of fall.

Records of a mild degree of this aberration were later found. German workers are aware of a failure of delineation between the cells-column and serum or an "unscharfe Zone" in bloods with a marked hastening of sedimentation, associated with severe anaemia.

The Influence of Variations in Physiological State.

1. Sex

The normal S.R. is higher in the female than the male:[2] this is usually attributed to the higher plasma-globulin noted in the female.

2. Age

The S.R. is diminished in the first month, and increased in old age.[74] Corresponding changes in blood fibrinogen have been recorded.

3. Pregnancy

The S.R. is increased from the third month.[2] This increase is said to correspond with fluctuations in the globulin and fibrinogen content. The S.R. during lactation changes from subnormal to rapid.

4. Menstruation

Changes in the S.R. are not well established. Variations have been observed, and attributed to

alterations in the blood viscosity by endocrine activity.[84]

5. Time of day; food; exercise.

These have no effect on the S.R.

6. Fever.

S.R. is increased, but BERNET[85] by producing fever in dogs, has shown that hyperthermia alone does not raise the S.R. This suggests that the underlying cause of the pyrexia is responsible for the change in S.R.

7. Ante-Mortem

Retardation of sedimentation is said to occur a few hours ante-mortem.

8. Cardiac failure

The influence of cardiac failure is not yet fully understood, but in some cases it is know to inhibit sedimentation.

The haematological and somatic factors influencing sedimentation have been summarily tabulated :

SEDIMENTATION

Hastened	No Effect	Retarded	Action Uncertain
Increase of fibrinogen Increase of globulin		Increase of Albumin	
		Hydraemia	
			Lipoids Electrolytes
Bence-Jones Proteose. Increase of N.P.N.			
Increase of Plasma viscosity (gelatine, gum arabic).		Increase of whole-blood viscosity (polycythaemia)	
	Bilirubin	Bile Salts	
	Haemolysis		
	Sugar		
		Excessive increase of $C O_2$	Variations in oxygen content of R.B.C.
Diminution of R.B.C. count. High colour index.		Increase of R.B.C. count. Low colour index.	
		Sedimentation in solutions (a) deprived of plasma proteins (NACL, HAYEM'S) (b) poor in proteins (lymph, C.S.F. transundates)	
Old age		First month.	
Pregnancy Lactation			Menstruation
Fever			
	Time of day; food; exercise		
		Ante-mortem Cardiac failure.	

V. A Further Study of the Sedimentation Rate: Its comparison with the White Blood Cell Count, with special reference to Lobar Pneumonia.

Before mention is made of the writer's results, which relate to the correlation of the white cell count and the sedimentation rate of the blood in Lobar Pneumonia, it is necessary to consider more exactly the prevailing circumstances, and to review previous work bearing on the subject.

In this section therefore, a summary will be given of reports concerning :-

A. The association of pyrexia with an increased sedimentation rate.

B. The plasma proteins and total base in Lobar Pneumonia.

C. The sedimentation rate in Lobar Pneumonia.

D. The white blood cell count in Lobar Pneumonia.

E. The connection between the white cell count and the sedimentation rate.

A. The association of Pyrexia with an increased sedimentation rate.

The frequent association of Pyrexia with a hastened sedimentation rate is undisputed; writers are agreed too that following the onset of Pyrexia, there is a noticeable lag before a change in the sedimentation rate is recorded, and that, following the abatement of fever, the sedimentation rate shows a still greater delay before beginning to return towards normal. (MOTZFELDT,[87] WESTERGREN,[88] HERRMANN,[89] HECKSCHER,[90] ROGATZ,[91] PULVER[92] and others). This observation has been confirmed experimentally by W. & H. LÖHR [93] who produced Pyrexia by parenteral injections of protein.

Clinical and experimental evidence indicates that there is no direct association between hyperthermia and changes in the rate of sedimentation; for with the Pyrexia of short-lived acute infectious

diseases without marked local manifestations (such as measles) the sedimentation rate remains normal despite high temperatures and in the exanthemata of long standing, variation of the sedimentation rate with the temperature occurs but little and only in the later stages. On the other hand, in febrile states associated with local complications, the sedimentation rate and temperature show a fairly marked degree of concomitant variation.

Experimentally it has been proved that hyperthermia alone is not responsible for increasing the sedimentation rate (LUMIERE and SONNERY, [94] HARADA and KUSAKA [95] and BERNET [96]).

BERNET found that the sedimentation rate was unaltered by the production of hyperthermia centrally (B-tetrahydronaphylamine-H Cl) and by physical means. Fever produced by the injection of

hay infusion resulted in a slight increase in sedimentation rate.

The view now held is that in those cases where an increased sedimentation rate is observed in association with a raised temperature, the changes of each arise from a common cause.

B. The Plasma Proteins and total base in Lobar Pneumonia.

(i) Plasma Proteins. Reports of the plasma protein changes in pneumonia are somewhat conflicting. ROWE [97] and GEILL [98] reported an increase in plasma globulin, which became more marked at the crisis. On the other hand LOEPER DE SEZE and LEBERT [99.100] found that the globulin was never greatly increased, and that indeed when the pulmonary condition was fully developed globulin was frequently diminished: it showed a definite increase however, just before the crisis, whereafter it again fell to a subnormal level.

The writers admitted that wide fluctuations from this standard were often found.

They observed that the increase of globulin at the crisis was paralleled by a rise in amino-acids, and attributed both phenomena to the action of leucocytic ferments associated with cytolysis and resolution of exudate.

MOEN and REIMANN [110] concluded that the total plasma protein was usually decreased during the febrile period, but that fibrinogen and globulin were increased. This increase occurred promptly after the onset and persisted far into the post-febrile phase.

(ii) Total Base. SUNDERMAN [101] and others, studying the total base and chloride content of the blood in Lobar Pneumonia have shown that both are diminished up to the time of the crisis and that thereafter they rapidly return to normal. (WIEMER [102]

and ENOCKSON [103] have demonstrated that chlorides tend to inhibit sedimentation).

JANSEN [104] found that calcium was lowered in the precritical and increased in the epicritical phase. GERSTENBERGER, BURHAUS, SMITH and WETZEL [105] reported a reduction throughout the febrile stage of both calcium and inorganic phosphorus, and PEABODY [106] found that calcium and magnesium were lower in the febrile stage than in convalescence. SUNDERMAN [101] found a diminished calcium and potassium content both before and after the crisis, and reported that the magnesium content was approximately normal throughout.

C. The Sedimentation Rate in Lobar Pneumonia.

MOTZFELDT [87] concluded from a study of fifty nine cases of Lobar Pneumonia that on the first day, the sedimentation rate was approximately normal: that it reached a maximum on the fourth day

and remained at about the same level for a further four days, before slowly returning to normal. There was, he observed, occasionally an additional increase at the crisis. He called attention to the delay of changes in the rate in accordance with alterations of the physical state and found that the rate sometimes remained high with normal physical signs and radiographic appearances.

When dyspnoea and cyanosis were present, he found that sedimentation was inhibited. He attributed this phenomenon to anoxaemia. This finding corresponds with those of BEREZELLER and WASTL, and WIEMER who showed that the addition of carbon dioxide to blood in vitro inhibited sedimentation. They considered this action was exercised either directly or indirectly through the electrolytes of the blood.

MOTZFELDT found that the average blood sedimentation rates of those cases surviving and those ending fatally were approximately the same, and concluded that the test was useless as a guide to prognosis.

WESTERGREN [107] agreed with MOTZFELDT'S observations, but commented on the S.R. figures between twenty and thirty reported in some cases by MOTZFELDT. He thought these were so low as to indicate that the diagnosis of Lobar Pneumonia was erroneous.

PULVER [92] observed that the time-lag in pneumonia between the onset of fever and increase in the S.R. was shorter than in other febrile conditions. He suggested that this was due to an unusually rapid change in the constitution of the blood.

STILLMUNKES and COSTE [109] noted in the report of a single case, a regular progression in the

S.R. up to the crisis, with a temporary retardation at the sixth day.

BROOKES [37.38] using his own technique, charted the results of five-minute readings and suggested that the type of curve was related to some extent to the gravity of the condition, and offered an indication of the prognosis.

D. The White Blood Cell count in Lobar Pneumonia.

The mechanism of leucocytosis in Lobar Pneumonia is similar to that in other inflammatory states.

The degree of leucocytosis varies according to the severity of the inflammatory process and the resistance of the individual. SONDERN [117] indicated that the degree of increase of the white cells bore no relationship to the intensity of the pathological process: where resistance is good, pronounced

leucocytosis may be found even with slight infections, but where it is poor, leucocytosis may be slight or absent whatever the severity of the infection.

In pneumonia, both leucopenia and excessive leucocytosis are considered indications of a bad prognosis . In most cases which recover, the white cell count shows a moderate increase (16,000-20,000 per cubic m.m.) and reaches its maximum during the early period of the crisis before falling. A second rise is indicative of a complication. TODD and SANFORD [113] showed that a sudden fall in the white cell count might be the first warning of a fatal issue, and that no matter how low the total count, an increase indicated a spread of the pneumonic process.

TÜRK, [111] GAITSKELL [112] and others elucidated the differential blood picture in pneumonia and found that there are three distinct phases; one, that of

neutrophil polymorph leucocytosis; the second, that of monocyte increase and the third, that of lymphocytosis. About the time of the crisis myelocytes often appear in the blood.

Studies of the ARNETH count have shown that it is sometimes of value in judging the severity of the infection and the resistance of the individual.[114.115.116.]

E. The connection between the White Cell Count and Sedimentation Rate.

No evidence of a definite correlation between the white cell count and sedimentation rate has so far been recorded.

GRESHEIMER, RYAN and JOHNSON[119] in a study of one hundred unselected individuals, found no significant relationship between the sedimentation rate and the total white count, and neutrophil and lymphocyte percentage.

MOTZFELDT[87] and GRAMS[118] denied a connection between the sedimentation rate and white cell count

in Lobar Pneumonia, and this opinion has been reiterated in respect of other inflammatory conditions by MORRISS,[120] MASSELL and JONES,[121] PULVER [92] HEIMANN [123] and others; but ERNSTENE [124] reported an approximate parallelism between the sedimentation rate and the white cell count in rheumatic fever and BEHR,[125] using MOMMSEN'S [126] stain, claimed a direct relationship between the sedimentation rate and the number of leucocytes containing basophil granules. These granules he took to be of the nature of globulins and he classed them morphologically with thrombocytes. Confirmation of this work is still lacking.

The white cell count follows the clinical condition more rapidly than the sedimentation rate, which shows a definite lag both at the onset of an inflammation and at the commencement of resolution (ERNSTENE,[124] HERRMANN,[89] ROGATZ,[128] PULVER [92] and others).

This delay has been attributed to the fact that whereas the bone-marrow is directly influenced by bacteria and their toxins, the sedimentation rate is first affected by the products of somatic reaction to inflammation.

Some writers have concluded that the sedimentation rate is a more reliable guide than the white cell count in inflammatory conditions (SCHUMACHER and VOGEL [129]; YATES, DAVIDOW, PUTNAM and ELLMAN; [130] BAER and REIS; [131] GRESHEIMER, RYAN and JOHNSON [119]). Others have stated that each form of test is fallible and that therefore both should be employed (HEIMANN, [123] MASSELL and JONES, [121] VOLK, [127] CHAIMOFF, [132] WESTERGREN, JUHLIN-DANNFELDT and SCHNELL. [60])

These conclusions indicate that any possible relationship between the white cell count and sedimentation rate is slight and inconstant.

Two factors mitigate against the accurate study of this relationship; first, as is well recognised, wide daily and diurnal variations occur in the white cell count,[133.134.135.136] whereas there is no similar variation in the sedimentation rate. Secondly, even in the hands of an expert, the assessment of the total number of white cells is by no means an exact form of estimation.[137.138.139.]

VI. A Report of Forty four cases of Lobar Pneumonia with special reference to the Sedimentation Rate, White Cell Count and Temperature.

The author has investigated the relationship between the sedimentation rate, white cell count and temperature in forty four cases of Lobar Pneumonia of which ten died.

A. Statistical Study.

(i) Method. Estimations were made daily throughout the febrile stage, and at regular intervals thereafter, so far as was practicable. Unfortunately, the admission of the majority of patients on the third, fourth and fifth days has prohibited the publication of an extensive number of investigations on the first few days of illness.

Observations of the three factors under consideration were made simultaneously and no one factor is quoted except the other two for that day are also reported.

The figure for the white cell count was obtained by taking the mean of three estimations. As far as possible, this was done at the same time each day, but as all patients were receiving a fluid diet, diurnal variation through the taking of food can have been only slight. For the purpose of statistical comparison, the mean of the four-hourly temperature readings on the day concerned has been adopted.

The three factors under consideration - the sedimentation rate, white cell count and temperature - have been compared statistically each day, from the second to the thirteenth day of illness.

(ii) Factors against statistical accuracy. The statistical report, while providing useful and suggestive results can not be accepted as being completely significant for the following reasons :-

a. The number of estimations is not sufficiently great to provide an entirely satisfactory study. This applies in particular to the tails of distribution.

b. The cases investigated were, to a certain extent, selected, for all the investigations were made on hospital patients who presumably were admitted for the most part either owing to the gravity of their condition or because they required special attention.

