ARTIFICIAL PNEUMOTHORAX

in the treatment of

ACUTE LOBAR PNEUMONIA.

St. Olave's Hospital,
London. S.E.16.

15th December, 1931.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

T H E S I S

presented by

MALCOLM STEEL PURVIS, M.B., Ch.B.

for the Degree of

DOCTOR OF MEDICINE.

UNIVERSITY OF GLASGOW.

SCOPE OF THE THESIS.

The following sequence in relation to the order into which the various portions of this thesis are subdivided was considered, in the circumstances, to give the clearest explanation and to present the subject in the most logical way. It must be realised that this was not the order of events in which the problem was undertaken. As much of the technique of the treatment was evolved from previous cases, and as some of the theoretical considerations were modified by the results, it seems more sensible to place the cases before the theory.

I. INTRODUCTION.

II. LITERATURE OF THE SUBJECT.

III. DESCRIPTION OF CASES TREATED.

IV. DISCUSSION OF CASES AND LITERATURE.

V. THEORY ADVANCED TO EXPLAIN THE RESULTS.

VI. CORRELATION OF CASES AND THEORY.

VII. CONTRA-INDICATIONS TO THE TREATMENT.

VIII. CONCLUSION.

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X. X-RAY PLATES IN POCKET OF BACK-COVER.

INTRODUCTION.

The work for the following thesis was carried out in St. Olave's Hospital, London.

On account of the fact that no satisfactory theory had ever been advanced to explain certain phenomena of pneumonia, and also that no treatment based on known pathological facts of undoubted value was at present in use, the theory later to be explained was first suggested and the practical work in support of it then undertaken.

It is acknowledged that the number of cases so far investigated is small, and that much more still requires to be done. However, the results were considered well worthy of further trial over a larger field and hence a sufficient justification for publishing and thus placing this mode of treatment at the disposal of others who might be interested.

The practical work was commenced in June, 1931, and is still being carried on.

I have to thank Dr R. Kelson Ford, Medical Superintendent, for permission to publish the

cases and the radiological photographs.

A large indebtedness is acknowledged later to Dr J.J. Coghlan, who also kindly lent the sections from which the lung photomicrographs were made.

LITERATURE.

After careful search of text-books and current literature, it has been impossible to discover any advocate for the treatment of acute lobar pneumonia, per se, by means of inducing artificial pneumothorax.

Introduction of gas into the pleural sac during pneumonia is not original, although when first used in the following cases this was not known to me. Its use, up to the present, appears to have been confined entirely to the relief of the concomitant pain of pleurisy. Wynn (1), in an article on the treatment of lobar pneumonia, dismisses the subject in two lines, by stating that .." in hospital cases immediate relief (from pain) can be given by the insertion of 400-500 c.cm. of oxygen between the pleural layers with a pneumothorax apparatus. "

Taylor (2) discusses fairly fully the cause of pain in pleurisy and quotes three cases treated for this symptom by artificial pneumothorax.

He found that by introducing oxygen between the pleural layers immediate relief of pain was obtained. This result, he states, is accompanied by slower and more regular respirations, which are often followed by sleep. He concludes that the artificial pneumothorax lasts and maintains its effect for three to four days. The effect to which he refers must be the absence of pain, as he remarks immediately afterwards that a few days later a pleuritic rub often returns.

In his first case, a pneumothorax by means of 400 c.cm. of oxygen was tried in a girl on the third day of her illness. A vaccine was also given. Her crisis occurred on the fifth day, but, as in his other two cases, he passes no special comment on this.

In the second case, artificial pneumothorax was done on the fifth day of the illness and the temperature fell by lysis on the fourth to fifth day.

In his third case, a woman was treated by means of an artificial pneumothorax on the sixth day,

as she had entirely failed to respond to orthodox treatment. The result was exceedingly good and on the following day she had her crisis.

Further reference to these results will be made when discussing the present cases.

Burrell (3) suggests a more widespread use of artificial pneumothorax than is at present done and suggests its application to the treatment of cases with dry pleurisy.

CASES.

The following cases of acute lobar pneumonia were treated by means of an artificial pneumothorax. These cases were consecutive ones and were not specially selected. All were definite uncomplicated cases of lobar pneumonia, showing the usual signs and symptoms.

CASE I. C.J.

Male. Aet. 20. Previous history - nil.
Admitted 12 midnight, 21.6.31. T.102°. P.96. R.32.
He had become suddenly ill, with pain in the right side of his chest, three days previously.
On examination the right lower lobe was consolidated and a pleuritic rub near the base behind was heard.
At 2 p.m. on the 22nd inst. he was worse and the above figures had risen to T. 102.4° F. P.124. R.46.
The middle lobe was now found also to be affected by consolidation.

An artificial pneumothorax was induced on the affected side by introducing 400 c.cm. of air.
Half-an-hour previously he had been given morphia Gr.¼

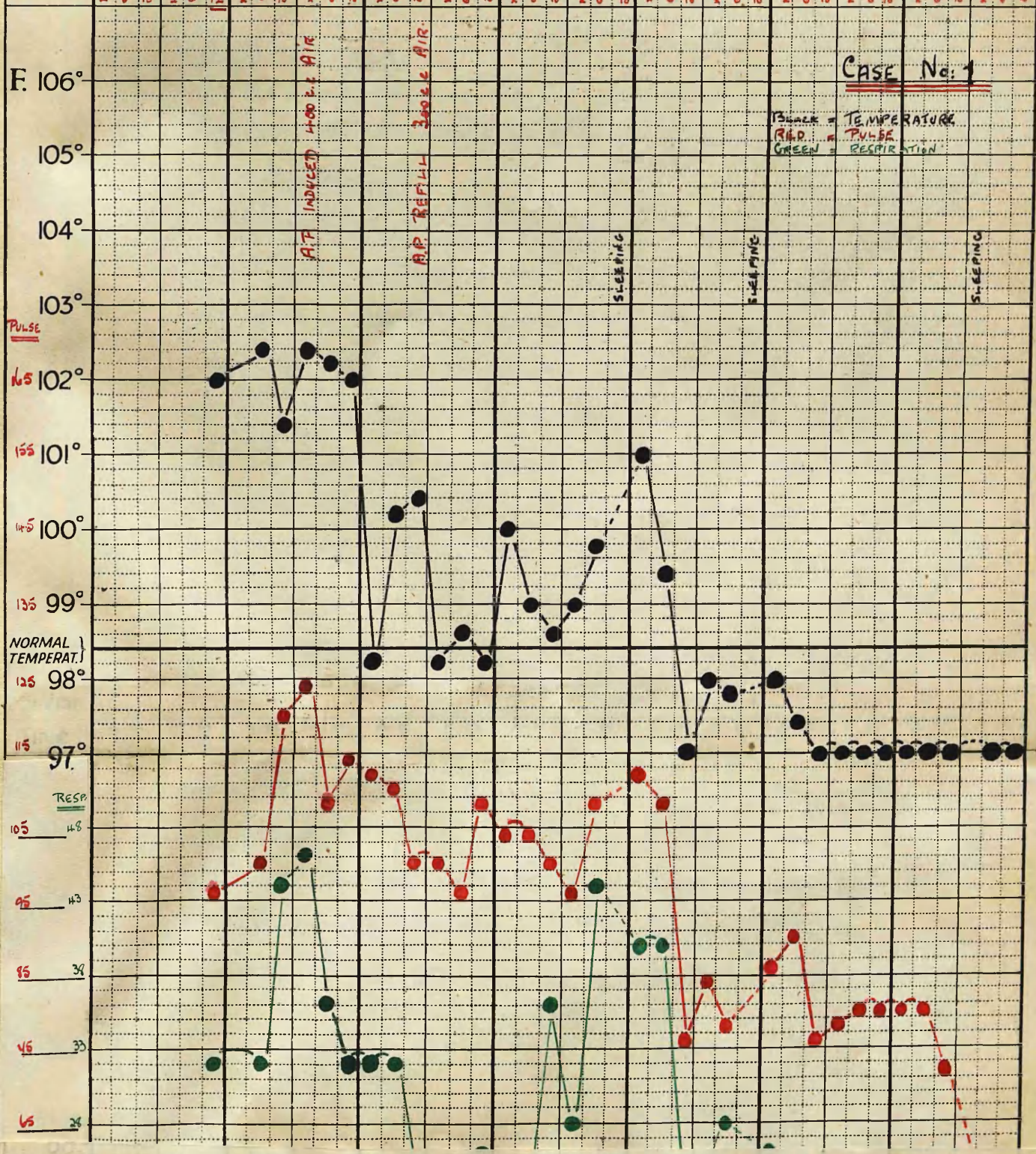
hypodermically and very thorough local anaesthesia was attempted with planocaine before the introduction of the needle. The manometer readings showed very wide variations and were of such a violent nature that further efforts to take them were finally abandoned. Despite the morphia and local anaesthetic, severe pain was experienced when the pleura was touched. The air entered easily and the lung apparently retracted, as it was no longer felt with the stilette. Within a few minutes the patient remarked on the diminution of the former pain, and soon afterwards his skin, from being hot and dry, was covered with a profuse perspiration. His cyanosis and dyspnoea also showed great improvement. Twelve hours later his condition appeared eminently satisfactory. His temperature was 98.6° F., pulse 112, and respirations 32. These, though rapid, appeared effortless; his colour was good and he felt fairly comfortable. Everything considered, he appeared to have had his crisis. He was next seen at 10 a.m. on the following morning and his condition

