A STUDY

of

ACUTE LOBAR PNEUMONIA.

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

In the construction of this thesis the cases were, for convenience, numbered consecutively from 1 to 120 and throughout the contents certain numbers will be observed, inserted within brackets, after certain statements have been made. These numbers indicate the cases on which such statements are based.

In Part I. the "essential" elements or features of the diagnosis of Acute Pneumonia are considered namely temperature, pulse, respiration, lung condition & expectoration.

In Part 2. are considered those features which are observed during the course of an Acute attack but are not essentially characteristic of the illness, namely, the nervous, blood and urinary conditions &c.

PART I

A STUDY OF ACUTE LOBAR PNEUMONIA.

The following thesis is based on the study of 120, more or less complete records of patients, admitted into the City Poorhouse Hospital of Glasgow, suffering from Acute Pneumonia. Some of these are very incomplete, and are included either for their general bearing on the general statistical conclusions, or to illustrate certain points herein afterwards dealt with.

These patients were nearly all drawn from the submerged tenth, the flotsam and jetsam of Society drifting between the Poorhouse, the Model Lodging House, the Prison and the Street. The alcoholic habit, either in the form of prolonged drinking at low pressure, as it were, or of periodical drinking bouts, can be obtained in fully 80 % of the cases. The investigation therefore derives some interest from this alcoholic factor being so prominent. One would naturally expect therefore that all or nearly all of these patients to be physical wrecks, yet it is astonishing what a number were admitted with the present being their first illness. In the others, the previous illnesses were the usual ills that flesh is heir to; the diseases of childhood and of adult life, viz ; Enteric, Typhus, Scarlet and Rheumatic fevers, Ague, Bronchitis, etc., but of 86 recorded cases, in 23, previous attacks of Pneumonia were noted. At first sight this large percentage (27 %) would appear to favour the idea, that a previous attack renders a patient more liable to a subsequent one. But, on going more carefully into these cases, this impression is not indicated with such certainty. Thus of 19 cases admitted with a history of one previous attack, in 9 only did the second onset affect the same lung, the shortest intervening period being two years and the longest ten. In 10 cases the opposite lung was the one attacked, the shortest period here being 2 months. Thus the facts, that in 53% of these cases, the opposite lung was the one

affected and that when the same lung was the seat of the disease, it was after the lapse of years, rather militates against the idea that one attack, per se, renders a patient more predisposed to a subsequent one. The same impression was also favoured by the histories of the 3 cases admitted with their 3rd Pneumonia attack.

If a patient suffering from Acute Pneumonia comes under observation, we may, from his appearance, form an impression of the probable diagnosis. We then instinctively enquire for the duration of the illness, its form of onset and also the alleged cause. This leads us into an investigation of the History of Onset.

HISTORY OF ONSET.

In the majority of cases the onset is more or less sharp and sudden: with headache, feverishness, shivering and very frequently rigors. Sooner or later pain is complained of in one or other side. This pain may indeed be the first symptom but more usually it sets in after the general symptoms have been in existence for some time. In other instances the onset seems to expend its energies on the gastro- intestinal tract ; the symptoms then being complicated with troublesome vomiting and diarrhoea. Sometimes it is impossible to fix with precision the day of onset, especially if the Pneumonia has supervened on some pre- existent catarrhal or pyrexial condition.

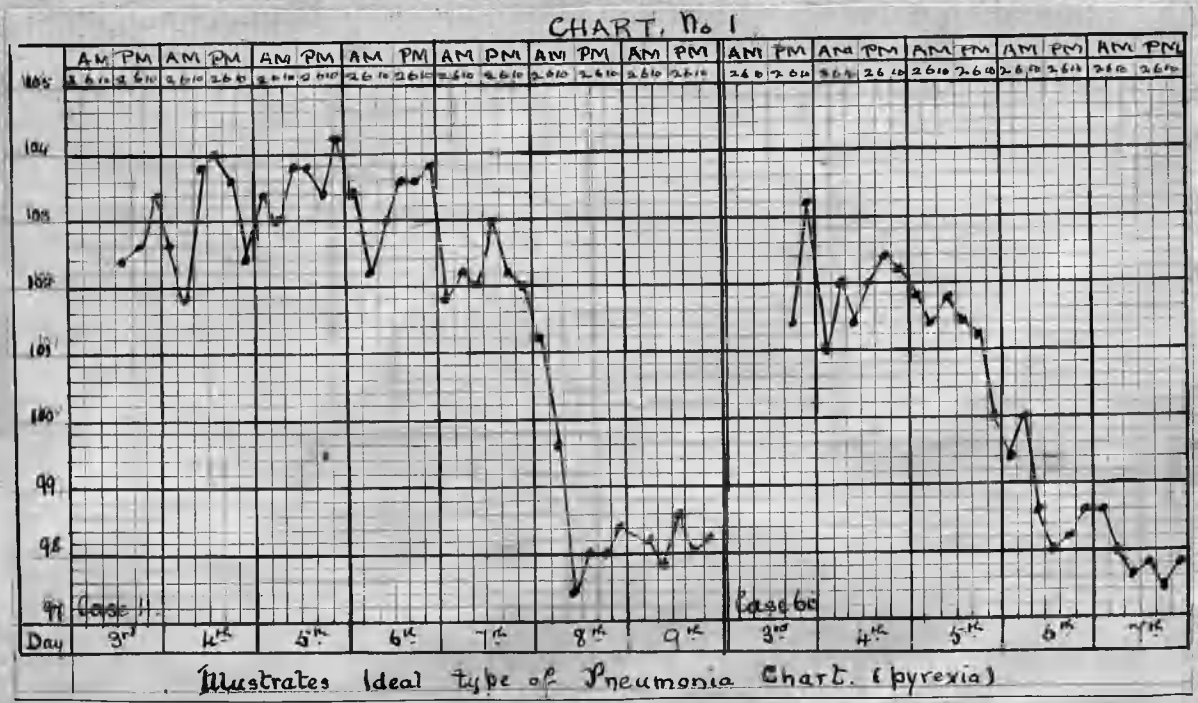
In numerous cases, exposure to damp or cold, either with or without alcoholism or other irregularity is adduced as the cause. In many instances the actual onset of the disease does not appear until 2, 3 or 4 days after the alleged exposure, but so frequently is this history of exposure voluntarily given, that it is difficult to exclude such, altogether, from being a potent factor in the causation of the disease.

Again a few patients attribute their present illness to some sudden

injury or violence. In this series four gave definite histories of injuries. One man was struck on the right side by a plank of wood which was being swung round by a crane. Next day he was feverish and ceased work. On admission on the 7th. day a bruised area was observed over a right apical pneumonia. Another stated thata blow on the right side was the commencing point of his pneumonia on the corresponding side. The remaining two cases are somewhat different. The first had his scrotum and penis lacerated with a block and tackle, whilst the second sustained a fracture of the 7th. left rib with the subsequent developement of a right sided pneumonia. Such types are generally explained by the accident of coincidence, but the histories given were so definite that it is difficult to exclude such injuries altogether from some causal relationship, very probably not proximate, but possibly predispositional. Owing to the shock, the vital activities of the nervous system may be so depressed, that an opportunity is given for the action of some potent proximate cause.

An idea of the onset having been obtained, an observer now proceeds to register the Temperature, Pulse and Respiration.

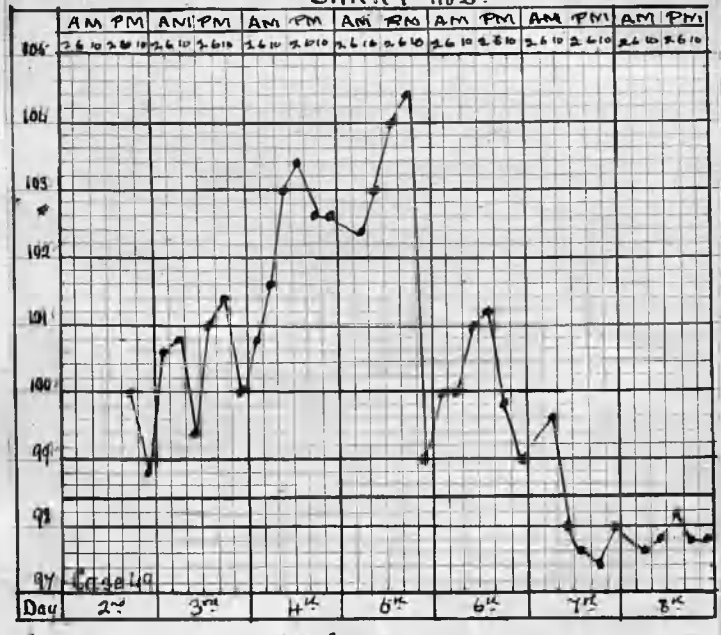
THE TEMPERATURE.