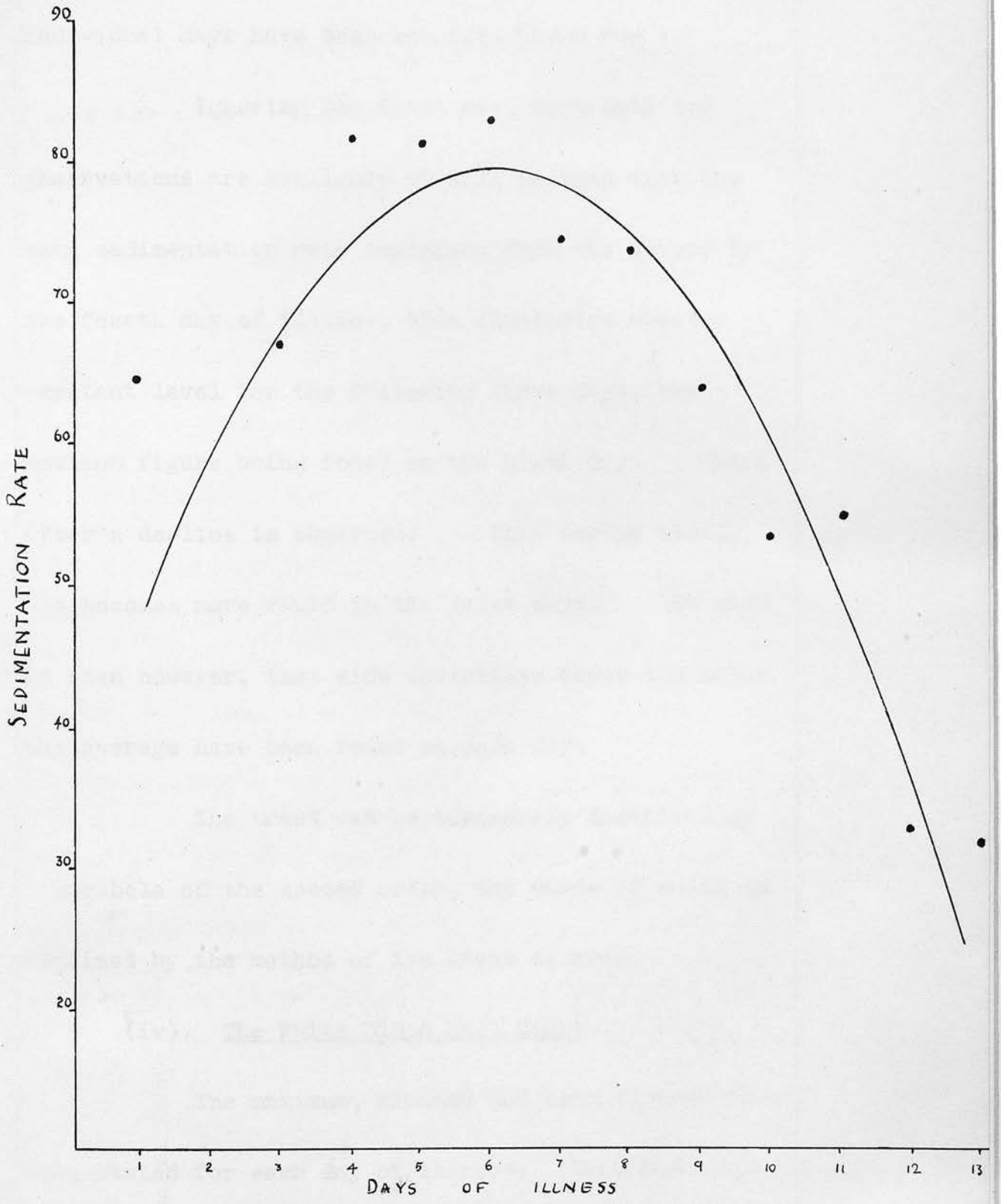
c. The mean figures reported for each day can not be taken as representing necessarily the average trend, for the course of the illness varies in each patient. In some of the cases reported, the pneumonic condition aborted as early as the third day; in others, the temperature fell by crisis on the fifth, sixth, seventh or eighth day, whilst in yet others, the temperature fell by lysis. This variability of normal standards mitigates against the success of a statistical investigation.

This variability of normal standards and the inclusion of cases which developed complications or ended fatally has obscured in the statistical report certain common trends which will later be illustrated by individual cases.

d. No allowance has been made for the effect of therapeutic agents: fourteen cases of those reported received four-hourly intravenous injections of collosol iodine throughout the febrile stage, and a further five were given anti-pneumococcal serum intravenously. No report is available of the effect of iodine injections on the sedimentation rate, but the writer formed the impression that it was negligible. ANGHELESCOW [122] found that therapeutic sera promoted sedimentation.

(iii) The Sedimentation Rate

The maximum and minimum figures and the mean of the observations of sedimentation rate on the



PARABOLIC REPRESENTATION OF THE TREND OF THE SEDIMENTATION RATE IN PNEUMONIA OBTAINED BY THE FORMULA $S.R = 35.3913 + 14.6484(D) - 1.2121(D)^2$ WHERE (D) REPRESENTS THE NUMBER OF DAYS OF ILLNESS

individual days have been set out. (Index Page i.)

Ignoring the first day, when only two observations are available it will be seen that the mean sedimentation rate increases from the second to the fourth day of illness, then fluctuates about a constant level for the following three days, the maximum figure being found on the sixth day. Thereafter a decline is observed. This begins slowly, but becomes more rapid in the later days. It will be seen however, that wide deviations above and below the average have been found on each day.

The trend can be adequately described by a parabola of the second order, the curve of which is obtained by the method of the least squares.

(iv). The White Blood Cell Count.

The maximum, minimum and mean figures have been stated for each day of illness. (Index Page iii).

It is seen that the mean results fall from the first to the sixth day, whereafter they continue to rise till the ninth day. They then tend to fall. Wide fluctuations from the average figure are found.

(v). The Sedimentation Rate, White Blood Cell Count and Temperature.

To facilitate the study of the relationship of these three variables, the data has been divided into three groups in accordance with the number of days of illness. The groups are from the first to the fourth, from the fifth to the ninth, and from the tenth to the thirteenth day of illness inclusive.

The mean and standard deviations together with the standard errors of the means for each variable in each of the three groups have been calculated (Index Page iv).

With the white blood cells and the temperature, the highest means occur in the first group,

and these means become progressively lower in the succeeding two groups. In each case, the difference between the means in consecutive groups of both variables is not statistically significant.

As has already been seen, the sedimentation rate follows a different trend, the maximum falling in the middle group of days five to nine. The difference between the means of the first and second groups is not statistically significant, but between the second and third groups a statistically significant difference is observed.

To establish the relationship, if any, between the three factors under discussion, the correlation coefficients were calculated for the S.R. and W.B.C., S.R. and temperature and W.B.C. and Temperature (Index Page v).

Consideration of the correlations with temperature shows that only one is statistically significant - that between the Sedimentation Rate and temperature in the last group. This correlation is not large, and all the other groups have definitely no correlation at all.

It would therefore appear that the temperature variable can not exercise much influence upon the degree of correlation between the remaining two variables: that this is true is seen from the partial correlations between the sedimentation rate and white blood cells, with temperature held constant, (Index Page vi). In every case, the partial correlations are of the same order as the total correlations which included the influence of temperature. It can therefore be concluded that temperature exercises no influence on the sedimentation

rate and white blood cell count.

(vi) The Sedimentation Rate and White Blood Cell Count.

The only statistically significant correlations between the sedimentation rate and white blood cell count occur in the first group (the first four days of illness), but the trend of the signs of these coefficients suggests that whereas in the early stages the two variables are inversely related, the tendency in the later days is towards direct relationship. In the intermediate group the coefficient approaches closely to zero, and in this stage the transition from inverse to direct relationship probably occurs.

An attempt has been made to establish exactly the point during the illness at which this transition occurs. For each day of illness the individual S.R. values have been plotted graphically against the

corresponding W.B.C. results, and from these graphs, correlation coefficients have been calculated for each day by SHEPPARD'S method:-

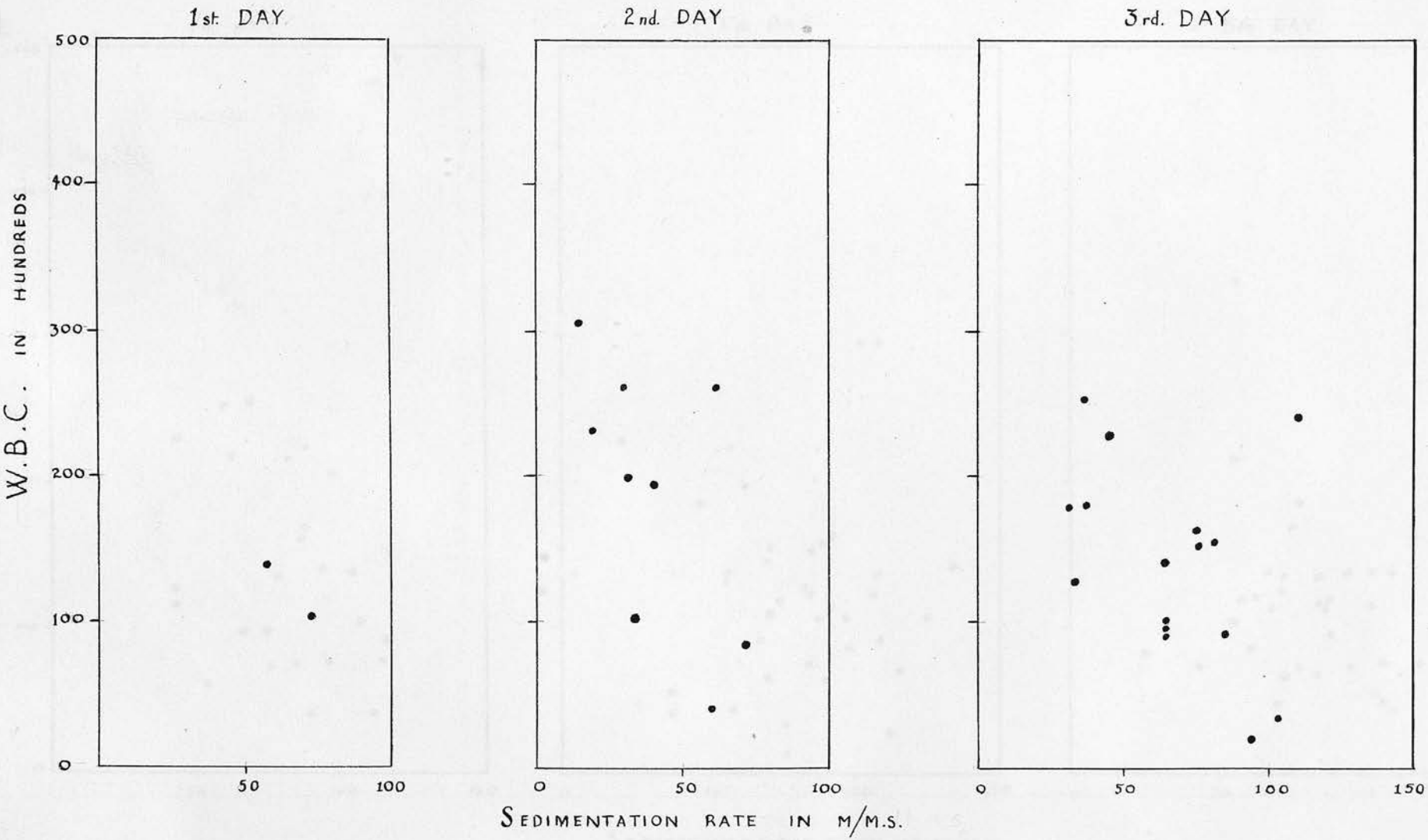
Days of illness.	Number of observations.	Sheppard's correlation coefficient.
2	9	-.7660
3	16	0.
4	26	-.1204
5	37	-.0425
6	34	-.1837
7	32	.1951
8	25	.2487
9	15	.5000
10	9	.1736
11	9	.5000
12	6	.5000
13	6	.5000

It is doubtful whether these coefficients are statistically significant and consequently little importance can be attached to their magnitude, but the constant trend of the signs presents an interesting sequence. Except for a zero correlation at day three,

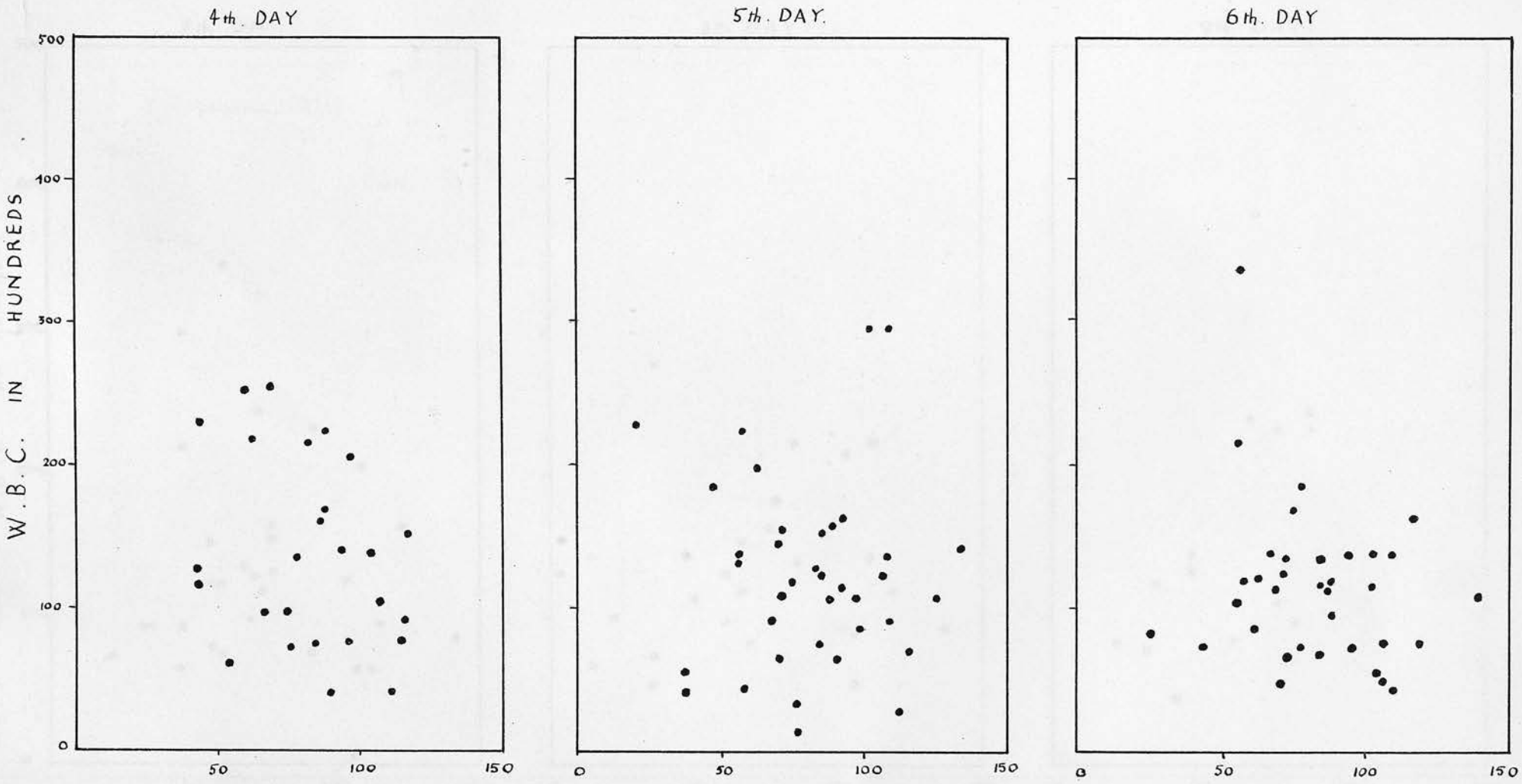
all the coefficients up to and including the sixth day are negative, while after the sixth day all are positive. This would suggest that the change from inverse to direct relationship between the S.R. and W.B.C. occurs between the sixth and seventh days.