had reverted to that found prior to inducing pneumothorax. T. 100.4° F. P.106. R.32. A refill of 300 c.cm. was given, no manometer readings being obtained, on account of the violent oscillation of the fluid levels. Soon afterwards he again commenced sweating and at 2 p.m. the T.P.R. were 98.2° F., 100 and 24, respectively. Apart from two small rises of temperature shown on the accompanying chart, his recovery appeared to differ in no great way from that of a case treated by the usual methods. Seven days after the artificial pneumothorax a radiograph (Plate 1.) was taken. This still showed a small collapse near the apex. Resolution of the lung appeared to be taking place satisfactorily. He was dismissed five weeks later, the lung then being resonant, with a normal respiratory murmur and clear of adventitious sounds. He was kept as an in-patient much longer than his condition warranted, in case some untoward event might arise. When seen two months later, no evidence of the pneumonia could be found.

Form GH 332.

JUNE 1931

Date	21st	22nd	23rd	24th	25th	26th	27th
Day of Dis.	4th	5th	6th	7th	8th	9th	10th
Time	A M 2 6 10 / P M 2 6 12	A M 2 6 10 / P M 2 6 10	A M 2 6 10 / P M 2 6 10	A M 2 6 10 / P M 2 6 10	A M 2 6 10 / P M 2 6 10	A M 2 6 10 / P M 2 6 10	A M 2 6 10 / P M 2 6 10



CASE II. C.R.

Female. Aet. 9. Previous history of measles and whooping-cough.

Admitted 20.6.31. with a history of two days illness. The T.P.R. rates were 103° F., 128 and 30. She was not seen by me until the 4th day of her illness; she was then very delirious and appeared almost to be dying. The question of an artificial pneumothorax was raised, and, in view of the previous result, it appeared reasonable to attempt the treatment. The T.P.R. rates were by this time 104° F. 140 and 36. About 150 c.cm. of air were introduced, but, owing to an incessant cough, the exact amount is doubtful, as some of it became extravasated into the surrounding tissues and a surgical emphysema was produced. About half-an-hour later the T.P.R. rates had fallen to 100.2° F. 100 and 28 respectively. The child was perspiring a great deal and appeared very much more comfortable. One and a half hours later the house physician in charge of the ward

reported the temperature to be 98.4° F. and the patient asleep. The next day the patient appeared much improved, although the temperature chart (Chart 2) was not yet normal. In consequence, nothing further was done. The following day the child again became slightly delirious, the breathing was more difficult and she again had the appearance of a pneumonic person. During the day the symptoms became more marked, and in the evening a refill of 100 c.cm. was given. On this occasion the child objected strenuously to the treatment and much of the air was forced into the thoracic wall. However, a short time later, her general condition appeared much more comfortable, and, as will be seen from her chart, she seemed to be improving. No further treatment was given; she was dismissed three weeks later, perfectly well.

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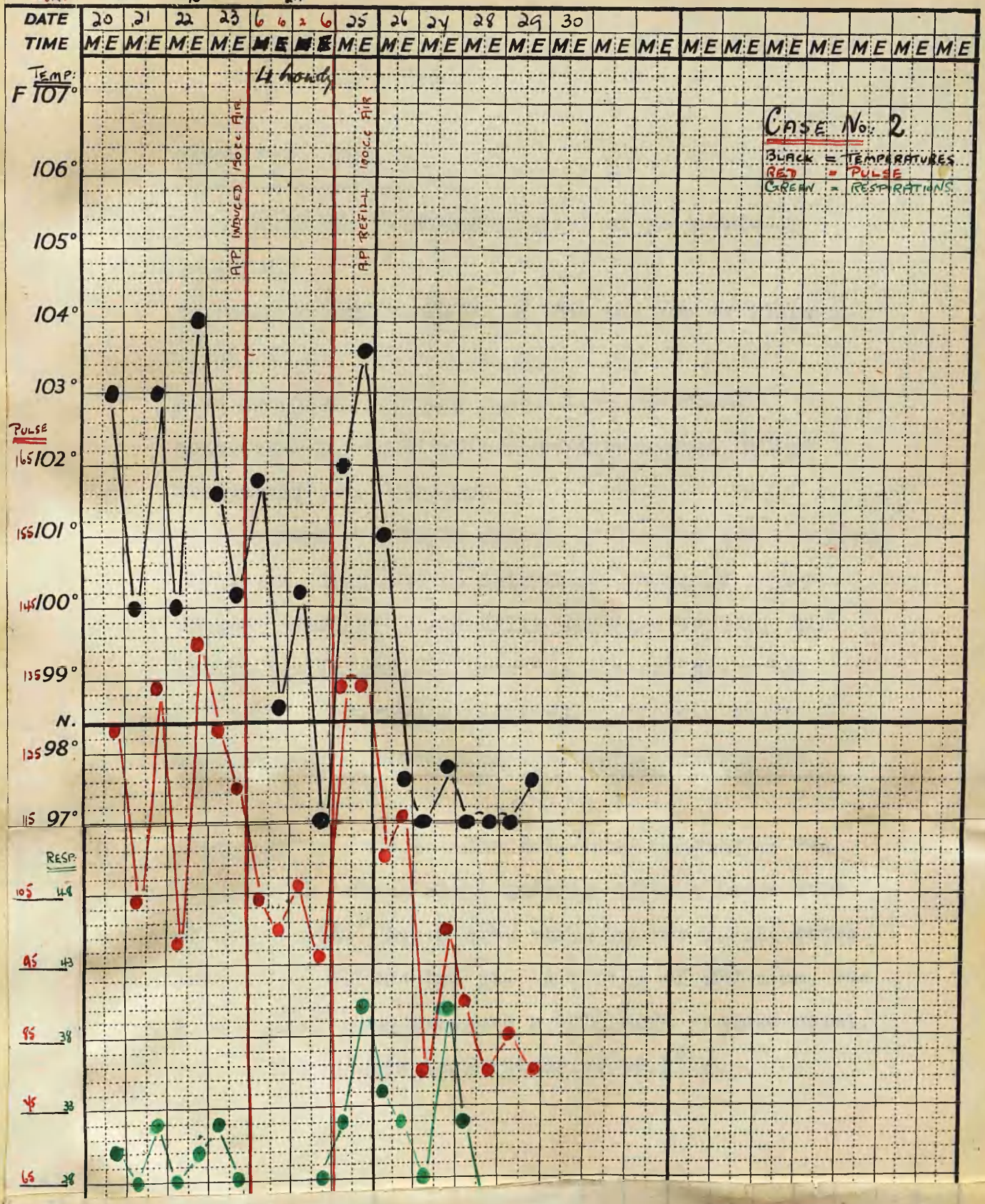
JUNE 1931



CASE III. G.A.W.

Male. Aet. 32. Previous history of pneumonia in childhood.

Admitted 6 p.m. 15.9.31. on the 4th day of illness.

T. 101.6° F. P.112. R.32.

When examined the left lower lobe was found consolidated, with typical tubular breathing and whispering pectoriloquy.

In this case a smaller amount of air for the induction was given, in order to determine the question of whether small or large fills were the better. The needle was introduced into the pleural space and 250 c.cm. of air were admitted. The pain abated considerably; almost immediately afterwards the patient started perspiring, though not to the same extent as in Case I. The following morning he was reported as having slept quietly quite a large part of that night. At 10 a.m. that day the T.P.R. rates were 101.2° F. 104 and 36. A refill of 200 c.cm. was given, which soon produced a much more profuse sweat, with a fall of his temperature to 99.8° F.

By evening the temperature had risen to its original level, but nothing further was done as he was sweating freely and did not feel unduly ill. This rise was quite probably of a post-critical nature. His further progress is seen from Chart 3, from which it can be seen that he settled finally on the following afternoon.