Illustrates Ideal type of Pneumonia Chart. (pyrexia)

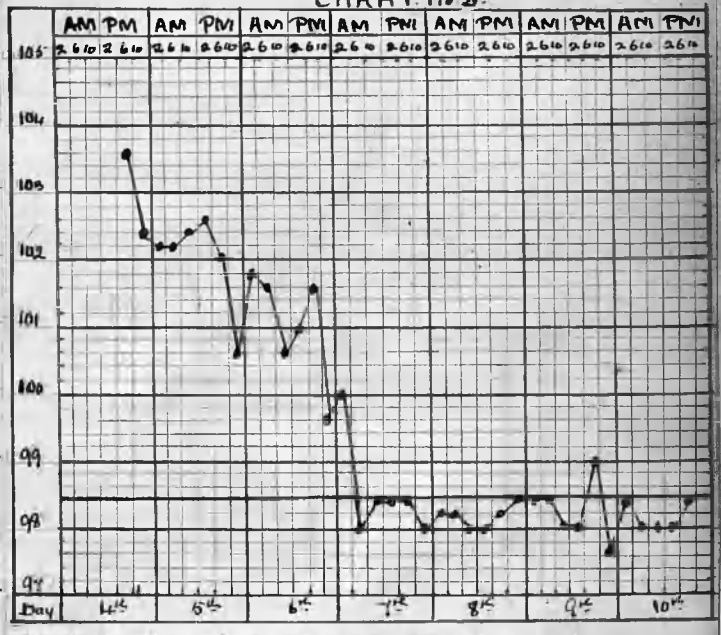
An analysis of these temperature records induces one to conclude that no general rule can be predicated of the rise progress and fall of the pneumonic fever. The ideal type (see chart 1. page 3) almost the only one I can produce) where the pyrexia sets in more or less suddenly and remains high with slight diurnal variations of 1° or 2° until the crisis, is exceptional, and my impression is, that cases characterized by such evenly sustained temperature generally prove fatal. (cases 48, 71, 78 etc.). During the fastigium the temperature varies considerably. The average mean level may be 103° or 104°, but, on the other hand, it may never exceed 101° even when both lungs are implicated. It may intermit one day : another day it may be sustained : then for the next day or two decidedly remittent. A few charts show remittency or intermittency throughout. In others again the temperature rises day by day to reach its maximum just immediately before the crisis (Chart 2) or it may reach its maximum early and then begin to fall

CHART No 2.



Illustrates a continuous rising temperature until crisis was reached.

CHART No. 3.

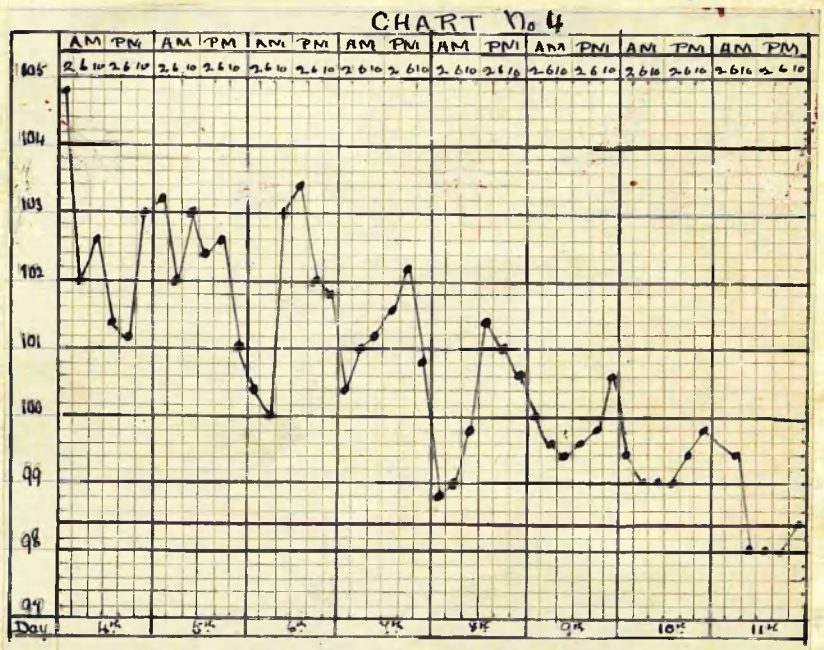


Illustrates falling temperature interrupted by a crisis

gradually. This declining temperature may be suddenly interrupted by a critical fall (chart 3), or it may fall steadily and gradually until the

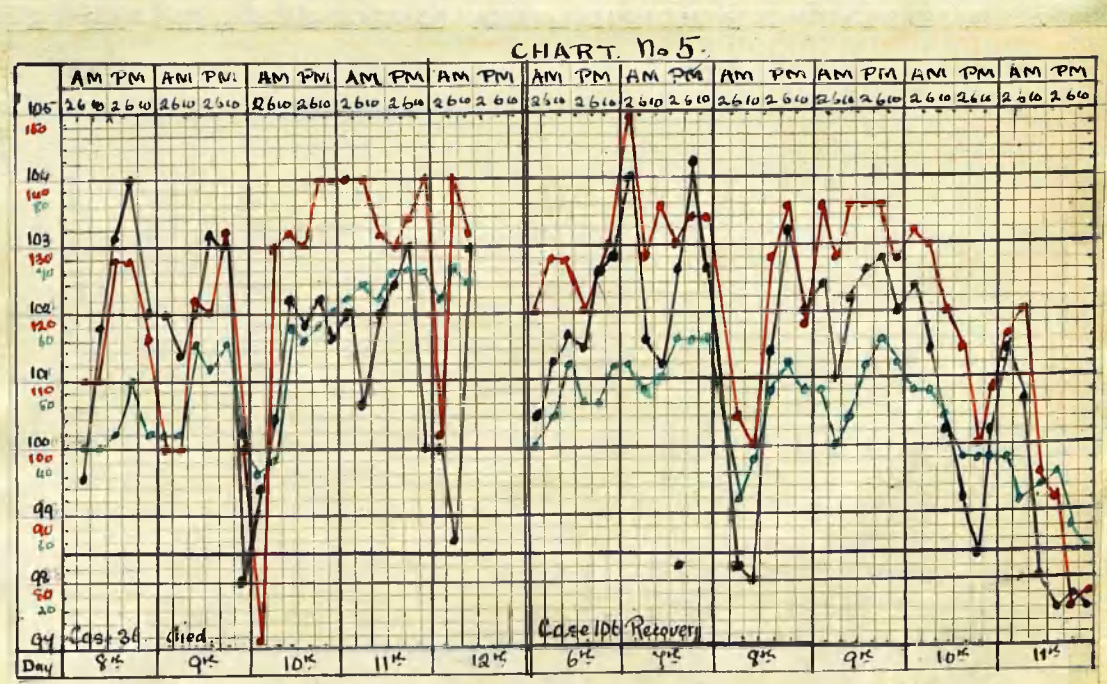
P.S. In the following Charts the temperature is registered in Black Ink
Pulse " " " Red "
Respirations " " " Green "

normal temperature level is reached (chart 4). Occasionally after the



Illustrates early maximum temperature with defervescence by continuous lysis

4th. day a considerable fall of temperature- it may be of 5° or 6° - to or near the normal level, with a subsequent increase of the pyrexia recorded. Such an incident is termed a False Crisis (see chart No. 5).