Confirmation of this conclusion is found when the trend of the S.R. variable is compared with that of the W.B.C. variable. It has already been seen that whereas the W.B.C. showed a successive decline in the three groups of one to four, five to nine and ten to thirteen days of illness, the S.R. showed a second order parabolic tendency, with a maximum about the sixth day. It seems therefore that the phenomenon of transition from an inverse to a direct relationship may be due to this parabolic trend of the S.R. variable.

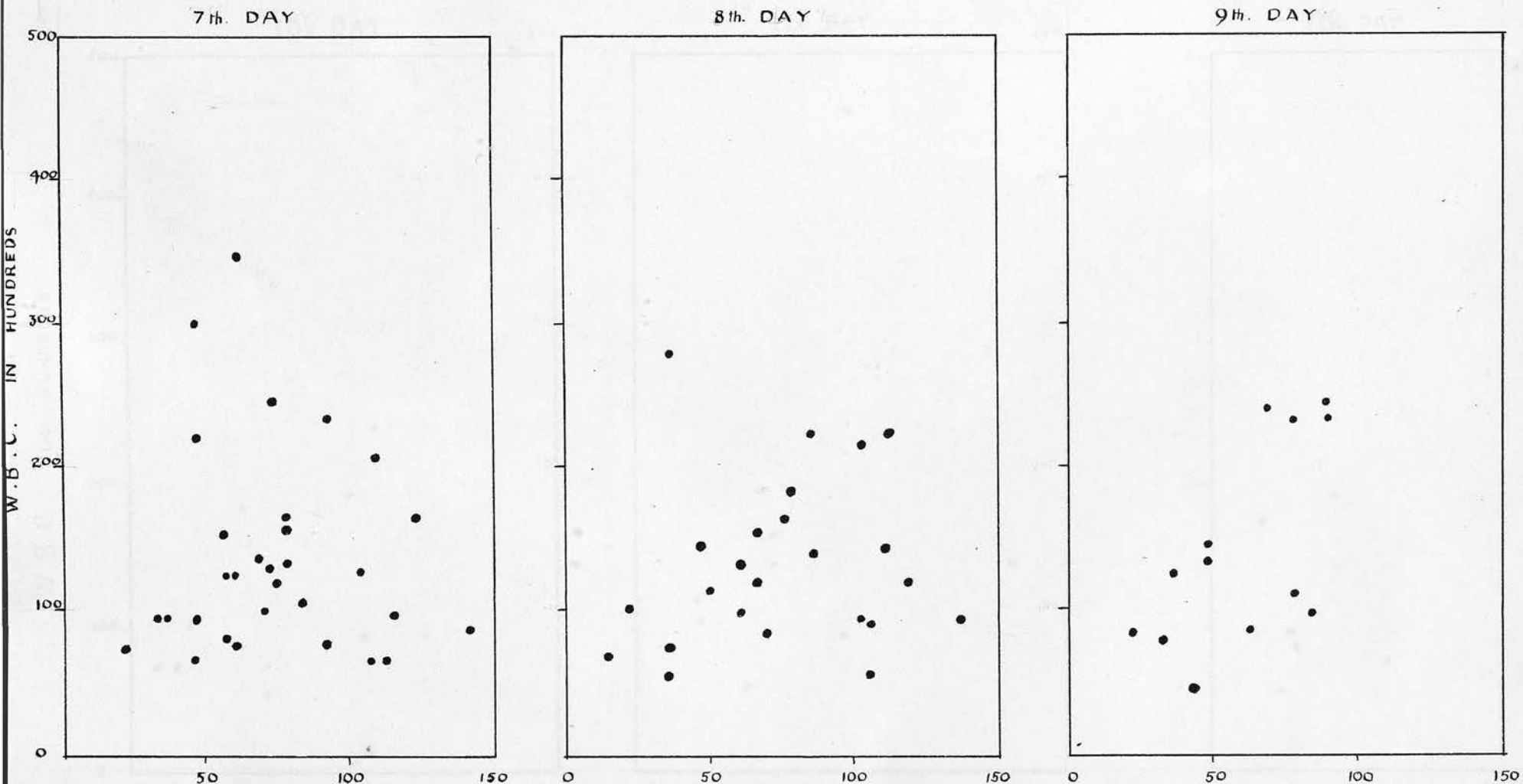
Charts of the frequency distributions of the S.R. and W.B.C. help to illustrate this tendency to



THE DISTRIBUTION OF SEDIMENTATION RATES AND WHITE BLOOD CELL COUNTS ACCORDING TO THE DAYS OF ILLNESS

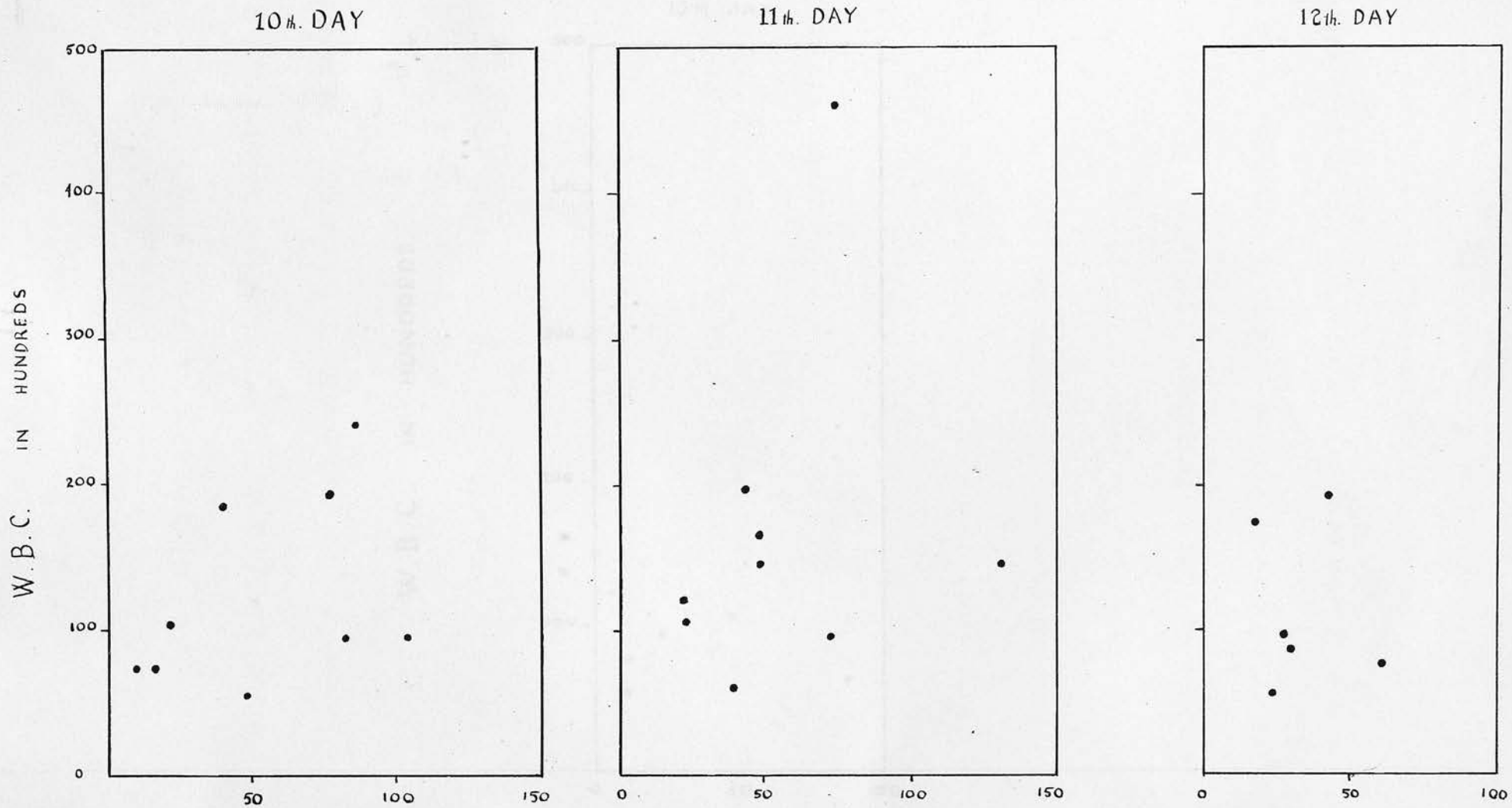


THE DISTRIBUTION OF SEDIMENTATION RATES AND WHITE BLOOD CELL COUNTS ACCORDING TO THE DAYS OF ILLNESS



SEDIMENTATION IN M/M.S.

THE DISTRIBUTION OF SEDIMENTATION RATES AND WHITE BLOOD CELL COUNTS ACCORDING TO THE DAYS OF ILLNESS



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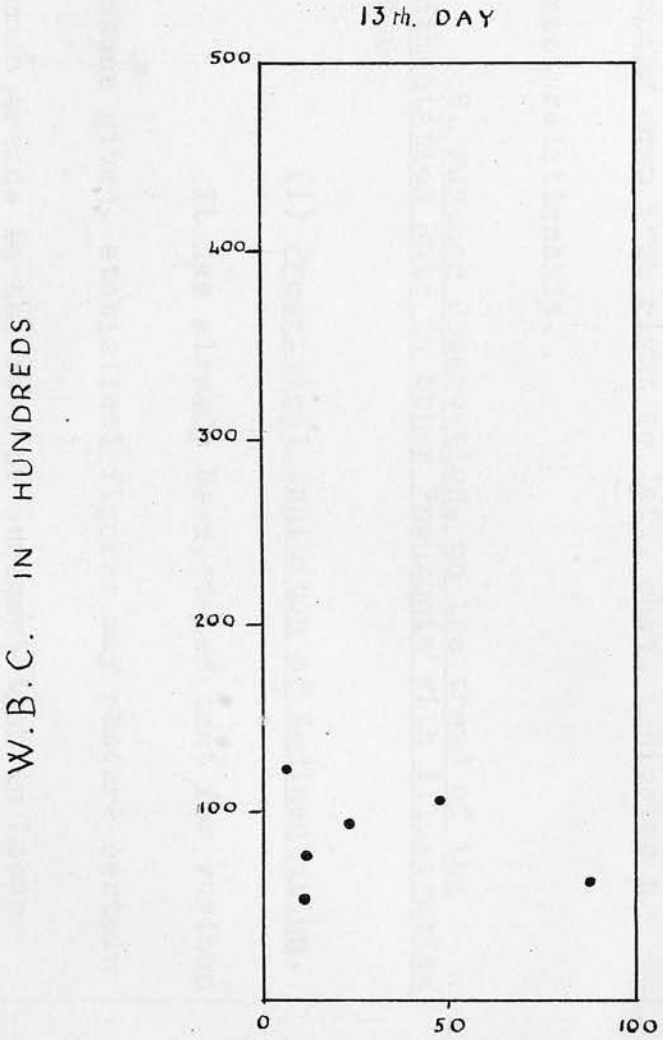


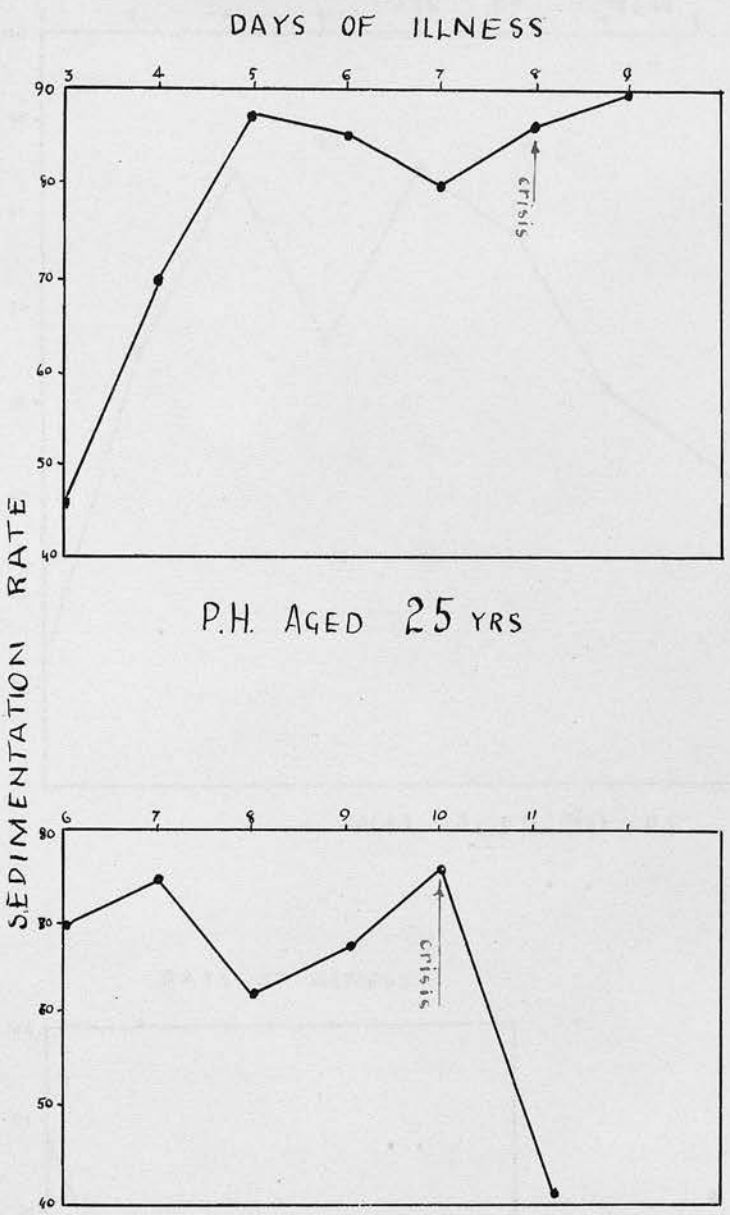
THE DISTRIBUTION OF SEDIMENTATION RATES AND WHITE BLOOD CELL COUNTS ACCORDING TO THE DAYS OF ILLNESS

inverse and later direct relationship. From these it can be seen that the individual results tend during each of the first five days to form a diagonal line sloping downwards from left to right, indicating an inverse relationship. No conclusion can be drawn from the chart for the sixth day, but from the seventh day onwards each record of distribution tends to form a diagonal line sloping down from right to left, which indicates a direct relationship.

