The Blood Sedimentation Reaction in General Practice, with Special Reference to Chronic Chest Diseases, particularly amongst Coal-miners.

presented as a

Thesis for the degree of M.D.

by

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This investigation was undertaken in order to ascertain the value of the Erythrocytic Sedimentation Reaction to the general practitioner in the diagnosis and prognosis of disease.

The work was carried out in the mining district of Shotts, Lanarkshire. During the past fourteen months 370 cases were examined and 804 individual sedimentation tests were made. Various diseases met with in general practice were studied, not only to estimate the significance of the sedimentation reaction in these conditions, but also to ascertain the pathological factors which influence the rate, e.g. it was found that asthma and other allergic conditions tended to retard the rate and so counteracted the accelerating effect of other diseases.

Particular attention was given to the study of chronic chest affections, owing to their high incidence locally, especially amongst coal-miners. In addition to other examinations (clinical, skiagraphic, sputa, and blood Wassermann reaction), the blood sedimentation reaction, hereafter named the B.S.R., was carried out in each case at regular intervals.

History.

Fåhraeus (2) states that blood-letting was practised from time immemorial up to the middle of last century, not only in the treatment of disease, but also as a general preservative/

preservative for those in health.

As long ago as the second century, Galen observed that when blood was withdrawn from a patient and allowed to stand, it would settle into two layers, an upper clear one and a lower solid one. The evacuated blood in many diseases was found to secrete a whitish substance of solid consistency in a layer above the clot. This substance was the so-called Crusta Sanguinis or Buffy Coat, and was not found in the blood taken from a healthy person. In 1771 Hewson (3) showed that when the coagulation of the blood is delayed, as by cold, neutral salts or otherwise, a coagulable plasma can be separated from the corpuscles and skimmed off the surface. Coagulation, in Hewson's view, was due to the formation in the plasma of this insoluble substance which he called "coagulable lymph", and which is now known to be fibrinogen. He was the first to show that the Buffy Coat consisted of fibrin and that the sinking speed of the erythrocytes was much greater in plasma than in serum from the same blood.

It was well known to the barber surgeons that blood taken from different patients would settle more quickly in one case than in another. In 1786 Hunter (4) said: "Indeed the/

the power of coagulation in the blood throws so much light on the nature of a disease, so far as the blood is concerned, that it is almost the only part we have recourse to in the examination of the blood after bleeding, when we look to see whether or not the blood is buffy.".

Two kinds of coat were noted: a small greyish white one, called the Crusta Inflammatoria, found in inflammatory conditions, and a loose bluish green one, the Crusta Gravidarum, found in pregnancy. In 1828 Blundell (5) wrote: "When women are pregnant the blood is more or less sizy, so that when you take away two or three ounces from the arm, the size on the surface of the crassamentum forms a valuable indication of pregnancy".

In 1918 Fåhraeus (1) noted an increase of the sedimentation rate of the red cells in citrated blood of pregnant women. At first, this was thought to be a new test for pregnancy, but it was soon found to be unreliable in the early months, when its value would have been greatest.

Theory.

Fahraeus (1) called the sedimentation rate the "Suspension Stability" of the blood. From his researches, he concluded that sedimentation is caused by an increased haemagglutination, the red cells when agglutinated falling down. In 1921, he (2) stated that "the degree of agglutination is

chiefly/

chiefly dependent on the properties of the plasma (fibrinogen, serum globulin, serum albumin). The globulin increase, i.e. an alteration in the colloidal state of the plasma, is the most important cause of the increased rouleau formation and of the increased sinking velocity of the red corpuscles, viz. to the reduction of the suspension stability of the blood". Gilligan and Ernstene (6) observed a close relation between the plasma fibrinogen and the sedimentation rate of the erythrocytes. In certain cases of liver damage, however, they found that the rate was increased out of proportion to the plasma fibrinogen. Aldred Brown and Munro (7) concluded from their investigations that the sedimentation rate had no connection whatsoever with the fibrinogen, globulin, or albumin of the plasma. They bring forward two theories on the nature of the reaction: 1/. that the agglutination of the red cells is caused by an adsorption phenomenon resulting from the lowered electrical burden of the suspended particles, and thus the sedimentation rate depends on alterations in height of the electrical burden of the particles; 2/. that the rate is controlled by the viscosity of the blood. Rossier and Basto (8) also believe that the acceleration of the B.S.R. is due to decrease of the electrical negative charges of the erythrocytes. This diminution of the electrical charges appears to be due to variations in the state of dispersion of the plasma/

plasma colloids. According to Griffin (9) variations in fibrinogen content, globulin, and the cholesterol-lecethin ratio are factors in influencing the B.S.R., probably by changing the electrical potential of the cells.

Stockes' law (10) states that "the sedimentation velocity of the corpuscles in a suspension of globular elements in fluid is proportionate to the square of their radius". Stockes' formula (10) is given as follows :

$$\frac{\frac{4r^3\pi}{3}(\varrho-\varrho_1)g}{6\pi\eta} = \frac{2}{9}\cdot\frac{r^2(\varrho-\varrho_1)g}{\eta}$$

r = radius of cells
ϱ = their specific weight
ϱ_1 = specific weight of the fluid
η = viscosity of the fluid
g = force of gravity.

However, as pointed out by Reichel (11), the formula takes no account of the electrical charges, nor of the friction of the cells which are not little round balls. He holds that the red blood cells have electric negatively charged potentials and keep themselves in suspension through repelling one another. He observed that in quickly sinking blood a clump of erythrocytes consists of 58,000 cells, and grows no more from the moment when the sinking rate is constant.

In spite of the many theories regarding the suspension-stability of the blood, it is generally agreed that the erythrocytes aggregate into clumps, that there is something in the plasma which is responsible for the agglutination, and that the number of red corpuscles influence the rate, the fewer/

fewer the corpuscles, the faster the sedimentation rate. According to Friedman (12), a method for correcting the B.S.R. for variations in cell volume percentage of blood has been devised by Rourke and Ernstene. The employment of the method was not found practical in this investigation, but where the B.S.R. was accelerated the Haemoglobin percentage was estimated by the Tallquist Scale. It is generally agreed that a Hb. content above 70% has little or no effect on the rate.

The white blood cells sediment more slowly than the red, and form a thin white ring between the plasma and the sedimented red cells. The leucocytic sedimentation reaction does not seem to be in use, as its practical diagnostic value is not yet established. Perhaps it may be useful in the diagnosis of leukaemia.

Technique.

Three principal methods are in use for estimation of the B.S.R. :

1/. The method of Westergren (13) in which the distance the red cells fall in a given time is measured. Readings are taken at the end of 1, 2 and 24 hours. The normal readings at the end of the first hour are 1 to 3 mm. for men and 4 to 7 mm. for women and children. I have seen this method used in hospitals, but do not consider it so useful for general practice. 2/.

2/. The graphic method of Cutler (14), by which the rate of sedimentation is measured at frequent intervals (every five minutes for one hour) in order that a curve may be drawn. The observations are recorded on charts, in which the horizontal lines represent the divisions of the tube, and the vertical lines the intervals of time. Four types of graph are recognized: - the horizontal line alone being normal; the diagonal line, the diagonal curve and the vertical curve always indicating abnormal findings in different degrees of intensity. This method is much in use in the United States of America.

3/. The method of Linzenmeier (15), in which the time required for the plasma to reach the mark 18 is noted. Normally this takes from 250-300 minutes. The obvious disadvantage of this method is that one must pay particular attention in order to avoid missing the reading at the appointed level. Greisheimer (16) states that the average sedimentation in one hour for "normal" subjects appears to be reasonably concordant for the three methods, despite the wide differences in tube width, anticoagulant concentration, and the length of fluid column, although the differences between the means for the three methods are significant statistically.

A modification of Linzenmeier's method, as recommended by Lafont(17), Scott (18), and Huie-Lan Chung (19), was used in/

in this investigation, owing to its suitability for general practice.

Tubes 3-4 mm. in diameter and 6·5 cm. high, with a mark 0 indicating the level of 1 c.c. of fluid were used. Below the 0, marks are made at 6, 12, 18 and 24 mm. respectively, with subdivisions at every 3mm. 0·2 c.c. of 3·8% sodium citrate solution, as recommended by Westergren(13), was drawn into a 1 c.c. syringe, preferably a Tuberculin one, owing to its greater accuracy. 0·8 c.c. of blood, taken from a vein in the antecubital space, was added. A number 17 stainless needle with a short point was found to be most suitable. The citrated blood was mixed by inverting the syringe several times after drawing in a bubble of air. The mixture was then placed in a tube and reached the mark 0. The tube was fixed VERTICALLY in a stand, and at the end of one hour, the upper level of the sedimented red cells was noted.

The following precautions were taken: the room temperature was 60-65°F., as cold was found to retard the rate, and warmth to increase it; the test was repeated if the blood were found to clot; a tourniquet, if applied in obese patients, was only allowed to remain on for a few minutes, otherwise the CO_2 of the blood would have increased and caused a slowing of the rate; the citrated blood was not placed in the direct sunlight./

sunlight. One is supposed to avoid doing the test during the menstrual period of a patient. I found that it did not make much difference to the rate. Lenzi (20) advises that the blood should be taken from patients before breakfast, i.e. while they are still fasting, but this was not found to be practicable.

Forty apparently normal individuals were tested in order to ascertain the normal values for each sex at different ages. The following Table indicates the result.

Males.		Females.	
Age (years)	B.S.R.(mm.)	Age	B.S.R.
14	2	12	3
19	3	15	3
21	2	16	3
21	3	17	4
22	1	24	3
24	3	28	4
26	1·5	28	3
26	2	29	7
27	2	31	3
32	1	35	3
32	0·5	35	5
40	4	38	2
44	2	39	3
46	2	40	4
52	3	40	3
54	3	51	4
57	1	53	7
58	1·5	55	5
60	2	60	3
79	4	67	8
average 38	2·2	36	4 .

It will be observed that 2 mm. (roughly) is the average B.S.R. per hour in males, and that the rate may vary from 1 to/

to 4 mm. In my opinion, a rate of 0·5 mm. is subnormal. In females the average is twice that of the males and the range varies from 3 to 8 mm. A reading of 2 is probably subnormal.

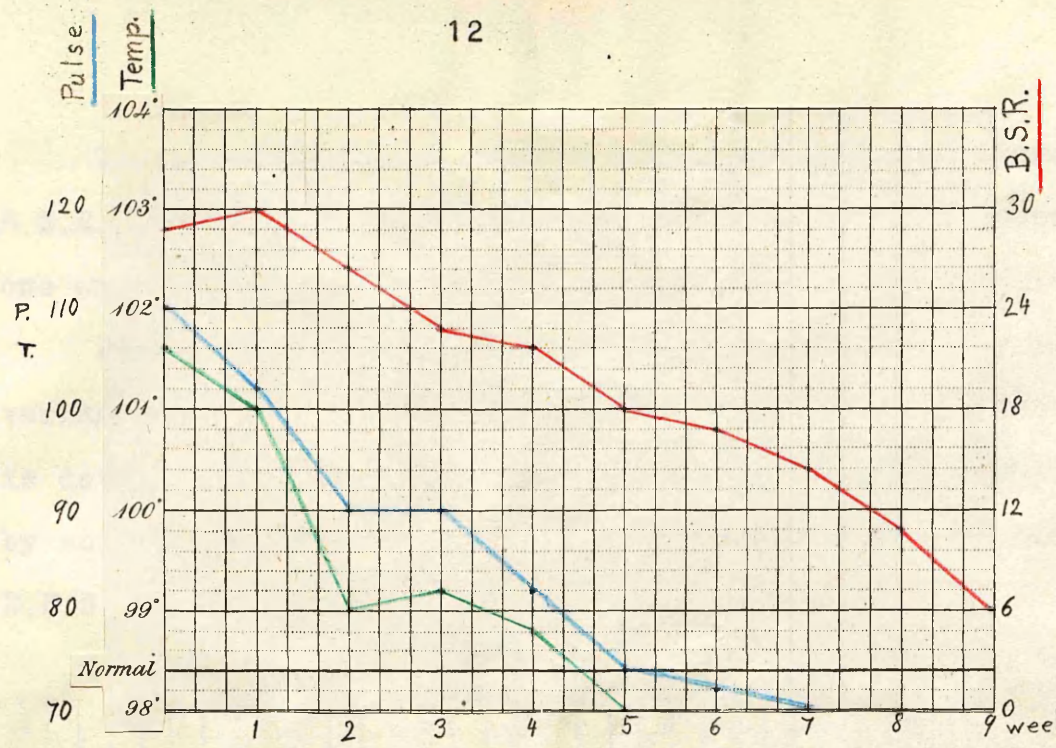
Dr. Scott (18), who uses the same method, informed me that in females the rate lies between 3 and 8 mm., but may be as great as 12 mm. at times.

The B.S.R. in Rheumatic Conditions.

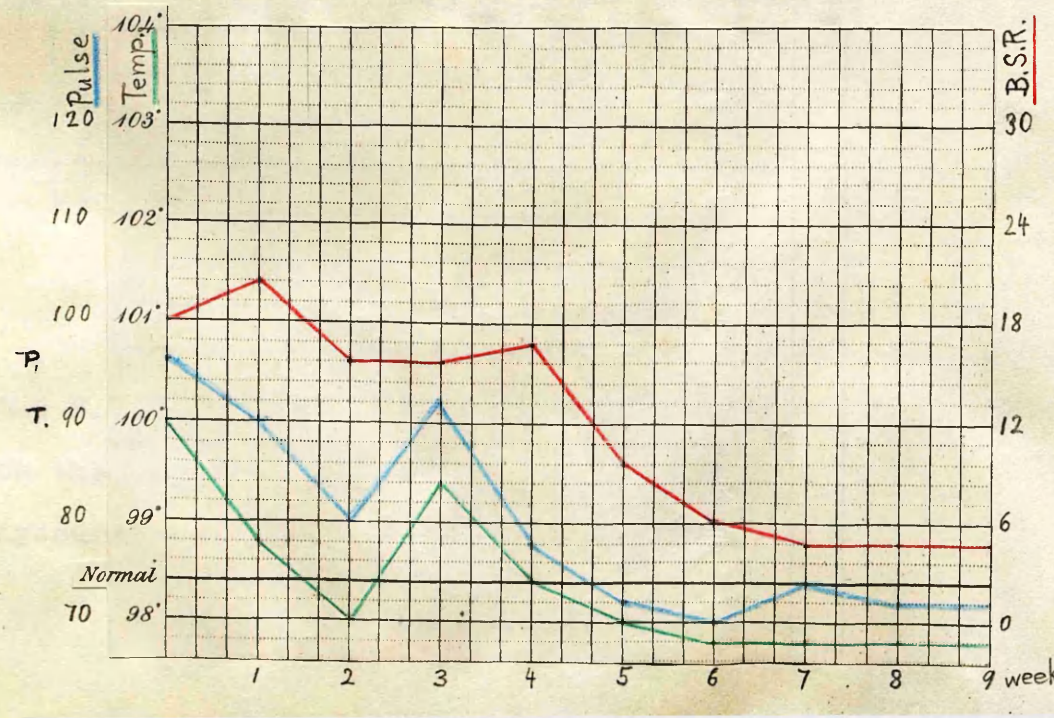
The test was found to be helpful in the diagnosis of articular and cardiac rheumatism. The rate was accelerated in all active cases and returned to normal after the acute or subacute symptoms subsided. Nine cases of subacute rheumatism occurred in my practice during the past fourteen months, six being adults and three children. It is difficult to account for the paucity in numbers, as it is generally known that rheumatism, especially the juvenile type, is so prevalent. Perhaps, better housing conditions may have contributed to this improvement. On the other hand, tonsillitis and influenzal chills were common. In these illnesses, salicylates (aspirin, etc.) were given not only during the active stage, but were continued for ten days or even longer. This was in accordance with the teaching of Collis (21), who advises that all cases of acute haemolytic throat infection should/

should be given large doses of aspirin, combined with a double quantity of sodium bicarbonate, for not less than ten days after the appearance of throat symptoms, in order to prevent or minimise the subsequent rheumatic manifestations, especially in already rheumatic subjects. He found that"when naso-pharyngeal infections occurred with haemolytic streptococci, a certain sequence of events subsequently took place. The naso-pharyngeal symptoms would clear up, the child apparently recovering, then after a silent period of ten to twenty days, a sudden acute rheumatic relapse would occur." Collis holds that these patients are allergic to certain products of the streptococcus (usually haemolytic) and that the "silent or incubation period" is in all probability an anaphylactic phenomenon. However, Wilkinson (22) considers that the finding of characteristic changes in the joints is in opposition to the allergic theory.