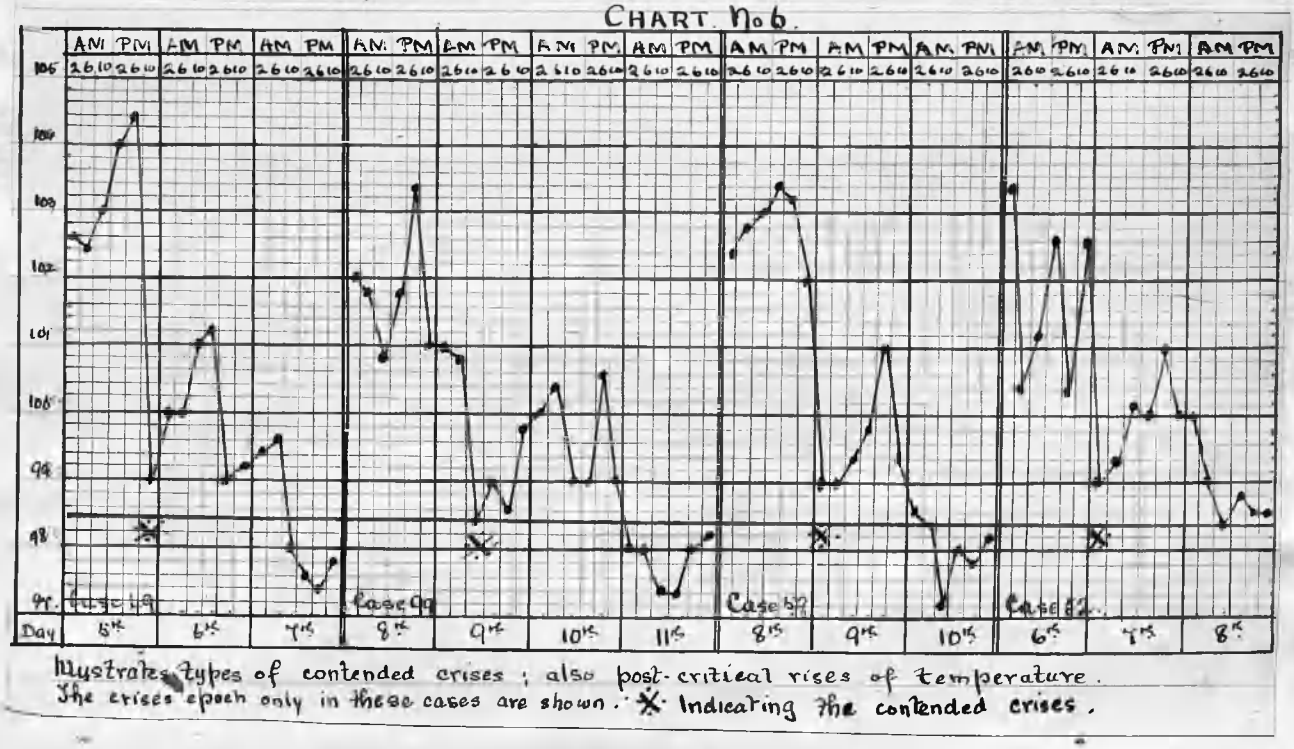


Illustrates two 'false Crises' both participated in by the temperature. pulse and Respiration.

Temperature is here indicated in Black.
Pulse . . . . Red
Respirations are . . . Green

With occasional examples it is difficult to assure one- self that such a critical fall is really false in its nature especially if associated with a decided diminution of the pulse and respiration rates , and a coincident appearance of a urate deposit in the urine, and also with an amelioration of the general symptoms without any exacerbation during the subsequent rise of temperature. In such a case, the question " May not this be a real crisis, followed by a post- critical rise due to some complicating and possibly unrecognized cause" ? is justifiably raised.

When a patient recovers the pyrexia most frequently disappears by a crisis, and by this is generally meant, a sudden and decided fall of temperature to the normal level 98°.4. In some instances the observer must be allowed some liberty in adopting for "crisis" purposes, the normal mean level, as occasionally, it would, otherwise, be somewhat difficult to determine the presence of a crisis, if 98.4 is rigidly adhered to. Thus in cases, 48, 52, 59 and 99 (see chart 6) the decided falls observed, although



Illustrates types of contended crises ; also post-critical rises of temperature.
The crises epoch only in these cases are shown. ※ Indicating the contended crises.

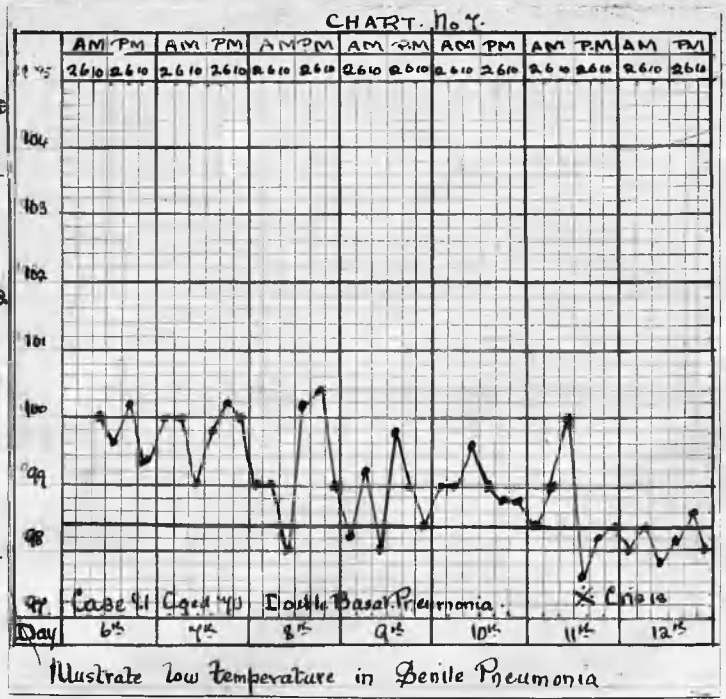
only to 99° in three of these, were in reality critical in their nature, and coincided with a marked improvement in the general condition of the patients. The subsequent slight rises would consequently thus be, post- critical in their relationship and of the nature of a rebound as it were.

With this reservation : of 71 patients who recovered, in 53 (74.5%) the termination was by crisis, the temperature falls varying from 2.5 to 7.5 in from 4 to 36 hours. A tabulation of these showed that the 7th. (8 cases 15.1 %) and the 9th. days (17 cases =32%) were the favourite critical days.

Table No1.

Day of Crisis	Total No. of Cases	Percentage Calculated
3rd	1	1·9%
4th	0	0·
5th	6	11·3%
6th	6	11·3%
7th	8	15·1%
8th	5	9·4%
9th	17	32· %
10th	5	9·4%
11th	4	7·5%
12th	1	1·9%
Total	53	99·8

The reliability of these figures however, cannot be trusted too much, as they are based on the histories as given by the patients themselves or their friends and relatives. In 44 of these cases (83%) the crisis commenced in the afternoon or evening, and seldom in the morning. In other charts the temperature curve is seen to fall more gradually. Then the temperature level of health is, generally not reached until a few days after the usual period. Indeed if by the 9th. day the pyrexia has not completely abated, one expects the defervescence to be by lysis (cases 96, 97 and 100). In old people the pyrexia itself may be very slight, but nevertheless it



Illustrate low temperature in Senile Pneumonia

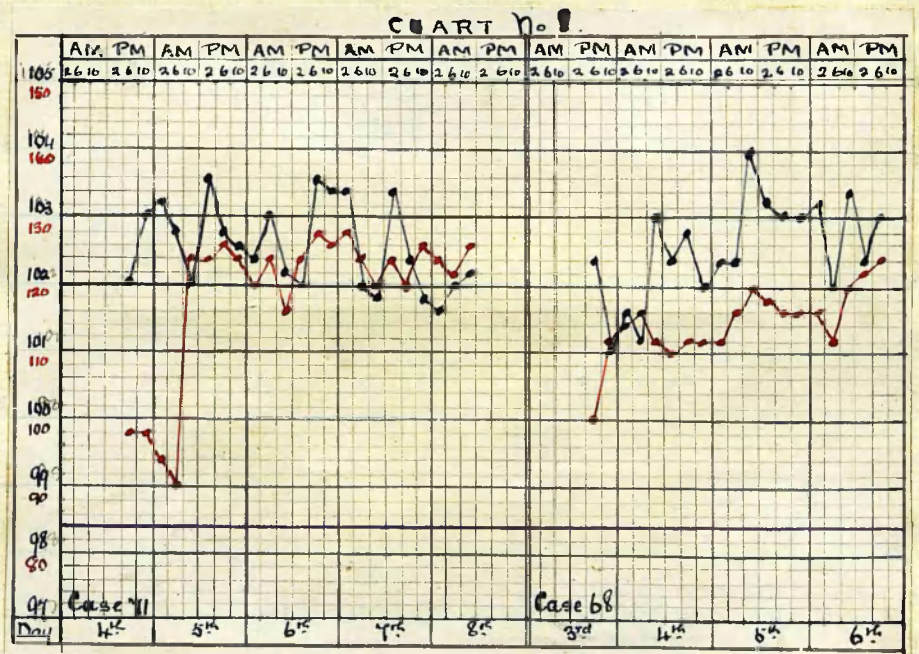
(See Chart 7. page 7)

it is perhaps the rule to find pneumonic senile patients with temperatures , averaging almost as high as in younger patients . (~~chart 7~~). One fact, however, stands out clearly in patients of all ages, viz " the pneumonic pyrexia is in no way proportionate to the lung implication."