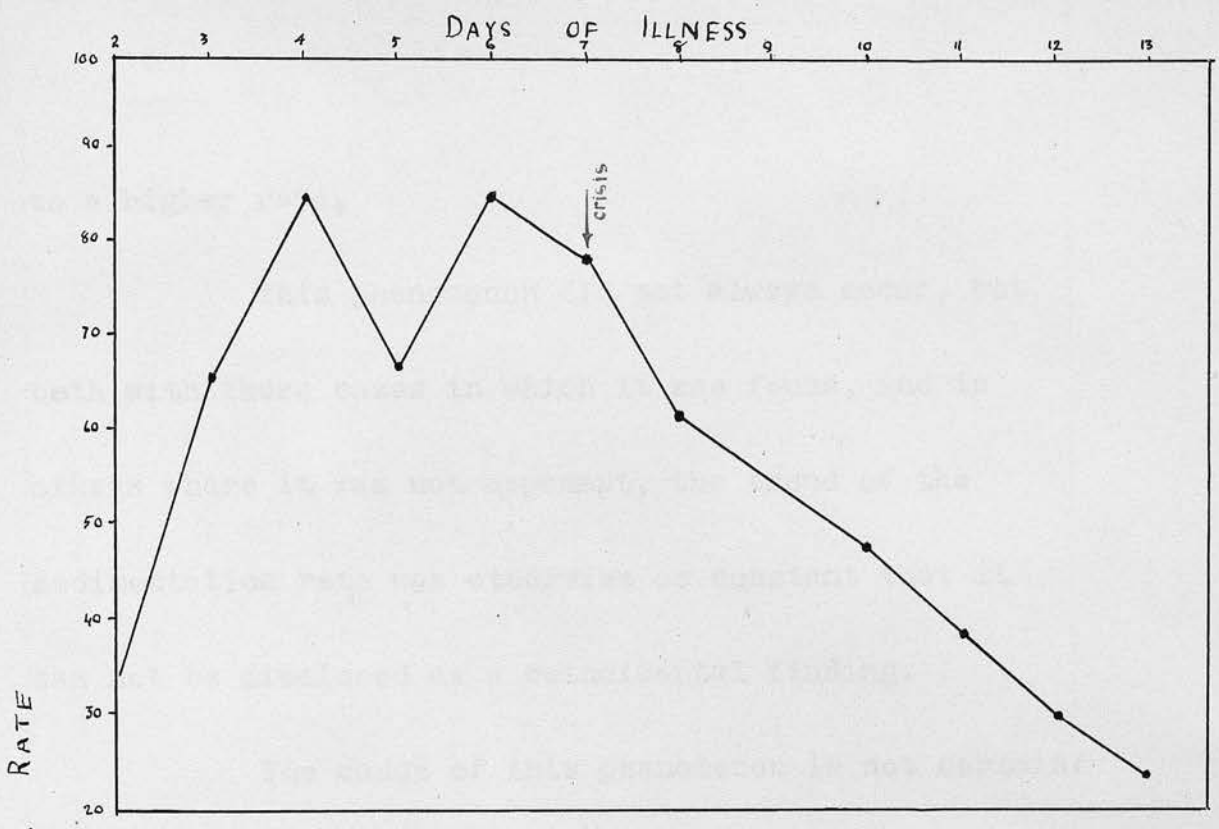
B. Further Observations on the trend of the Sedimentation Rate in Lobar Pneumonia with Illustrative Cases.

(i) Precritical Inhibition of Sedimentation.

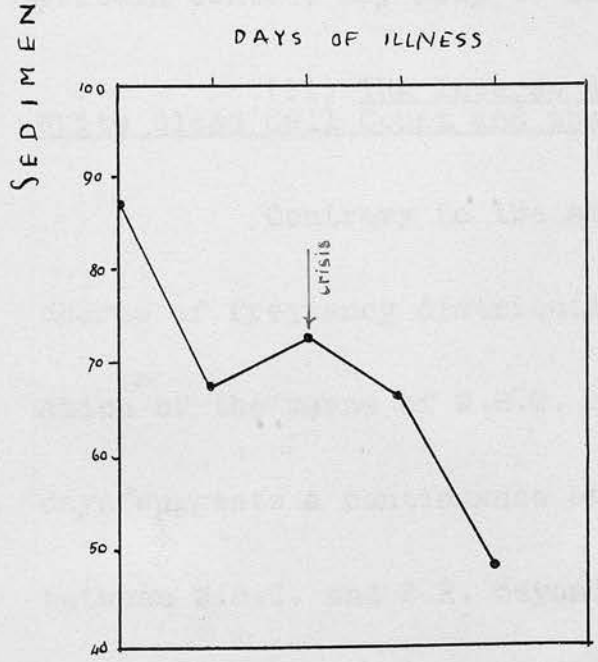
It has already been stated that for various reasons given, statistical figures may obscure certain common trends in the rate of sedimentation in Lobar Pneumonia. One such trend observed by the author, is a precritical diminution in the rate of sedimentation followed by a return to the former high, or even



EXAMPLES OF PRACTICAL INHIBITION OF SEDIMENTATION IN LOBAR—PNEUMONIA



W.M. AGED 40 YRS



J.M. AGED 37 YRS.

EXAMPLES OF PRACTICAL INHIBITION OF SEDIMENTATION IN LOBAR PNEUMONIA.

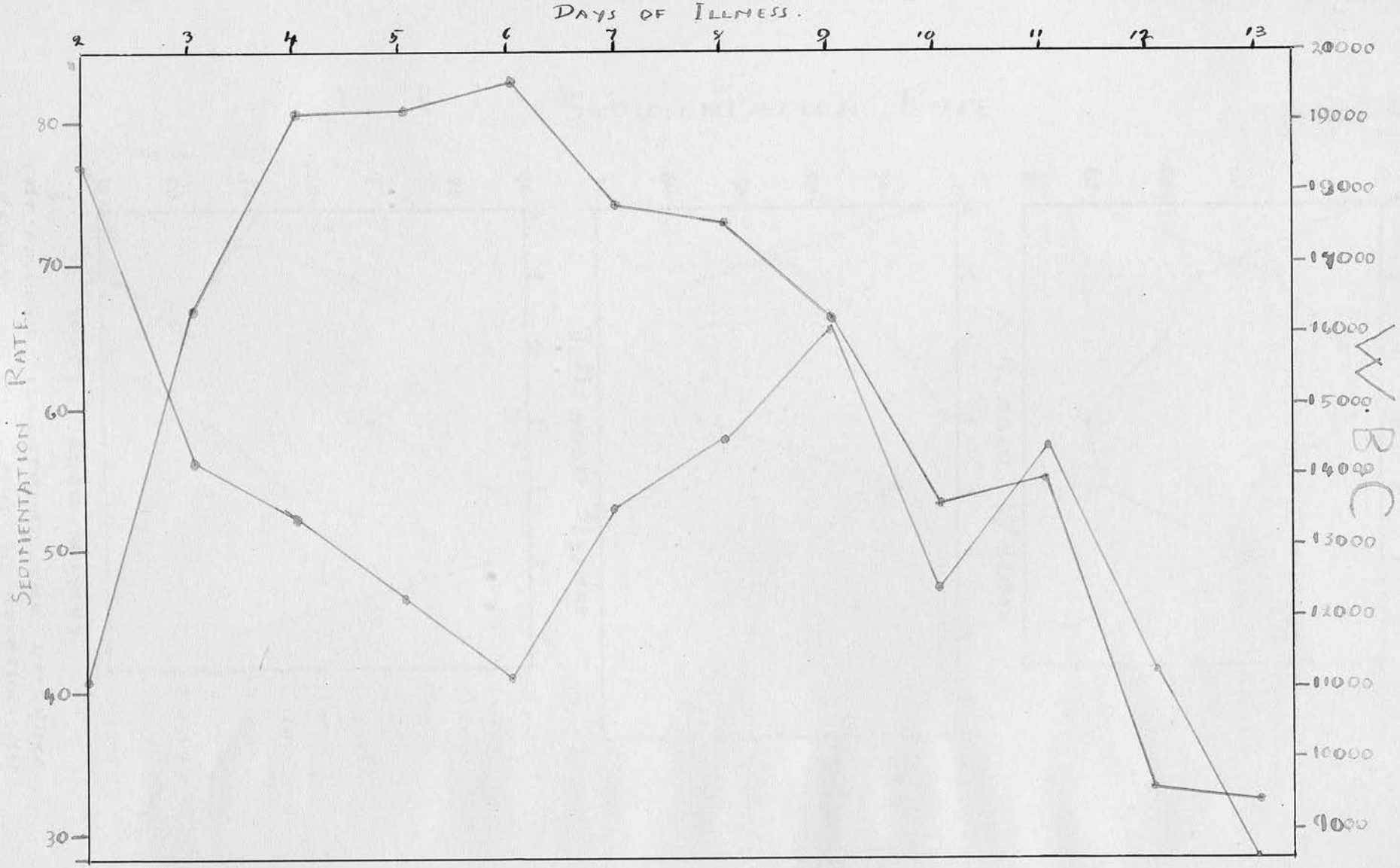
to a higher rate,

This phenomenon did not always occur, but both with those cases in which it was found, and in others where it was not apparent, the trend of the sedimentation rate was otherwise so constant that it can not be dismissed as a coincidental finding.

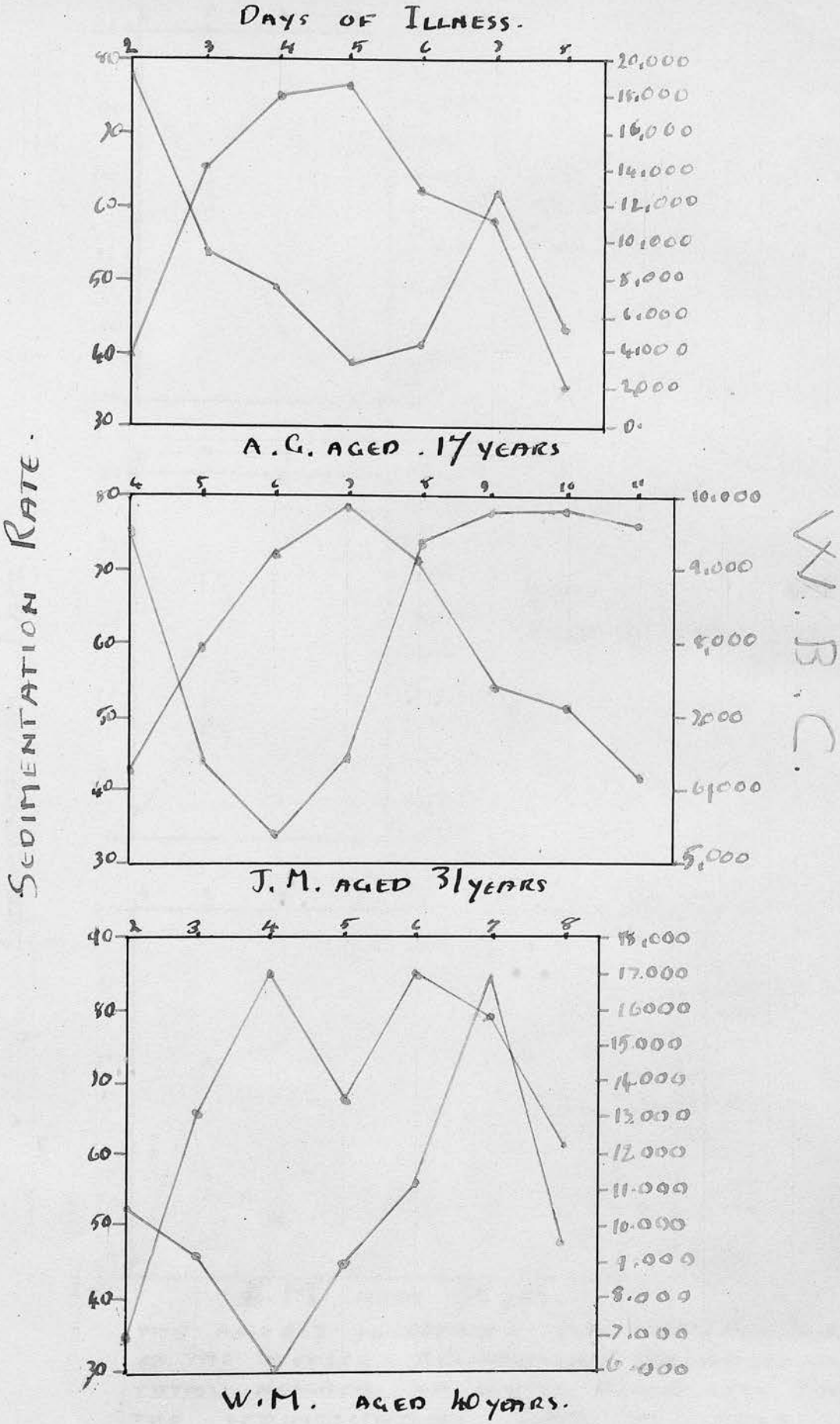
The cause of this phenomenon is not certain: a close study of the blood concentration and plasma protein content may help to elucidate the question.

(ii) The Inverse Relationship between the White Blood Cell Count and the Sedimentation Rate.

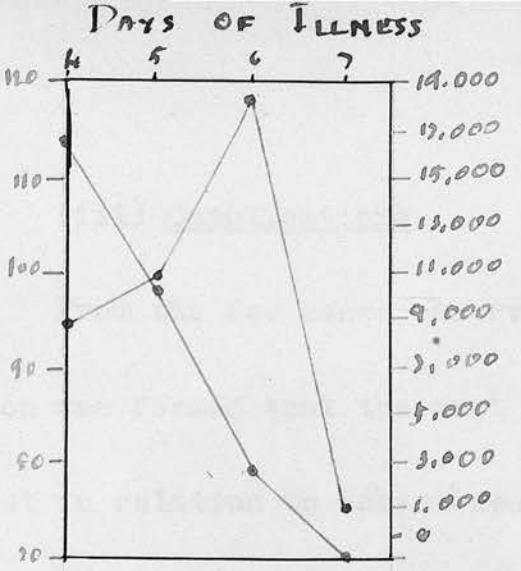
Contrary to the statistical results and charts of frequency distribution, the graphic presentation of the means of W.B.C. and S.R. on individual days suggests a continuance of inverse relationship between W.B.C. and S.R. beyond the sixth day. This was in fact observed in some cases.