A radiograph (Plate 2), taken six days after admission, is shown. The left side shows a small pneumothorax near the base and a resolving lower lobe. During the rest of his stay in Hospital a very slight rise of temperature at nights (99°) was noticed, but clinically he appeared fit. With regard to this rise, it is of interest to note the suspicious shadows shown in his right lung and those in a further plate (Plate 3) taken 2½ months later. These are probably of a tuberculous nature, in which case, these or his small amounts of air may have been factors influencing a quicker reaction to his treatment.

September 1931

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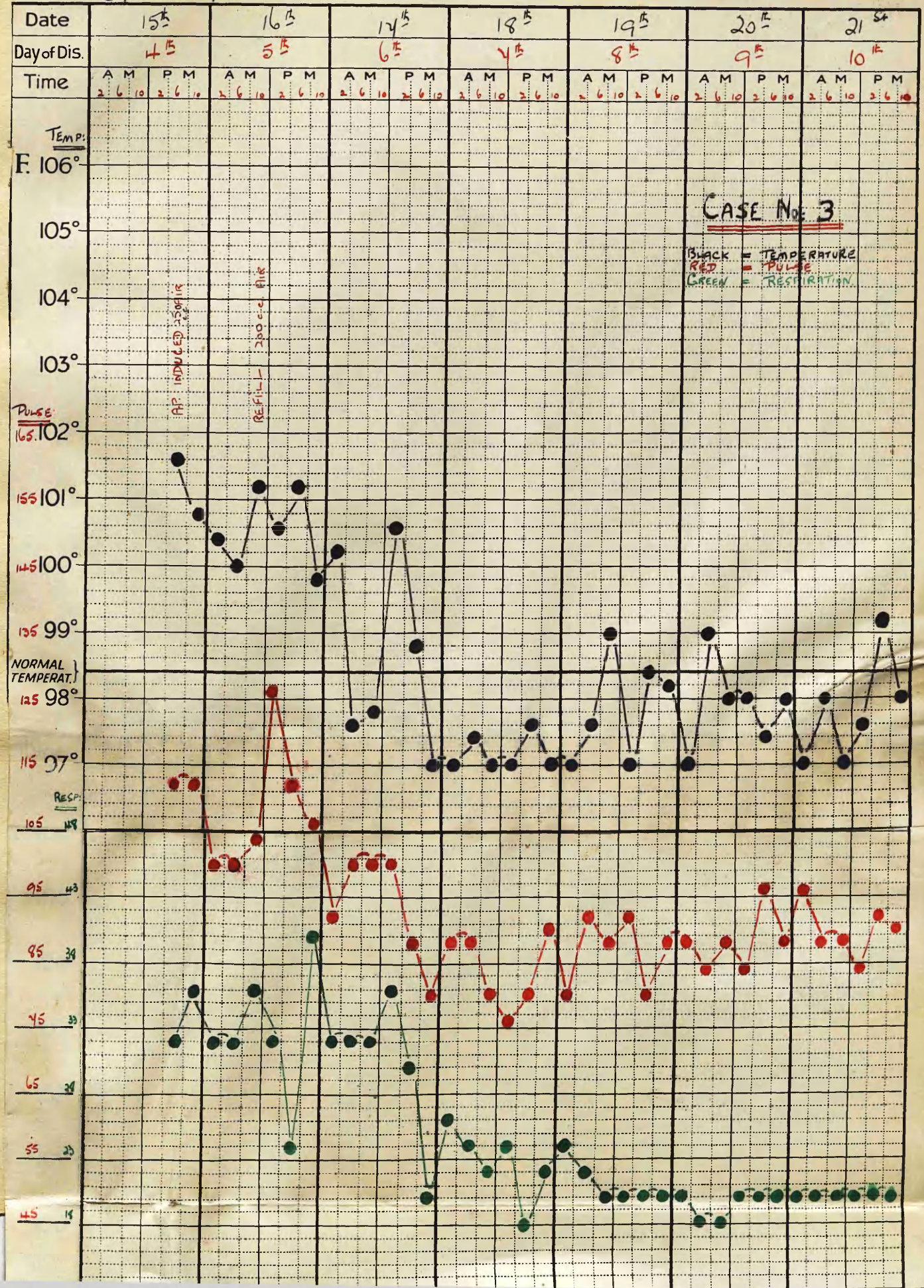

CASE IV. J.I.

Male. Aet. 55. History of cough every winter for many years previously.

Admitted 18.9.31. Appeared very ill with T.P.R. rates 99.8° F. 132 and 36 respectively. The day of illness was uncertain.

He was a thin emaciated man with great cyanosis and dyspnoea. His heart was enlarged to the right and the sounds were of very poor quality. The left lower lobe was consolidated and the remainder of his lungs contained a large number of crepitations and rhonchi. He was given some glucose and insulin, in order to nourish him, and half-an-hour later an artificial pneumothorax was induced by admitting 750 c.cm. of air. He commenced soon afterwards to perspire markedly and showed signs of improving. His cyanosis became much less marked and his breathing was much quieter. About four hours later he was not so well and his pulse was noticed to be very weak and rising in rate. The right lung was also much more congested around the base and, despite various cardiac stimulants,

he died about sixteen hours after treatment was commenced.

It is probable that the amount of air admitted was too large. The " crisis " may have been too great a strain on a heart which was in a presumably damaged state. A better mode of treatment might have been to introduce a small amount of air, and if, or when, necessary, to refill with further quantities.

It is unfortunate that no post-mortem examination was obtainable.

CASE V. H.N.

Male. Aet. 25. Previous history of several attacks of pneumonia.

Admitted 3.10.31. at 6 p.m. which was his first day of illness. On admission the T.P.R. rates were 104° F. 128 and 38 respectively. He was suffering from pain in the right side of the chest, just below the scapula. Nothing definite could be found.

By the 5th inst. the right lower lobe was obviously consolidated and no doubts remained as to the diagnosis. His T.P.R. rates were now 105° F. 124, and 32 respectively.

An artificial pneumothorax was induced, but, owing to the irritability of the patient, who was also of a very nervous temperament, only 250 c.cm. of air were admitted. The patient perspired well and in two hours the T.P.R. rates were 99.8° F., 100 and 22 respectively, while two hours later they had further dropped to 98° F. 88 and 20. Twenty hours later these had risen to 104.4° F. 112 and 28 respectively.

Here an attempt at refilling was made, but the patient was so antagonistic and restless that no more than 100 c.cm. could be admitted. This small refill caused a fall to 100.4° F. 100 and 28 of the T.P.R. rates respectively. He would allow no further treatment and his pneumonic condition returned. On the seventh day he had a normal crisis.

This case was of great interest and value, when compared with others of the series.

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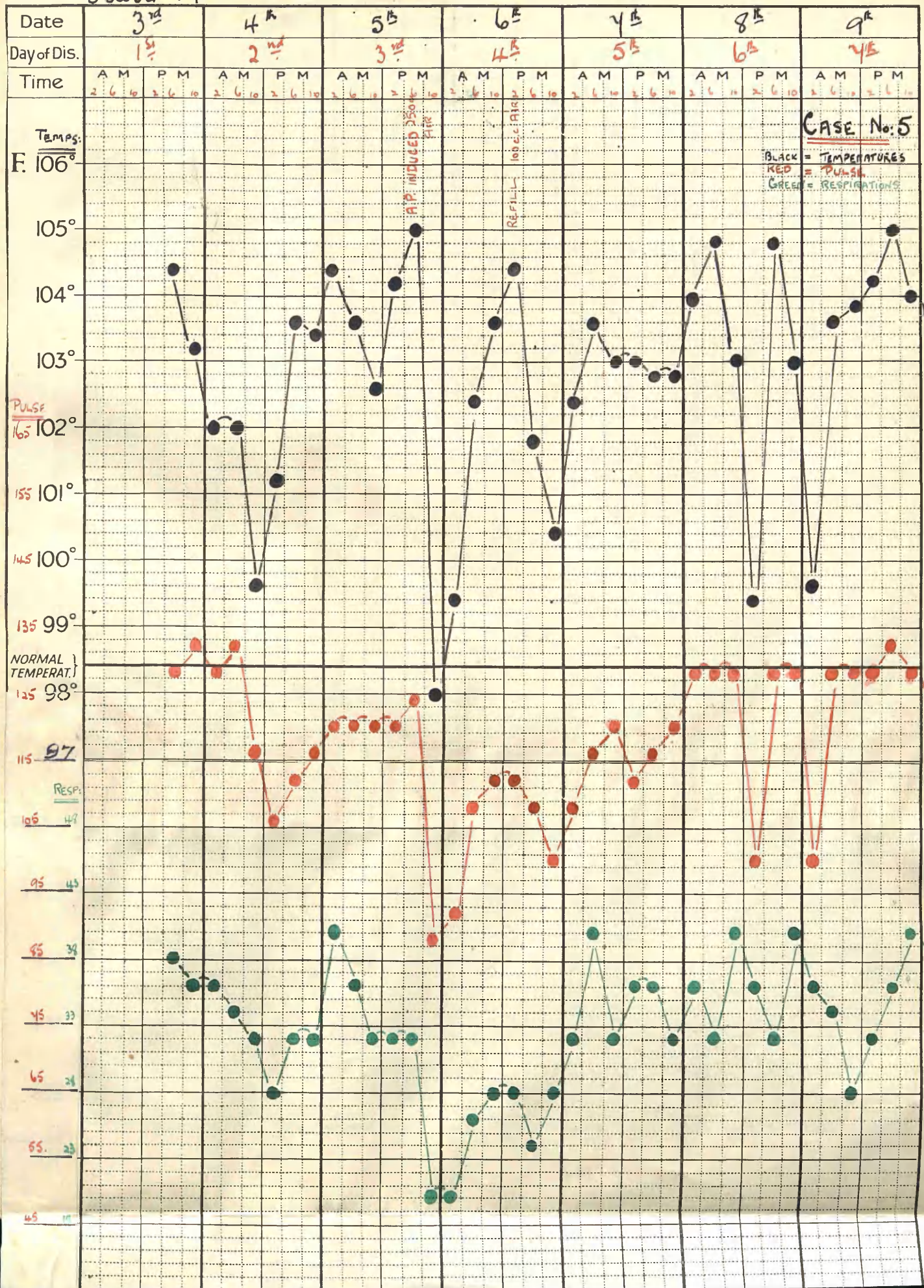
October 1931