THE PULSE.

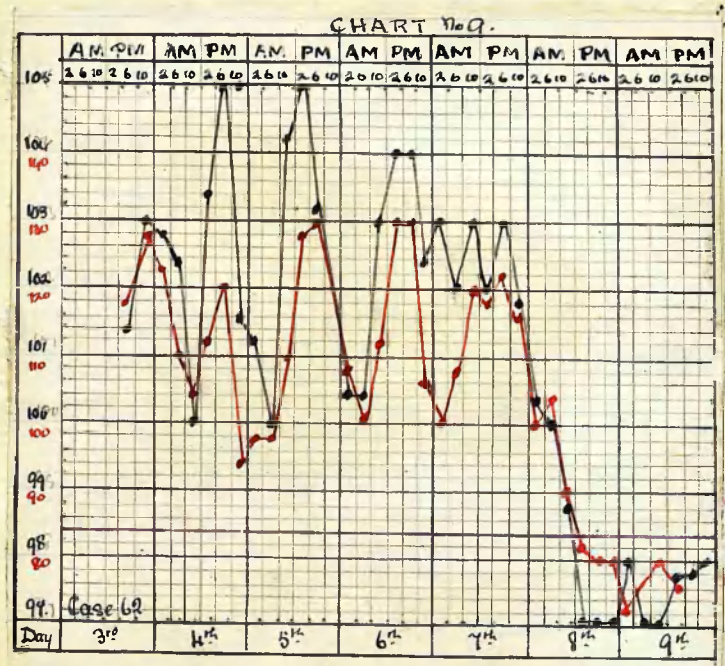
As a rule during the initial stages , the pulse is firm, rapid, and of increased tension, but when the disease is fully established it becomes full, soft, and easily compressible, and in a few instances dicrotism can be made out by digital examination. In later stages, pulse irregularity and even its diminution or disappearance during inspiration when cardiac failure is setting in, is observed. These statements can all be verified by sphygomographic tracings.

After studying the pulse records, it is observed that the pulse rate

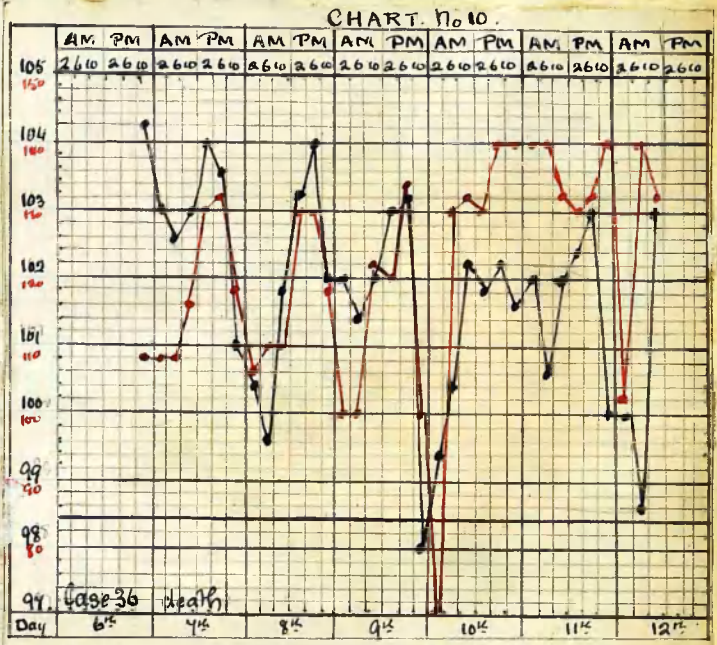


follows the temperature variations with astonishing regularity. If the pyrexia is sustained so also is the pulse (chart 8). The same correspondence is observed whether the temperature is intermittent, remittent or irregular .

(see charts 9 and 10 page 9)



Illustrates correspondence between remittent pulse & Temperature.



Illustrates irregular pulse coinciding with irregular temperature.

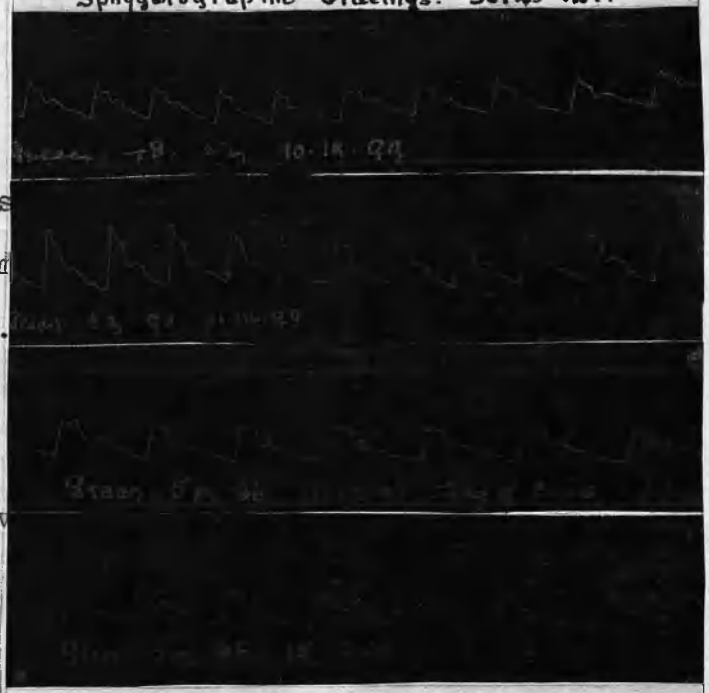
It is only in odd cases, where marked pulse variations independent of the temperature changes are noted. The same co-relationship is observed, during defervescence, whether the pyrexia falls by crisis (cases 11.91, and 117) or by lysis (cases 3 and 97). A high temperature however, does not necessarily indicate a high pulse rate, although the variations of both may still closely correspond. It.would thus appear that these two phenomena are either closely interdependent or related to the action of some cause common to both. The temperature and pulse mechanisms are each closely related to, and controlled by the nervous system, and possibly it is through this agency that they are influenced. Sphygmographic tracings show many facts about the condition of the pulse, much more clearly and in greater detail, than is possible by the digital palpation. Thus after examining these records it would appear to be the rule for dicrotism to be present even in cases which recover. It may exist for two or three days, but again it may only be noted on day before the crisis, but in others again , although the pulse tension is low, it is not

fully dicrotic (tracing No. 1). If the signs of Cardiac failure set in, then this is reflected in the sphygomographic tracings by increased rapidity and marked dicrotism with occasional hyper dicrotism of the pulse (see series No. 2.). Irregularity in the rythm also appears and occasionally the influence of inspiration is observed. On the day of death, when mucous rattling is heard in the tracheas, great differences may be noted in the character of the tracing as shown in series No. 3. in which 5 tracings are contrasted, and in one of these (McAlpine. 1st. of series) dicrotism was scarcely present. When recovery sets in, it is remarkable how quickly the pulse tension improves (tracing series No. 4). It was astonishing to find that dicrotic tracings were almost always obtained at some period in the great majority of cases associated with subsequent recovery. Thus, dicrotism, although it indicates low pulse tension, cannot always mean cardiac failure or insufficiency per se. It is possible that a reasonable explanation of this can be deducted from the experiments of Schülein, who showed, that although in the majority of pyrexial conditions, a

page 11.

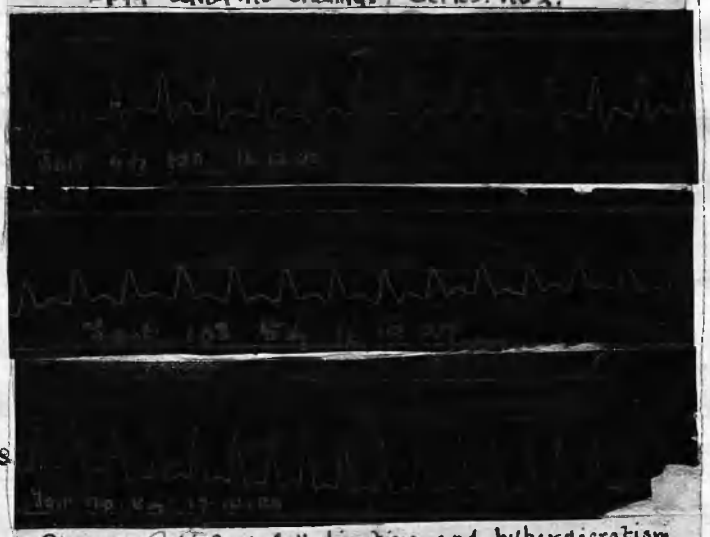
page 11.