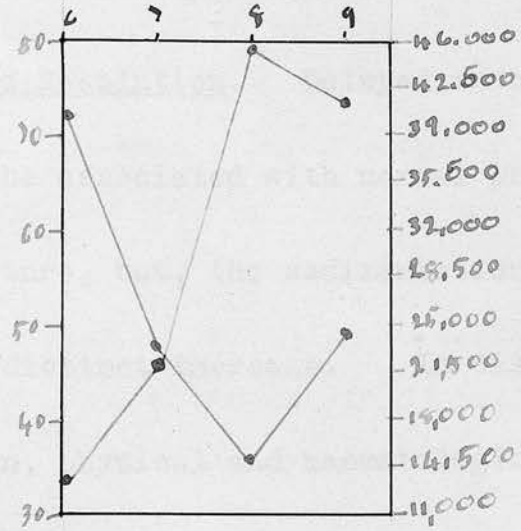
THE MEAN FIGURES OF THE WHITE BLOOD CELL COUNTS AND SEDIMENTATION RATES IN FORTY FOUR CASES OF LOBAR PNEUMONIA, PRESENTED GRAPHICALLY.



THE FIGURES ILLUSTRATE THE VARYING DURATION OF THE INVERSE RELATIONSHIP BETWEEN THE TOTAL NUMBER OF WHITE BLOOD CELLS AND THE SEDIMENTATION RATE IN LOBAR PNEUMONIA.



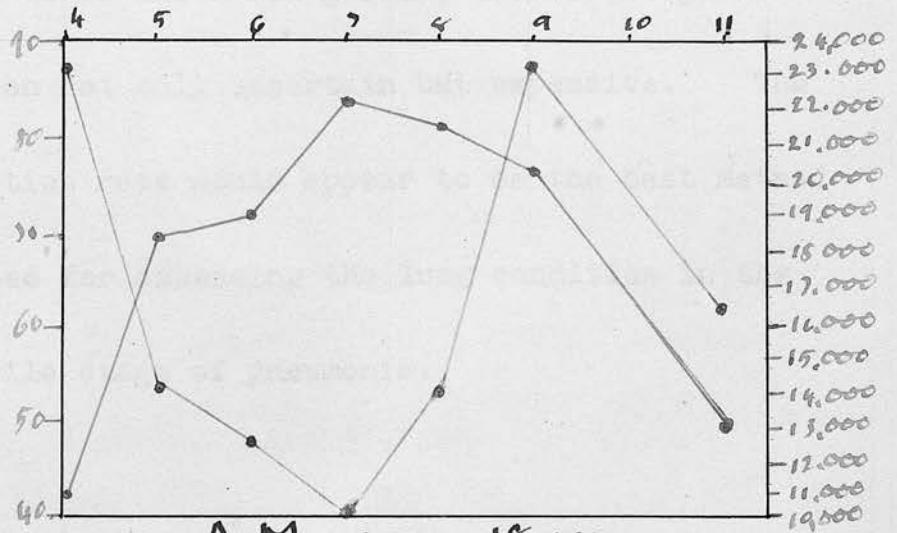
W. B.
AGED 49 YRS



E.W.
AGED 15 YRS.

W.B.C.

SEDIMENTATION RATE.



A. M. AGED 18 YRS.

THE FIGURES ILLUSTRATE THE VARYING DURATION OF THE INVERSE RELATIONSHIP BETWEEN THE TOTAL NUMBER OF WHITE BLOOD CELL AND THE SEDIMENTATION RATE. IN LOBAR PNEUMONIA.

(iii) Complications.

From the few cases observed by the writer, the opinion was formed that the most useful application of the test in relation to Lobar Pneumonia is in connection with complications.

a) Delayed Resolution. Delayed resolution was often found to be associated with normal physical signs and blood picture, but, the sedimentation rate invariably showed a distinct increase. In distinguishing delayed resolution, physical and haematological examinations are known to be uncertain guides, and radiological examination not only uncertain but expensive. The sedimentation rate would appear to be the best method yet devised for assessing the lung condition in the post-febrile stage of pneumonia.

CASE 14 - HISTORY.

J. H. aged sixty six, an old army man, was admitted on the third day of illness. He was collapsed, delirious and apparently moribund. There were signs of consolidation of the left lower lobe. His condition remained much the same until the sixth day, when the temperature fell by crisis.

He made a rapid recovery and three weeks after the inception of the illness felt quite fit; but physical and radiographic examination at this time suggested that the lung condition had not fully resolved.

Five weeks after the onset of illness, the patient felt his old healthy self. The physical signs and haematological picture were within normal limits and he was about to be dismissed. The sedimentation rate, however, was found to be 26 and radiological examination made in consequence of this finding revealed that the lung condition had not fully resolved.

(b) Delayed Resolution and Empyema.

CASE 30 - HISTORY

W.W. aged 30, was admitted on the fifth day of illness. He was extremely ill and

anxious. Signs of left lower lobe consolidation were present. Type III Pneumococci were found in the sputum.

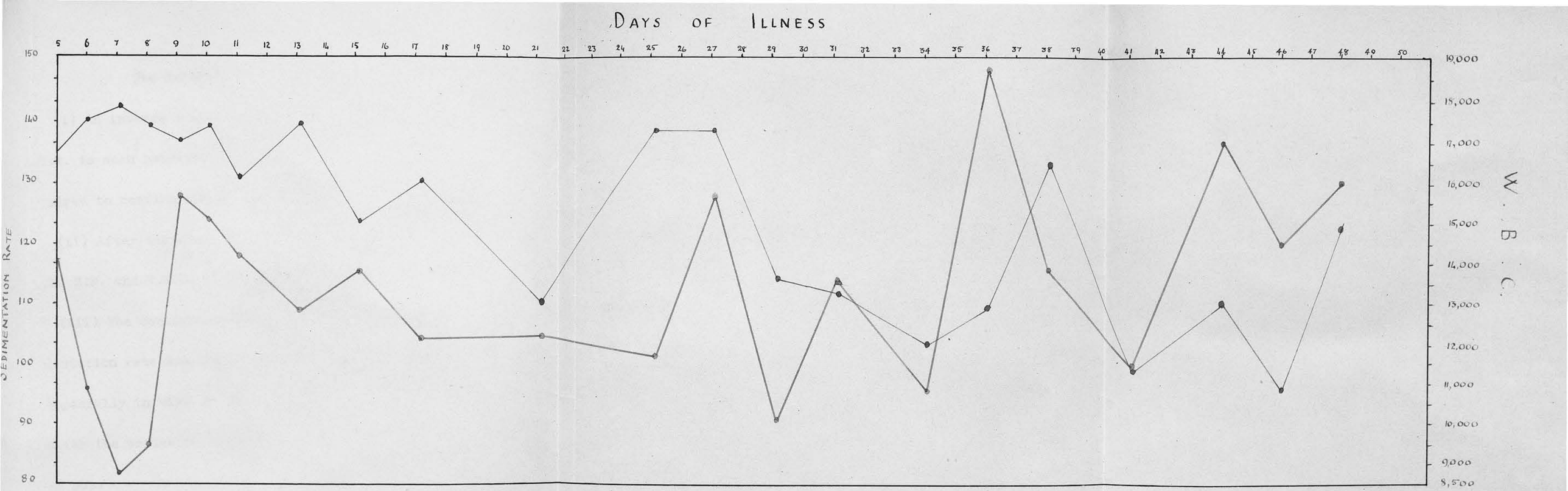
After admission, intermittent pyrexia persisted, as did the signs of consolidation. The patient continued to look ill and toxic.

On day fourteen, X-ray examination showed a consolidation of the left lower lung field.

On the fortieth day, the left pleural cavity was needled and a small amount of pus obtained.

X-ray examination on the forty fourth day suggested a continued consolidation of the left lower lobe, and small localised effusion in the upper part of the left pleural cavity. There was probably a little fluid in the lower part of the chest. The opacity might have been accounted for by consolidation alone, and carcinoma of the left lower bronchus was considered.

On the forty eighth day, the patient was transferred to a surgical ward, where operation revealed a fairly fresh pneumococcal empyema.



W. W. AGED 30 YEARS

DELAYED RESOLUTION AND EMPYENA. GRAPHIC COMPARISON OF THE SEDIMENTATION RATE AND WHITE BLOOD CELL COUNTS WHICH ARE INVERSELY RELATED UP TO THE TENTH DAY

The features observed in this case are :-

(i) An inverse relationship between the W.B.C. and S.R. is seen between the fifth and ninth day. This helped to confirm the diagnosis of Lobar Pneumonia.

(ii) After the ninth day the relationship between the S.R. and W.B.C. is totally irregular.

(iii) The continuance of an extremely rapid sedimentation rate was suggestive of a pyogenic process, especially in view of the wide fluctuations of S.R., which the writer has come to associate with collections of pus.

(iv) Carcinoma alone, as suggested by X-ray examination could certainly not account for such a high fluctuating S.R.

(v) Certain of the W.B.C. results were inconclusive or almost within limits of normal. Each S.R. result was far removed from normal.

The writer believes that in the assessment of complications single readings of the S.R. are of much greater value than single estimations of the W.B.C.

(iv) Fatal Cases

A study of the maximum, minimum and mean results of the S.R. and W.B.C. counts in fatal cases shows that these do not deviate greatly from those including the cases which survived, (Index Page ii).

Ten cases with fatal terminations were observed (Nos. 35-44). This number is not sufficiently great to warrant firm conclusions but the following tendencies were noted :-

(i) Leucopenia indicated a fatal outcome.

(ii) Moderate and even increasing leucocytosis were observed ante mortem, although the average W.B.C. count was below that of the mean, which included those cases recovering.

(iii) The S.R. was usually below average and in every case where more than one reading was obtained, began to diminish one, two or three days ante mortem.

Of the ten cases which died, all but one death occurred in the febrile stage, during which no decrease in the sedimentation rate is normally anticipated.

Two of these cases died before a second reading could be obtained. Amongst the remaining eight, the S.R. of one began to diminish three days ante mortem; of four, two days ante mortem and of three, one day ante mortem.

Inspection of the white blood cell counts in these eight cases shows that four exhibited an absence of leucocytosis or definite leucopenia, but the remaining four a normal leucocytosis.

A decreasing sedimentation rate associated

with a high or rising pulse rate,with or without leucopenia, resulted on several occasions in the accurate forecast of a fatal outcome.

VII The Sedimentation Rate as an Aid to Diagnosis in Lobar Pneumonia

Mention has been made of the inverse relationship between the S.R. and W.B.C. count in the febrile stage. The author believes that this relationship is a real aid to diagnosis, but the relationship can not be confirmed for several days by which time the diagnosis is usually apparent by other means.

The practical importance of the test, if any, is in the confirmation or refutal of the diagnosis of Lobar Pneumonia by single results.

The writer had the opportunity of seeing two cases in which the diagnosis of Lobar Pneumonia proved to be erroneous.

CASE 45 - HISTORY

S.R. aged 27 was admitted as a right basal Lobar Pneumonia.

Three days prior to admission he felt cold and shivery. He also complained of malaise, headache, sweating, a sore throat and

a dry cough, with a sharp pain over the precordium on coughing and deep breathing. Sputum was moderate in amount, white and muco-purulent.

When admitted on the fourth day of illness, the fauces were seen to be congested and there were no signs indicative of pneumonic consolidation.

The sedimentation rate was twenty four, and W.B.C. 13,400.

The following day a swab was taken and found to contain numerous diphtheria bacilli. The S.R. was seventeen m.m. less than the minimum observed in any case of Lobar Pneumonia on the fourth day.

CASE 46 - HISTORY.

E.R. aged 37. Two days before admission to hospital the patient had a rigor after playing golf in rain, and went to bed.

On the day of admission, he felt better, and got up, but was seized with acute abdominal pain. He was admitted as a case of perforation to a surgical ward, but no operation was performed.

The following day, signs of left basal

consolidation were found, and he was transferred as a case of Lobar Pneumonia to the medical side where he died twentyfour hours later.

On the day of transference to the medical ward (the fifth day of illness) the S.R. was thirty nine.

Except in those cases where the pneumonic state had aborted, (which it plainly had not done in this case) and in one other fatal case where the S.R. rate was thirty five, (Case 44) no figure was found on the fifth day of Lobar Pneumonia, which was nearly as low as that quoted in the present case.

The writer therefore mistrusted the diagnosis of Lobar Pneumonia, but failed nevertheless to discover the true diagnosis.

Post mortem examination revealed a septicaemic state following a boil on the back of the neck: a recent infarct was found in the left lower lobe, and the staphylococcus aureus abounded both in the infarct and the abdominal organs.

On one occasion, the sedimentation rate was of help in establishing a diagnosis of Lobar Pneumonia in a case previously thought to be tuberculous -

CASE 5 HISTORY

A.H. aged 23 gave a history of five months malaise and cough with white sputum.

Three days before admission, he complained of shivering and pain in the right side of the chest, with exascerbation of the cough and expectoration.

On admission, the patient's face was flushed. Restricted movement and dullness were found over the whole left lung field: breath sounds were faint and there were a few coarse crepitations. No fluid was obtained on aspiration of the pleural cavity.

X-ray examination showed calcification at the right apex and consolidation of the left lung.

The condition was diagnosed as tuberculous on the grounds of the long history of cough and malaise, the flushed appearance and the old tuberculous focus at the right apex revealed radiologically.

This view was maintained until the eighth day when the S.R. diminished sharply. In the absence of a rising pulse rate it was possible to state that this marked decrease indicated an

abrupt turn for the better; and, although the clinical examination and chart readings did not at the time support this view, the diagnosis of tuberculosis was refuted and a rapid clinical improvement forecast. This prognostication was verified by the subsequent rapid recovery of the patient.

VIII. The Sedimentation Rate in other Respiratory Conditions

A brief investigation has been made of the Sedimentation Rate in spontaneous pneumothorax, intrathoracic neoplasm, tuberculous pulmonary infiltration, pulmonary oedema and broncho-pneumonia. The results are given as affording an interesting contrast with those in Lobar Pneumonia.

A. Spontaneous Pneumothorax. (Cases 47 and 48).

Both cases gave a typical history and clinical picture of pneumothorax, which had in each case occurred within two days of the estimation of the Sedimentation Rate. In both instances, the S.R. was well within normal limits.