CONTD

Form GH 332.

Date	10th	11th	12th	13th			
Day of Dis.	8th	9th	10th	11th			
Time	A M 2 6 10 P M 2 6 10	A M 2 6 10 P M 2 6 10	A M 2 6 10 P M 2 6 10	A M 2 6 10 P M 2 6 10	A M 2 6 10 P M 2 6 10	A M 2 6 10 P M 2 6 10	A M 2 6 10 P M 2 6 10

TEMP: F. 106° 105° 104° 103° PULSE 102° 101° 100° 99° NORMAL TEMPERAT. 98° 97°

RESP: 105 48 95 43 85 38 75 33 65 28 55 23 45 19

CASE VI. C.N.

Male. Aet. 23. Previous history - pleurisy 18 years previously.

Admitted at 2 a.m. on 6.11.31., this being the third day of illness. It had started early in the morning of the 4th inst. with stabbing pain in his right axilla. He was thoroughly examined about eight hours after admission and the right lower lobe was found consolidated. He was slightly cyanosed and appeared to suffer considerable pain. The urinary chlorides were diminished (400 mgms %) and he had a polymorphonuclear leucocytosis of 19,200.

An artificial pneumothorax was induced by admitting 600 c.cm. of air. The pain was markedly relieved practically at once, and when seen four hours later he was sitting up in bed reading a newspaper. He slept well that night and no further temperature was taken after 10 p.m. At 10 a.m. the next morning the T.P.R. rates were 100.8° F. 100 and 28 respectively. He complained of a return of the pain but not so severe as it had previously been.

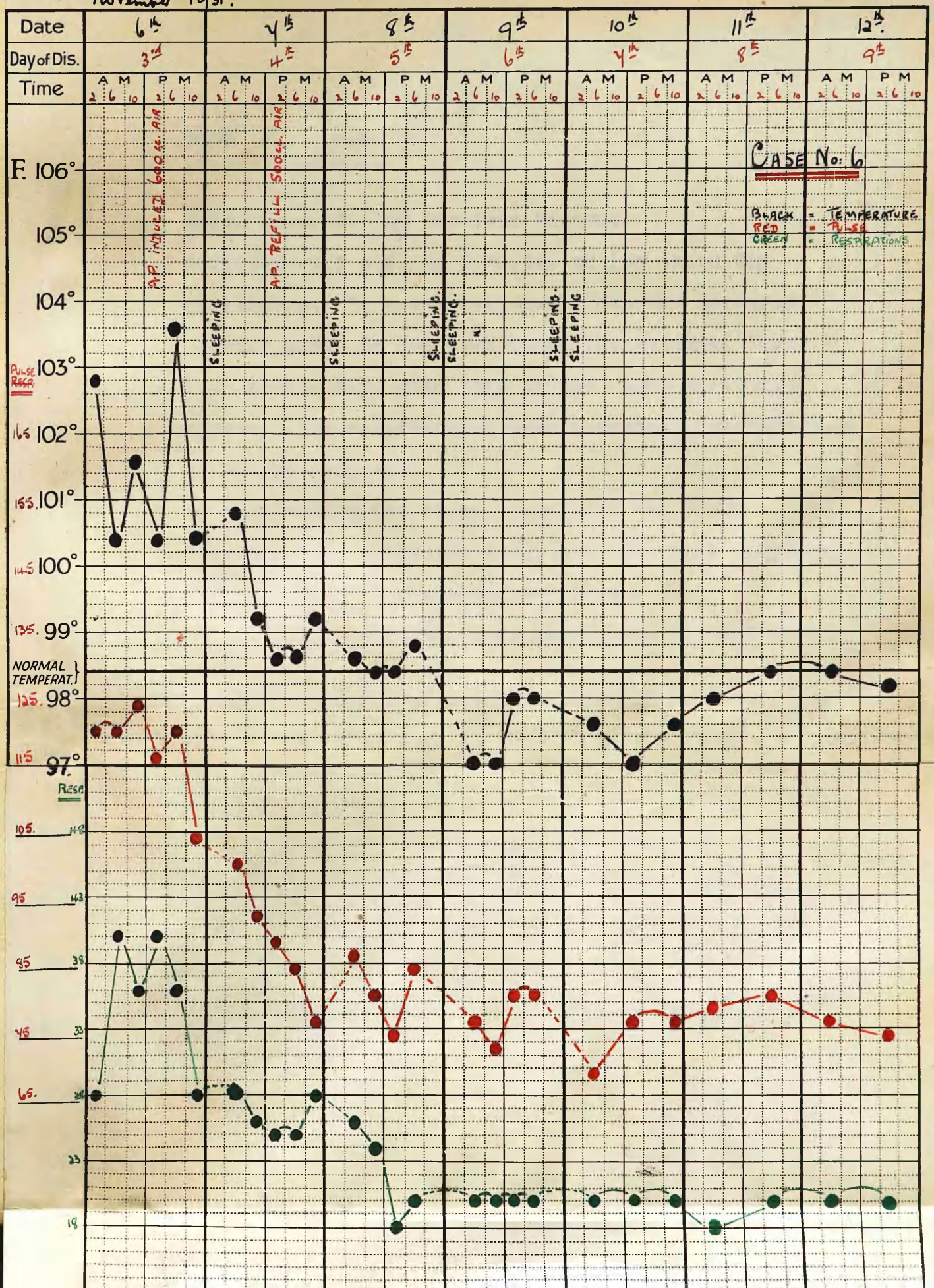
Twenty four hours after his initial fill a refill of 500 c.cm. of air was given. His temperature etc. from this point continued to fall, so that 48 hours from the commencement of treatment it was normal. On the 7th inst. his chlorides had risen to 640 mgms % and his leucocytosis had fallen to 14,200. The two following days the urinary chlorides were respectively 460 mgms and 510 mgms %.

A radiograph (Plate 4) taken on the fifth day of illness shows an artificial pneumothorax with consolidation at the right mid zone most marked at the periphery and traces of peribronchial thickening throughout the rest of the lobe. Resolution is well advanced.

A further plate (5) a week later, shows that the solid patch at the mid zone has now disappeared and the whole lung is clear, except for slight traces of peribronchial thickening, especially near the hilum. The artificial pneumothorax has completely disappeared and the whole lung shows a diffuse uniform shadowing, possibly due to the pleura.

Form GH 332.

November 1931.



CASE VII. J.R.

Male. Aet. 38. No previous history.
Admitted at 5 p.m. on 3.12.31. on the third day of illness. He was a well-built man who appeared sharply ill. He had been taken ill suddenly three days previously, with pain in the right axillary region. The respirations were increased, accompanied by slight cyanosis.
On examination the right lower lobe was found consolidated, while the lower lobe was very congested. The left side of the chest was clear.
The following day the right lower lobe was definitely solid.
On the 5th inst. 350 c.cm. of air were admitted to the right side, more being impossible owing to his excitable nature. The pain immediately disappeared, and he commenced perspiring. He slept very well that night and appeared much better the following day. As his " crisis " was not maintained, a further 650 c.cm. of air were introduced, and this caused very profuse sweating, with a great increase in

comfort.