Sphygmographic Tracings. Series No 1.

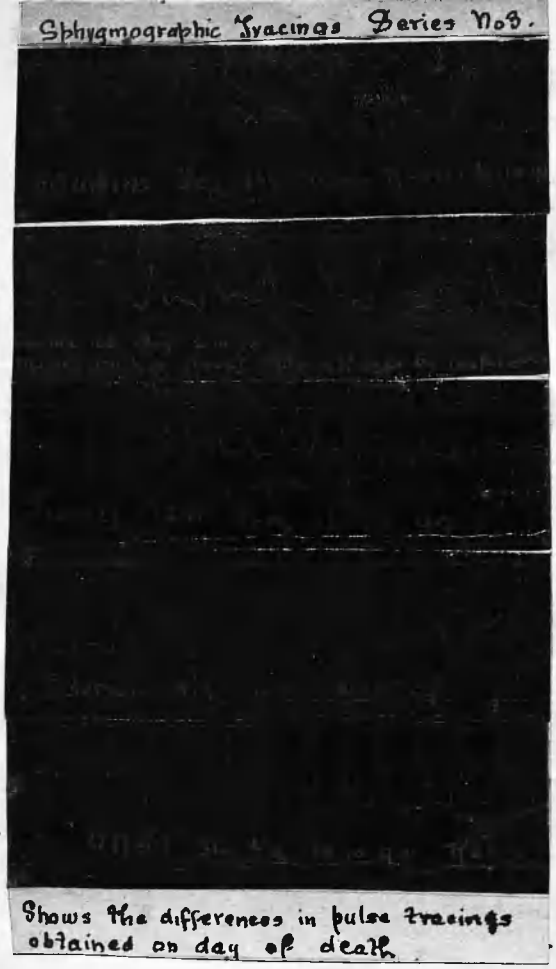


Showing a series of tracings in a case of pneumonia where the pulse was not fully dicrotic. Note however the rapid return of tension after the crisis.

Spygmographic Tracings. Series. No 3.

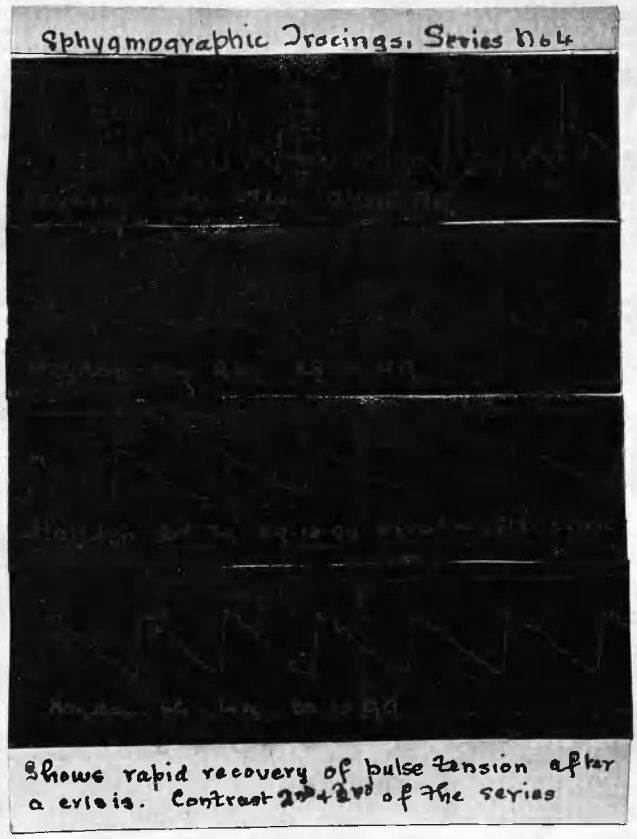


Showing Rapidity & full dicrotism and hyperdicrotism associated with cardiac failure and death.



Sphygmographic Tracings Series No3.

Shows the differences in pulse tracings obtained on day of death



Sphygmographic Tracings. Series No4

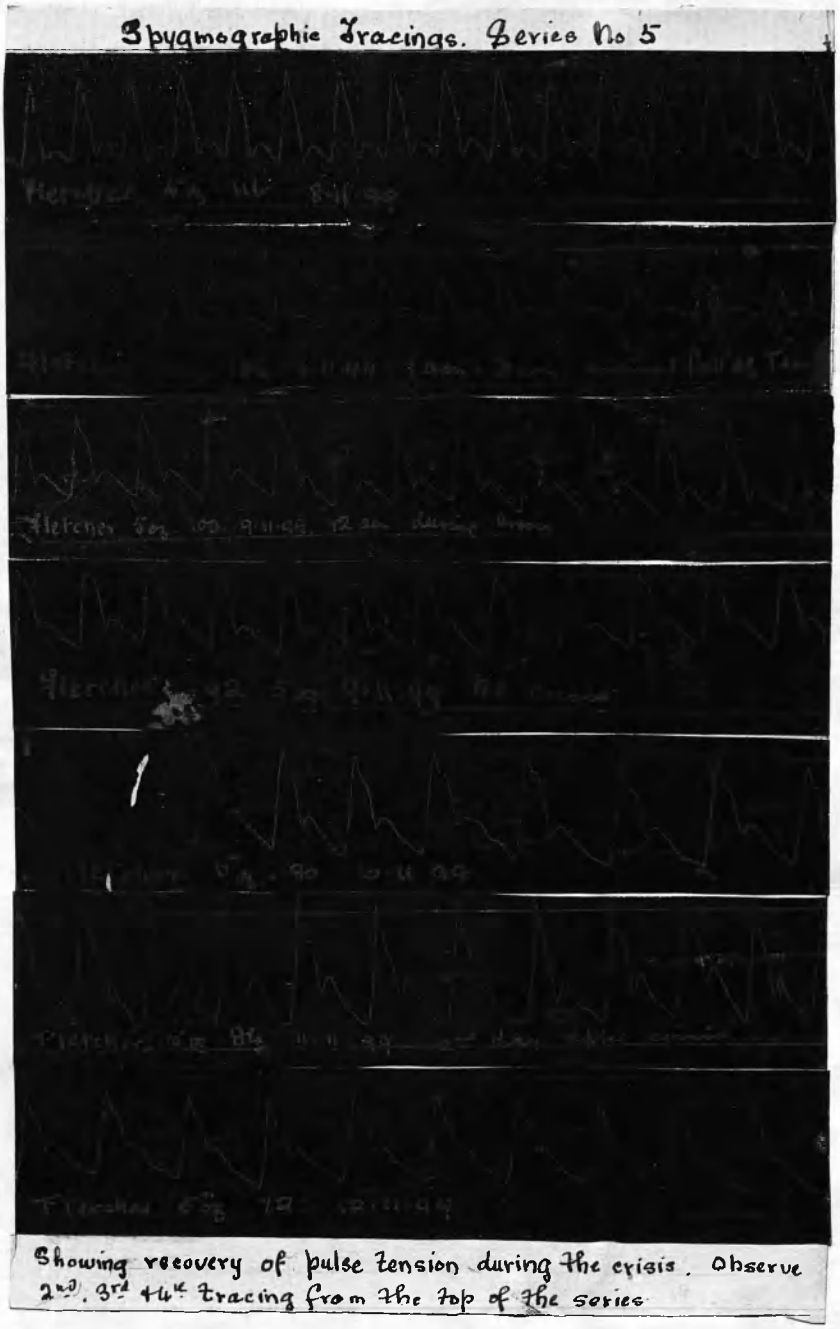
Shows rapid recovery of pulse tension after a crisis. Contrast 2nd & 3rd of the series

--------oOo----------

a high internal temperature is usually associated either with a cold or moderately warm skin, in Acute Pneumonia the surface or skin temperature almost equals the internal temperature, and the variations of both coincide. (see Fagge- Smith's system of Medicine). This great external heat- the probable cause of the hot pungent skin of pneumonia - can only be kept up by great radiation, and possibly to some extent by increased local generation. . This is probably associated with a considerable dilatation of the cutaneous capillaries, and perhaps the capillary circulation generally. The dilated capillaries by drawing a large quantity of blood from the arteries, would thus tend to lower the arterial blood tension. Such a hot pungent skin must also be associated with a greatly increased loss of moisture from cutaneous evaporation as well as that lost through the breath, which in this disease is both hot and rapid. This loss of fluid will tend to diminish the total volume of blood in the circulation; so further lessening the arterial tension.