There are two possible applications for the S.R. when it is found to be normal in this condition. First, it helps to confirm the diagnosis by excluding inflammatory states and secondly, the diagnosis

established, it is of aid in the exclusion of a tuberculous etiology.

B. Bronchial Carcinoma. (Case 49).

In the case reported, an extensive haemorrhagic exudate, requiring repeated aspiration was present.

Despite this feature, the S.R. is seen to fluctuate around a constant figure. This observation of constancy is in accord with previous observations, and may be a helpful feature in distinguishing an intrathoracic neoplasm from an inflammatory state of the lung.

C. Tuberculous Infiltration. (Case 50).

This patient was admitted with signs of consolidation of the left lung and of a left pleural effusion. The consolidation cleared up completely and the effusion partly, but his general state at the

time of dismissal to another hospital suggested an early demise.

The figures are included for the purpose of illustrating that the relationship between the W.B.C. and S.R. is irregular, and that the S.R. affords a more constant guide than the W.B.C. count which was frequently normal.

D. Pulmonary Oedema. (Case 51).

The patient was admitted with a history typical of pulmonary oedema of four hours duration. The diagnosis was confirmed by physical examination and copious frothy, blood-stained sputum.

Comparison of the S.R. and W,B.C. shows that there is no tendency to inverse relationship, and indeed that in the early stages, the trend is towards direct variation.

E. Broncho-Pneumonia (Cases 52-55).

From the records of the four cases observed it will be seen that the Sedimentation Rates are of the same order as in Lobar Pneumonia, but the rate remains high in the post-febrile stage in each case for a longer time than in the corresponding period of Lobar Pneumonia.

An inverse relationship between W.B.C. and S.R. is found in Case 52. up to the eighteenth day, and in Case 54. until the fourteenth day; but the number of cases is insufficient to warrant a conclusion concerning this relationship in broncho-pneumonia.

SUMMARY

Subject.

The sedimentation rate, white blood cell count and temperature have been investigated and compared in forty four cases of Lobar Pneumonia.

Method.

WESTERGREN'S method of estimating the sedimentation rate has been employed. Charts constructed from readings taken every five minutes showed that the curve of fall was almost constant for each given end-point. The conclusion was reached that no advantage is gained by the taking of such frequent readings or by the use of the continuous photographic technique.

In two cases during the process of sedimentation, the supernatant plasma was coloured pink and contained minute red particles. In each case, the sedimentation rate was grossly increased, and the

blood profoundly anaemic. The phenomenon was attributed to the formation of erythrocyte aggregations of unequal size with dissimilar rates of fall.

The Sedimentation Rate in Lobar Pneumonia.

The mean sedimentation rate in a series of cases of Lobar Pneumonia has been calculated for each day of illness. This rate is increased from the first day, rises till the fourth day, and fluctuates about a constant level for the next three days, before slowly returning to normal. Where resolution proceeds normally, the sedimentation rate is found to be within normal limits, about three or four weeks after the onset of the illness. The persistence of an increased sedimentation rate may be the sole guide to a complication. The sedimentation rate is especially useful in the assessment of delayed resolution, being always raised, whereas physical signs and the blood

picture may be normal.

In some cases, a precritical diminution of the sedimentation rate has been observed.

Wide fluctuations from the mean have been recorded, but the sedimentation rate is usually so greatly increased in Lobar Pneumonia that it may be of help in the diagnosis or exclusion of this condition, as was found in the cases reported of erroneous diagnosis.

In cases ending fatally, the sedimentation rate invariably diminished ante mortem. This retardation in association with a high or rising pulse rate, with or without leucopenia, is an excellent guide to the prognosis.

<u>The Sedimentation Rate, White Blood Cell Count and Temperature, in Lobar Pneumonia</u>.

By calculation of the correlation coefficients between the temperature and the

sedimentation rate and white blood cell count in the cases studied, it has been shown that body temperature exercises no influence on either the sedimentation rate or white blood cell count. This conclusion has been confirmed by repeating the calculations with temperature held constant. The coefficients of this partial correlation are in every case, almost identical with those of the total correlations.

Statistical comparison of the sedimentation rates and white blood cell counts on individual days has revealed that they tend to vary inversely up to about the sixth day, and thereafter, to vary directly with each other.

The Sedimentation Rate in other Respiratory Conditions.

For purposes of comparison, mention has been made briefly, of the sedimentation rate in other chest conditions. In broncho-pneumonia, a tendency

to inverse relationship between the sedimentation rate and white blood cell count during the febrile phase, has been noted. In bronchial carcinoma, the sedimentation rate varies around a constant figure.

CONCLUSIONS.

The Sedimentation Rate in Lobar Pneumonia.

1. The sedimentation rate is greatly increased throughout the febrile stage.

2. The sedimentation rate tends to vary with the white blood cell count, inversely till about the sixth day, and directly thereafter.

3. Body temperature exercises no influence on the sedimentation rate or white blood cell count.

4. The sedimentation rate is of help in diagnosis.

5. The sedimentation rate is of great value in the assessment of complications, especially of delayed resolution.

6. Ante mortem retardation of the sedimentation rate invariably occurs.

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52-55 Broncho-Pneumonia.

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THE SEDIMENTATION RATE IN A SERIES OF CASES OF LOBAR PNEUMONIA.

Day of Illness	No. of observations.	Sedimentation Rate Max.	Min.	Mean
1	2	71	58	64·50
2	9	71	15	40·00
3	16	109	31	67·00
4	26	115	41	81·38
5	37	135	20	81·35
6	34	140	25	82·91
7	32	142	23	74·28
8	25	139	15	73·48
9	15	136	21	63·93
10	9	103	10	53·33
11	9	75	21	55·00
12	6	60	17	32·67
13	6	89	7	31·83

THE SEDIMENTATION RATE IN TEN FATAL CASES OF LOBAR PNEUMONIA

Day of Illness	Sedimentation Rate		
	Max.	Min.	Mean.
2	61	19	46·6
3	109	34	78·2
4	115	41	83·0
5	116	35	80·3
6	110	55	73·8
7	114	34	73·0
8	104	21	52·0
9	21	21	21·0

THE WHITE BLOOD CELL COUNT IN A SERIES OF CASES OF LOBAR PNEUMONIA

Day of Illness	No. of Observations.	W.B.C. in hundreds		
		Max.	Min.	Mean.
1	2	140	106	220·0
2	9	306	40	187·1
3	16	254	24	142·0
4	26	258	40	133·6
5	37	298	18	123·2
6	34	336	42	111·8
7	32	324	64	136·4
8	25	456	54	145·4
9	15	410	78	161·3
10	9	244	54	124·0
11	9	460	60	144·2
12	6	194	58	113·3
13	6	126	52	86·0

THE MEANS, STANDARD DEVIATIONS AND STANDARD ERRORS OF THE MEANS OF THE SEDIMENTATION RATE, WHITE BLOOD CELL COUNTS AND TEMPERATURES IN A SERIES OF CASES OF LOBAR PNEUMONIA.

Days of Illness.	Number of Observations.	S.R. Mean	S.R. S.D.	W.B.C. in Hundreds Mean	W.B.C. in Hundreds S.D.	Temp. in Degrees F. Mean	Temp. in Degrees F. S.D.
1 - 4	53	69.38±3.63	26.44	144.83±9.80	71.32	101.25±0.190	1.38
5 - 9	143	76.81±2.37	28.30	132.02±6.09	72.86	99.81±0.135	1.62
10 -13	30	45.40±5.60	30.65	126.53±14.51	79.47	99.67±0.151	0.826

THE CORRELATION COEFFICIENTS BETWEEN THE SEDIMENTATION RATES, WHITE BLOOD CELL COUNTS AND TEMPERATURES IN A SERIES OF CASES OF LOBAR PNEUMONIA.

Days of Illness	Number of Observations	Correlation coefficient, (r ± standard error.) S.R. & W.B.C.	S.R. & Temp.	W.B.C. & Temp.
1 - 4	53	-.4435 ± .110	.0067 ± .137	.0697 ± .137
5 - 9	143	-.0964 ± .083	.1928 ± .081	.1198 ± .082
10 -13	30	.2878 ± .167	.4117 ± .152	.1993 ± .175

TOTAL CORRELATIONS BETWEEN THE SEDIMENTATION RATES AND WHITE BLOOD CELL COUNTS, AND PARTIAL CORRELATIONS WITH TEMPERATURE HELD CONSTANT, IN A SERIES OF CASES OF LOBAR PNEUMONIA.

Days of Illness	Number of Observations	Correlation coefficients (r ± S.E.) S.R. & W.B.C.	S.R. & W.B.C. keeping Temperature constant.
1 - 4	53	-.4435 ± .110	-.4450 ± .110
5 - 9	143	-.0964 ± .083	-.1227 ± .082
10 -13	30	.2878 ± .167	.2304 ± .173

THE CORRELATION OF THE SEDIMENTATION RATE AND THE WHITE BLOOD CELL COUNT IN A SERIES OF CASES OF LOBAR PNEUMONIA BY SHEPPARD'S METHOD.

Day of Illness	No. of Observations	Sheppard's correlation coefficient.
2	9	-·7660
3	16	0·
4	26	-·1204
5	37	-·0425
6	34	-·1837
7	32	·1951
8	25	·2487
9	15	·5000
10	9	·1736
11	9	·5000
12	6	·5000
13	6	·5000

Day of Ilness	Pulse M.	Pulse E.	Temp. M.	Temp. E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R.
3		102·		102·6		30				
4	104	108	102·2	100·2	32	38	108/70	90	13 600	78
5	106	108	101·6	101·2	40	50	128/80		11 000	70
6	120	104	102·8	100·	60	48	132/70	84	6 800	75
7	108	104	100·4	101·	42	48	120/78		12 600	60
8	102	96	101·	98·4	50	40				
9	92	96	100·4	99·4	50	42	140/70	78	14 600	48
10	76	90	97·	97·8	38	40				
11	74	78	97·	98·	34	30	126/74		10 200	22
13	78	66	97·8	97·2	24	22		62	7 800	12
15	72	60	97·	97·2	20	20		52	6 000	10
19	76	80	98·	97·	20	20	120/70		9 200	16
22	76	74	97·4	97·	20	20		52	4 400	12
25	70	70	97·	97·	20	20		48	1 800	10
28	70		97·4		20		120/70	52	6 400	5

CASE 1.
Lobar Pneumonia
A.W. Aet. 23.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R. 1.	S.R. 2.	S.R. 24.
2		136		103·5		34	112/80	81	10 400	34	64	106
3	120	128	99·4	100·2	30	32	118/86	76	9 200	66	88	110
4	104	128	99·	102.	26	32	105/68	78	6 000	85	122	137
5	126	128	100·8	100·	40	38	144/90	82	9 000	67	92	114
6	116	120	100·	100·4	40	36	106/60	78	11 200	85	121	139
7	112	100	99·9	99·4	32	32	108/70	92	16 800	79	121	136
8	104	84	98·5	97·	28	30	94/60	72	9 600	62	111	133
9	86	80	97·4	96·8	28	30						
10	80	72	97·	97·	30	26	108/64	58	5 400	48	89	134
11	72	70	97·2	96·8	24	24	104/66		6 000	39	76	127
12	70	64	97·2	96·8	24	22	100/66		8 400	30		
13	76	70	97·4	97·	20	20	100/50	86	9 200	24	51	120
14	72	70	97·2	97·	20	22			7 400	17	38	115
15	72	70	97·	97·	20	20	108/52	46	5 800	14	29	101
16	68	70	97·	96·8	20	20	110/56		6 200	13	34	85
18	76	74	97·	97·	20	20	108/52	48	8 600	10	23	99
21	72		97·		20		96/54	48	6 600	6	14	

CASE 2
Lobar Pneumonia
W.M. Aet.40

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R. 1.	S.R. 2.	S.R. 24.
4		100		102·2		28	122/84	90	9 600	73	109	131
5	108	112	101·3	101·4	26	28	94/65	86	6 400	90		130
6	100	104	100·4	100·2	28	28			5 400	103	125	132
7	88	84	99·2	98·2	20	22	92/64		6 400	109	128	138
8	78	68	97·2	96·4	22	22	92/66	76	9 400	102	129	137
9	78	72	98·	99·	20	28	100/64	51	9 800	84	121	135
10	78	72	98·2	98·4	26	28	104/68	86	9 800	82	117	130
11	78	72	97·4	97·	24	26	122/74	76	9 600	71		
12	76	64	97·2	97·	20	20			7 600	60		
13	78	68	97·	97·4	20	20	126/84	73	10 400	47	85	127
14	74	60	97·8	97·	20	20	110/74	64	4 200	47	80	124
15	60	84	97·2	98·4	20	20	100/68	80	9 600	41	78	124
18	72	76	97·	97·2	20	20	98/70	78	11 800	43	82	125
21	68		97·4		20			51	8 600	33		124

CASE 3
Lobar Pneumonia
J.N. Aet. 31

CASE 4
Lobar Pneumonia
H.D. Aet. 24.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R. 1.	S.R. 2.	S.R. 24.
1		108		102.2		24	104/74	86	10 600	71	104	133
2	100	118	100.6	102.7	24	24	106/74					
3	108	112	101.	102.2	24	24			3 800	103	126	134
4	100	112	100.8	100.6	24	28	88/66	81	4 200	109	129	138
5	100	96	102.	99.4	26	26	118/84		9 000	109	121	128
6	100	104	99.6	100.8	22	24	108/78	78	9 200	90	122	136
7	106	96	99.6	100.4	22	28	102/68	90	13 800	68	106	132
8	88	90	97.2	97.2	22	20	100/66	78	11 400	51	-	-
9	86	72	97.7	97.	20	20		67	7 800	34	-	112
10	84	72	96.6	97.2	20	20	100/70	72	10 200	21	46	105
11	76	68	97.	97.8	20	20			12 000	21	49	107
12	68	84	97.	96.8	20	20	98/54	66	5 800	22	-	-
15	72	84	97.2	98.	20	20	92/58	80	9 000	24	61	114
20	64		97.		20			52	7 000	17	-	-

Day of Illness	Pulse M.	Pulse E.	Temperature. M.	Temperature. E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R.
4		108		98·7		30	100/65		7 600	95
5	118	108	101·	102·	30	30				
6	112	120	100·	100·	28	36		84	11 800	101
7	118	118	103·	99·6	28	36			13 000	105
8	102	118	98·2	101·2	28	28	114/75	96	18 600	80
9	108	94	98·8	99·	28	28				
10	100	94	98·4	98·8	20	26		80	18 400	38
11	82	72	98·	98·8	20	22	110/62			
12	78	76	98·2	98·	22	22	102/60	80	17 600	17
13	72	70	98·2	98·	22	22	102/60			
14	80	74	97·2	98·2	20	20		48	11 200	15
16	68		97.2		20		116/55	76	7 800	11

CASE 5
Lobar Pneumonia
A.H. Aet. 23

CASE 9

Lobar Pneumonia.