That evening he was convinced by a priest of his imminent decease, and, owing to his consequent emotion, he would allow of no further treatment. His temperature finally fell by lysis two days later, and he made a quick and uneventful recovery. Two drops in his temperature, following the admission of the air, are clearly seen on his chart.

December 1931.

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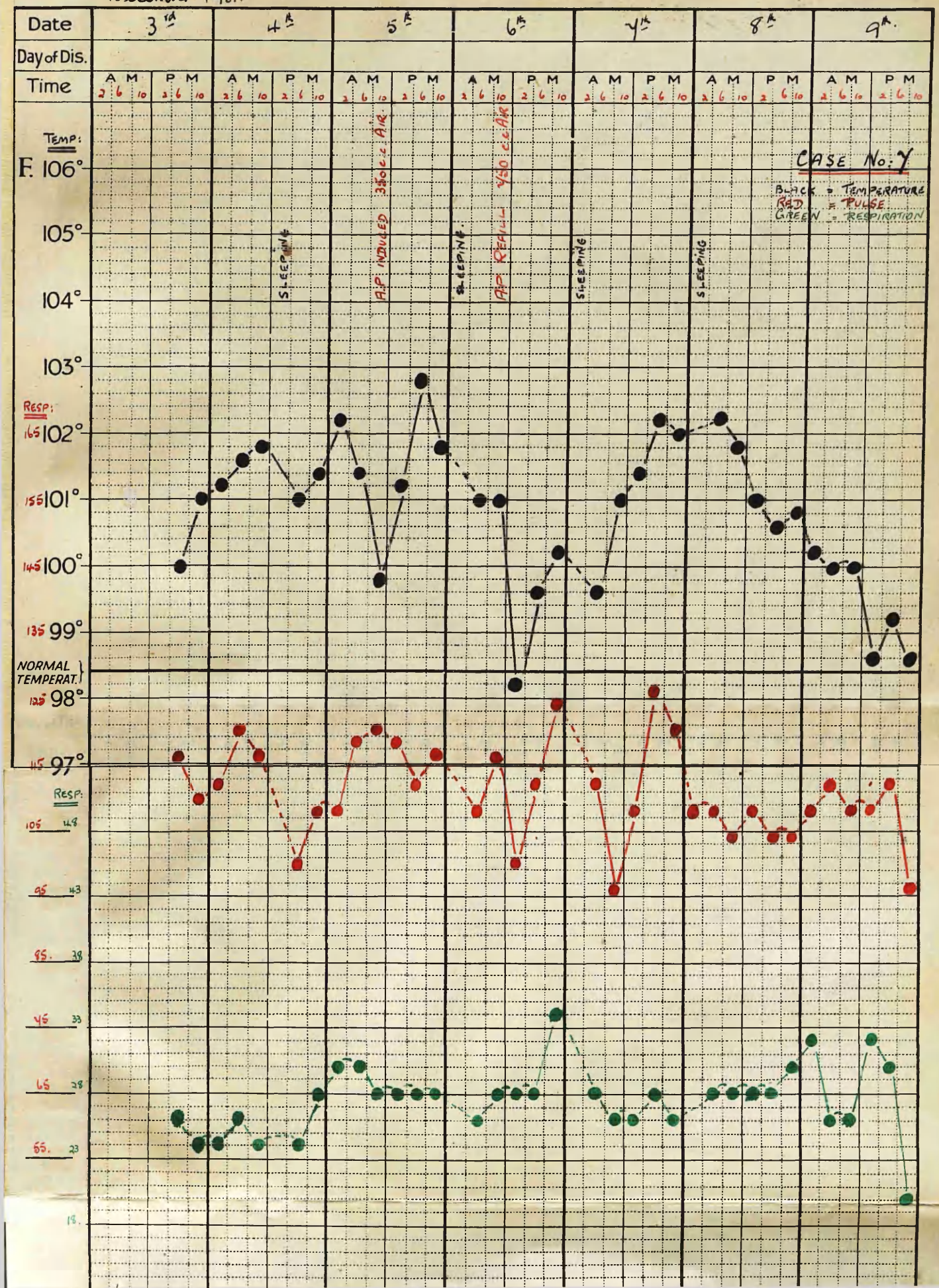


DISCUSSION OF CASES AND LITERATURE.

The cases which have just been described, along with those recorded by Taylor, when carefully studied and reviewed, give useful information as to the value of the treatment, the benefits to be derived from it, and the best technique.

Before starting to discuss these points, certain facts noticed, common to all the cases, will be mentioned.

The first thing of practical consideration met with is the pain which all the patients evinced on having the artificial pneumothorax induced. The pleura appears to be exquisitely tender in these cases, a point not to be wondered at, when it is remembered that it is in a state of acute inflammation. No matter how careful and thorough an attempt to anaesthetise the pleura is made, the pain is still considerable. It has to be recollected that local anaesthetics are supposed to have little or no effect in inflamed areas and, in consequence, it is thought advisable to give morphia.

The next point is that, when once the needle has been inserted, the manometer readings are so rapid and violent that they would appear to be of little practical value. Readings such as Taylor (2) quotes may be obtainable in some cases. With regard to this it may be mentioned that at the refills readings very like those seen in a normal person were found.

In considering the results which have been described, the first to be mentioned is the immediate and great relief from pain to be derived from introducing air into the pleural sac. This point is not the one of prime importance, but it is the first obvious and noticeable one. The fact of greatly abating or stopping the pain places the patient at once in a position more conducive to recovery. The worry and mental upset produced by the knowledge that at each breath or cough a sharp lancinating pain will occur is obvious. There is nothing more lowering than the presence of pain, and by its relief the patient, wearied by the fight taking place within, is likely to fall asleep, which, actually, often takes place.

The second, and more important, point is the production of a crisis, that is, a fall in temperature, pulse and respirations, and an obvious improvement generally. It may be argued that the falls in temperature obtained are in no way due to the artificial pneumothorax, but that these are coincident with a normal crisis. This view is untenable when the cases are taken seriatim and collectively.

In Case I a crisis was obtained after the introduction of air. Unlike a true crisis, this did not continue and two days later he was nearly as ill as he had been. A further fill of air produced another crisis, this time permanently. The reason for the relapse will be fully explained later, but, for the moment, it will suffice to state that it was due to the very rapid absorption of the air and release of the pleural pressure. Rapid absorption might be expected from the greatly increased blood supply to the inflamed pleura.

Case II closely parallels the above case, as here, also, the process started again when the air was absorbed.

In the third case a comparatively small amount of air was given. The effect was not so striking as in the two previous cases, and the effect wore off very much more quickly.

Obviously, if absorption accounted for the other " relapses ", then a case with a small amount of air would show this unfortunate occurrence sooner. The refill produced the desired results, a fact strongly favouring the conclusion that the fall in the temperature, pulse and respirations were due to the introduction of air.

Case V is of great importance from this point of view. Here was a man who, four hours after the induction of the artificial pneumothorax, appeared to have had his crisis. The amount of air used was again small and, by the following day, his symptoms had returned. A further smaller amount produced a transitory amelioration of his symptoms, and when nothing further was done for him he went on to the seventh day of his illness and then had a normal crisis.

Reverting to Case IV, a large amount of air was given. Without doubt a mistake was made in doing this. He was a man in a very poor state of health, and by introducing this large amount a sudden crisis, such as his heart could not be expected to stand, was encountered, but this has already been discussed.

In the sixth case reported, a moderately large amount of air was given with amazingly good results. A suspicion of a relapse was satisfactorily countered with a refill, after which nothing further required to be done. This case also helped to prove, by means of blood and urinary findings, that the crisis obtained was very comparable to a normal one.

Another conclusion which must be drawn is that the pneumothorax has only a temporary effect in holding the crisis. The air, as already stated, is rapidly absorbed, and, in order that the pneumonic process may not regain the upper hand, it is necessary to refill the pleural cavity. The necessity for this will be explained later.

This point, of necessity, raises the question as to the amount of air to be admitted and the length of time the lung is to be kept collapsed, but, unfortunately, owing to the small number of cases, this is quite impossible to answer accurately. It would appear that each case must be considered separately and on its own merits. In Case II, that of a child, a small amount produced a remarkable effect, while a similar amount, given as a refill, in Case V produced only a very little change. Again, the largest amount given was 750 c.cm. and here the only fatality which could be attributed to the pneumothorax was encountered. In Case VI two moderately large amounts were given and appeared in every way to be eminently satisfactory.