The following chart illustrates the course of the B.S.R. in a case of subacute articular rheumatism (female, aet. 32 yrs). It will be noted that the temperature and pulse-rate returned to normal before the B.S.R. Patient was confined to bed until the latter reached 6mm.

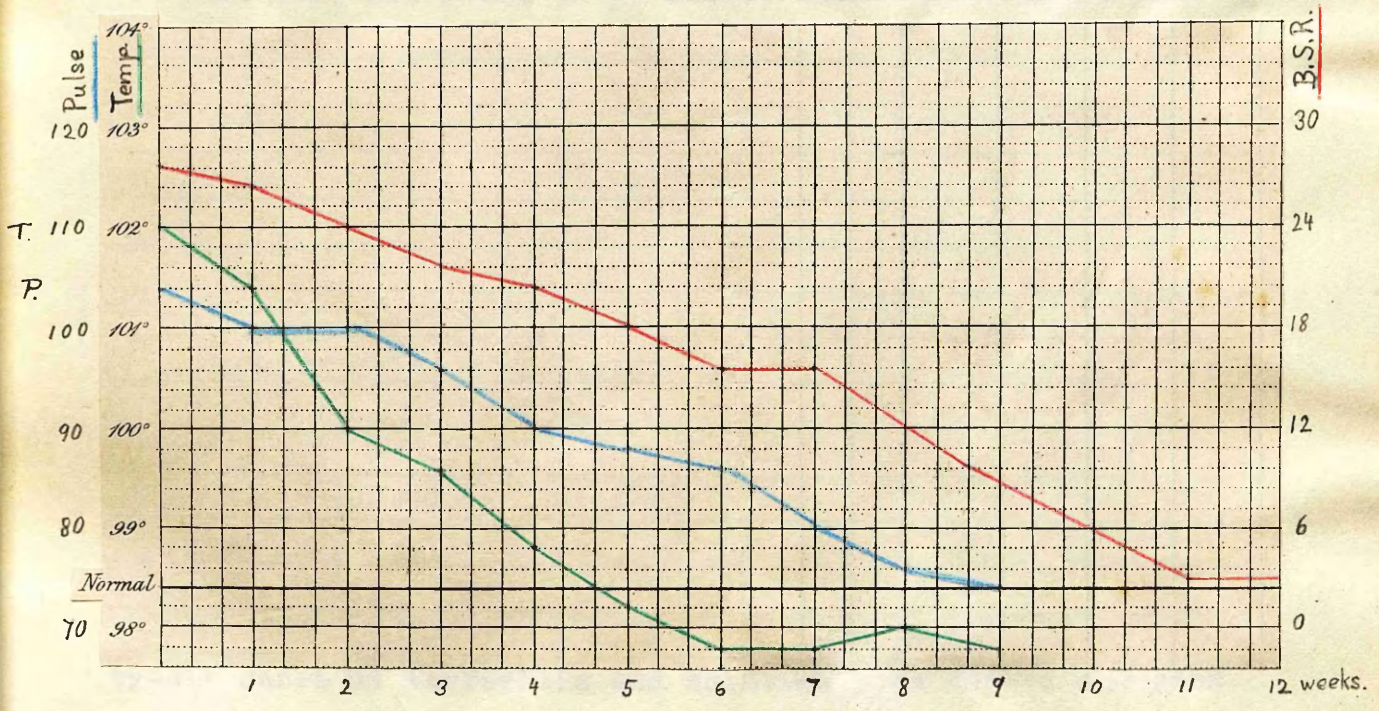


In the next case, patient (female, aet.36) rose from bed with the disappearance of the articular pains and temperature at the end of the second week, but with a raised B.S.R. One week later, she was again confined to bed, due to a recrudescence of the subacute rheumatism.



The continuation of infection, as shown by the increased B.S.R. after the first attack, indicates that the subsequent one was not in the nature of a true relapse.

Ritchie (23) states that in many cases apparently recovering from uncomplicated rheumatic fever, a latent carditis is developing. Two cases of cardiac rheumatism were observed by me. The following chart illustrates the course of the B.S.R. in one of them (male, aet. 14 yrs.).



The temperature returned to normal many weeks before the B.S.R. and the pulse-rate. The latter becomes accelerated on the least exertion, owing to the weakened state of the myocardium./

myocardium. The apex beat is in 6th interspace outside the nipple line and a mitral systolic murmur is heard. Patient was kept resting until the B.S.R. returned to normal, i.e. until all infection had gone.

From a study of the preceding cases, I am of opinion that the B.S.R. is useful in the diagnosis of rheumatic infection and that it is more sensitive in assessing the degree of infection than are the temperature and pulse-rate.

The test was found to be little influenced, if at all, by dental caries. In one case of chronic rheumatism, a molar tooth which had been "crowned" twelve years previously, was skiagraphed and showed rarifying osteitis around it, due to apical infection. The tooth was extracted and yielded on culture the Streptococcus Viridans. The patient was not agreeable to have autogenous vaccine treatment, but in a few months the pains had gone.

Fibrositic conditions (lumbago, pleurodynia, torticollis, muscular rheumatism, etc.) were very common during the past winter, aggravated possibly by the inclement weather. Thirty-six cases of fibrositis and sciatica were tested and each gave a normal B.S.R. (3 to 6 mm.). This finding is important in that it assists one to differentiate true rheumatic infection, possibly manifested only by a mild febrile disorder or by "growing pains" in children, from inflammation of the white fibrous/

fibrous tissue, which is less serious.

The B.S.R. was found to be normal in three cases of acute chorea. Elghammer (24) recorded low readings in twelve cases and concluded that chorea was not a manifestation of an active rheumatic process, but was either the expression of the damage of previous rheumatic infection, or due to some cause as yet unknown.

In two cases of rheumatoid arthritis I observed accelerated rates (18, 20 mm.), but in two of osteoarthritis the rates were normal (7,4 mm.). The test is thought to be of value in differentiating the two conditions and in assessing treatment in a case of rheumatoid arthritis, where improvement shows itself by a lowering of the rate.

According to Aldred-Brown and Munro (7) the B.S.R. is slightly accelerated in gout and is within normal limits in fibrositis.

The B.S.R. in Phlebitis.

The test was employed by me in cases of phlebitis complicating varicose veins or pregnancy. The rate was found to be raised in cases of septic origin and normal in simple thrombotic ones. The finding conformed to the classification of Payne (25), who considers that there are two main groups of phlebitis in varicous veins: 1./ A purely thrombotic process of/

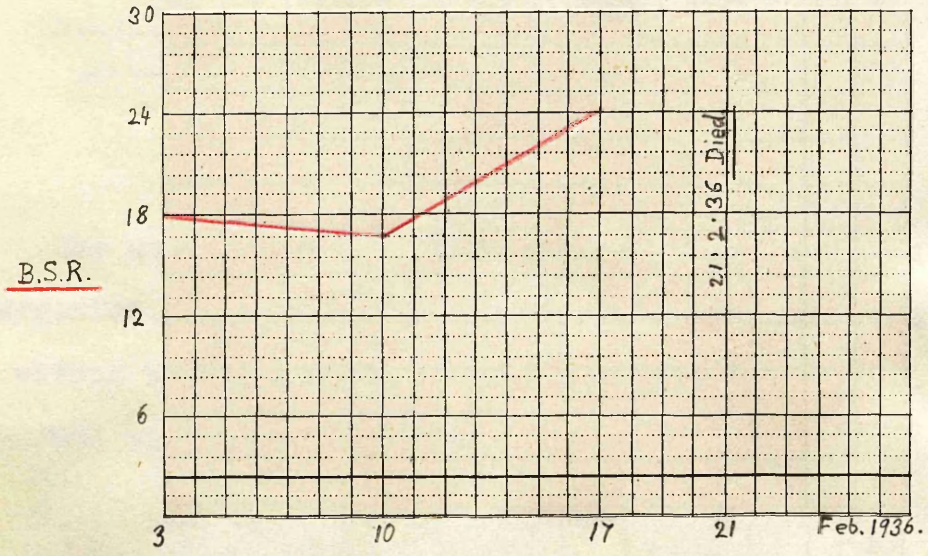
of non-bacterial origin,secondary to changes in the damaged endothelium,and in which local inflammatory changes are negligible and constitutional disturbance is absent. 2/. A septic bacterial phlebitis,characterized by a rapidly spreading septic thrombosis,associated with marked local inflammatory signs,together with fever and severe constitutional disturbance,and possibly with embolic phenomena.

Cases of phlebitis coming within an intermediate group are common in general practice. In such cases,particularly,I found the B.S.R.to be helpful. If the rate were normal,an Elastoplast bandage was applied to the limb and the patient allowed to walk about. If the rate were accelerated,a bandage was also applied,but the patient was confined to bed until the B.S.R.was at or near normal.

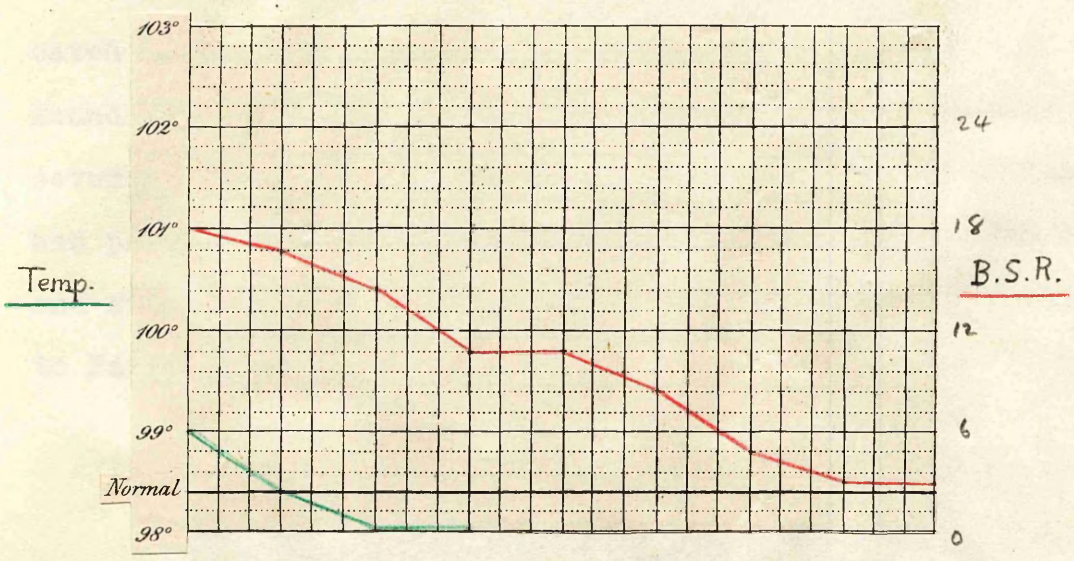
Payne advocates rest in cases of spreading septic phlebitis and says:"it is often found that arrest of the condition is not brought about,unless the patient is in bed." Dickson Wright (26),on the other hand,states;"I do not even regard pyrexia as an indication to lie up,and think that the more severe the superficial phlebitis,the more essential it is to keep the patient out of bed to prevent embolism and deep thrombosis."

The/

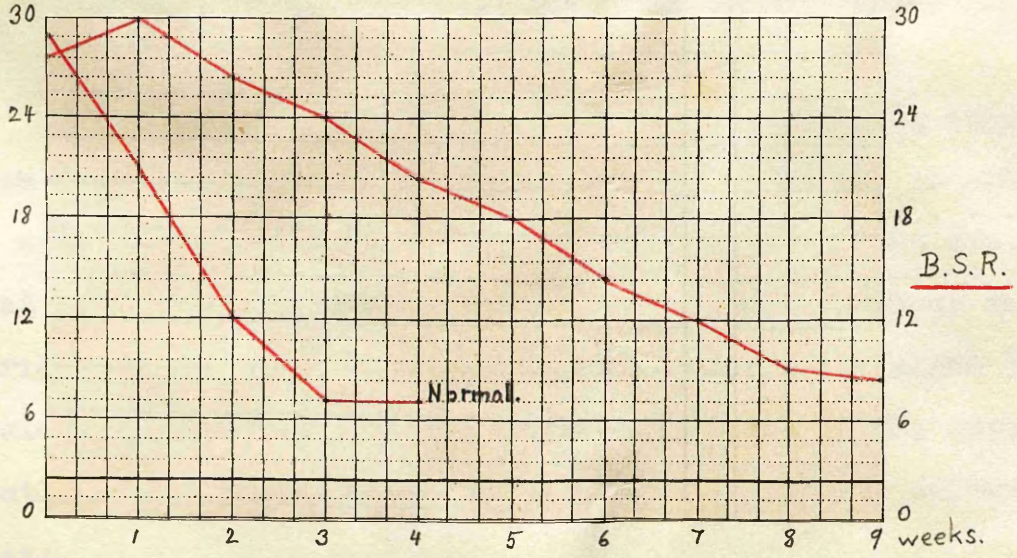
The following chart indicates the course of the B.S.R. in a case of septic superficial phlebitis under my care. Against advice, patient commenced to do housework, while the rate was still raised. Two days later (17-2-36), she had severe pain in right side of chest with dyspnoea and slight haemoptysis, due to pulmonary infarction caused by an embolus. On 21-2-36 cardiac complications developed and patient died.



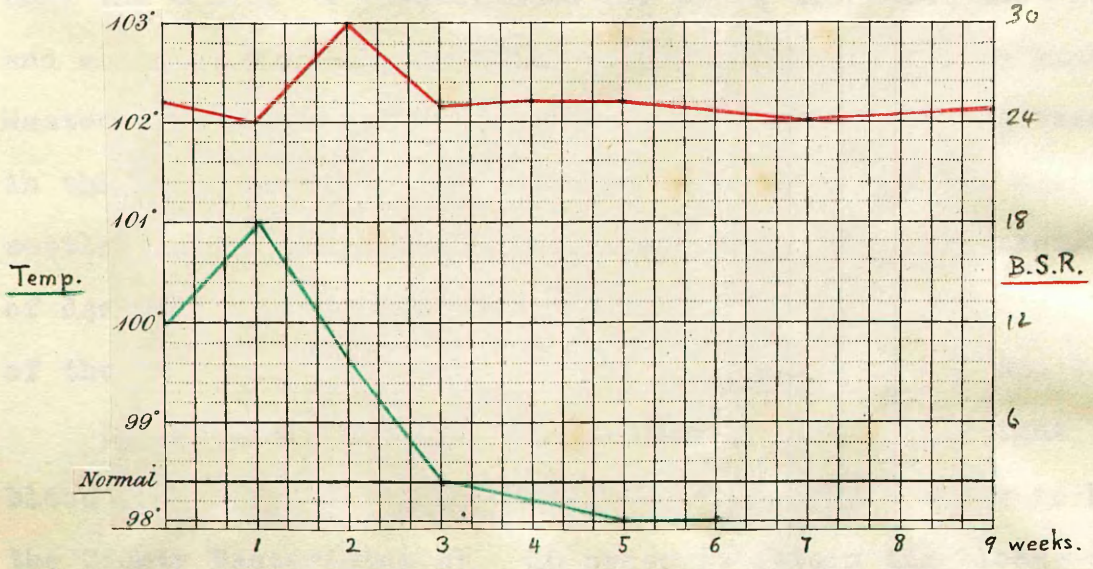
Eight cases of phlebitis were examined during this investigation. The next chart illustrates the usual B.S.R. curve in a case of superficial septic phlebitis complicating varicose veins.



The next chart is from a case of phlebitis during the puerperium. Patient was made to rest until the rate returned to within normal limits. For comparison, the B.S.R. curve in a normal puerperium is given.



In one case of infective phlebitis of the leg complicated by abscess formation (staphylococcal), the B.S.R. was found to remain high even after the limb had completely recovered. This was accounted for by the fact that patient had paratyphoid infection three years previously. The blood and stools were again examined and gave positive reactions to Paratyphosus B.