A.B. Aet. 20.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	1.	S.R. 2.	S.R. 24
1	92	96	99.	103.	26	24	108/65	91	14 000	58	-	132
2	104	100	103.6	102.4	28	26	108/64	86	8 600	71	110	135
3	78	78	96.4	97.5	24	20	127/90		9 600	67	109	132
4	78	68	97.	97.	20	20	118/78		6 000	52	88	132
5	76	64	96.8	97.5	20	20	90/65	56	4 200	36	-	127
6	80	76	96.6	96.6	20	20	90/64		8 000	25	48	122
7	78	68	97.8	96.4	20	20	90/62	67	7 200	23	-	107
8	80	84	98 2	97 6	20	20	110/78		6 800	15	32	95
9	80	86	97.2	98.4	20	20	136/86					
10	78	80	97.2	98.4	20	20	136/86	60	7 600	10	24	98
13	68		97.		20		142/100	71	12 600	7	16	96

CASE 7

Lobar Pneumonia

J.G. Aet. 25.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R.
5		120		103.		48				
6	112	110	100.8	102.	40	44	90/62	96	11 200	70
7	116	120	102.	102.4	38	53		92	12 000	75
8	110	120	101.2	102.2	40	54	100/64	92	13 600	63
9	108	120	99.8	101.3	32	42	98/70	96	24 000	68
10	100	80	98.4	98.4	30	36	94/62	80	19 200	76
11	88	88	96.4	98.2	31	38	90/55	88	19 800	41
12	84	86	97.8	99.	38	24	68/50	80	19 400	41
14	88	78	97.2	98.4	21	28	90/50	76	16 200	31
16	76		97.8		24			72	8 600	25

CASE 8
Lobar Pneumonia
J.H. Aet. 36.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	W.B.C.	S.R.
5	92	88	102.5	102.	20	22		23 000	20
6	96	88	101.	100.6	24	20	120/70		
7	96	92	100.6	99.	24	24	148/80	9 800	47
8	88	98	99.	100.	24	24		12 000	67
9	68	68	97.2	99.	20	22	128/80	8 600	62

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	Pol.%	W.B.C.	S.R.	
4	104	104	102.3	103.	28	24				
5	100	90	100.4	100.2	24	24	78	13 400	56	

CASE 9
Lobar Pneumonia
D.T. Aet. 24.

CASE 10

Lobar Pneumonia Ty

D.N. Aet. 15.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	W.B.C.	S.R.
2	134	136	103.	102.4	30	36	26 200	29
3	128	120	102.8	103.6	28	40	14 200	65
4	110	120	101.8	103.	30	32	16 600	88
5	110	120	102.2	101.	34	30	7 600	84
6	100	104	100.5	102.	32	28	6 800	84
7	108	112	100.2	101.2	30	26	13 400	78
8	100	84	98.8	99.8	26	26	16 400	77
9	84	80	97.	98.2	20	22	11 000	77

CASE 11

Lobar Pneumonia Type 1.

JC. Aet. 35.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	W.B.C.	S.R.
5				101.		36	16 400	92
6	84	96	98.4	98.	24	24	14 000	103
7	68	82	97.3	98.4	22	20	13 000	72
		80						

CASE 12

Lobar Pneumonia Type 1.

M.S. Aet. 20.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	W.B.C.	S.R.
2	106	116	101.	100.8	56	40	19 600	31
3	112	120	99.4	100.6	34	32	15 600	81
4	98	88	98.4	98.8	28	26	20 800	97

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	W.B.C.	S.R.	
4	96	104	102·	101·5	20	28	110/72	22,000	61	
5	104	116	99·8	98·	28	28		14,000	57	
6	108	92	98·4	99·	26	22	108/74	11,600	60	
7	86	92	96·	98·	20	20		12,200	36	

CASE 13
Lobar Pneumonia
Type 1
M.W. Aet. 47

CASE 14

Lobar Pneumonia Type

J.H. Aet. 66.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	BP.	W.B.C.	S.R.
3	110	118	99.4	103.	40	40	95/70	18 000	31
4	88	92	101.6	101.	32	32		7 200	82
5	96	96	101.2	100.2	30	30	98/76	11 400	91
6	84	68	99.	98.4	30	24		7 800	107
7	64	64	97.4	98.	30	28		7 600	92
36	76	78	97.6	98.2	20	20		5 600	28

CASE 15
Lobar Pneumonia
Type 2
L.A. Aet. 40

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R.
5	112	102	104 2	102 8	38	36	116/78	96	12 200	106
6	108	100	100 3	100 4	36	28	100/60	92	13 600	110
7	88	80	99 2	100 2	26	24		76	9 800	115
8	76	76	99	98 8	24	28	98/66	56	9 000	106
9	72	72	98 6	98 4	24	26				
10	76	68	98 6	98	24	24		76	9 200	103
11	72	68	98	98 3	24	24				
13			98	98 4	22	24		72	6 400	89
16			97 2	97 2	20	20		92	11 000	25
22			97 1	97 1				48	6 200	15

CASE 16.
Lobar Pneumonia
Type 2
A.G. Aet. 17

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R.
2	100	100	101·8	103·	28	32		68	19 400	40
3	96	84	100·	102·	28	32	114/54		9 800	66
4	80	92	101·	102·6	24	40		96	7 600	75
5	84	80	102·2	99·4	32	32			3 400	77
6	68	88	98·4	102·4	24	44		72	4 200	63
7	78	62	101·3	99·	36	34		88	12 600	58
8	58	44	98·4	96·6	22	22		84	5 400	35
9	40	44					110/50			
10	48	44	97·8	97·2				48	7 400	16
13	40	60	97·	97·2				32	5 200	12
16	56	72	97·4	97·8				36	4 200	10
19	64	64	97·7	98·				84	8 800	12
21	72	88	97·6	98·2				56	6 600	11

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	Pol.%	W.B.C.	S.R.
2		120		102·2		28			
3	110	112	102·	103·2	28	32	80	15 400	77
4	112	112	101·8	102·4	28	30	82	13 800	103
5	94	82	100·5	100·	26	28	82	29 800	110
6	70	68	97·8	97·4	22	28		7 000	98
7	72	68	97·2	99·	20	20			
8	48	56	97·	97·6				8 400	70
9	50	52							
10	56	56							
12	48	54	97·2	97·				9 200	26

CASE 17
Lobar Pneumonia
Type 2
A.M. Aet. 21

CASE 18

Lobar Pneumonia Type 2.

J.L. Aet. 42.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	W.B.C.	S.R.
8	128	124	100.	100.2	32	36	22 400	112
9	130	136	100.	101.8	36	44	24 400	89
10	152	126	101.4	100.	44	48	24 400	86
11	120	130	96.8	96.	38	42	46 000	75

CASE 19

Lobar Pneumonia Type 2

J.M. Aet. 37.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	W.B.C.	S.R.
5	118	120	103.	101.	34	36	10 600	88
6	130	134	102.2	100.6	40	44	14 000	68
7	130	138	100.1	100.1	36	36	24 400	73
8	112	100	98.4	99.	34	28	15 400	67
9	104	116	98.8	99.8	32	28	13 200	48
11	96	98	98.4	99.	26	24	14 400	48

CASE 20

Lobar Pneumonia Type 2

E.W. Aet. 15.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	W.B.C.	S.R.
6	124	116	102.	100.6	36	38	13 400	72
7	124	130	102.	101.8	44	48	22 000	47
8	138	152	101.	100.5	44	44	45 600	36
9	120	132	98.	100.	40	60	41 000	49
10	104	108	98.	97.6	40	48		

CASE 21

Lobar Pneumonia Type 2.

J.N. Aet. 56

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	W.B.C.	S.R.
5	120	128	101.5	102.	28	32	29 800	103
6	124	126	102.	101.4	28	30	16 200	118
7	128	108	98.4	99.	36	32	16 400	123
8	96	104	97.	99.	24	28	12 000	120

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	W.B.C.	S.R.
4	120	124	99.8	99.	24	26	25 200	60
5	84	80	97.6	98.6	24	22	15 600	72
6	76	80	97.6	99.	22	20		
7	76	72	98.	98.4	20	20	7 600	60
8	70	60	97.6	98.	20	20		
9	60	76	97.	98.8	20	20	4 400	41

CASE 22
Lobar Pneumonia Type 2.
R.G. Aet.25.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	W.B.C.	S.R.
3	116	98	102.6	100.8	48	36	9 200	85
4	108	96	102.	100.2	34	36	9 000	115
5	96	92	99.	98.4	30	26	10 800	125
6	72	80	96.4	97.	24	20	7 600	120
7	76	80	98.8	97.	22	22		
8	78	80	98.2	98.	22	20	14 400	112

CASE 23
Lobar Pneumonia Type 2.
B.M. Aet 37.

CASE 24
Lobar Pneumonia Type 2
P.H. Aet. 25.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	W.B.C.	S.R.
3	128	112	103.8	100.	26	30	23 000	45
4	150	120	96.8	101.2	30	30	25 800	69
5	120	116	100.4	101.6	30	20	15 200	87
6	114	104	101.	99.6	28	26	13 400	85
7	100	120	100.6	100.	32	28	16 000	79
8	116	120	100.8	98.4	28	30	22 000	86
9	96	98	98.4	98.	24	24	23 400	89

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	W.B.C.	S.R.	
3	92	98	100.	102.2	20	26	122/66	25 400	36	
4	92	100	101.8	102.4	22	24	120/70	22 200	89	
5	88	88	101.	99.8	22	20	114/70	8 600	98	
6	76	76	99.8	99.	20	20	114/70	4 400	73	
7	82	100	99.	97.8	20	20	114/68	7 800	61	
27	68	70	98.1	98.2	20	20		6 400	6	

CASE 25
Lobar Pneumonia Type 2
J.J. Aet. 32.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	W.B.C.	S.R.
4	98	82	103.4	102.	26	24	21 600	82
5	86	70	99.8	98.8	24	22	15 600	88
6	72	60	100.	96.4	24	22	11 800	88
7	60	60	96.2	97.	20	20	8 000	58

CASE 26
Lobar Pneumonia Type 2.
D.M. Aet. 14.

CASE 27

Lobar Pneumonia Type 2.

R.F. Aet. 29.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	W.B.C.	S.R.
2		112		101.4		40	122/92	30 600	15
3	108	106	99.4	101.	36	32	114/80	18 000	37
4	94	78	100.2	99.	34	28	114/80	11 600	41
5	92	88	99.	98.4	28	24	114/82	18 400	47

CASE 28

Lobar Pneumonia Type 3.

W.W. Aet.37.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	W.B.C.	S.R.
4	112	100	103.8	101.2	28	28	118/68	13 600	91
5	100	108	100.5	101.2	24	24	110/68		
6	100	88	102.	100.	28	28	110/55	7 000	78
7	100	72	100.2	98.8	32	24	100/55		

CASE 29
Lobar Pneumonia Types 3.
W.B. Aet. 49.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	W.B.C.	S.R.
4	88	100	100.4	101.1	24	28	128/76	8 800	114
5	108	92	100.	99.4	28	28	138/70	10 800	98
6	84	88	99.	98.	24	24	136/80	18 400	79
7	80	88	98.	98.2	24	20	140/90	10 000	70

CASE 30
Lobar Pneumonia Type
W.W. Aet. 30.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R. 1.	S.R. 2.	S.R. 24.
5		104		99.4		40	104/70	84	14 000	135	-	149
6	100	80	98.8	99.8	34	22	94/58		10 800	140	-	153
7	100	84	98.	100.6	17	22	128/96	70	8 800	142	-	152
8	88	76	97.2	99.	20	20	90/66	72	9 400	139	144	150
9	84	88	97.6	99.4	20	26	138/96	92	15 600	136	-	150
10	84	92	97.6	98.	24	32	92/60		15 000	139	-	-
11	84	84	97.	99.	22	26	114/82	82	14 200	130	-	144
13	86	96	98.4	101.5	24	28		82	12 800	139	145	150
15	84	96	98.	99.2	22	24		90	13 800	123	144	150
17	92	90	98.	98.4	24	20	100/56		12 000	130	-	-
21	80	80	98.	98.	20	20			12 200	109	-	-
25	82	80	98.2	99.	20	20		76	11 600	138	-	-
27	80	74	97.2	97.2	20	20			15 600	138	-	-
29	80	96	97.4	97.6	20	20		88	10 000	114	-	-
31	88	82	98.	98.4	20	20		76	13 600	111	-	-
34	84	88	97.2	98.2	20	20	118/70		10 800	103	-	-
36	90	100	98.	101.	20	24			18 800	109	-	-
38	96	88	98.2	98.4	22	20			13 800	133	-	-
41	88	100	98.5	97.	21	21		60	11 400	99	-	-
44	92	80	97.4	98.	20	20	104/68	88	17 000	110	-	-
46	100	108	98.	98.	20	20		72	14 400	95	-	-
48	92		97.8		16			80	16 000	122	-	-

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	W.B.C.	S.R.	
5		112		100.8		28	22 800	57	
6	100	100	102.	100.6	24	26	33 600	57	
7	100	108	102.6	99.4	26	24	30 000	48	
8	80	76	97.6	97.8	26	24	28 000	37	

CASE 31
Lobar Pneumonia Group 4
W.W. Aet 53.