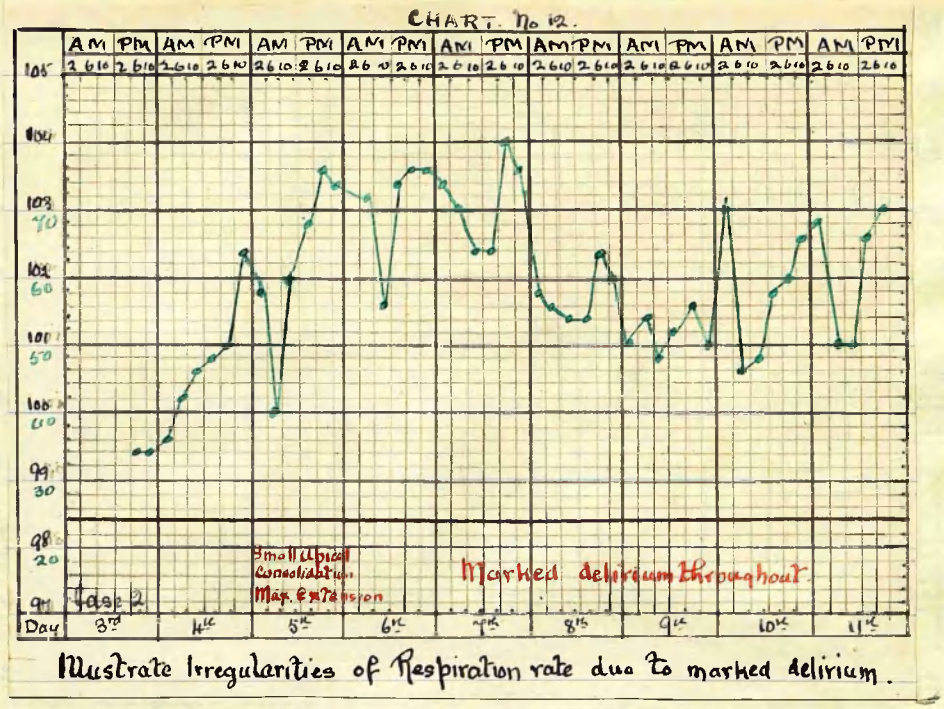
This explanation is rendered possible if not probable by the pulse tracings of one case I was fortunate enough to obtain (see tracing No. 5 page 13) Here at the commencement of the crisis the pulse was dicrotic; during the critical fall this was lessened, and finally when the crisis was completed the dicrotism had disappeared. One can suppose that at the crisis, the pneumonic poison and its influence on the nervous system had been overcome. With this return of nervous control, the capillaries would contract. The capillary capacity would thus be lessened, and in this manner the volume of blood in the arterial system would be augmented and so raise the arterial pulse tension as shown by the sphygmograph. If dicrotism really indicated cardiac weakness per se, it is unlikely that such a debilitated heart would recover so rapidly as to exert so decided an influence on the arterial tension within so short a period. Although the arterial tension is greatly diminished, the muscular power of the heart, may still be good, and although unable to overcome this lowered arterial tension, is still able to respond sufficiently to the necessary requirements of the system. Thus when dicrotic pulse is noted , indicating a low tension in the systemic circulation, an accented second pulmonic sound, and epigastric pulsation with co-related loudness of the cardiac sounds of the same area , may be observed. This shows that such a heart is still able to respond sufficiently when the blood tension (as in part due to the contraction of the capillaries and arterioles themselves- in this instance the pulmonic) is normal or increased, although it may coincidently be unable to throw sufficient blood into the arterial circulation to make good that taken into the dilated systemic capillaries. It is even questionable owing to the obstructed pulmonic if the heart could overcome this if it would, as it itself must be receiving circulation a smaller supply of blood into its own chambers. Moreover at a crisis the pulmonary consolidation and obstruction does not disappear at once, yet the dissappearance of dicrotism is rapid and complete. To sum up therefore it

would appear that dicrotism (full) is not necessarily due to cardiac weakness or failure per se, but is a reflex of the physical conditions brought about by the systemic capillary dilatation and diminished total volume of blood due to the loss of fluid from the skin and from the breath.



Showing recovery of pulse tension during the crisis. Observe 2nd. 3rd + 4th Tracing from the top of the series

with the exertions of a delirious patient. (see chart No. 12).



Illustrate Irregularities of Respiration rate due to marked delirium.

The reduction of the normal pulse respiration ratio from the proportion of 4 to 1 to anything from 1 to 1, 2 or 3 is commonly observed but as the respiration is somewhat stable in its course, and the pulse apt to be very irregular, the pulse- respiration ratio is subject to very considerable variations and may vary from 1 to 1 up to 3 and ½ during the acute stage of the illness.

Apart from the discomfort and restraint caused by the presence of pleuritic pain the respiration rate seems to be influenced mainly by three conditions.

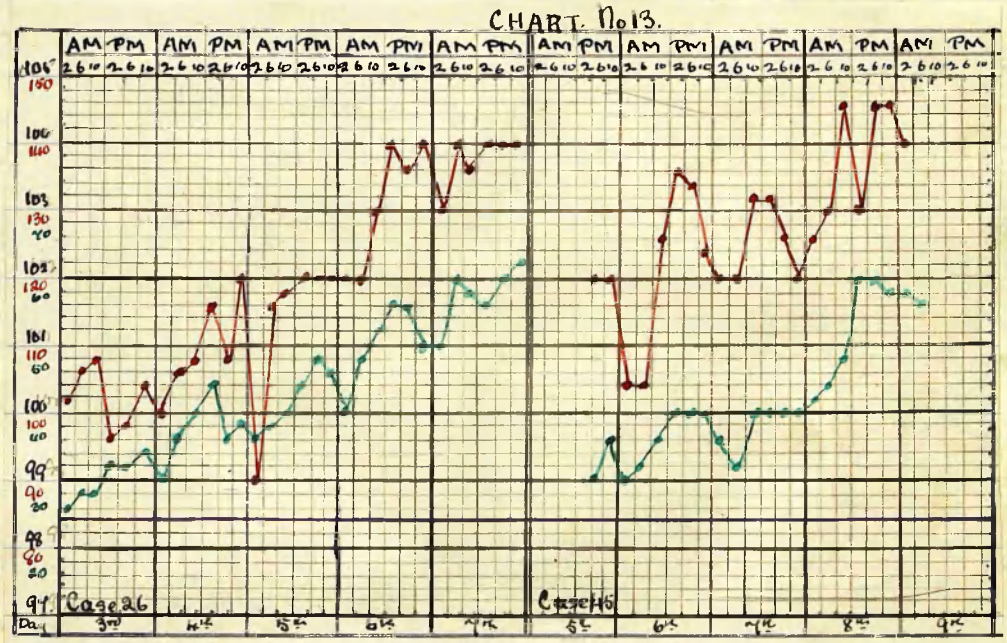
(1) During the early stage, when the signs of lung implication are absent, the augmented oxygenation required ~~with~~ from the existence of pyrexia is met by quickened respirations.

(2) The onset and spread of consolidation causes the respiratory rate to rise, so that the influence of the temperature is lost or submerged, but as already stated , this increase is not necessarily proportionate to

to the extent of the lung involved.

(3) When cardiac failure sets in as evinced by pulmonary congestion and rapidity or irregularity of the pulse, this is almost invariably accompanied with greatly increased respirations which will continue high even in the face of a rapidly falling temperature, until death closes the scene . (see case 26 and 45. Chart 13).