The test was employed by me prior to commencing injection treatment of varicose veins in four patients, in order to exclude the presence of sepsis. Biegeleisen (27) states that it is possible to get so-called latent infections in varicose veins which have practically no physical signs that would help in the diagnosis. According to him, many apparently normal varicosed extremities harbour latent infection that/

that may be aroused by chemical irritation, e.g. injection treatment. The test is especially valuable before commencing treatment in a case, where there is a history of recent phlebitis. Pennoyer (28) states that emboli can usually be traced to cases, where the injections have been given in the presence of a pre-existing phlebitis.

Biegeleisen observed that varicose ulcers do not influence the B.S.R. I investigated six cases and found the rate and also the blood Wassermann reaction to be normal in each. Masten (29) says that "the presence of a destructive process in the body increases the rapidity with which the red cells settle". Perhaps it would be better to add that the products of destruction must be carried into the blood-stream, in view of the fact that varicose ulceration does not affect the B.S.R.

During the course of this investigation thirty-eight blood Wassermann reactions were kindly carried out for me by the County Bacteriologist. In order to obtain the blood, the following procedure was adopted: 2 c.c. were withdrawn from the Median Basilic Vein. Into a B.S.R. tube containing 0·2 c.c. of 3·8% sol.sod.citrat. sufficient blood was added to bring the fluid to the mark 0. The remainder of the blood was placed in a B.W.R. tube and sent for examination. Thus, the one withdrawal of blood sufficed for the two tests.

The B.S.R. in Diseases of the Heart.

The following Table indicates the rates in cases under my care:

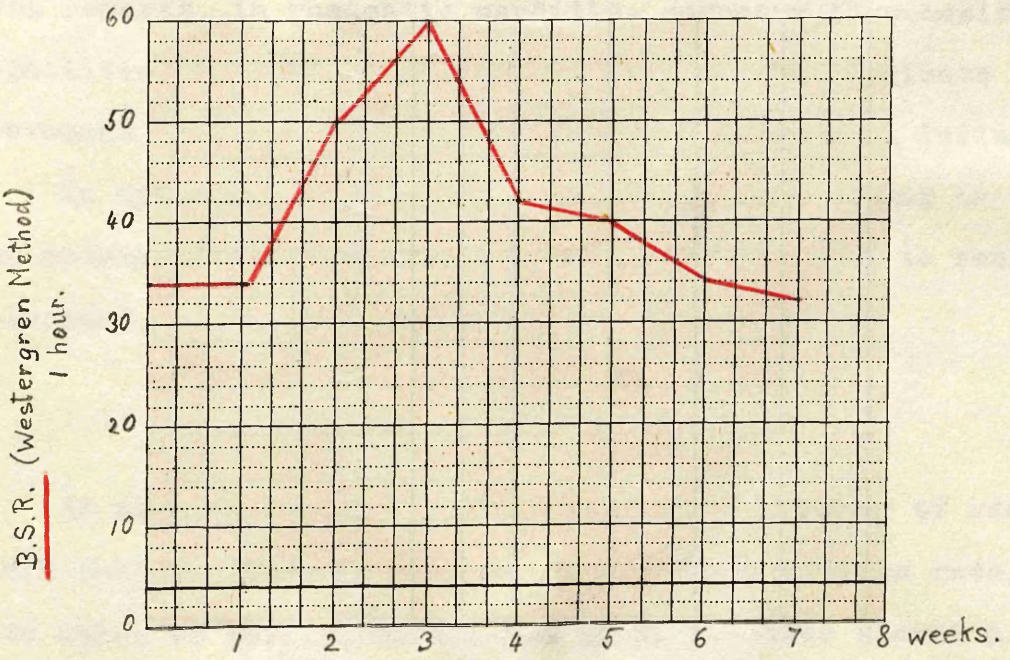
Disease.	No. of cases.	B.S.R.
Valvular disease (mitral)	4	3,1,3,4 mm.
Auricular fibrillation	2	4,2 " .
Persistent tachycardia	2	1,1 " .
Myocardial degeneration	4	1,2,2,1 " .
Congestive heart failure	2	0·5, 1 " .
Rheumatic carditis	2	28, 22 " .

It will be noticed that the readings are within normal limits, except in one case of congestion, where the rate was subnormal, and in two cases of rheumatic carditis, where it was much accelerated.

According to Wood (30) increased rates are found in active rheumatic carditis, syphilitic aortitis and myocardial infarction. In one case of syphilitic aortitis with a negative blood Wassermann reaction after treatment, I found a normal reading (4 mm.).

Wood observed that the test is useful in the prognosis of coronary thrombosis. He states that the rate is not raised immediately, but only after a day or two, and then increases steadily to a maximum at the end of the third week, after which it/

it slowly returns to normal in six to eight weeks, or it may remain at a slightly increased level. It is held that the B.S.R. may be of value in the diagnosis of coronary occlusion, e.g. when a patient is not seen until some time after an attack ,and where the history leaves the diagnosis doubtful, especially if no electrocardiogram is available. The following chart by Wood illustrates the B.S.R. curve in a case of coronary thrombosis, the clinical diagnosis being confirmed by the electrocardiograph. The initial reading was taken the day after the thrombosis occurred.



The rate is said to increase during softening of the myocardium in the region of the ischaemia, and it decreases when the infarct is healing. Therefore, in such a case, the patient/

patient should remain in bed, until the B.S.R. is normal or on a slightly increased level. Bickel and co-workers (31) found the rate to be considerably accelerated in five cases of myocardial infarction and held that "the analysis of the serum proteins suggests that the rapid sedimentation is due to an increased concentration of fibrinogen and is not correlated with the serum protein changes occurring in congestive heart failure".

It is important to notice that congestive heart failure retards the rate regardless of the pathology. Therefore, as Wood remarks, in rheumatic carditis, coronary thrombosis and syphilitic aortitis, a decreasing rate may not indicate improvement, but may signify the onset of myocardial failure.

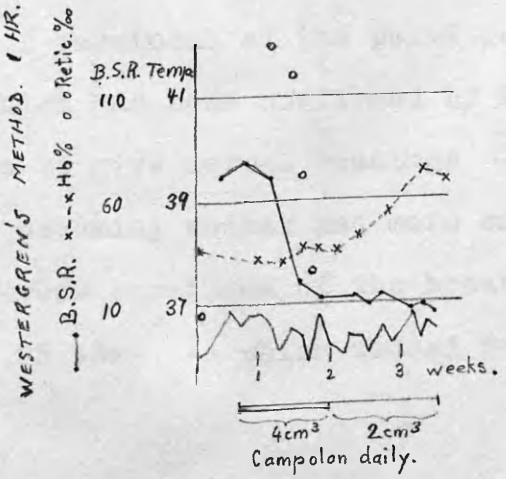
In hyperpiesis (6 cases), the B.S.R. was normal in three and moderately raised in the others, due possibly to renal involvement, although albuminuria was absent.

The B.S.R. in Pernicious Anaemia.

As already stated, a diminution in the number of red blood cells causes an increase in the sedimentation rate. Five cases of pernicious anaemia in my practice showed a high rate when the erythrocyte count was low, and gradually gave normal readings as the blood picture improved with liver therapy intramuscularly. The following data is from one of the cases.

R.B.C.	Hb.	C.I.	B.S.R.	W.B.C.
1,400,000	40%	1.4.	22 mm.	5,500.
3,210,000	65%	1.	15 mm.	7,000.
4,820,000	90%	.9	4 mm	8,400.

Reichel (32) states that a grave anaemia is always indicated if the B.S.R. is rapid and the upper limit of the erythrocyte column is hazy. He considers decrease of the rate a more important diagnostic factor of a remission than the reticulocyte crisis, for the latter may be overlooked unless numerous counts are made, and occasionally a crisis may occur without being followed by a remission. If, when liver therapy has been instituted, no decrease in the rate occurs, he assumes that the diagnosis of pernicious anaemia is wrong, or that the dosage of liver is insufficient. If, at first, a decrease occurs, but the rate does not become normal, a complication must be looked for. The following diagram by Reichel illustrates the course of the B.S.R. at the beginning of a remission in a case of pernicious anaemia.



The B.S.R. in Abdominal Conditions.

The test was not found to be of much assistance in diseases of the stomach or intestines. The rate was normal in two cases of gastric and two of duodenal ulcer. Lesser and Goldberger (33) state that the rate increases if the ulcer perforates. Normal readings were observed also in six cases of gastritis. Three patients suffering from chronic cholecystitis had accelerated rates -- 20,18,17 mm., two being subject to attacks of biliary colic due to gall-stones.

Liedberg (34) examined 145 patients suffering from acute cholecystitis and noted high rates in all of them. He found that the B.S.R. continued to rise even after the temperature was subsiding and concluded that the temperature was an index of the clinical condition of the patient as a whole, but that the B.S.R. was an expression of the local pathological changes. He states that it is better not to operate until the acute symptoms have subsided and advises repeated tests as a guide to the local and general condition.

One case of carcinoma at the pelvi-rectal junction, the diagnosis of which has been confirmed by X-ray examination, etc., continues to give normal readings -- 2mm. --, although the patient is becoming weaker and more emaciated. Three females with scirrhus carcinoma of the breast gave accelerated readings -- 24,28,18mm. -- which tended to increase as the disease/

disease progressed. In each case the lungs were affected owing to metastasis. Kessler (35) investigated the B.S.R. in one hundred cases of malignant disease which chiefly concerned the digestive tract. In 53% the rate was not so high that any importance could be attached to it in the differential diagnosis of malignant and non-malignant disease. He found, however, that seven out of eight cases of malignant disease of the lungs showed an accelerated rate. Cutler (14) also observed a raised reading in pulmonary carcinoma.

In four cases of acute appendicitis I obtained normal rates. I cannot state, what the B.S.R. is where the disease is advanced, as most of my patients are seen at an early stage and are sent to hospital as soon as diagnosed. Lesser and Goldberger (33) concluded from a study of two thousand cases of acute abdominal conditions that appendicitis, not complicated by abscess formation or generalized peritonitis, gives practically normal rates, whereas salpingitis, cholecystitis and inflammatory conditions of the genito-urinary tract have moderate to high rates. Smith, Harper and Watson (36) observed that the rate tends to increase in appendicitis after forty-eight hours, but is accelerated in salpingitis within twenty-four hours, because the infection has been there many hours before it began to cause symptoms.

The B.S.R. does not seem to be of practical use in the diagnosis/

diagnosis of abdominal carcinoma, but might be employed in differentiating other abdominal conditions, e.g. where the rate is normal, salpingitis, cholecystitis and inflammatory conditions of the genito-urinary tract may reasonably be excluded, whereas with a raised B.S.R. gastric or duodenal ulcer and early appendicitis (uncomplicated) are not likely to be present.

In renal disease I found readings as follows :

Disease	No. of cases.	B.S.R.
acute glomerular nephritis	1	26 mm.
chronic parenchymatous nephritis	2	16,22 mm.
cystic disease of kidney	1	23 mm.
renal calculus	3	3,2,2 mm.
pyelitis	1	20 mm.

It will be noted that the rates are increased, except in the three cases of stone in the renal pelvis. Wood (30) quotes Fahr and Swanson as stating that the plasma fibrin is increased in all forms of renal disease. This, no doubt, accounts for the raised B.S.R.

Ljungstroem (37) found remarkably high rates in forty-two cases of hypernephroma, thirty of which gave readings over 72 mm. per hour (Westergren's method), whereas this was not so with forty-two cases of abdominal carcinoma examined by him.

Boughard-Potocki (38) observed that the B.S.R. was accelerated in/

in all cases of tuberculous disease of the genitals or urinary tract. He has never found tuberculous lesions of these parts with a normal rate. In one patient with acute gonorrhoea I observed a high B.S.R. which gradually returned to normal as the condition responded to treatment. One case of urethritis, found on microscopical examination not to be due to Neisser's organism, gave a normal reading of 3 mm. In this district, venereal diseases are almost conspicuous by their absence, but illegitimate births and premarital conception are quite common. Two cases of tertiary syphilis were tested and both gave high readings -- 18, 20 mm. --, but with anti-specific treatment the rates gradually diminished. Rossier and Basto (8) state that a lowering of the B.S.R. towards normal in syphilis indicates either recovery or a lack of general reaction as the result of which may occur all the torpid happenings so frequent in such an illness. In four cases of congenital syphilis I found the B.S.R. to be normal or only a little raised, and the blood Wassermann reaction to be faintly positive in one.

The B.S.R. in Diseases of the Thyroid & Pancreas.

The following Table indicates the results obtained during the investigation. All the patients were females.

Disease.	No.of cases.	B.S.R.
Hyperthyroidism	2	15, 18 mm.
Cystic goitre	3	3, 4, 3 mm.
Myxoedema	3	1·5, 2, 3 mm.

Those patients with hyperthyroidism responded to rest and small doses of Lugol's Iodine (m.v.b.i.d.) and this was accompanied by a gradual subsidence of the B.S.R. The Basal Metabolic Rate was determined in one case which was treated in hospital. The B.S.R. was normal in cystic goitre, but tended to be subnormal in myxoedema.

According to Wood (30), Tschernozatonskaia studied ninety-seven cases of Thyrotoxicosis and found an increased rate in all of them. The latter considered that the test could be used as a guide to the degree of activity of the thyroid gland. Mora and Gault (39) noted an increased rate in all of their thirty cases, but found no correlation between it and the Basal Metabolic Rate. Masten (29) and Cutler (14) both observed that hyperthyroidism accelerated the B.S.R., but Van Antwerp (40) concluded from his investigation that persons with hypothyroidism had an increased rate, while those with hyperthyroidism had an abnormally slow rate. Reichel (11) cites four cases of typical Basedow's disease and two of hypothyroidism with normal B.S.R. From the above conflicting results it would seem that the application of the test in diseases/

diseases of the Thyroid is unreliable.

In five cases of diabetes mellitus I found the B.S.R. to be normal in four, but in the fifth the rate was as high as 30 mm. owing to the presence of pulmonary tuberculosis. In all five, Insulin treatment did not affect the rate although the glycosuria disappeared. According to Reichel (11), opinions differ regarding the B.S.R. in uncomplicated diabetes mellitus, some finding an acceleration, others a normal reaction in all their cases. From a study of 366 diabetic patients, Kramer (41) concluded that the blood sugar per se had no influence upon the rate, but that focal sepsis was the most likely explanation of the high B.S.R. in diabetes, 67·8% of his cases showing abnormal readings. Analysis of the patients observed showed a high incidence of the minor infections, particularly in the teeth, tonsils, upper respiratory tract, urinary tract and gall-bladder.

Tulipan and Director (42) investigated the B.S.R. in diseases of the skin and found that the rate was normal in lupus erythematosus, erythema multiforme (including bullous type) and the common dermatoses, but was increased in tuberculosis of the skin, erythema nodosum, dermatitis herpetiformis and pemphigus. They state that the test may be used to differentiate between pemphigus and erythema multiforme bullosum.

The following Table gives the B.S.R.results obtained by me in skin diseases.

Disease	No.of cases	B.S.R.
erysipelas	3 males	16,18,14 mm.
erythema nodosum	3 females	14,20,18 mm.
furunculosis	3 males	8,12, 6 mm.
eczema	3 males	½, ½, 8 mm.
lupus nasalis	1 female	10 mm.
alopecia areata	2 males	4, 3 mm.
dermatitis herpetiformis	1 male	12 mm.
erythema multiforme	1 male	2 mm.

As will be noticed,increased rates occurred in all the diseases,with the exception of alopecia areata and erythema multiforme which were normal,and of two cases of eczema with subnormal readings.In the third case of eczema the B.S.R.was raised owing to the skin becoming infected. As allergic conditions are usually accompanied by a subnormal rate,the test might be employed to indicate the presenee of hypersensitivity.