CASE 32

Lobar Pneumonia Group 4

A.M. Aet. 18.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	W.B.C.	S.R.	
4	100	118	100.8	101.4	20	30	112/64	23 000	42	
5	100	120	99.8	101.8	18	40	110/64	14 400	69	
6	110	128	102.2	103.	32	32	108/64	12 600	72	
7	120	120	100.8	101.	30	32		10 400	84	
8	128	120	103.4	102.	28	30	112/66	14 200	81	
9	128	122	103.	101.	40	30	112/70	23 200	76	
10	122	122	102.8	99.8	36	32	110/66			
11	90	88	98.8	97.8	28	22	108/66	16 200	48	

CASE 33
Lobar Pneumonia Group 4.
M.D. Aet. 19.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	W.B.C.	S.R
5	120	116	101.	102.	34	28	110/68	12 000	75
6	132	128	102.8	101.	36	32	106/68	12 000	64
7	120	100	100.8	96.	28	26	104/60	15 600	56
8	100	86	99.	98.	24	28	100/58	14 800	48
9	88	84	97.2	97.2	26	24		12 800	37

CASE 34
Lobar Pneumonia Group 4.
A.S. Aet. 58.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	W.B.C.	S.R.
6	120	122	101.	101.8	34	40	12 000	88
7	88	106	98.	99.2	28	38	23 200	94
8	96	100	98.	97.6	32	34	14 000	86

CASE 35

Lobar Pneumonia Type 2 DIED

DM. Aet. 56.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R. 1.	S.R. 2.	S.R. 24
3	88	132	102.8	102.8	36	34	75/50					
4	112	112	98.8	98.4	32	32	55/38					
5 DIED	118		98.8		36			84	4 400	56	84	97

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	W.B.C.	S.R.
5	144	142	104.8	102.3	42	48	134/94	12 800	82
6 DIED	?		106.2		48				

CASE 36
Lobar Pneumonia DIED
G.S. Aet. 26.

CASE 37

Lobar Pneumonia Type 2 DIED

A.T. Aet. 54.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R.
5		104		103.		22				
6	104	106	102.	102.6	24	36	110/60	70	3 000	111
7	108	112	103.2	102.	50	38	140/70		4 200	110
8	118	108	101.8	100.2	46	44	114/54	75	6 600	114
9 DIED	144		102.4		50		122/74	72	5 600	106

CASE 38

Lobar Pneumonia Type 3 - DIED

W.E. Aet 63.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R
5	112	114	100.2	101.2	40	40	110/70	88	19 600	62
6	112	112	100.4	101.4	36	32	95/70	80	21 200	56
7 DIED										

CASE 39

Lobar Pneumonia Group 4 DIED

P.B. Aet. 46.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	W.B.C.	S.R.
4	104	102	101.	102.	28	26	9 800	65
5	108	98	101.	99.6	32	36	6 800	70
6	108	100	99.4	100.2	28	32	10 200	55
7	108	128	98.4	100.4	28	40	6 600	46
8	120	124	98.8	98.4	32	44	7 800	35
9 DIED	156		98.3		42		8 200	21

CASE 40

Lobar Pneumonia Type 2 DIED

H.T. Aet. 24

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	W.B.C.	S.R.
2	114	110	101.6	100.	40	34		26 200	61
3	102	108	102.	103.	28	32	100/60	24 200	109
4	120	110	100.2	102.4	32	32	100/60	10 200	107
5	120	120	102.	100.6	32	38	96/66	13 400	109
6	124	108	102.8	100.8	38	32	104/66		
7	116	120	102.2	100.2	32	32	112/60	20 600	110
8	128	128	102.8	102.4	30	52	112/70	21 400	104
9 DIED									

CASE 41

Lobar Pneumonia Type 2 - DIED

W.C. Aet. 26.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	W.B.C.	S.R.
2		124		101.4		32	100/65	4 000	60
3	128	128	102.8	100.8	30	30		2 400	94
4	140	128	104.	102.	36	36		4 000	87
5 DIED	140	152	104.	103.4	36	48		1 800	77

CASE 42

Lobar Pneumonia Type DIED

G.R. Aet. 54.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	W.B.C.	S.R.
5	132	120	100.4	100.6	34	36	104/66	12 200	85
6	130	128	99.	99.6	32	32		16 800	75
7	124	124	98.8	101.	36	38	110/70	32 400	61
8 DIED	160		102.		42				

CASE 43

Lobar Pneumonia Type 2 DIE

J.T. Aet. 21

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	W.B.C.	S.R.
3		128		103.6		40		16 400	76
4	96	120	100.8	101.	32	30	120/72	15 000	115
5	104	128	101.	102.	26	30	130/70	7 000	116
6	116	124	102.2	103.4	38	38	128/72	5 000	105
7 DIED									

CASE 44

Lobar Pneumonia Type 2 DIED

G.M. Aet. 29

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	W.B.C.	S.R.
2	104	104	102.8	101.8	26	28	126/68	23 400	19
3	112	100	102.	101.2	24	22	114/70	13 000	34
4	96	104	102.	101.4	20	24	104/60	12 600	41
5	120	100	101.6	100.	36	32	110/74	5 600	35
6	132	120	101.2	100.6	32	36	84/54	7 000	42
7	108	120	99.8	101.	28	36	120/66	9.800	34
8	140	120	100.	100.8	44	40	132/68	10 000	21
9	DIED		99.8		48				
	172								

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R.	
4		86		100.6		32	112/70	88	13 400	24	
5	80		97.8		24						

CASE 45
Diphtheria
S.R. Aet. 27.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P	Pol.%	W.B.C.	S.R. 1.	S.R. 2.	S.R. 24.	
5		104		101.7		36	118/78	77	4 800	39	-	121	
6	160		105.6		48								

CASE 46
Septicaemia.
E.R. Aet. 37.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R.	
2	76	82	96.2	98.2	20	20	118/70	65	7 600	7	

CASE 47
Spontaneous Pneumothorax
L.M. Aet. 25.

CASE 48

Spontaneous Pneumothorax

J.W. Aet. 28.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R.
1		66		96.6		20				
2	66	66	97.	97.8	20	20	118/80	56	5 400	4
3	68		97.6		20					

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	Pol.%	W.B.C.	S.R. 1.	S.R. 2.	S.R. 24
3	88	96	99.2	99.4	20	32	67	10 200	41	-	132
11	78	82	97.7	98.8	24	30		4 200	35	70	126
15	80	96	98.	99.6	26	32		9 400	30	-	134
19	80	94	97.	98.8	20	20	56	8 600	36	-	-
23	86	92	98.4	100.	22	28	66	4 000	34	-	-
28	84	100	97.6	99.	28	30	64	4 600	42	-	-
32	88	92	98.2	98.2	18	22		9 200	44	-	-
35	92	102	98.	99.4	22	24		7 800	41	-	-
50	92	98	98.8	99.8	20	28	60	5 600	45	-	-

CASE 49
Bronchial Carcinoma
P.S. Aet.21.

CASE 50
Pulmonary Tuberculosis
J.M. Aet. 27.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R. 1.	S.R. 2.	S.R. 24
44	128	140	102.	102.2	40	46	118/80		14 000	69	107	138
45	120	110	100.2	100.	34	36	116/78	70	13 200	87	126	138
46	114	102	98.6	100.8	32	32			8 400	83	-	138
47	96	102	98.4	99.8	30	32		78	8 200	76	116	140
48	96	106	99.	100.2	34	38	106/74		8 000	66	-	-
49	104	110	98.	99.8	32	34	116/70		7 800	57		
50	102	102	99.2	99.4	36	38	110/80		8 200	54		
52	102	106	99.2	101.	28	28		88	7 400	54		
54	120	110	101.4	101.2	28	26			17 200	59		
56	108	120	99.	98.8	26	28		86	11 200	68		
58	100	110	99.3	99.2	26	28		76	9 800	57		
60	102	110	98.4	97.2	26	24		76	9.400	42		
62	110	106	98.4	98.6	24	24		88	8 800	40		
64	96	98	98.4	97.6	22	24			7 000	40		
67	102	124	98.5	100.2	22	26			7 200	48		
71	108	104	98.6	99.4	22	30		76	9 200	59		
73	114	112	98.2	99.2	26	24		88	4 800	57		
75	108	108	98.5	98.5	26	28		70	5 400	50		
78	110	116	96.8	98.	22	22		76	7 800	54		

CASE 51
Pulmonary Oedema.
J.D. Aet. 43.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R.
1	120	90	97.4	98.6	30	26	150/100	84	9 400	77
2	96	88	96.	97.4	24	26			12 400	85
3	90	86	98.	97.	22	20	170/90	80	11 000	102
4	84	90	97.2	98.	20	20				
5	83	90	97.4	97.	20	20		40	7 800	90
6	92	88	97.	97.	20	20				
7	88	88	97.	97.	20	20	164/100	66	11 000	109
8	80	84	97.	97.6	20	20	164/100			
9	78	78	97.2	97.	20	20		74	7 200	83
10	84	76	97.	97.	20	20				
11	88	84	97.	98.2	20	20		58	8 600	76
13	84	84	97.	98.4	20	20			6 000	68
15	82	78	97.	96.8	20	20				
16	80	88	96.8	97.6	20	20			6 800	68
20	82	80	97.6	98.	20	20	170/112	64	7 000	72
22	80	84	97.6	98.	20	20			6 800	64
24	80	80	97.8	98.	20	20		70	6 800	72
26	78	82	97.6	98.	20	20	174/100	66	5 400	60
28	76	78	97.8	97.8	20	20			7 000	54
30	80	82	97.2	98.	20	20		56	7 400	54
32	82	86	97.6	98.4	20	20	124/90	36	7 800	58
34	84		97.8		20	20	132/90	64	6 800	60
								76	7 400	53

CASE 52
Broncho-Pneumonia
J.K. Aet. 43.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R. 1.	S.R. 2.	S.R. 24
14	124	112	99.4	99.6	28	26	100/62	78	5 600	97	115	128
15	100	110	99.	99.	28	28	138/88	78	8 200	74	-	120
16	112	96	98.8	98.	24	22	104/55	80	9 000	71	111	124
17	108	96	98.4	98.4	24	22	106/58	82	11 200	52	84	116
18	88	74	98.2	97.	22	20	98/54	70	12 400	52	84	112
19	76	80	97.2	97.	22	20	102/54	62	9 200	37	70	110
20	80	76	97.	97.	22	20	112/54	80	5 600	30	63	112
21	78	76	97.	97.4	20	20			6 600	24	58	96
22	72	76	97.	97.	20	20	96/30		3 500	22	53	93
23	72	68	97.4	97.2	20	20	90/38	68	9 600	23	52	108
24	66	72	97.	97.2	20	20			4 400	19	44	107
25	70	70	97.4	97.2	20	20	98/46	70	8 800	16	42	110
27	72	70	97.	97.4	20	20			5 800	21	51	112
28	70	70	97.	97.	20	20	98/48	54	5 400	18	42	113
29	70	68	97.	97.	20	20			5 600	20	47	105
30	70	72	97.	97.	20	20	102/56	66	7 600	18	43	114
32	70	80	97.2	97.6	20	20	98/56	56	8 600	20	47	-
34	68	68	97.	97.	20	20	128/82		9 800	19	47	111
39	64		98.4		20		115/70	42	9 200	23	53	112

CASE 23.
Broncho-Pneumonia
W.H. Aet.30.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	Pol.%	W.B.C.	S.R.
5		92		102.4		32			
6	92	96	100.2	103.	28	32	70	12 400	85
7	82	96	99.6	103.2	28	32			
8	84	104	98.4	102.2	28	28			
9	82	80	99.	99.	32	32	78	10 200	101
10	100	104	103.	97.	30	28			
11	80	88	97.6	98.4	28	28	76	12 600	114
12	88	90	97.6	98.4	28	30		15 400	98
13	86	90	98.4	100.	28	36	84	14 400	107
16	86	84	97.2	97.	32	38	86	15 200	102
18	92	84	99.6	97.	24	24	84	13 200	97
20	80	78	97.2	97.4	22	22	86	11 000	87
22	80	80	97.	98.	20	22	92	13 200	66
24	76	80	97.2	97.	20	20	82	12 600	65
26	80	76	97.	97.6	20	20	88	10 600	46
28	74	84	97.2	97.2	20	20		14 200	53
30	80	80	97.	98.	20	20		8 000	33
31	80	90	97.2	97.4	20	20	68	11 800	34
32	80	86	97.	97.	20	20		10 200	27
33	80	88	97.	97.2	20	20		11 600	31
35	82	84	97.	97.4	20	20	72	7 200	26

CASE 54
Broncho-Pneumonia
T.K. Aet. 53.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R. 1.	S.R. 2.	S.R. 24
11	116	120	101.2	102.5	32	26	145/102	83	9 200	53	82	94
12	100	84	99.6	101.	24	26	125/85	90	8 000	64	96	122
13	84	86	98.4	99.4	24	28	118/86	83	9 200	60	91	108
14	88	76	98.	99.6	24	24	135/90	76	13 400	47	79	110
15	84	72	97.8	98.4	24	24	102/76	76	9 400	35	71	101
16	78	68	97.4	97.6	24	24	140/98	74	16 000	33	63	109
17	80	70	97.	98.4	20	22	148/105	76	10 400	34	67	115
18	68	72	97.6	98.4	20	20	152/102		8 600	31	66	117
19	88	72	97.2	97.2	20	22	85/55		4 000	37	67	120
20	84	68	97.	96.8	20	20	110/76	64	6 600	36	67	112
21	84	72	96.4	96.8	20	20			7 600	31	67	116
22	84	76	96.8	97.4	20	20	116/86	50	5 000	31	67	118
24	76	78	97.	97.	20	20			7 600	35	70	122
25	78	80	96.6	97.	20	20	116/88	66	10 200	44	79	126
26	92	80	96.4	97.	20	20	100/ 74		7 200	48	85	129
27	88	80	97.2	97.	20	20		66	10 000	50	87	135
29	84	80	96.4	97.2	20	20	112/78	59	8 600	52	89	134
30	84	80	96.8	96.	20	20			8 800	49	-	129
31	68	76	96.4	97.	20	20			8 600	54	94	130
32	72	72	97.	96.6	20	20	104/70	72	9 400	50	-	-
36	88	84	96.4	96.8	20	20	106/72	60	6 800	46	88	135
58	75	76	97.	97.2	20	20		68	6 800	25	-	-

CASE 52
Broncho-Pneumonia
P.B. Aet. 53.

Day of Illness	Pulse M.	Pulse E.	Temperature M.	Temperature E.	Resp. M.	Resp. E.	B.P.	Pol.%	W.B.C.	S.R.
1	112	102	100.2	101.2	28	30	110/75	88	5 200	19
2	100	100	98.4	99.4	26	24				
3	96	94	98.	98.8	24	28	114/78	80	14 200	90
4	96	88	98.5	97.8	26	28		72	10 200	97
5	98	88	97.	98.	26	24	112/70			
6	84	78	97.2	97.6	22	22		68	5 400	75
7	76	84	97.8	97.5	24	24				
8	80	72	97.4	98.2	22	22	120/70	68	4 600	58
9	76	72	97.6	96.4	20	20			4 200	47
10	72	70	97.8	97.8	20	20				
12	76		97.		20			80	6 600	29

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