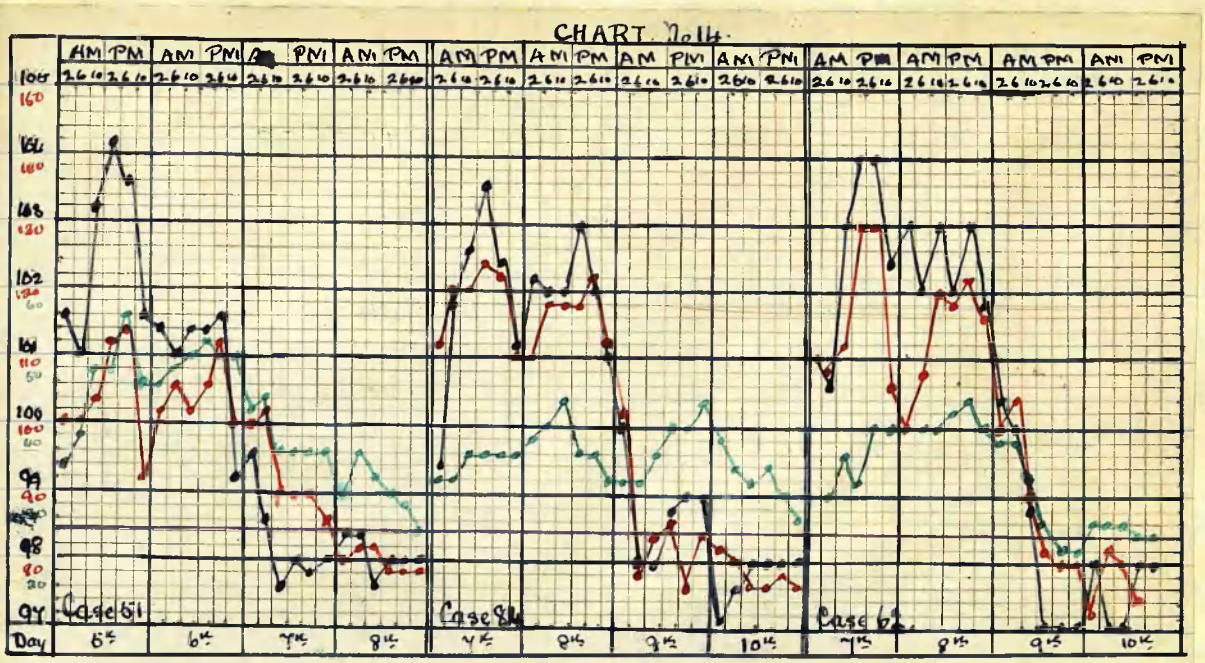
CHART. No13.



Illustrates the rise of pulse and respiration rates resulting from Cardiac Failure.

When a crisis is taking place it is exceptional for the respiratory curve to show a critical fall. The defervescence is mostly by lysis, even when the other phenomena have terminated suddenly. In the majority the respiratory "defervescence" is by a lysis and 30 per minute must be regarded as the critical mean minimum level, as it is usually three or four days after the crisis before the respirations reach the normal of health. (20 per minute) although during this period the patient may be making a rapid recovery.

At the critical period the relationship of the pulse, respiration and temperature to one another varies considerably, but these almost all can be arranged under three types (see chart 14).



Illustrates the three types of relationship of the pulse. respiration & temperature to each other at the crisis.

Thus in the 1st. the temperature reaches normal first, to be followed in a few hours by the pulse, and this again in a few hours later by the respiration . In the 2nd. type pulse and temperature fall together by a crisis, whilst the respirations become normal by a lysis in the course of a day or two, and in the last, all three reach the normal level together by a crisis.

THE PULMONARY SIGNS AND SYMPTOMS.

The provisional diagnosis of Acute Pneumonia having been made from the appearance and history of the patient, and after the observance of the pulse, respiration and temperature ; one instinctively examines the lungs for local implication, and an observer's diagnostic instincts are always on the alert until such evidence is obtained. This opens up an intensely fascinating chapter of the natural history of Pneumonia, viz the lung consolidation, its appearance, development, and disappearance.

In Hospital practice the patients have generally been ill for a few

days before admission, so that the opportunity for personal observation of the lung condition in the first day or two of the disease, is seldom obtained. Of 106 patients admitted, where the date of onset was more or less accurately ascertained, 5 only (4.7 %) were admitted on the first day of illness. In two of these, no characteristic pulmonary physical signs had yet appeared, but crepitus appeared on one (case 79) on the second and the consolidation on the fourth day. In the other case ((93) crepitus set in on the fourth day and consolidation on the fifth. In the remaining three cases, crepitus was the only sign detected on admission, and in one of these, crepitus and percussion dulness alone were observed throughout the illness and although tubular breathing was not at any time detected, the post- mortem examination revealed the presence of consolidation (case 88). In the second case (No. 107) the consolidation appeared on the second day and in the last, crepitus and crepitus only were detected throughout the illness with recovery. Thus from this rapid survey of these five cases alone we see at a glance what differences there may be in the day of onset and rate of progression of the characteristic pulmonary physical signs. We see that crepitus may appear on the first, , or be delayed until the fourth day ; also, how it may be the only physical sign observed in a patient who recovers. We also note that a consolidation may appear as early as the second day, or its appearance may be delayed until the fifth day, and lastly it may be present and yet be not revealed by the classical physical signs.

By an extended study of the condition of the lungs on admission, other interesting facts are brought to light. In the contiguous table (No. 2. page 106 cases are classified. The first column represents the day of illness on admission, and the second shows the number of patients admitted opposite their corresponding days of illness, from which it will

Table No 2.

Day of Admission	Total No. of cases admitted	Total percentage of these cases	Preconsolidation Stage: Subsequent Appearance	Preconsolidation Stage: No Subsequent appearance	Consolidation present on Admission	% of these from Total admitted.
1st	5	4.7%	4	1	0	0 %
2nd	15	14.1%	12	3	0	0 %
3rd	28	46%	11	2	10	43 %
4th	26		7	4	15	58 %
5th	15	14.1%	5	2	8	53 %
6th	11	10.3%	1	1	9	82 %
7th	10	9.4%	0	0	10	100 %
8th	1	.94%	0	0	1	100 %
	106		40	13	53	
106		=	53		53	
			106			

be seen how comparatively few sought admission during the first two days of the illness, 4.7 % on the first day, and 14.1 % on the second ; a total percentage of 18.8 for the two days. Considering the suddenness of the onset and the acuteness of the disease, this is a comparatively small proportion, and probably , no doubt, is explained by the well- known habit of the poorer classes to attempt to "work off" any acute attack. It will be seen, however, that a large proportion (46%) have to "give in" during the third and fourth days and that by the fifth day 80% of all these cases had sought medical assistance. In the remaining three columns opposite their corresponding days, the condition of the lungs on admission are classified. From these it will be observed, that in none of the twenty patients admitted on either of the first two days of the illness was consolidation present, and that only in ten cases out of twenty eight admitted on the 3rd. day was it detected ; a total percentage of 20.8 of all cases admitted during the first three days. From these facts alone it is reasonable to conclude that it is unusual for a consolidation to set in before the third day. In the last column is recorded the percentage of cases, calculated from the total admitted on each day, in which consolidation was present on admission, and this shows that each subsequent day of the illness is accompanied by an increasing proportion of instances where solidification has appeared, so that, one expects to find it in all cases by the seventh day.

That this is true is also shown by the observation and classification of those records in which consolidation appeared after the patient had come under observation. Of this group, 53 were admitted, but from this 13 must be deducted as they came under that equivocal class in which consolidation is never present, or at all events not discovered, thus leaving a total of 40 cases in which solidification of lung tissue subsequently ensued. A classification of these (see table 3.) again shows that in no case did consolidation appear on the first day and only once had it developed by the second day (2.5%). In four instances (10%) it appeared on the third day : thus only in 12.5% of all these cases had consolidation developed by the third day. This Table also indicates that consolidation makes its appearance with almost equal frequency on the fourth, fifth and sixth days ; the fourth slightly predominating. Here again the expectancy that in all or almost all cases, that solidification will certainly be present by the seventh day is rendered reasonable if not in fact proven, as only in 2 cases (5%) was its appearance delayed until the eighth day.

Table No 3.

Day of Appearance of Consol.	No. of Cases where Consol appeared	Percentage of Total
1st	0	0%
2nd	1	2.5%
3rd	4	10.0%
4th	10	25.0%
5th	8	20.0%
6th	9	22.5%
7th	6	15.0%
8	2	5.0%
Total = 40		100.0.

page 20

In table No. 4 the cases are tabulated in decades, and according to the part of the lung or lungs diseased, after the maximum extension had been reached. Here it will be seen that exactly 60 patients (50%) were attacked between the ages of 30 and 50 years. It is also noted that the right lung had a greater tendency to be attacked than the left, almost in proportion of two to one. The right base was only slightly more frequently affected than the left (37 to 33). The greatest difference was revealed in the apical consolidation. The right being more frequently affected than

Table. No 4. Portion of Lung affected.