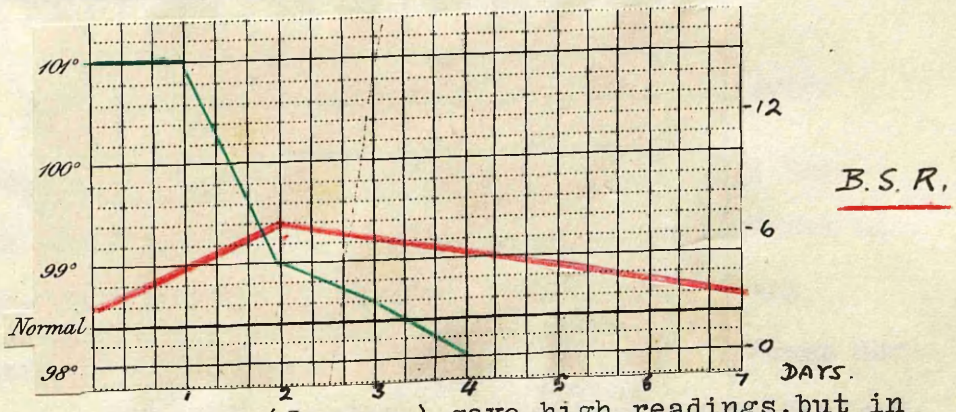
The B.S.R. and blood Wassermann reaction were found to be normal in six cases of major epilepsy,two of disseminated sclerosis and one of spastic paraplegia. In six cases of neurasthenia and psychogenic disorder the B.S.R.was within normal limits.

Four patients suffering from chronic otitis media had increased rates. As the ear condition improved more normal readings were observed. The test might therefore be useful in indicating prognosis and in excluding the development of complications, e.g. mastoiditis.

Normal readings were found in four cases of parotitis epidemica, but Gallagher (43) noticed acceleration of the rate if orchitis supervened. One of my patients with bilateral pyogenic parotitis had a reading of 4 mm. i.e. within normal limits.

The rate was moderately increased in three cases of tubercular cervical adenitis.

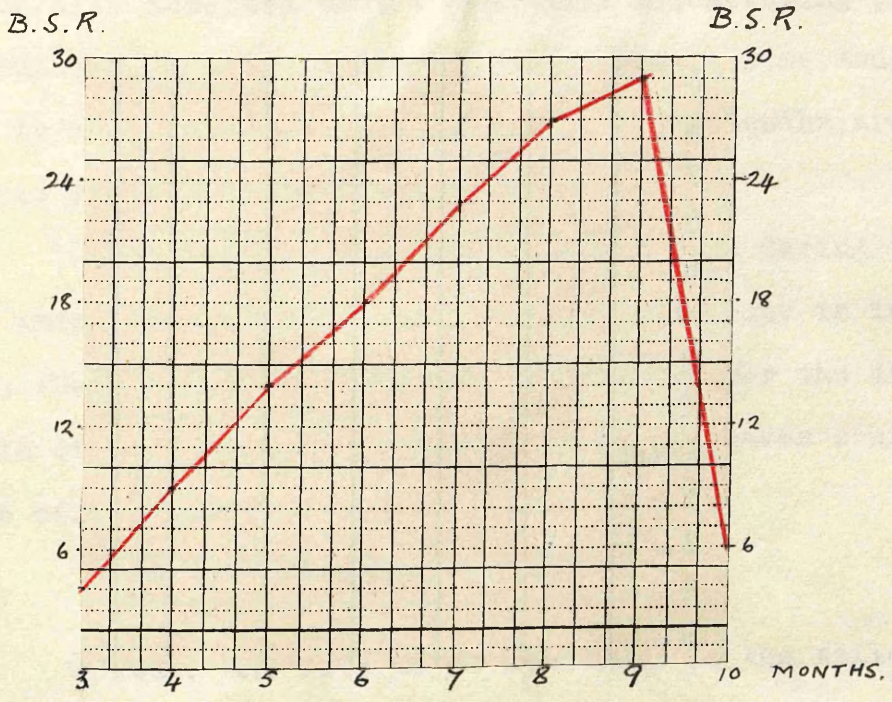
In influenzal chill (6 cases) the B.S.R. increased but little in comparison to the temperature, and lagged behind the latter as the chart indicates.



Tonsillitis (5 cases) gave high readings, but in coryza (4 cases) the rate was normal or only slightly raised.

The B.S.R. in Pregnancy.

It is interesting to notice that pregnancy is the only physiological condition in which the rate is accelerated. This chart indicates the B.S.R.curve during a normal gestation occurring in my practice.



In pregnancy the rate increases gradually from the fourth month until the time of delivery and then returns to normal by the end of the fourth week of the puerperium.

Fåhraeus (2) considered that the globulin increase during pregnancy and also in disease may possibly be in the nature of a protective reaction. He found that the plasma from the fourth month of gestation gradually became richer in fibrinogen/

fibrinogen. Cherry (44) quotes Sakae and Tsutsumi as stating that during pregnancy the ratio of the blood volume to the erythrocytes is greater and that the corpuscles agglutinate more rapidly,because of the increase of serum globulin and fibrinogen or because of a decrease in the albumin contents. It is generally accepted that the anaemia accompanying pregnancy resulting in a lower percentage of cell volume,and the increase in the fibrin,especially in the later months,are responsible for the increased B.S.R.

Reichel (11) considers that the test during pregnancy is unreliable,as such great differences occur in individual women that no normal value can be obtained for the different months of gestation. My examination of ten cases confirms Reichel's conclusion.

Factors retarding the B.S.R.

I found the rate to be subnormal in the following conditions: hay fever,bronchial asthma,chronic bronchitis with emphysema,congestive heart failure,polycythaemia,whooping cough, jaundice,and eczema.

As the rate in pertussis becomes subnormal in the early stage,the test may therefore be helpful in diagnosis before the characteristic " whoop " has developed.The withdrawal of blood from the veins of young children was sometimes found/

found to be difficult. Therefore, the Linzenmeier-Raunert micro-method as mentioned by Elghammer (24) would probably be more suitable, the blood being obtained from the tip of the finger or lobe of the ear.

As already noted on page 23, a decreasing rate in rheumatic carditis, coronary thrombosis and syphilitic aortitis may not indicate improvement, but signify the onset of myocardial failure.

Roesler and Meisel (45) observed that in cancer the injury to the liver through metastasis may have a stronger effect in slowing the rate, than the tumour has in accelerating it. They state also that a CO_2 increase in the blood raises the suspension stability through hypoventilation, as for example, in collapse therapy of the lung, cardiac failure and moribund states.

Förster (46) noted that the rate was slowed in persons with rachitic stigmata. I have not had an opportunity of confirming this as rickets is uncommon in Shotts.

It is well known that a sojourn in high altitudes increases the red cell count of the blood. Thus, the B.S.R. becomes subnormal as was observed by Raponsky (47). He believes that the electric discharges of the atmosphere, atmospheric depression, winds, rain, heat and climate all have an accelerating effect on the rate.

It is interesting to note that the rate is remarkably slowed in the blood of a newborn infant. I found that the umbilical blood in four cases took 48 hours, on the average, for the erythrocytes to fall to the mark 18, whereas the mothers' blood took only half an hour to reach the same mark. According to Ellenberg(48), Bruchsaler studied the relative B.S.R. and blood fibrinogen in infants and pregnant women and found that the maternal blood showed from 250-500 units of fibrinogen, while the blood of the newborn infants contained from 64-125 units. That the smaller amount of fibrinogen in the baby's blood is not the only cause of the extreme slowing of the rate is, in my opinion, explained by the fact that the normal infant at birth usually has a blood count of six to seven million red cells per c.mm. As Parsons (49) remarks," the infant, in utero, is living in a medium of low oxygen tension, and, exactly as in adults who have to live at high altitudes, it is necessary for the red blood cells to be present in increased numbers so that tissue respiration may be maintained."

Chronic Chest Diseases in General Practice.

Particular attention was paid to the investigation of pulmonary affections owing to their high incidence in the district. The majority of the cases occurred in miners, coal-mining being the chief local occupation. According to a Report of/

of the Department of Health for Scotland (50), the mortality from respiratory diseases is heavy in the industrial belt of Scotland, a statement which applies to both males and females. As is pointed out, geographical and climatological factors have to be taken into account, especially in such diseases as bronchitis, pneumonia and possibly phthisis. The following Table from the Report (page 38) represents the rate per 100,000 for miners and other males in Coalfields for selected causes of incapacity. As will be seen, the incidence of tuberculosis is less in miners than in other males in Lanarkshire, while that of bronchitis, pneumonia and upper respiratory diseases is greater in miners. This suggested to me the need for an investigation of this widespread cause of ill-health and incapacity.

As far as I am aware, no systematic inquiry has yet been carried out regarding the causation of pulmonary affections in coalminers in Scotland. Most of the cases are certified as suffering from " chronic bronchitis ", but the etiological factor is still indefinite. In this investigation I considered the various causal factors, including the inhalation of dust in mines. Special attention was paid to the B.S.R. in chronic pulmonary diseases in order to ascertain its diagnostic value and the reaction of the patients throughout the year. The majority of the cases examined were coalminers, the the others being females and non-miners.

Rate per 100,000 for Miners and other Males in Coalfields for selected Causes of Incapacity.

Coalfields.

Causes of Incapacity.	Lanarkshire.			Ayrshire.			Fife.			West Lothian and Stirling.			East Lothian and Midlothian.			All Scotland.		
	Miners.	Other Males.	Ratio. Miners : Others.	Miners.	Other Males.	Ratio. Miners : Others.	Miners.	Other Males.	Ratio. Miners : Others.	Miners.	Other Males.	Ratio. Miners : Others.	Miners.	Other Males.	Ratio. Miners : Others.	Miners.	Other Males.	Ratio. Miners : Others.
All Tuberculosis	99	150	0·7	138	140	1·0	114	107	1·1	84	76	1·1	148	134	1·1	117	132	0·9
Malignant Tumours	44	63	0·7	48	50	1·0	30	52	0·6	34	45	0·8	44	50	0·9	42	57	0·7
Diabetes	14	18	0·8	5	18	0·3	9	19	0·5	7	26	0·26	6	22	0·3	12	18	0·7
Anæmia	57	49	1·2	138	55	2·5	173	64	2·7	168	63	2·7	118	54	2·2	117	57	2·1
Infectious Diseases	123	273	0·5	204	385	0·5	186	412	0·5	164	254	0·64	199	340	0·6	164	311	0·5
Cerebral Hæmorrhage	25	36	0·7	42	51	0·8	38	28	1·4	17	35	0·5	28	41	0·7	29	36	0·8
Veins	327	227	1·4	371	203	1·8	433	259	1·7	530	214	2·5	494	228	2·2	413	239	1·7
Influenza	6,389	3,362	1·9	10,331	3,983	2·6	12,075	5,905	2·0	12,145	4,222	2·9	10,452	3,638	2·9	9,487	4,261	2·2
Bronchitis and Pneumonia	2,242	1,539	1·4	2,120	1,080	2·0	2,615	1,175	2·2	2,593	1,282	2·0	2,693	1,332	2·0	2,446	1,391	1·8
Upper Respiratory	1,880	1,648	1·1	2,789	2,082	1·3	2,922	2,517	1·2	3,801	2,326	1·6	2,389	3,054	0·8	2,544	2,031	1·3
Gastric and Duodenal Ulcers	262	251	1·0	286	240	1·2	378	299	1·3	532	219	2·4	315	271	1·2	337	271	1·2
Appendicitis	276	262	1·1	416	356	1·3	466	380	1·2	401	249	1·6	366	264	1·4	373	311	1·2
Hernia	141	153	0·9	148	106	1·4	149	149	1·0	159	133	1·2	238	146	1·6	163	145	1·1
Gastritis	1,720	880	2·0	1,971	951	2·1	2,910	776	3·8	2,641	774	3·4	3,015	784	3·8	2,330	1,141	2·0
Kidney	190	210	0·9	215	207	1·0	266	212	1·3	241	232	1·0	164	240	0·7	218	208	1·0
Inflammatory Skins	3,094	1,190	2·6	3,787	1,434	2·6	4,871	1,564	3·1	4,532	1,818	2·5	5,639	1,455	3·9	4,133	1,621	2·6
Skin Diseases	386	338	1·1	554	274	2·0	659	282	2·3	629	331	2·0	689	369	1·9	545	350	1·6
Rheumatic and Joint Conditions	4,577	2,116	2·2	5,251	2,287	2·3	6,156	2,301	2·7	6,136	2,467	2·5	7,389	2,639	2·8	5,658	2,543	2·2
Violence	4,615	1,599	2·9	6,835	1,846	3·7	6,431	2,214	2·9	7,787	2,122	3·7	8,618	2,173	4·0	6,342	2,326	2·7
Cardiac Debility	61	44	1·4	93	13	7·1	78	19	4·1	99	69	1·4	97	66	1·5	80	45	1·8
Nervous Debility	50	117	0·4	37	65	0·6	89	56	1·6	91	76	1·2	82	109	0·8	69	83	0·8
Neurasthenia	103	106	1·0	77	87	0·9	133	175	0·8	75	72	1·0	151	84	1·8	110	96	1·1
D.A.H. and Tachycardia	52	36	1·4	64	35	1·8	91	37	2·5	84	31	2·7	102	24	4·2	74	37	2·0
Undefined Debility	67	111	0·6	156	80	1·95	157	73	2·2	177	77	2·3	223	83	2·7	136	83	1·6
All Causes	305	172	1·8	400	186	2·1	464	217	2·1	442	216	2·0	540	210	2·6	405	190	2·2

At the International Congress on Silicosis in Johannesburg,1930 (51),Silicosis was defined as " a pathological condition of the lungs due to the inhalation of silicon dioxide (SiO_2)." Jones (52) does not agree with this definition and holds that silicosis is due to the inhalation of sericite,the compound silicate of aluminium and potassium. This view is strengthened by the statement of Davidson (53) who says: " It is a fact of no little significance that cases of silicosis do not occur among the mineworkers in the Kolar Goldfield,India,where the rock contains more quartz than that of the South African mines,but no sericite."

The air passages provide a natural defensive mechanism against the entrance of dust into the lungs. Legge (54) states: " the dictum is true that all dusts,in excessive amount,must be injurious if not to the lungs,then to the upper air passages,causing atrophy of that mucous membrane." It is generally agreed that chronic bronchitis causes a shedding of the ciliated epithelial cells lining the bronchi and bronchioles,thus reducing the power of resisting the entry of dust particles. The modern view is that the main damage to the lung tissue is not caused by the physical properties of the particles inhaled,but is due to the fact that silica is soluble in the tissues and acts as a chemical poison.

According to Lyle Cummins (55),pneumonoconiosis may be due to the inhalation of two kinds of dust: 1/ dusts which/

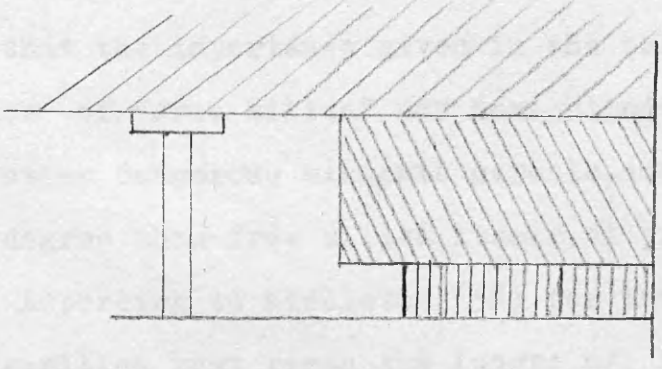
which are chemically active and cause pathological changes in the lungs owing to their solubility in the body fluids. As a result,two types of lesion may be produced,first,toxic due to local necrosis and cell death,favouring growth of Tubercle Bacilli; second,sclerotic,nodular and diffuse fibrosis,typical of silicosis. 2/. Dusts which are inert owing to their insolubility,but may cause a certain amount of diffuse fibrosis around the dust deposits.

The inhaled particles of dust are taken up by the phagocytic cells and are either expectorated or are carried into the lymphatics of the lung where they become distributed, especially along the peribronchial lymphatics and at the junction of the interlobular septa and in the pleura. There,the particles become encapsulated,the amount of fibrosis depending on the nature of the dust inhaled. In anthracosis,Muir (56) says that there is generally very little fibrous overgrowth. " It would seem,in fact" he continues," that carbon particles in a pure condition have very little irritating effect and do not in themselves lead to any serious damage to the lung tissue. The fibrous nodules and diffuse fibrosis,which are sometimes met with,are due,we believe,to the concomitant presence of stone dust."