It must, however, be confessed that the amount to be used is still open to the method of trial and error. Certainly the small amounts appear to be safer than larger ones.

With regard to the length of time during which pressure has to be kept up on the lung, further

investigation may show the following to be incorrect, but in the present series a refill at the end of twenty four hours was found to be sufficient. Possibly, with smaller amounts of air, it might be necessary to refill more frequently and to continue the treatment over a longer period. Radiography shows pneumothorax to be present after the lapse of two days in Case VI.

With regard to Taylor's cases, it must be noted that oxygen was used, whereas in this series ordinary air was introduced. It has already been pointed out that the air appeared to be rapidly absorbed, and the likelihood is that oxygen would be even more so. This would release the pressure and allow the continuation of the pneumonia, as happened in his second case. The crisis in the first case may possibly have been due to the exhibition of the vaccine. In the third case the patient possibly was on the eve of having a crisis, which would be helped materially by the artificial pneumothorax performed.

The commendable points in the treatment noticed in these cases have been mentioned. It is also necessary to consider possible dangers. The most likely one is empyema. To minimise any risk of this, great care must be taken in the asepsis of the induction and refills. It was also considered politic to try not to injure the visceral layer of the pleura. Refills were performed in a different place from the initial puncture, in order not to irritate and injure the pleura at one particular point too much. No cases of either pleural effusion or of empyema were encountered.

The second serious danger is cardiac failure, due to the sudden crisis and the forcing of great strain upon the heart. The hoped-for event in pneumonia is the crisis, which must always be a severe strain, and if it is possible to bring on the crisis sooner than usual, then the heart ought to be in a state more fit to withstand the shock. One fatality is recorded and has already been discussed.

Of less likely occurrence, but possible are:-

a) Pleural shock. As this has been described as occurring when artificial pneumothorax was being done for pulmonary tuberculosis, it would have to be regarded as a possibility.

b) Pulmonary abscess and gangrene.

c) Pulmonary embolus.

None of these misfortunes was encountered, and it is thought that they should not be any more common than in cases left alone or treated on more orthodox lines.

The advantages of this treatment were obviously manifest, both to the patient and to the clinician. In every case, except that the child, the patients expressed their relief from pain, this occurring soon after the active treatment with the needle was finished. Their skin, from being hot and dry, became, almost within minutes, bathed in perspiration. Cyanosis, when present, either

disappeared or became greatly alleviated and the breathing, though often still rapid, was no longer rather painful to watch. Two of the cases slept moderately well their first night, an index to their comparative degree of comfort.

Another point of interest was that resolution, as evidenced by redux crepitations, tended to be more rapid than in ordinary cases, due, probably, to the shorter course of the illness, with, consequently, less damage to the lung. The sixth case returned home apparently fit and well fourteen days after admission to hospital, while a brother, one year younger, who was admitted the following day in the process of having his crisis (seventh day) was not fit for dismissal for three weeks.

From these cases the following technique has been formulated. It is inserted to serve as a guide to any who may wish to investigate the treatment, and must be used in conjunction with a full consideration of the case.

1. Morphine gr.¼ about half to one hour before commencing.

2. Thorough local anaesthesia. It is very difficult, if not impossible, to anaesthetise the parietal pleura.

3. Induction fill of 450-500 c.cm. of air. This would have to be lowered for (a) children (b) old persons.

4. A second fill, in about twelve hours, of 300-400 c.cm. of air.

5. Further fills if necessary and depending on the further course of the case.

In conjunction with this treatment one may give diaphoretic stimulant or expectorant mixtures. Such a therapeutic measure as Felton's Serum, in suitable cases, would probably prove of value. None of these measures was used in this series in order not to confuse the results.

THEORY ADVANCED TO EXPLAIN THE RESULTS.

A few preliminary remarks regarding the present treatment of pneumonia may be permissible. In no other disease can so many and varied forms of treatment be found. Many of these are contradictory, or at least appear to be so. Fashions come and go in treatment, and with the advent of each new therapeutic measure so a swing in that direction occurs. Most clinicians are in agreement over general nursing rules and attention to the principles of hygiene. In any scheme of treatment one finds that the room should be freely ventilated, the clothing warm but light, daily sponging should be given, and, along with that, a bland diet should be given. The bowels must have proper attention.

The above rules are those necessary for the treatment of any disease. No fault can be found with them. It is on further investigation that one finds great differences of opinion. Some people advise poultices and pneumonia jackets, which, it must be

admitted, appear to be comforting to the patient, while others are equally strong in their condemnation, and may suggest ice-bags, leeches, hydrotherapy or vaccine treatment. The value of any of these is doubtful. Anti-serums all have their advocates. Various so-called specific combinations of drugs regularly appear in medical journals, along with statements that since their use the prescriber has had no further deaths with pneumonia cases. Some teachers even preach a masterly inactivity. The fact of all this conflicting opinion, and that none of it is based on anything further than general broad principles, goes to prove that, up to the present, no satisfactory treatment has been evolved.

It is essential for the treatment of any illness that first the physiology of the part affected, and, second, the pathology of the disease be known, before any rational form of treatment can be suggested. In the explanation of the facts observed with the pneumonias, it will therefore be necessary to review the situation both from the

physiological and from the pathological points of view. The physiological aspects of the case will be treated first.

In considering the physiology of the lungs, it is essential to recollect that, in these organs, there is a blood supply from the heart, which is quite different from that found anywhere else. The blood is carried by means of two arterial streams, one, the smaller, being from the left side of the heart, while the larger amount is derived from the right side. The blood from the left side of the heart is arterial, and is distributed by means of the bronchial arteries which run in the substance of the bronchi and bronchioles. The other supply is carried by means of the pulmonic arteries and is venous. These latter arteries are found outside the connective tissue of the larger bronchi and they finally break up into a very fine capillary network, running between the alveoli. This network must be very large, as it supplies an area over a hundred times greater than the surface area of the body. (Howell) (3)(a)

Owing to the small length of the arteries and the large area into which they break up, the resistance to outflow from the right side of the heart is very small. Another point of importance regarding the outflow is the peculiar pressure to which the capillaries are exposed. On the one side, namely, the alveolar surface, the pressure is, for practical purposes, atmospheric. During inspiration it falls slightly and the reverse takes place during expiration. Again, the alveolar ducts are probably able to contract and raise the pressure slightly. On the other side the capillaries are exposed to the pressure in the pleural sac. This is always negative, except during forced expiration, when it can be raised to a greater force than that of one atmosphere. The capillaries are, therefore, for the most part, under a negative pressure, which is another important factor in explaining why the pulmonic pressure is so considerably smaller than the systemic. Howell gives the pulmonary pressure as somewhere about 25 mms. Mercury, or about one-fifth of the systemic pressure.

Another aid to the pulmonic circulation is the aspiratory effect produced by the thorax during inspiration.

The essential point of all this is that the pressure in the pulmonic system is low, and that only a small force, provided that it can be applied, is necessary to cause its arrest.

The main function of the blood is to act as a carrier from one organ to another. From the lungs it carries oxygen to all parts of the body and on its return gives off carbon dioxide. These are not the only substances with which it is capable of dealing. It is well known that, when certain drugs are administered, at least a portion of them is excreted via the lungs. Instances of these are creosote, alcohol and ether. This last substance suggests the fact that the lung can also absorb certain substances. The best known, by reason of their daily administration, are inhalation anaesthetics. The point of importance with regard to these is their potency when absorbed by the lungs.

McEwan (4) states that Fl.Oz.vi of ether may be given per rectum to induce anaesthesia. Such an amount volatilised and given at once would almost certainly produce death. The same thing is seen with chloroform. Glaister (5) cites the survival of a man who had swallowed Fl. Oz.v of pure chloroform, with intent to commit suicide. It would be impossible without fatal results to pour this amount into the lungs.

The significance of this is that anything taken up by the lungs has a very quick and marked effect on the brain. This is due to the fact that the substances absorbed by the lung when neurotoxic reach the brain quickly and undiluted by blood from any organ, as happens when drugs are exhibited in other ways.

As has been mentioned, the blood serves the purpose of carrying oxygen to the tissues, but as the main supply to the lungs is venous, it is difficult at first to see how the alveolar cells respire. It is possible that the venous blood still contains enough oxygen to supply the alveolar cells and these

cells may take up oxygen from this source. Haldane advances a suggested secretory activity for the pulmonary epithelium so far as the exchange of oxygen is concerned, comparing it to that found in the swim-bladder in deep-sea fishes, and if this is the case, there can be no reason for the cells not taking up oxygen for themselves.