According to Lemon and Higgins (57),the enzyme or soluble substance,elaborated by the phagocyte,fails to dissolve the particles of carbon,which are therefore bland and/

and non-toxic. The phagocyte,by chemical action of its enzyme or soluble substance,seems to produce a tissue poison from the particles of silica and this process continues as long as silica remains in the lung. It gradually stimulates hyperplasia of fibroblasts and constant addition to the amount of fibrous tissue. Thus,these investigators hold that men may lose respiratory competency years after having been engaged in work where the atmosphere contained a high percentage of silica.

The following diagram illustrates the section of a Seam in a Shotts colliery.



It will be observed that the upper stratum consists of hard stone,which an ordinary coal-cutting machine would be unable to cut. Therefore,this stone must be bored by a special machine and dislodged by explosives. The ordinary machine is used,however,for cutting the coal and the lower layer of fireclay. Thus,the atmospheric dust consists mostly of these two layers. Samples of atmospheric dust may be collected for enumeration by means of the Thermal Precipitator and the particles/

particles may be recognized by means of the petrological microscope. These modern methods were not able to be utilised in this investigation,but I had the dust from the masks,worn by machinemen in three coalmines,examined by an analyst. The composition of each specimen was found to be as follows:

	Silica	Carbon
1/	53·9%	46·1%
2/	31·4%	68·6%
3/	26·7%	73·3%

The analyst was unable to state if the silica was free or combined. However,as Lyle Cummins (55) says: " It may well be that the importance given in the past to the presence or absence of "free silica" may have blinded us to the existence of other dangerous elements capable,equally with or in greater degree than free silica itself,of producing silicosis."

According to Middleton (58),for the production of silicosis,silica must reach the lungs: a/. in a chemically uncombined condition,although it might be mixed with other dusts;b/.in fine particles of the order of less than 10 microns in diameter; c/.in sufficient amount and over a certain period of time. He states that " it is generally agreed that the majority of dust particles found in the lungs are under 5 μ and very few exceed 10. There are exceptions,for example,in the asbestos industry and in coal-mining,where the length of particles found in the lungs may greatly exceed 10μ."

According to Jones (59), the mineral sericite is present in the sandstones of the anthracite coal-field in England, while in those of well-known Scottish colleries sericite is either absent or rare. The following Table, quoted by Middleton (58), indicates the number of certificates issued for silicosis and silicosis with tuberculosis in Scottish coal-mines, by the Medical Board during period June 1st. 1931 to December 31st. 1935:

District	No. of wage-earners on Dec. 14, 1935	No. of certificates issued for Death.	Total disablement	Suspension
Fife & Clackmannan	21,625	-	-	1
Lothians (Mid. & East)	12,472	-	2	-
Lanarkshire	38,830	-	1	3
Ayrshire	11,311	-	-	-
total	84,238	-	3	4

In England and Wales for same period the numbers were respectively:

531,694	22	66	35
139,228	147	512	198

It will be noticed that the incidence of silicosis is greatest in the Welsh coal-mines.

The number of cases examined in this investigation were as follows: females 24; males (non-miners) 20; males (miners) 64. The ages of the females ranged from 12 to 69 years, with an average of 33 years; the non-miners from 18 to 67, with an average of 42; while that of the miners varied from 20 to 67, with an average of 45 (Vide Tables at end of Thesis).

Twenty of the females, mostly married, did housework. Two were in business and two still at school. No children under twelve years of age were taken into consideration. Several trades were represented among the non-miners, labouring being the chief. The miners were classified according to the work they mostly undertook while in the pits. The majority of them had been doing underground work since leaving school. The Table below indicates their various occupations.

	No.
Machinemen(work coal-cutting machines)	21
Strippers(load coal into hutches)	16
Brushers(prepare coal-face and roads)	14
Oncostworkers(repair roads)	6
Stonemineworker(makes new roads through)	1
Bottomers(move hutches at roads through stone/t)	2
Gummer(removes dirt from newly-cut coal)	1
Drawer(pushes hutches)	1
Putter(assists drawer)	1
Surfaceman(works at pithead)	1
total	64

It will be noticed that most of the men were machine-men, strippers and brushers, i.e. men who were exposed to much atmospheric dust and explosive gases.

Clinical Examination

A thorough physical examination of the chest was carried out in each case. The general condition of the females and the non-miners was found to be much better than that of the miners. Many of the latter were debilitated and below normal weight. It was not possible to have all the cases weighed, so definite figures cannot be given.

Cyanosis was not marked amongst the females, occurring only in four cases, whereas this sign was present in five of the non-miners and in fourteen of the miners.

Clubbing of the fingers and especially incurving of the nails were noticeable, the number of cases in the three classes (females, non-miners, miners) being 6, 6, 18 respectively.

The shape of the chest was next examined. As emphysematous changes were so common, the typical barrel-shape was frequently found, the number of cases in which it was present being 8, 7, 20 respectively. Flattening of the chest was noticed in some of the younger patients. The measurement of the chest was not done in all the females, but was carried out in the other two classes. It was observed that the average readings for the non-miners were 32 inches expiration and 33⅜ inches inspiration, while in the miners the readings were 34 and 35.

The range of expansion was often below the normal (2-3 inches),being in many of the miners as low as ½ inch, this being due,no doubt,to the pulmonary fibrosis and associated emphysema. Most of the chests yielded hyper-resonance on percussion,but many miners had patchy areas of dullness, which,according to Williams (60),is a sign commonly found in pneumonoconiosis.

On auscultation,râles and rhonchi,together with weak breath sounds and prolongation of expiration,were audible in most of the bronchitic cases,whilst in the others, signs of the pathological condition present were usually discernible.

The area of cardiac dullness was difficult to define owing to the presence of emphysema,but five skiagraphic reports contain remarks regarding enlargement of the heart shadow(female No.9; non-miners 9 & 15; miners 15 & 26). No female suffered from valvular disease of the heart,but this condition was present in four of the non-miners(Nos. 9,14,17,18). V.D.H.was absent in the miners,six of whom (Nos.5,11,15,26,40,55),however,showed evidence of myocardial decompensation,whilst several had visible pulsation in the epigastric region.

Dyspnoea was present in almost all the cases,but was most noticeable among the miners,many of whom were breathless on slight exertion,even in the absence of bronchitis and/

and were unable to hold the breath for more than fifteen seconds. Williams (60) found,on examining 100 old and retired coalminers in South Wales,that dyspnoea was more marked in those under 60 years of age. In her opinion,this was due to the fact that a greater amount of dust,both of coal and stone,is breathed by miners nowadays,on account of the advance in mechanical methods of getting the coal and ripping the rock. The results of this investigation tend to confirm Dr.Williams' opinion.

Haemoptysis occurred in five females: Nos.2,5 & 17 suffered from pulmonary tuberculosis; No.8 from chronic bronchitis and probably dry bronchiectasis; while No.9 had myocardial degeneration. Two cases of haemoptysis were found in the non-miners: No.3 suffered from bronchiectasis and No.9 from bronchitis and V.D.H.(mitral and aortic). Four cases are recorded in the miners: Nos.12 and 39 having pneumonoconiosis and Nos.52 and 57,chronic bronchitis. No.27 gave a history of haemoptysis in 1926 & 1933 and was diagnosed as a case of bronchitis and/?bronchiectasis. No.36,with quiescent pulmonary tuberculosis,had haematemesis due to gastric ulcer.

The sputum was examined for Tubercle Bacilli by the County Bacteriologist,Dr.T.Gow Brown,twice or thrice in each of the 108 cases,with results as follows:

	Negative	Positive
24 females	22	Nos.5,11
20 non-miners	19	No. 17
64 miners	61	Nos.44,56,63

Nineteen cases in all were diagnosed as having pulmonary tuberculosis,but,as will be seen,only six had positive sputa.

The blood Wassermann reaction was done by Dr.Brown in all the cases where the B.S.R.was raised in order to exclude the presence of syphilis. Only one positive reaction was found,namely,in miner No.8,who suffered from low grade pulmonary tuberculosis.

The past history was then examined. Three females(Nos. 2,5,8) had been in Sanatoria,but No.8 was thought to be non-tubercular. The Mother of Nos.10 and 11,who are twins,died of pulmonary tuberculosis,and the former is receiving sanatorium treatment for a similar complaint. No.1,also,had a bad family history,but was not considered to be tubercular. There was a history of pneumonia and/or pleurisy in Nos.1,3,7,12 & 19, but none has developed pulmonary tuberculosis. No.3 had pleuro-pneumonia and empyema in 1932 and subsequently bronchiectasis. Her condition has improved considerably with medical treatment in a convalescent Home. Nos.2,5,8 & 17 gave a history of haemoptysis,2,5 & 17 being tubercular. Many of the other cases, mostly asthmatics,were subject to attacks of dyspnoea and coughing especially during the misty or frosty weather.

Amongst the non-miners,only two (Nos.14 & 17) of the four having V.D.H.had a history of rheumatic infection. The Mother of No.11 died of pulmonary tuberculosis, but after clinical/

clinical,skiagraphic and sputum examinations,he was thought to be non-tubercular and was finally diagnosed as a case of bronchiectasis. A history of haemoptysis or pleurisy was not present in the non-miners,but chronic coughs and colds were common particularly during the winter months. This applied to most of the miners also. It is interesting to notice that only three miners(Nos.10,14,30) attributed their bronchial trouble to "gassing" during the late War. This may be accounted for by the fact that miners as a whole were exempt from military service.

Ten miners were or are still receiving sanatorium treatment,but four(Nos.14,22,51,29) are thought to be non-tubercular. Pneumonia and/or pleurisy are recorded in Nos. 9,13,39,43 & 53,but only 43 has,so far,been diagnosed as suffering from tuberculosis.

The B.S.R. in pulmonary diseases.

This test is now an established method in the diagnosis and particularly in the prognosis of pulmonary tuberculosis. In order to ascertain the reaction of the individual patient,the test was carried out in this investigation at frequent intervals throughout the year. Special attention was paid to coal-miners,as it is generally accepted that the danger of dust inhalation is the risk of superadded tuberculosis.

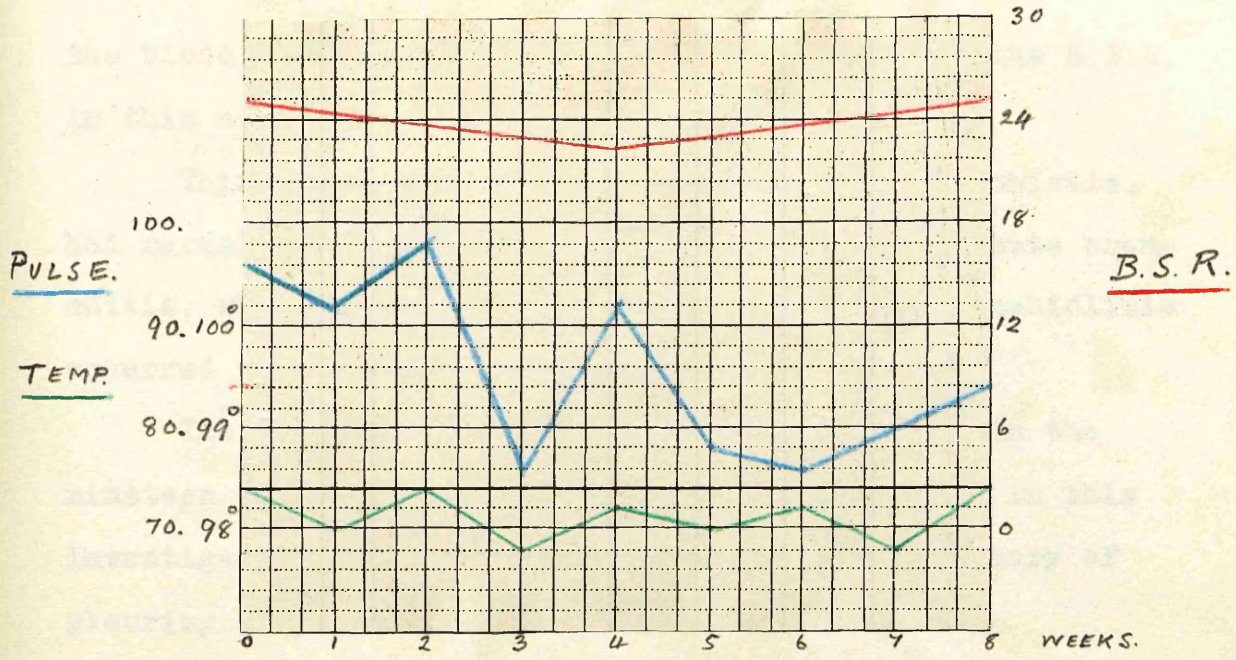
No test was done in the presence of intercurrent affections,e.g.furunculosis,tonsillitis,etc.as these were found to accelerate the rate.

Four females(Nos.5,11,17,22) gave readings above 12 mm.per hour.In the first three cases pulmonary tuberculosis was diagnosed,while in the fourth,a bronchitic,the raised rate was attributable,no doubt,to the presence of thyrotoxicosis. Subnormal readings were found in nine females, Nos.6,13,15,18 suffering from asthma(bronchial),Nos.2,4,10, 12,16 from bronchitis. Normal rates,varying from 3 to 12 mm. occurred in six bronchitic and three asthmatic patients. The B.S.R.did not vary much in bronchitis,even during a subacute attack,unless the inflammation spread to the bronchioles (Nos.1,7,10). It will be noted that No.3,with bronchiectasis, gave normal readings.

Seven non-miners had persistently high rates: Nos.2, 10,17 suffered from pulmonary tuberculosis; No.1 had bronchial asthma and polycystic disease of the left kidney,the raised B.S.R.being due to the latter condition; No.5 was a case of bronchitis with foul smelling expectoration resulting from secondary infection. It is well known that the latter cause may be partly responsible for an acceleration of the rate even in pulmonary tuberculosis; No 9 suffered from high blood pressure(200/120 mm.Hg.) and arteriosclerosis,the raised rate being probably due to renal involvement, although/

although no albumin or casts were found on examination of the urine. No.11 had bronchiectasis with secondary infection and gave moderately high B.S.R.readings. It is interesting to compare this case with No.3 and with female No.3,in both of which low rates were obtained and were accounted for by the absence of infection. Subnormal B.S.R.occurred in Nos. 7 and 15,the former having bronchial asthma,the latter,myocardial degeneration. As with the females,the rate was more or less constant in the ten bronchitic non-miners,unless bronchiolitis supervened(vide Nos.12,20),in which case the B.S.R.became accelerated.

High readings were found in fourteen miners,as shown by double underlining in the Table. Ten of the cases suffered from pulmonary tuberculosis,six of whom received or are receiving sanatorium treatment. Nos.44 and 56 were in Hairmyres Colony,East Kilbride. Dr.Johnstone,the Superintendent, kindly sent me their B.S.R.readings(Westergren's method) and skiagraphic reports. In the case of No.56,the rate gradually increased and reached 116 mm.per hour shortly before death. Of the remaining four miners with raised B.S.R.No.7 suffered from bronchiectasis,Nos12 & 34/from pneumonoconiosis,No.30 from chronic bronchitis.Each of the four cases had foetid expectoration resulting from secondary infection. The temperature, pulse and B.S.R.rates of No.7 are recorded overleaf.



It will be seen from the chart that the B.S.R.remained high,the temperature being 98·4 F.or under,while the pulse-rate was increased even at rest.

Miner No.43 had normal rates,although he suffered from pleurisy with effusion,Tubercle Bacilli being found in the fluid. It is generally accepted,however,that the B.S.R.is raised in pleurisy,but perhaps the low readings in this case were due to the infection being of a low grade.For example, No.62 was diagnosed as a case of pulmonary tuberculosis,but had only moderately raised readings.

Subnormal rates were observed in eleven miners with asthma and/or bronchitis. No.33 gave three readings of 0·5 mm. and was found to have 6,300,000 erythrocytes per c.mm.and 110% haemoglobin. Polycythaemia and increased CO_2 content of the/

the blood doubtless accounted for the slowing of the B.S.R. in this case of bronchitis and emphysema.

Thirty-nine miners, bronchitic or pneumonoconiotic, had normal rates, unless during an attack of subacute bronchitis, when higher readings were obtained if bronchiolitis occurred (vide Nos.2,3,6,32,42,61).

The following Table indicates the findings in the nineteen cases of pulmonary tuberculosis examined in this investigation. Only one, miner No.43, gave a history of pleurisy or pneumonia.

	No.	Sputum	Haemoptysis	B.S.R.
FEMALES.	2	-	+	normal
	5	+	+	raised
	11	+	-	"
	17	-	+	"
NON-MINERS.	2	-	-	"
	10	-	-	"
	17	+	-	"
	18	-	-	normal
MINERS.	5	-	-	raised
	8	-	-	"
	18	-	-	"
	36	-	-	"
	43	-	-	normal
	44	+	-	raised
	48	-	-	"
	56	+	-	"
	60	-	-	"
	62	-	-	"
	63	+	-	"

It will be seen that the B.S.R. was raised in sixteen cases. In the other three, the normal readings were due to the infection being quiescent or of low grade.

The B.S.R. results in fifty-eight cases of asthma or bronchitis are tabulated as follows:

	No. of cases	B.S.R.
Females	9	subnormal
	9	normal
	1	raised
Non-miners	2	subnormal
	9	normal
	3	raised
Miners	3	subnormal
	21	normal
	1	raised

Therefore, one may conclude that the rate, in asthma or bronchitis, is either normal or subnormal, but is raised in the presence of secondary infection.

In twenty-seven cases of pneumonoconiosis in miners, the B.S.R. may be recorded thus:

No. of cases	B.S.R.
8	subnormal
17	normal
2	raised

That is, a normal or subnormal rate is usually found in this condition.

The following findings were obtained in four cases of bronchiectasis:

	No. of cases	B.S.R.
Female	1	normal
Non-miners	1	normal
	1	raised
Miner	1	raised

The conclusion arrived at was that the rate remains normal in bronchiectasis in the absence of infection.

Although no case of tuberculosis occurred in this investigation,in which the B.S.R.was counterbalanced by the retarding influence of asthma or other allergic conditions,it is important to remember such a possibility. Repeated tests would,however,show whether the rate remained stationary or tended to increase.

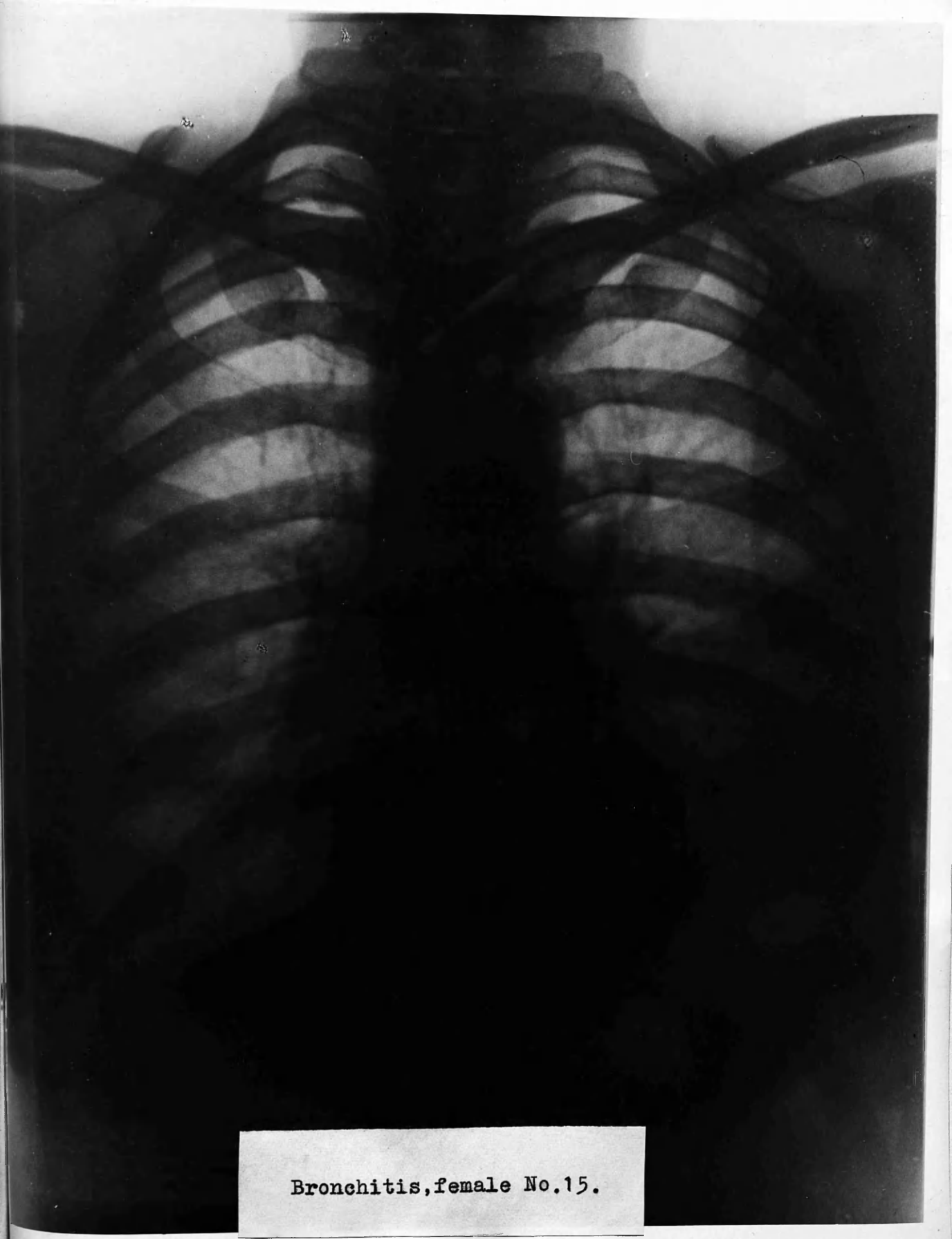
Skiagraphic Reports of the Chest

In this investigation,96 of the 108 cases were examined by X-rays. This was done mostly at the County Hospital,Motherwell,the skiagrams being interpreted by Dr.C.B.Wilson,Superintendent,Shotts Sanatorium.

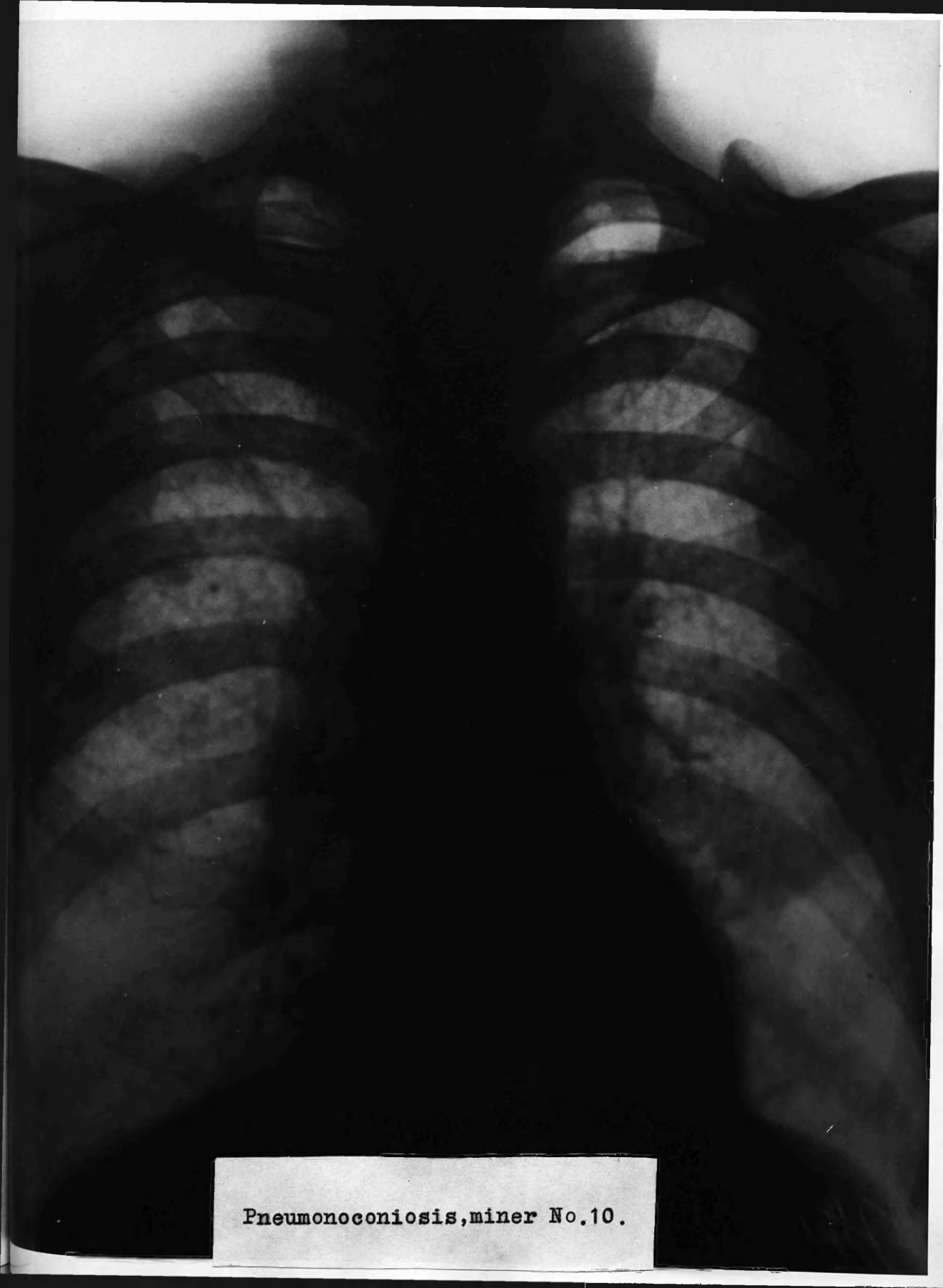
Signs of pulmonary tuberculosis were observed in four females,while fifteen others had fibrotic changes mostly typical of bronchitis. No.3 showed a bronchiectatic cavity in left lower lobe,and No.9,broadening of the mediastinum and heart shadow.

Four non-miners gave evidence of tuberculosis and seven had peribronchial fibrosis. Nos.3 and 11 had bronchiectatic cavities,while the lungs of No.5 had a honeycombed appearance due to fibrotic changes. Enlargement of the heart shadow was noted in Nos.9 and 15.

Amongst the miners,seven had tubercular changes,while miliary nodulation and diffuse fibrosis,occupational in type, were noted in twenty-seven cases,and peribronchial fibrosis in eighteen.



Bronchitis, female No. 15.



Pneumonoconiosis,miner No.10.

No.7 showed bronchiectasis of of left lung and No.43, a dense opacity at right base due to pleurisy.

The two skiagrams,accompanying this Thesis,indicate the changes generally found in bronchitis and pneumonoconiosis. In the former(female No.15),root and peribronchial fibrosis is present,while in the latter(miner No.10),miliary nodulation and fibrosis,occupational in type,are seem.

In the tubercular cases,the apices were especially affected and showed asymmetrical nodulation,the nodules tending to aggregate.

Although realizing the value of Röntgen rays,the final diagnosis was based on all the methods applied,especially the clinical.

Diagnosis of Cases

This was made from the data collected and may be tabulated thus:

		No.of cases.
Females(24)	chronic bronchitis	11
	bronchial asthma	7
	pulmonary tuberculosis	4
	bronchiectasis	1
	myocardial disease	1
Non-miners(20)	chronic bronchitis	10
	bronchial asthma	4
	pulmonary tuberculosis	4
	bronchiectasis	2
Miners(64)	pneumonoconiosis	27
	chronic bronchitis	22
	pulmonary tuberculosis	11
	bronchial asthma	3
	bronchiectasis	1

Chronic bronchitis was the most frequent disease, occurring in 45·8 % of the females, 50 % of non-miners and 34·4 % of miners. Two of the females (Nos.16 & 22) had diabetes mellitus and thyrotoxicosis respectively. 29·2 % of the females suffered from bronchial asthma, 20 % of the non-miners also, but only 4·7 % of the miners.

The incidence of pulmonary tuberculosis in the three classes was about the same, namely 16·7, 20 and 17·2 % respectively. Williams (60) found 6% of the old and retired coalminers examined by her to be tubercular and a further 26% to present a blood picture of the disease.

Lyle Cummins (55) believes that the carbon accumulations in silico-anthracotic lungs are capable of adsorbing toxic substances, resulting in a relatively low incidence of clinically active tuberculosis in silicotic coal-miners. He has shown experimentally the power of coal dust to adsorb the active principle of tuberculin in vitro, and it seems probable that a similar adsorption may take place in vivo in colliers. He holds that the "tuberculophile" effect of silicosis is neutralized through the detoxicating effects of the coal dust inhaled in large quantity throughout the life of the collier. The tuberculosis mortality of the several industries involving exposure to silicious dust, without simultaneous exposure to coal dust, is very high, as e.g. in the gold mines of South Africa.

It is interesting to note that bronchiectasis was found in 4·2% of the females, 10% of the non-miners, but in only 1·6% of the miners.

One female and six miners suffered from myocardial degeneration. Two of the latter (Nos. 11 and 15) developed cardiac failure with congestion and died. Unfortunately, permission for post-mortem examination was not granted. Not one of the miners had valvular disease of the heart, but this condition was present in four non-miners.

Pneumonoconiosis, probably silicotic in origin, was diagnosed in 42·2% of the miners, most of whom suffered from chronic bronchitis also. Williams (60) found 32% of her cases to be pneumonoconiotic and another 31% probably so on clinical grounds alone.

According to Cooke (61), mouth breathing whether due to chronic naso-pharyngeal disease or not is said to accelerate the development of silicosis in persons exposed to silica dust. It is generally accepted that damage to the respiratory system, as the result of bronchitis, interferes with the dust eliminating function of the lungs. Kettle (62), however, states that, as far as he knows, there is no alteration in the surface epithelium of the bronchi in an ordinary chronic bronchitis persistent for years. He believes that the very pronounced secretion of mucus helps to entangle dust particles on the walls of the bronchus, and that the current upwards from the lung is, if anything, enhanced in chronic bronchitis and definitely/

definitely not diminished. In my opinion, sufficient dust is inhaled to bring about pathological changes in the lungs, as are shown in the skiagrams of my mining patients, many of whom have suffered from chronic bronchitis for years.

Haynes (63) states that " injection of Lipiodol or some other substance, opaque to X-rays, down the bronchi shows that on coughing the content of the lower portions of the bronchi may be and generally is thrown back, only the upper content of the/portions of the bronchial tree being rendered available for expectoration. If the cilia be in part destroyed, as they are in bronchitis, it seems to be obvious that the dust will be less easily removed from the alveoli, and that there will be more chance of solutes passing into the lung parenchyma."

On discussing the subject with Professor E.H.Kettle, he held the view that bronchitis was microbic in origin and might be transmitted through contact from a bronchitic patient to other members of the household.

It appears to me that the predisposition to bronchitis is inherited, just as there is a tendency in some families to certain diseases, e.g. obesity, varicose veins, etc. It is generally accepted that the prognosis in phthisis pulmonalis is not so good if there is a familial diathesis to the disease.

The family histories of the 64 miners concerned in this investigation were examined and compared with those of

64/

64 healthy miners,most of the families being known to me during the past fourteen years. It was observed that the incidence of pulmonary affections in the blood relations of the incapacitated miners was much greater than in those of the healthy miners,being 42·2% and 18·8% respectively. A family was considered positive in the calculation of the percentage where one or more members suffered from chest complaints. The striking difference in the results would lead one to conclude that the incapacity for work of the miners with chest affections was due not only to the pulmonary changes resulting from the inhalation of the various dusts,but also to the associated changes resulting from chronic bronchitis. Davidson's Manual (53) contains radiograms of the chest in cases of silicosis,where the men had few symptoms or abnormal physical signs and were able to continue their occupation. I have had X-rayed two miners who have worked coal-cutting machines for over twenty years. The skiagrams showed marked pneumonoconiosis,yet the men were not incapacitated. They had never worn masks while at work and were not subject to bronchitis. Lyle Cummins (64) states that of 127 "healthy" miners,a selected group with not less than twenty years work at the coal-face,examined by the Medical Staff of the Welsh National Memorial Association,over 50% showed "mottling" on X-ray examination.