Another possibility would be a supply from the bronchial arteries, as stated by Zoethout (6) though this seems improbable. The oxygen from the air has to pass through the alveolar cells in order to reach the blood, and as these are in direct contact with a source from which they may absorb this necessary gas, it seems most likely and probable that they are able to take it up for themselves.

Against this suggestion that the alveoli are supplied from the systemic system are sections obtained from a case treated for Tuberculosis by means of artificial pneumothorax, which died of an intercurrent intussusception. The lung had been

kept fully collapsed for eight months. Three sections are shown, each with a bearing on the present discussion.

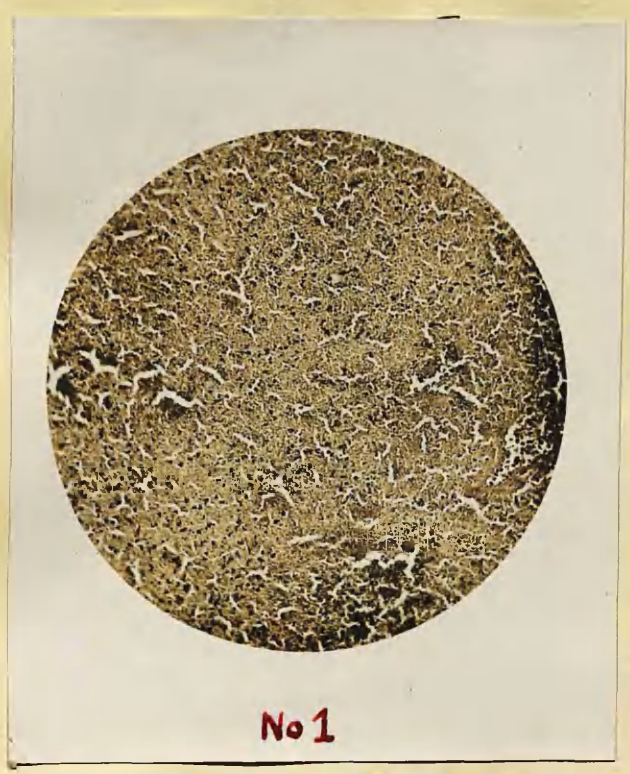
No. 1 shows an area of collapsed lung not much infiltrated by tubercle.. The alveoli are folded up, but the spaces are still represented by tiny interstices capable of holding air. The cells all appear fairly healthy, although little evidence of blood can be seen.

In the second section a bronchus is seen and is in a state of degeneration, being reduced to a line of columnar epithelium. The lumen is obliterated by cellular débris. Near by is a healthy pulmonic artery which is out of focus.

The last shows a group of cartilage cells, representing a broken-down bronchus. It is embedded in reasonably healthy alveolar tissue.

These sections alone are of very little value, but, taken in conjunction with other facts, they are at least slightly suggestive that pulmonary alveolus does not depend on an arterial blood supply

Sections from a Collapsed Lung.



No 1

No 2

No. 3

for its oxygen. It would appear, however, to be begging the question that these cells derive their oxygen from any other source than the abundant one of the air, except, perhaps, where this may not be available. Again, in support of this mode of respiration, infarctions may be considered. Normally, when the blood supply to an area is cut off, the cells die, the highly specialised ones dying first. Infarcts in the lungs are difficult to produce, due, without doubt, to the excellent collateral blood supply and also to the fact that the cells can survive owing to their direct supply of oxygen very much longer than in other organs. These infarcts, when obtained, are of the haemorrhagic type. Occasional anaemic infarcts are found in people with chronic bronchitis, and this can be explained by the increase of fibrous tissue shutting off the direct supply of air to some particular area, so that the cells are no longer able to obtain the oxygen direct.

This digression into the realms of pathology leads to a consideration of the process taking place in acute lobar or croupous pneumonia.

Pneumonia is a disease often looked upon as a septicaemia, accompanied by pulmonary lesions. By the word septicaemia is understood a condition where the blood is invaded by micro-organisms which multiply therein, giving rise to generalised symptoms and also local ones, the latter depending upon the organs which are attacked. Such a state of affairs is of grave occurrence, and the patient generally succumbs. In opposition to this, with pneumonia the symptoms are essentially local, and the general symptoms appear to be secondary to the pulmonic lesion. Pneumococci are sometimes to be found in the circulation of pneumonia patients, but a close distinction must be drawn between their presence, bacteriaemia, and their multiplication, septicaemia. In straightforward cases there is no evidence of this last occurrence.

Muir (7) defines lobar pneumonia as an " inflammation (which) starts at one place and then spreads by direct continuity involving the various structures in its course and leading to extensive consolidation of the lung substance."

The word " lobar" refers to the anatomical distribution and " croupous" to the fibrin which is found in large quantities in all pneumococcal processes, no matter where they occur. The disease is described in four stages, that is the inflammation, namely:-

(1) Acute congestion
(2) Red hepatisation
(3) Grey hepatisation
(4) Resolution.

Acute congestion is the first stage seen in any inflammation. Cohnheim, who gives us the classical description of this, describes first an increased rapidity in the flow of blood in the affected part, and this is accompanied by dilatation of the capillaries. In the lungs, therefore, it may be

expected that, owing to some debilitating factor, pneumococci gain entrance to an alveolus and commence to multiply. The surrounding area is stimulated so that more blood is brought to the region.

In the next stage of inflammation the blood slows down and the cells within wander out from the axial into the peripheral stream, and are found accumulated along the vessels' walls, through which they later pass by a process of diapedesis. This stage in the lungs is the commencement of red hepatisation. At this point of a lobar pneumonia the affected area is congested with blood which is circulating very slowly. Cells have commenced to creep out into the alveolar spaces and the interstitial tissue. Accompanying this there is an exudate rich in fibrin, which will allow of coagulation and further solidification. In the meshes of this fibrin are found great numbers of white cells, chiefly polymorpho-leucocytes. It is easily understandable that the increased blood and the distension of all

available parts with cells and fibrin soon produce a state in the usual sponge-like texture of the lung, which makes it comparable to that of liver. The phenomenon continues until such time as the alveoli are full of fibrin which has coagulated, forming plugs found in and blocking up completely the alveoli, atria, the alveolar ducts, and even some branches of bronchioles. The tissues surrounding these plugs are distended with the products poured forth into them. The inflammatory process is shared by the pleura, more intense over the actual area of involved lung. Except for differences of detail, the exact same condition is seen here.

The further stage in inflammation is final stasis of the blood, with death of tissues, due to deprivation of oxygen, the toxins of the invaders, and their own katabolic processes.

The next stage in pneumonia is that of grey hepatisation, and whether this ever occurs in cases which recover is a moot point. Some pathologists

deny it, but Muir (7) explains this difference of opinion by stating that, in a sense, there are two types to be found. In the grey hepatisation the alveoli and bronchioles are still found plugged with coagulated fibrin, but this has commenced to retract from the walls. The fibrin is also found to be in a state of proteolysis with an increased number of pus cells present. The red cells are in various states of haemolysis, many having disappeared. The surrounding parenchyma is much less congested. This stage of pneumonia corresponds to stasis in inflammation. With the cessation of blood to the part, death of the affected area would be expected, but, if correct, that the alveolar cells are capable of absorbing oxygen for themselves, then this event would not take place with such rapidity as in other areas. It is granted that the passages are blocked by a coagulum, but the nature of this being a gel, oxygen would still permeate to the imprisoned cells and help to keep them alive for a longer time than cells which depended on the blood for their oxygen.

It is also seen that by this stage retraction of the plugs has commenced, which would allow of the entrance of air. At the termination, or resolution, the plugs have still further retracted. Many have disappeared, epithelium is proliferating, and there is a diminution of the leucocytes. The autolysed products are absorbed by the alveolar walls, a small part is coughed up, and the lung returns to normal as in any other inflammation which ends favourably.