I know many miners in Shotts who have worked underground for twenty or more years and yet show no signs(clinical) of respiratory disease.

The dust which contaminates the atmosphere in a coalmine is caused by drilling,shot-firing,shovelling and handling of broken rock and coal. It is said that the dust remains longest suspended in the air after shot-firing. Efficient masks may be worn to minimize the inhalation of such dust. However,chronic bronchitis is aggravated not only by the inhalation of dust,but also by gases,wet conditions and draughts,which are to be found in most pits in this district. Injurious gases arising from the use of explosives, have an irritating effect on the bronchial mucous membrane and so induce attacks of bronchial catarrh.

Ivon Graham (65) states: " The question of fine dust-production during cutting is,to my mind,obviously connected with gas-production,as we know that the finer the coal is broken,the more readily will gas be given off." This does not seem to be of much importance in the Shotts colleries, where the miners are able to work with naked lights.

In a mining district,the sons usually follow in the footsteps of their father regarding occupation,and,in my experience,many suffer from bronchitis after a few years underground,or even before commencing work at all. In such cases,there is often a family history of bronchitis or other chest/

chest affection,but it is difficult to dissuade such youths from becoming miners,as alternative work is hard to obtain.

In order to lower the incidence of respiratory diseases in the mining industry,I am of opinion that the workers should be recruited from families in whom bronchitis and other chest complaints are absent.

Underground work,however light,should be abandoned by those partially incapacitated. In this investigation, thirty-eight of the sixty-four miners were totally unable to resume their occupation.

Means may be used to improve the general health,but will have no effect in removing the pathological changes already resulting from bronchitis and the inhalation of the various dusts. In all probability,these changes progress even after the men have ceased work and gradually increase the respiratory incompetency.

Summary of the B.S.R. Results

1/. Diseases in which the rate was raised:

articular and cardiac rheumatism.
septic phlebitis.
syphilitic aortitis.
coronary thrombosis.
pernicious anaemia.
cholecystitis.
pulmonary carcinoma.
salpingitis.
inflammatory conditions of genito-urinary tract.
renal disease(including pyelitis).
active syphilis.
hyperthyroidism.
erysipelas,erythema nodosum,pemphigus,furunculosis,
lupus nasalis,dermatitis herpetiformis.
otitis media.
pulmonary tuberculosis.
tubercular adenitis.
tonsillitis.
rheumatoid arthritis.
bronchitis or bronchiectasis,with secondary infection.

2/. Diseases in which the rate was normal or only moderately raised:

varicose ulcer.
gastric or duodenal ulcer.
cystic goitre.
diabetes mellitus.
appendicitis(uncomplicated and in early stage).
sciatica,fibrositis.
osteoarthritis.
gout.
chorea.
certain heart conditions(V.D.H. auricular fibrillation, tachycardia,myocardial degeneration).
hyperpiesis(without renal involvement).
microcytic hypochromic anaemia.
abdominal carcinoma.
congenital and latent syphilis.
alopecia areata,erythema multiforme.
epilepsy,organic nervous disease,psychogenic affections.
parotitis epidemica.
dental caries.
coryza.
influenza.
bronchitis(chronic or acute,if not involving bronchioles).

3/. Conditions in which the rate was subnormal:

congestive heart failure.
myxoedema.
allergic diseases(bronchial asthma,hay fever,eczema).
pertussis.
bronchitis(with much emphysema or fibrosis).
jaundice.
rickets.
sojourning in high altitudes.
umbilical blood.

As already mentioned,pregnancy is the only physiological condition in which the rate becomes raised.

Conclusions.

The blood sedimentation reaction is a useful guide in the diagnosis and particularly in the prognosis of disease, where there is breaking down of tissue with absorption. By comparing the readings,taken at frequent intervals,one obtains an indication of the progress or retrogression of the disease. Acceleration of the rate is never normal,except in pregnancy,and may appear before the pathological process manifests itself otherwise. The test is not a substitute for other means of diagnosis,but should be used in conjunction with modern laboratory methods and careful clinical examination. The reaction is not specific for any disease,but,like the temperature and pulse-rate,it may indicate the presence of an abnormal state. A slowing of the rate may assist one in the diagnosis of allergic and certain other conditions. As a normal reaction occurs in many organic diseases,its limitations/

limitations must be considered. Also,acceleration may be affected by influences which retard the rate,therefore proper interpretation of the results is essential.

The test is useful in the diagnosis and prognosis of chronic chest diseases. Where the rate is normal,one may reasonably exclude active pulmonary tuberculosis or secondary infection. Unfortunately,the B.S.R. does not help in differentiating between these two conditions.

The incidence of respiratory diseases in this district is high. Climatological and occupational factors are contributing causes,but the influence of family predisposition must be considered.

Pulmonary tuberculosis occurs in less than one fifth of the cases investigated,and the infection is mostly quiescent or of low grade,particularly amongst the coal-miners. In the latter class,pneumonoconiosis and chronic bronchitis are prevalent,tending to incapacitate and lead to respiratory incompetency.

Bibliography.

1 Fåhraeus,R: Biochem.Zeitschrift,1918,LXXXIX,355.

2 " ": Acta Med.Scand.1921,LV,3 & 156.

3 Hewson,W: Garrison's Hist.of Med,1922,3rd.Ed.331.

4 Hunter,J: quoted by No.2,31.

5 Blundell: " " " ",32.

6 Gilligan,D.R.& Ernstene,A.C: Amer.Journ.Med.Sci.1934, April,No.745,552.

7 Aldred-Brown,G.R.P.& Munro,J.M.H: Lancet,1934,June 23,1333.

8 Rossier,P.H.& de Castro Basto,R.A: Rev.Méd.de la Suisse Rom.1934,Mai 25,531-543.

9 Griffin,R.J: Amer.Journ.Obst.& Gynec.1934,XXVIII,532.

10 Stocke: quoted by No.9 and No.15.

11 Reichel,H: Blutkörperchensenkung,Wien.1936,34.

12 Friedman,S: Amer.Rev.Tuberc.1934,Feb.198.

13 Westergren,A: Brit.Journ.Tuberc.1921,XV.72.

14 Cutler,J.W: Amer.Journ.Med.Sci.1932,CLXXXIII.643.

15 Linzenmeier,G: Münch.Med.Wchnschrt.1923,Okt.5,1243.

16 Greisheimer,E.M.et alia: Amer.Journ.Med.Sci.1934,Feb.213.

17 Lafont,J:Le Progrès Méd.1935,Fev.23,No.8,321.

18 Scott,E: Supplement to B.M.J.1935,Sept.7,128.

19 Chung,H.L: Journ.Lab.Clin.Med.1935,XX.633.

20 Lenzi,M: La Riforma Med.1934,L.1306-1312.

21 Collis,W.R.F: The Practitioner,1935,CXXXIV.617-626.

22 Wilkinson,K.D: B.M.J.1935,July 20,99.

23 Ritchie,W.T: B.M.J.1936,April 4,679.

24 Elghammer,E.W: Arch.Pediat.1934,LI.281.

25 Payne,R.T: B.M.J.1936,May 2.877.

26 Wright,A.Dickson: B.M.J.1936,May 9,959.

27 Biegeleisen,H: Annals Surg.1934,IC.661-667.

28 Pennoyer,G.P: " " " " .997.

29 Masten,A.R: Amer.Rev.Tuberc.1934,XXIX.690-696.

30 Wood,P: Quart.Journ.Med.1936,V.No.17,Jan.1.

31 Bickel,G.et alia: B.M.J.1935,June 15,95(Epitome).

32 Reichel,H: Klin.Wochschrt.1935,Nov.23,1679.

33 Lesser,A.& Goldberger,H.A: Surg.Gynec.& Obst.1935,LX.157-166.

34 Liedberg,N: B.M.J.1935,Feb.2,22(Epitome).

35 Kessler,R: Deutsch Med.Wchnschrt.1936,Feb.14,258.

36 Smith,C.T;et alia: Amer.Journ.Med.Sci.1935,CLXXXIX.383.

37 Ljungstroem,C.E: B.M.J.1936,July 18,12(Epitome).

38 Boughard-Potocki,R: Journ.d'Urologie.1935,XXXIX.45-54.

39 Mora and Gault: quoted by No.30.

40 Van Antwerp,L.D: Amer.Journ.Dis.Child.1934,IIL.814-818.

41 Kramer,D.W: Journ.Lab.Clin.Med.1935,XXI.37-42.

42 Tulipan,L.& Director,W: Abstract from Journ.Lab.Clin.Med. 1933,XIX.215.

43 Gallagher,J.R: Amer.Journ.Med.Sci.1934,CLXXXVIII.450-455.

44 Cherry,T.H: Journ.Lab.Clin.Med.1934.XX.257-271.

45 Roesler,G.& Meisel,J: Deutsch Med.Wchnschrt.1934,LX.1677-78.

46 Förster,E: Med.Klinik.1935,XXXI.446-448.

47 Raponsky,A.I: Lyon Méd.1934,CLIV.139 & 351.

48 Ellenberg,S.L: Journ.Lab.Clin.Med.1934,IXX.944-947.

49 Parsons,L.G:B.M.J.1936,May 16,1009.

50 Dept.of Health(Scot):Report on Incapacitating Sickness in the Insured Populat.of Scot.during July 1.1933 to June 30,1934.

51 Middleton,E.L:Report on Internat.Conf.on Silicosis, Johannesburg.Aug.1930.

52 Jones,W.R: quoted by No.53.

53 Davidson,M: Diseases of the Chest.1935,Oxford.256.

54 Legge,Sir T: Industrial Maladies.1934,Oxford Med.Pub.181-197.

55 Cummins,S.L: B.M.J.1935,287,290.

56 Muir,Sir R: Textbook of Path.1936,4th.ed.416.

57 Lemon,W.S.& Higgins,G.M: Amer.Journ.Med.Sci.1932,CLXXXIII.153.

58 Middleton,E.L: Lancet.1936,July 4 & 11.

59 Jones,W.R: quoted by No.58.

60 Williams,E.M: Health of old & retired Coal-miners in South Wales,1933,Cardiff,71,44,92.

61 Cooke,W.E: The Practitioner.1936,CXXXVII.314-323.

62 Kettle,E.H: Trans.Institute Mining Engineers.1934/35, LXXXVIII.391.

63 Haynes,F: " " " 401.

64 Cummins,S.L: Amer.Rev.Tuberc.1934,No.1,Jan.,17.

65 Graham,J.I: Trans.Inst.Min.Eng.1935/36,XC.355.

FEMALES.

No.	Initials.	Age.	Occupation.	Clinical Examination.	Previous History.	B.S.R.	X-ray Report.	Diagnosis.
1	E.R.	13	school	Bronchitis	Pleurisy 1935; mother died of pul. tuberculosis.	3,10,9,6	Old root fibrosis, non-tubercular.	Bronchitis
2	Mrs.S.	29	home	Bronchitis, haemoptysis	Haemoptysis 1922; sanatorium treatment.	2,3,3,2	Pulmonary fibrosis, general haziness both lungs, calcified nodules at R.base.	Bronchitis, ?quiescent pulmonary tuberculosis
3	C.O'H.	12	school	Bronchiectasis	Pleuro-pneumonia and empyema 1932.	3,4	Bronchiectatic cavity in left lower lobe.	Bronchiectasis
4	S.D.	33	home	Bronchitis	Cough & colds.	2,3,3	Not X-rayed.	Bronchitis
5	Mrs.M.	38	home	Pulmonary tuberculosis, haemoptysis, pleurisy.	Haemoptysis 1930; sanatorium treatment.	9,7,10,11,13,12	Pulmonary tuberculosis, much peribronchial fibrosis, old calcified root deposits.	Pulmonary tuberculosis, low grade
6	Mrs.S.	23	home	Bronchitis	Cough & dyspnoea.	2,4,2	Peribronchial fibrosis.	Asthma & bronchitis
7	M.D.	20	home	Bronchitis	Pleurisy with effusion 1933.	3,4,10,3	Peribronchial fibrosis, non-tubercular.	Bronchitis
8	Mrs.G.	45	home	Chronic bronchitis, haemoptysis	Haemoptysis 1930; sanatorium treatment.	7,8,12,7	Basal fibrosis, no parenchymatous deposits, non-tubercular.	Bronchitis, ?bronchiectasis
9	Mrs.M.	52	home	Haemoptysis, Myocardial weakness	Haemoptysis 1935.	7,6,5,5	Broadening of mediastinum & heart shadow, non-tubercular.	Myocardial degeneration
10	A.C.	18	home	Chronic bronchitis	Cough; mother died of pul. tuberculosis.	2,6,9,3	Peribronchial fibrosis.	Bronchitis
11	C.C.	18	home	Pulmonary tuberculosis	Cough & colds; sister of No.10.	20,16,10,24,20	Pulmonary tuberculosis, generalized infiltration both lungs, opacity left base.	Pulmonary tuberculosis
12	E.K.	17	shop assistant	Bronchitis	Pneumonia 1931; sanatorium treatment.	3,3,4,2	Peribronchial fibrosis, calcified hilus glands, non-tubercular.	Bronchitis
13	Mrs.R.	40	home	Asthma, bronchitis	Cough & dyspnoea.	2,2,2	Not X-rayed.	Asthma & bronchitis
14	Mrs.C.	37	home	Asthma, bronchitis	Cough & dyspnoea.	7,4,3,3	Root & peribronchial thickening.	Asthma & bronchitis
15	Mrs.B.	44	home	Asthma, bronchitis	Cough & dyspnoea.	1,5,5,2,3	Root & peribronchial fibrosis.	Asthma & bronchitis
16	Mrs.R.	53	home	Bronchitis, diabetes mellitus	Cough & asthenia.	2,2,3	Peribronchial fibrosis, non-tubercular.	Bronchitis & diabetes mellitus
17	M.K.	35	pianist	Pulmonary tuberculosis, haemoptysis	Haemoptysis 1935.	20,12,13,7,11,7	Pulmonary tuberculosis right apex, old nodules left base.	Pulmonary tuberculosis, low grade
18	Mrs.C.	39	home	Asthma, bronchitis	Cough & dyspnoea.	2,6,2,2	Root & peribronchial thickening, non-tubercular.	Asthma & bronchitis
19	Mrs.R.	43	home	Bronchitis	Pneumonia 1935.	6,9,5,5	Peribronchial fibrosis, non-tubercular.	Bronchitis
20	Mrs.G.	44	home	Asthma, bronchitis	Cough & dyspnoea.	7,8,7	Peribronchial fibrosis, old calcified deposits upper half both lungs.	Asthma & bronchitis
21	Mrs.M.	69	home	Asthma, bronchitis	Cough & dyspnoea.	6,8,6	Not X-rayed.	Asthma & bronchitis
22	M.L.	20	servant	Bronchitis, thyrotoxicosis	Influenza 1934.	18,22,12,16,11,7	Peribronchial fibrosis, non-tubercular.	Bronchitis & thyrotoxicosis
23	Mrs.M.	32	home	Chronic bronchitis	Cough & colds.	12,12,11,10	Peribronchial fibrosis & root thickening, non-tubercular.	Chronic bronchitis
24	Mrs.S.	21	home	Chronic bronchitis	Cough.	3,3,4	Peribronchial fibrosis, non-tubercular.	Bronchitis

NON-MINERS.