Somewhere near the termination of all this, the patient experiences, in favourable cases, what is known as a crisis. This is shown by a sudden fall of temperature, accompanied by a fall in pulse and respiration rates. Along with these, the skin, from being hot and dry, becomes cool and moist, the urinary output, along with the chlorides, is greatly increased, and the leucocytosis in the blood drops to more normal levels. The patient is suddenly changed from a person dangerously ill into one who appears on the highway to recovery.
The phenomenon is one of the most dramatic and spectacular in medicine. No entirely satisfactory

explanation is known, the most universally accepted being one based on Anaphylaxis. Cotoni, Trouche & Raphael (8), crystallising this theory, state that the crisis is the result of a conflict between the antigen and the antibodies, therefore a manifestation of sensitivity. The antigens stimulate the body to produce antibodies, which at first are not obtained in sufficient quantities. The concentration of antigen increases to such an extent that the body is forced to protect itself by drastic means, which it does by pouring forth lysins. These split up the albuminoid pneumococcal antigen, thus giving rise to a toxin which acts in an anaphylactic manner by giving rise to a crisis. This explanation all appears very difficult of comprehension. It would be imagined that the antibodies on their production would commence neutralising the toxins until they finally came to a point of balance, by a gradual process. Rather in favour of this, and against the above explanation, is the fact that in no other form of pneumoccal infection, such as occurs in the meninges,

or peritonaeum, is the termination by means of a crisis.

It would therefore appear that the explanation of the crisis lies in the lungs and not in the infection itself. The present conception of the termination of lobar pneumonia is the sudden mastery obtained by the antibodies over the antigens, this conquest also serving to explain the crisis. With such a crowning victory for the body forces, as evidenced by the complete change in the patient, it is curious that various complications, notably empyema thoracis should be comparatively common. The authorities quoted above state that during pneumonia they could find no evidence of an increased bacteriacidal power in the blood. The power of killing these organisms is low and remains so. With regard to this, it is interesting to quote Armstrong (9), who cites three cases: the first, where despite a high protective power developed in the patient's serum, an empyema supervened; the second, where although the protecting substances

made a delayed appearance, they nevertheless crept up to neutralisation point, only to be followed by pericarditis; and, lastly, where a fortnight after clinical recovery, virulent pneumococci were obtained from the patient.

From his experiments this same observer found that protective powers in the blood did not develop until the fifth to sixth day of illness, and then increased rapidly up to a maximum. As a clinical method of determining their appearance, he suggests that the fall in leucocytes, associated with the crisis, be taken as an indication. This fall was found in Case VI at a time before they were supposed to be present. These powers do not appear to be bacteriacidal, in which case, the body, as it has only a low power of killing off pneumococci, must find some means of disposing of large numbers at the crisis.

With these facts and suggestions in mind, it is now possible to advance the following theory to explain the termination by crisis of pneumonia, and the logical use of artificial pneumothorax.

In this theory I am entirely indebted to Dr J.J. Coghlan, an ex-colleague of mine, for the conception of the crisis and blood stasis taking place simultaneously.

It is on this assumption that the whole theory turns and the various facts are explained.

Given suitable conditions, the pneumococci enter the lung and cause an inflammation. The actual exciting factor causing the entry is immaterial here, the only difference being that it is particularly rich in fibrin which "clots", and the resulting coagulum fills up the alveoli and the terminal bronchioles and helps to distend

the interstitial tissues.

In and around this, multiplication of the bacteria occurs, with a rapid absorption of the toxins by the circulating blood, and the production of marked symptoms more severe than the amount of toxin would warrant, on account of the site of the absorption being the pulmonary epithelium, as already explained.

The circulation through the lungs is, ordinarily, easily stopped, as the pulmonary pressure is a low one, but, here, on account of the driving force exercised by the wide variations of intrapleural pressure being so great, the circulation continues until such time as the pressure brought about by the inflammatory products has become very marked. However, there comes a time when the pressure of exudate is sufficient to bring the pulmonary circulation

to a standstill, through the affected portion of lung.

This actual stasis of the blood would account for the crisis by the immediate fall in absorption of the toxins. The amount now possible of absorption would be so small as compared with the previous doses that the symptoms of toxaemia would practically cease, as they do when the crisis occurs. An analogous condition to this occurs in a suppurative process, as when absorption ceases the symptoms rapidly subside, although the actual infection may still be in a high state of activity. When this pressure stasis occurs, the process in the lungs is still active, the bacteria are still multiplying, and the stage of red hepatisation is at its height. Owing to arrest of circulation and

the increase of imprisoned toxins, the leucocytes are broken down and liberate proteolytic enzymes and alexins. These, along with the accumulating toxins, kill off the bacteria, which, like any other cell, cannot survive without removal of its katabolic processes. This last event is represented by the stage of grey hepatisation, which, therefore, is post-critical.

The coagulum is next liquefied, and, by the release of pressure, circulation recommences. Further absorption with proliferation takes place and this is the stage of resolution.

CORRELATION OF CASES AND THEORY.

Applying the facts observed in the treatment of pneumonia by artificial pneumothorax, it is now seen how this theory explains them all. Fairly soon in the course of pneumonia, it is found that the lung has become solid. This is generally about the second to third day. Until such time the signs in the lung are rather indefinite and suggest only congestion. Immediately solidification has occurred, the lungs must be in the stage of red hepatisation. This means coagulation present throughout the affected area, but not sufficient, on account of the aid given by the respiratory force, to cause a stoppage of the pulmonary circulation. When an artificial pneumothorax is induced, several things take place. Firstly, the inflamed pleural surfaces are separated and pain therefore ceases. Secondly, ~~as~~ WHEN the inflamed surfaces are no longer rubbing together, owing to the entrance of air into the pleural cavity, the lung can no longer expand as before, and one of the aids to the increased pulmonary circulation is removed.

The entrance of air would act in two ways; firstly, by ceasing to allow a high negative pressure to develop, thus stopping that aid to the increased blood flow, and, secondly, in the purely mechanical way of allowing the lung to contract down upon the contained coagulated material. The resistance to the pulmonary circulation would now be so great as to cause its stoppage and, by stopping all further absorption of toxin, an artificial crisis would be obtained. It has been seen that, unless a refill is given in certain cases, the symptoms all return. This is easily understood, if it is remembered that the pleura is greatly inflamed with consequent increased blood supply, and so a quicker absorption than normal can take place. Absorption of the air would remove the force which is keeping up the pressure, and by releasing the pressure the circulation would become restored with fresh absorption of toxins and continuation of symptoms. The pseudo-crisis of an ordinary pneumonia could similarly be explained by first the cessation of circulation, sudden release,

In this explanation of the termination of a lobar pneumonia, no attention has been paid to the part played by protective antibodies. The theory given is practically a mechanical one. It would appear that these antibodies neutralise the toxins and only make their demonstrable appearance in the blood at a time when the absorption of toxins must be slowing down. The appearance of the protective bodies is therefore to be considered as being due to decreased amounts of toxin which would require neutralising, rather than as a sudden increase of antibodies.

and then stasis again. A pseudo-crisis takes place just before the ordinary crisis and at such time the intrapulmonic pressure must be considerable, but probably not quite enough to hold up the blockage long enough for the body to gain the upper hand. The release of the pressure allows continuation of the process and soon afterwards enough pressure is obtained to stop the pneumonia finally.

The artificial method of stopping the circulation and so producing a crisis cannot be compared with the natural way, so far as efficiency goes; the former is permanent and the latter only transitory. By the artificial method variations are obtained with each breath, and as the air is constantly being absorbed, the pressure is becoming less and less, which explains the gradual return of symptoms found in the patients so treated. Again, by admitting air between the layers of the pleura, healthy portions of lung are collapsed and put out of action, which would explain the comparatively higher respiration rates of patients so treated.

CONTRA-INDICATIONS TO THE TREATMENT.

Having considered the theory and in conjunction with the cases, certain points bearing on this form of treatment can now be profitably put forward. For convenience, these will be tabulated.

1. The treatment is only of use in certain cases. It is definitely contra-indicated

   a) before consolidation has taken place. Prior to this event an artificial pneumothorax would not produce a stasis, as the lung has nothing on which to contract and therefore stasis could not be produced.

   b) in cases with pleural adhesions.

2. The most obvious dangers are:-

   Overfilling the pleural sac

   a) in old or very debilitated persons this may produce a crisis, the strain of which their hearts are not capable of withstanding.

   b) in normal people, as it may hold the circulation up too long, and the alveoli pass into a non-viable state. This would result in

      (1) Gangrene of lung or
      (2) Abscess of lung or
      (3) Eventual fibrosis of lung.

Gangrene and abscess really only differ in degree of size. Both are due to arrested circulation. In fibrosis the specialised epithelium would not be able to regenerate and the affected part would become replaced by fibrous tissue.

CONCLUSIONS.

In properly selected cases artificial pneumothorax is of immense value in the treatment of acute lobar pneumonia.

The advantages claimed for it over any other form of treatment are these :-

1. Very rapid amelioration of pain.

2. A crisis several days before it would normally occur when the treatment commenced at the beginning of consolidation.

3. A shorter stage of resolution.

4. Probably less liability to the formation of pleural adhesions.

Whether the theory is acceptable or not, the facts of the cases remain, and are worthy of further trial and consideration.

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