No.	Initials	Age	Occupation	Clinical Examination	Previous History	B.S.R.	X-ray Report	Diagnosis
1	W.L.	27	farmworker	Asthma, bronchitis, abdominal tumour	Cough & dyspnoea.	23,15,15,16	Non-tubercular	Asthma, bronchitis, polycystic disease of L. kidney
2	J.C.	43	labourer	Pulmonary tuberculosis	Cough; influenza 1933.	22,26,23,10,12	Tubercular infiltration both lungs	Pulmonary tuberculosis
3	A.P.	56	shopkeeper	Haemoptysis	Cough.	4,4,2,3	Bronchiectatic cavity base of left lung, Lipidol was injected	Bronchiectasis
4	J.R.	58	labourer	Bronchitis	Cough.	7,5,4,3	Not X-rayed	Chronic bronchitis
5	T.D.	51	locomotive worker	Bronchitis with foetid expectoration	Cough & colds.	24,24,18,22,22,23,21	Honeycombed appearance both lungs from fibrosis, much root thickening, deposits in L. lung	Foetid bronchitis
6	J.C.	36	none	Asthma, bronchitis	Cough & dyspnoea.	1,2,1	Not X-rayed	Asthma & bronchitis
7	W.C.	44	labourer	Asthma, bronchitis	Cough & dyspnoea.	0·5,0·5,0·5	Slight peribronchial fibrosis	Asthma & bronchitis
8	J.O.	67	labourer	Bronchitis	Cough & colds.	2,8,2,1	Peribronchial fibrosis, accentuation of aortic curve	Chronic bronchitis
9	H.B.	67	labourer	Bronchitis, haemoptysis, B.P. 200/120 mm.Hg.	Cough.	10,10,13,11	Peribronchial fibrosis, haziness R. apex, non-tubercular, heart enlarged	Bronchitis, V.D.H. (mitral & aortic)
10	A.S.	21	engineer	?Pulmonary tuberculosis	Cough & colds.	20,12,4,4	Fine nodulation in R. lung, opacities both upper lobes, little root thickening	Pulmonary tuberculosis
11	W.S.	18	none	Purulent expectoration, ?bronchiectasis	Cough; mother died of pul. tuberculosis.	18,18,16	Bronchiectatic cavity R. lung, non-tubercular	Bronchiectasis
12	J.K.	18	grocer	Bronchitis	Cough, influenza.	4,10,4,4	Peribronchial fibrosis	Chronic bronchitis
13	E.C.	51	blastfurnace-man	Bronchitis	Cough.	1,2,12,2	Peribronchial fibrosis	Chronic bronchitis
14	C.H.	44	gatekeeper	Bronchitis, V.D.H. (mitral)	Rheumatic fever in childhood.	3,2,2	Not X-rayed	Bronchitis, V.D.H. (mitral)
15	P.L.	63	foundry man	Bronchitis, myocardial degeneration	Cough & dyspnoea.	1,1,0·5	Peribronchial fibrosis, old calcified root deposits, enlarged heart shadow	Bronchitis, myocardial degeneration
16	T.S.	26	clerk	Bronchitis	Cough, influenza 1934.	1,1,1	Apices quite clear, slight root thickening, non-tubercular	Bronchitis
17	S.L.	24	painter	Pulmonary tuberculosis, diabetes mellitus, V.D.H.	Rheumatic fever 1930.	28,23,26,25,30	Tubercular infiltration & cavitation both lungs	Pul. tuberculosis, diabetes mellitus, V.D.H. (mitral)
18	J.F.	25	vanman	Pulmonary tuberculosis & V.D.H. (mitral)	Cough & colds.	3,3,3,3	Tubercular infiltration of lungs	Quiescent pul. tuberculosis, V.D.H. (mitral)
19	J.C.	46	baker	Asthma & bronchitis	Cough & dyspnoea.	3,1,1	Not X-rayed	Asthma & bronchitis
20	J.C.	52	blastfurnace-man	Bronchitis	Cough.	14,3,2,5	Peribronchial fibrosis, non-tubercular	Chronic bronchitis

MINERS.

No.	Initials.	Age.	Occupation.	Clinical Examination.	Previous History.	B.S.R.	X-ray Report.	Diagnosis.
1	F.L.	43	stripper	Bronchitis, emphysema	Cough & colds.	7,18,7,7	Miliary nodulation, fibrosis-occupational, both lungs, root thickening.	Pneumonoconiosis
2	J.M.	49	stripper	Bronchitis, emphysema	Cough.	3,18,3,1·5,4	Miliary nodulation, fibrosis-occupational.	Pneumonoconiosis
3	J.H.	43	stripper	Bronchitis, emphysema	Cough, influenza 1934.	3,22,4,4	Much peribronchial thickening, calcified deposits both roots.	Chronic bronchitis
4	P.M.	35	stripper	Bronchitis, emphysema	Cough.	0·5,0·5,0·5	Miliary nodulation, fibrosis-occupational, calcified nodules, pleural thickening.	Pneumonoconiosis
5	J.L.	58	machineman	Bronchitis, emphysema, myocardial weakness	Cough & colds.	22,4,11,24,11,6	Pulmonary fibrosis, superadded tuberculosis low grade.	Pul. tuberculosis, low grade
6	P.K.	46	machineman	Bronchitis, emphysema	Cough.	20,6,4,6	Peribronchial and root fibrosis.	Chronic bronchitis
7	R.R.	48	machineman	Bronchitis, emphysema, purulent expectoration	Cough.	26,15,26,23,26,28	Bronchiectasis left lung; much fibrosis of both lungs.	Bronchiectasis, bronchitis
8	R.T.	46	machineman	Pulmonary tuberculosis, Blood Wassermann +	Cough & colds.	20,18,22,19,18,23	Infiltration of right upper lobe, cavitation and infiltration in left lung.	Pul. tuberculosis, low grade
9	S.M.	44	brusher	Bronchitis, emphysema	Pneumonia 1923, pleurisy 1928.	1,3,1,3	Peribronchial fibrosis.	Chronic bronchitis
10	R.M.	47	stripper	Bronchitis, emphysema	Gassed in late War.	1,1·5,11,3	Miliary nodulation, fibrosis-occupational.	Pneumonoconiosis
11	P.B.	40	machineman	Asthma, bronchitis, myocardial weakness	Cough & dyspnoea.	1,0·5	Not X-rayed.	Asthma & bronchitis, died 15/3/36
12	J.M.	60	stoneminer	Pneumonoconiosis, haemoptysis	Haemoptysis 1935.	9,10,10,11,11,11	Miliary nodulation, fibrosis-occupational, pleural thickening.	Pneumonoconiosis, ?pul. tuberculosis
13	R.F.	22	putter	Bronchitis	Pleuropneumonia 1935.	1,1,1,1	Peribronchial fibrosis, root thickening; no parenchymatous deposits.	Bronchitis
14	R.G.	38	brusher	Bronchitis, emphysema	Gassed in late War, sanatorium 1924.	1,1,1,1	Peribronchial fibrosis.	Chronic bronchitis
15	A.C.	58	stripper	Pneumonoconiosis, myocardial weakness	Dyspnoea & cough.	2,2	Fine diffuse mottling bases both lungs; increased root shadows, heart enlarged.	Pneumonoconiosis, died 29/1/36
16	W.C.	44	machineman	Bronchitis, emphysema	Cough.	2,1,1,1	Miliary nodulation, fibrosis-occupational.	Pneumonoconiosis
17	P.D.	47	gummer	Bronchitis, emphysema	Cough.	3,0·5,2	Miliary nodulation, fibrosis-occupational.	Pneumonoconiosis
18	T.M.	64	machineman	Bronchitis, emphysema	Cough & colds.	13,14,17,18,16	Much pulmonary fibrosis, small deposits both lungs, large deposits right apex.	?Quiescent pulmonary tuberculosis
19	J.M.	54	machineman	Asthma, bronchitis, emphysema	Cough & dyspnoea.	2,1,2,2	Miliary nodulation, fibrosis-occupational.	Pneumonoconiosis
20	J.W.	42	brusher	Bronchitis, emphysema	Haemoptysis; tubercular family history.	4,[illegible],2,2	Miliary nodulation, fibrosis-occupational.	Pneumonoconiosis
21	J.K.	38	brusher	Bronchitis	Cough & dyspnoea.	3,2,2,3	Miliary nodulation, fibrosis-occupational.	Pneumonoconiosis
22	J.L.	52	stripper	Bronchitis, emphysema	Sanatorium treatment.	3,2,4,3	Miliary nodulation, fibrosis-occupational, hilum root shadows.	Pneumonoconiosis
23	J.A.	35	oncost	Bronchitis	Cough.	2,0·5,0·5,1	Miliary nodulation, fibrosis-occupational.	Pneumonoconiosis
24	J.M.	67	brusher	Asthma, bronchitis, emphysema	Cough & dyspnoea.	4,3,2,2	Peribronchial fibrosis.	Asthma & bronchitis

MINERS (2).

No.	Initials.	Age.	Occupation.	Clinical Examination.	Previous History.	B.S.R.	X-ray Report.	Diagnosis.
25	R.E.	40	stripper	Bronchitis, duodenal ulcer	Cough & colds.	1,2,0·5,1	Root and basal fibrosis.	Bronchitis
26	W.B.	48	oncost	Bronchitis, emphysema, myocardial weakness	Cough & dyspnoea.	4,2,3,3	Peribronchial fibrosis, heart enlarged.	Chronic bronchitis
27	P.M.	37	stripper	Bronchitis	Haemoptysis 1926, 1933.	4,4,7,4	Peribronchial fibrosis.	Bronchitis, ?bronchiectasis
28	B.W.	33	machineman	Asthma, bronchitis	Chronic otitis media.	2,1,0·5,1	Miliary nodulation, fibrosis-occupat. peribronchial thickening, old calcified deposits.	Pneumonoconiosis
29	B.Q.	42	machineman	Bronchitis	Sanatorium treatment.	3,2,2,3	Miliary nodulation, fibrosis-occupat. root thickening, basal fibrosis.	Pneumonoconiosis, ?quiescent pul.tuberc.
30	M.M.	45	surfaceman	Bronchitis, emphysema	Gassed in late War.	6,12,8,16,11,12,7	Much root and peribronchial fibrosis.	Chronic bronchitis
31	H.M.	35	machineman	Asthma, bronchitis	Cough & dyspnoea.	1,1·5,3,3	Peribronchial fibrosis, root thickening.	Asthma & bronchitis
32	E.C.	59	stripper	Bronchitis, emphysema	Cough.	6,12,6,7	Root and peribronchial fibrosis.	Chronic bronchitis
33	P.M.	43	machineman	Bronchitis, emphysema, marked cyanosis, dyspnoea	Cough & marked dyspnoea.	0·5,0·5,0·5	Peribronchial fibrosis.	Chronic bronchitis, polycythaemia
34	W.H.	59	machineman	Bronchitis, emphysema	Cough.	22,16,12,12,10,12	Gross miliary nodulation, diffuse fibrosis-occupat.	Pneumonoconiosis
35	J.H.	26	brusher	Asthma, bronchitis	Cough & dyspnoea.	1,1,0·5,1	Miliary nodulation, fibrosis-occupat.	Pneumonoconiosis & asthma
36	S.H.	63	machineman	Bronchitis, haematemesis	Haematemesis 1935.	13,7,9,12,20,18	Old tubercular mottling upper left lobe, shadow right lower lobe.	Quiescent pul.tuberculosis, gastric ulcer
37	D.G.	56	stripper	Bronchitis, emphysema	Cough.	1,0·5,0·5	Nodulation, root and basal fibrosis-occupat.	Pneumonoconiosis
38	W.M.	57	brusher	Bronchitis, emphysema	Cough; son died of pul.tuberculosis.	1,1,1,0·5	Marked miliary nodulation, fibrosis-occupat., chiefly lower halves.	Pneumonoconiosis
39	J.M.	44	machineman	Bronchitis, haemoptysis	Pleurisy 1935.	5,5,11,5	Miliary nodulation, fibrosis-occupat.	Pneumonoconiosis
40	J.W.	37	machineman	Bronchitis, myocardial weakness	Cough & colds.	2,2,1	Peribronchial fibrosis.	Chronic bronchitis
41	A.W.	42	stripper	Bronchitis, emphysema	Cough.	5,7,6,6	Miliary nodulation, fibrosis-occupat.	Pneumonoconiosis
42	G.F.	61	brusher	Bronchitis, emphysema	Cough, influenza 1935.	2,12,2,2	Not X-rayed.	Chronic bronchitis
43	G.G.	20	bottomer	Pleurisy	Pleurisy 1934, sanatorium treatment.	2,3,2,2	Dense opacity right base.	Tubercular pleurisy
44	J.M.	24	oncost	Apical tuberculosis, synovitis of left shoulder	Sanatorium treatment.	60,45,40,52,70,45,30, 48,43,67,49,32,36	Coarse infiltration both lungs, vomica of left lung queried, fuzziness of head L humerus.	Pul.tuberculosis, tubercular synovitis (shoulder)
45	P.M.	35	machineman	Bronchitis	Cough & colds.	1,4,2,2	Peribronchial fibrosis, L ventricle enlarged.	Bronchitis
46	J.L.	43	machineman	Bronchitis	Cough.	3,2,2	Miliary nodulation, diffuse fibrosis of both lungs--occupat.	Pneumonoconiosis
47	W.M.	53	brusher	Bronchitis	Cough.	3,2,6,2	Miliary nodulation, diffuse fibrosis of both lungs--occupat.	Pneumonoconiosis
48	R.M.	45	brusher	Bronchitis	Sanatorium treatment.	8,9,9,10	Tubercular infiltration both upper lobes, pulmonary fibrosis.	Quiescent pul.tuberculosis

M I N E R S (3).

No.	Initials.	Age.	Occupation.	Clinical Examination.	Previous History.	B.S.R.	X-ray Report.	Diagnosis.
49	J.S.	56	stripper	Bronchitis	Cough.	3,2,2	Not X-rayed.	Bronchitis
50	J.G.	62	stripper	Bronchitis, emphysema, hyperpiesis (B.P. 180/120)	Cough.	2,2,2	Root thickening, peribronchial fibrosis, aortic curve & upper mediastinum enlarged.	Chronic bronchitis, hyperpiesis
51	J.S.	47	machine-man	Bronchitis, emphysema	Sanatorium treatment.	4,8,5,4	Peribronchial fibrosis, root thickening.	Chronic bronchitis
52	W.S.	53	oncost	Bronchitis, haemoptysis	Cough & colds.	6,12,9,3	Root & peribronchial fibrosis.	Chronic bronchitis
53	J.K.	54	stripper	Bronchitis, pneumonia	Pneumonia in 1935 & 1936.	2,3,2,4	Not X-rayed.	Chronic bronchitis
54	J.C.	51	stripper	Bronchitis, emphysema	Cough.	4,3,0·5	Miliary deposits & fibrosis all over both lungs--occupat.	Pneumonoconiosis
55	P.M.	50	brusher	Bronchitis, myocardial weakness	Cough, influenza 1935.	6,4,4	Peribronchial fibrosis.	Chronic bronchitis
56	R.K.	26	bottomer	Tuberculosis, both lungs	Sanatorium treatment.	61,84,78,56,78,83,72,87, 104,109,102,88,106,116	Tubercular infiltration & cavitation both lungs.	Pul. tuberculosis, died April 1936.
57	J.F.	20	oncost	Bronchitis, haemoptysis	Haemoptysis 1935.	3,3,3,2	Peribronchial fibrosis, calcified root glands.	Chronic bronchitis, ?bronchiectasis
58	J.H.	42	machine-man	Bronchitis, emphysema	Cough.	9,5,7,9	Much peribronchial & root fibrosis.	Chronic bronchitis
59	W.M.	67	machine-man	Bronchitis	Cough.	9,5,6	Miliary nodulation & diffuse fibrosis both lungs--occupat.	Pneumonoconiosis
60	W.J.	60	brusher	Bronchitis	Cough & colds.	20,15,12,10	Haziness R apex, deposits R upper lobe & L middle area, diffuse fibrosis.	Pul. tuberculosis, ?low grade
61	T.R.	59	brusher	Asthma, bronchitis	Cough.	10,2,2	Miliary nodulation, diffuse fibrosis-occupat.	Pneumonoconiosis
62	W.B.	56	oncost	Bronchitis	Sanatorium treatment.	8,12,8,9	Chronic opacities both lungs, worse upper halves & R side.	Pul. tuberculosis, low grade
63	P.C.	20	drawer	Tuberculosis, both lungs	Sanatorium treatment, mother died of phthisis.	27,28,28,26,24	Not X-rayed; too ill.	Pul. tuberculosis
64	P.M.	55	brusher	Bronchitis	Cough.	3,2,2	Marked miliary mottling, diffuse fibrosis all over both lungs -- occupat.	Pneumonoconiosis