Age in Decades	No. of cases in these decades	Right Lung Apical	Basal	Median	Left Lung Apical	Basal	Median	Double.
0 - 9	6	2	1	0	0	1	0	2
10 - 19	9	1	5	1	0	1	0	1
20 - 29	16	5	3	0	0	4	0	4
30 - 39	29	7	5	1	3	12	0	2
40 - 49	31	7	10	0	1	10	1	1
50 - 59	16	6	6	0	0	4	0	0
60 - 69	11	3	6	0	0	1	0	0
70 - 79	2	0	1	0	0	0	0	0
Total	120	31	37	2	4	33	1	12
		70			38			12
Total	120	=			120			

the left, nearly in the proportion of eight to one (31 to 4 cases). In 12 instances both lungs were affected. The strictly median consolidations were the least frequently observed. In the order of greatest frequency these cases would thus be classified as follows :-

Right Basal -----	37 cases	---- 30.8 per cent.
Left Basal -----	33 do.	---- 27.5 do.
Right Apical-----	31 do.	---- 25.8 do.
Dble. Consoli----	12 do.	----- 10.0 do.
Left Apical. ----	4 do.	---- 3.3 do.
Right median ----	2 do.	---- 1.6 do.
Left Median ----	1 do.	---- .8 do.
Total	120.	99.8.

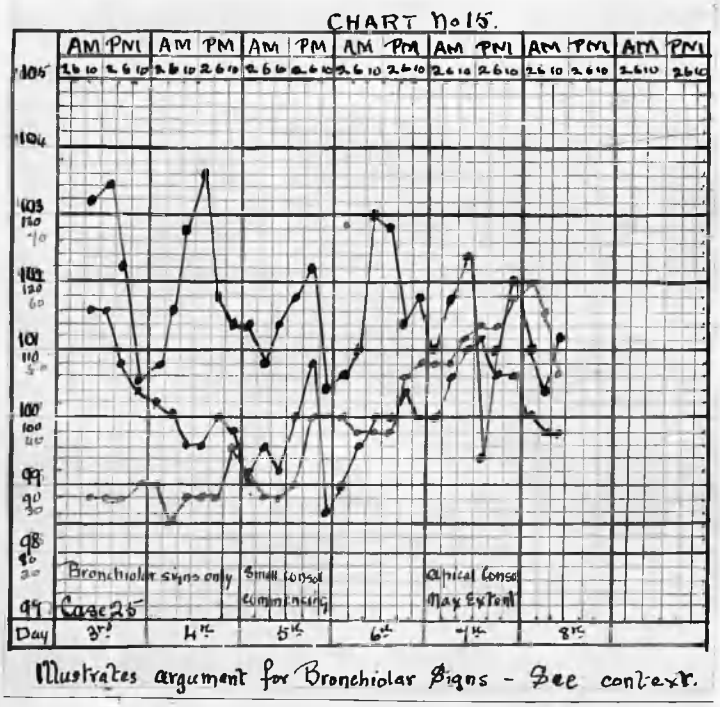
With this introduction it now remains to follow in detail the onset pregsess and disappearance of the characteristic signs observed in the lungs. Generally, the first sign to appear is a very fine crepitus, inspiratory in its character : universally known as Pneumonic crepitus. This is a very constant and characteristic phenomenon, but too great reliance cannot be placed on this sign alone. A certain woman, an habitue of the

Poorhouse frequently left this institution for a few days drinking, and was often brought in, in a day or two, in a feverish condition. Coincidently fine inspiratory crepitus appeared at the posterior bases. The first time I examined her, she was sent into hospital with the suspicion of commencing pneumonia. It is interesting to note that the resident physician who preceded me , and also my successor, both fell into the same error with this patient, but this diagnosis was invariably negatived by a few days observation. Pneumonic crepitus may not set in for a few days after the onset. Its duration is usually evanescent and generally persists alone for one or two days only, but it may exist for five or six days before consolidation is observed. In the vast majority of cases, in the area of crepitation, signs of supervening consolidation(commencing tubularity of the Respiratory murmur or pure tubular breathing : increasing V.R. or pure bronchophony etc.) ultimately set in. But although crepitus is first detected in a certain area, this does not necessarily indicate that the solidification will subsequently appear here. Not infrequently indeed, crepitus is first observed at the extreme posterior base of one or other lung and spreads upwards to the scapula, and here the consolidation is first noted (cases 12, 66 and 79). Again crepitus may even begin at the extreme base and extend upwards to the extreme apex with the subsequent developement of a consolidation which remains strictly apical throughout the illness (cases 45 & 90). Generally speaking, therefore, the first physical signs detected in the diseased lung are either inspiratory crepitus, or signs of commencing consolidation, and it must be admitted that in odd cases the latter may come into existence without any pre- existent crepitus having been observed.

In a few cases there is still another form of onset, of which there is some reason to admit, but of which I am not aware of any previous mention, unless perhaps those instances where Stokes speaks of a peculiar roughness

of the respiratory murmur preceding the appearance of crepitus, belong to this category. If, strictly speaking, crepitus or evidence of commencing solidification are the first true local signs of lung implication, then , in these curious instances of pneumonia, the following contention must be for a pre- crepitant state of the lung , but still pneumonic, and for want of a better name might be called a " BRONCHIOLAR CONGESTIVE STAGE".

In case 25 (see chart 15) the decided and persistent rise of the respirations after the fifth day will be seen. The temperature per se, was in all probability not sufficiently high to cause the respiration to rise thus to sixty per minute. Nor was there undoubted evidence of cardiac failure to account for this, as at this period the pulse rate only averaged about one hundred per minute. On the fifth day a small consolidation appeared between the third and fourth ribs, and by the next day this had spread upwards to the scapular spine : its maximum extent, as verified by post- mortem examination. This is a very small consolidation to be the direct cause of such a high respiratory rate. But in this patient, previous to the onset and recognition of solidification, and during which period no rusty expectoration was observed to indicate its possible central existence, certain signs were present and recorded which might offer a reasonable explanation. Thus on admission, dimished R.M. and slight distant inspiratory wheezing ((sibilant rhonchi) at the right posterior base, associated with doubtfully diminished lateral movement of the chest were recorded.



Illustrates argument for Bronchiolar Signs - See context.

Next day these physical signs were observed all over the right lung, but there was now present distinctly diminished movement of the whole of the right side, with impairment of the percussion note, but above all, increased respiratory movements of the left lung had now appeared. Finally on the top of these physical signs pointing to mischief affecting the whole lung, a small strictly apical consolidation supervened, whilst the remaining portion continued as before. This diminished lateral movement and diminished R.M. over the right, with coincident increased respiratory movements of the left lung, obviously indicates obstruction to the entrance of air into the right lung which could not possibly be this purely apical consolidation, as these signs were not local, but affected the whole of the right side. The absence of moist or viscid crepitus, and the presence of distant sibilant rhonchi points to the causation being of a dry origin. Possibly, if not probably, this is due to swelling and congestion of the smallest bronchial tubes and of the bronchioles. By obstructing the free passage of air, this would reasonably explain the presence of the diminution in the volume of the respiratory murmur, and thus also, of the diminished lateral movement of the affected side.

The air passing over the presumably congested and consequently irregular surface of these tubes would account for the presence of the distant sibilant rhonchi. If at any portion, the presumed congestion becomes extreme and exudation ensues, this may result in the appearance and detection of pneumonic crepitus.

This contention is also markedly illustrated in case 26. In this patient these bronchiolar congestive signs- diminished R.M. and distant wheezing etc- were detected over the right lung bosteriorly from apex to base, and anteriorly down to the third rib, whilst at the extreme anterior base, the breathing tended to be puerile. This continued during the second and third days, and it was not until the fourth day that consolidation set in, which, after its maximum extent had been reached was strictly apical in distribution, the lower borders

being anteriorly the fourth rib, and posteriorly the midscapula, whilst pulmonary oeduma supervened in the remaining portion.

In case 72 the patient was admitted on the second day of illness when the presence of diminished R.M. prolonged expiration and high pitched distant wheezing, — the bronchiolar signs — along with marked diminished expansion of the right lung were recorded. These signs were strictly unilateral and were associated with slight cardiac epigastric pulsation, strong presumptive evidence, in the absence of cardiac valvular disease, of the existence of pulmonary obstruction. Next day crepitus appeared between the second and fouth rib, and on the fourth day a consolidation about one inch square was observed between the third and fourth ribs. Coincidently cardiac pulsation over its apical region and also of the vessels in the neck were superadded to the previously recorded epigastric pulsation, indicating a degree of obstruction not explicable by such a small consolidation. That this small area observed did not denote a central implication of the lung is probable as previously to this the respiration rate was not much increased, but with the subsequent increase of the consolidation as shown by physical signs the respiratory became correspondingly increased. Here again we have strong presumptive evidence of some pulmonary obstruction not explained by the extent of the solidification.

In case 27, the signs recorded on admission were, diminished movement, R.M. and V.R. and slight distant wheezing extending from the right posterior base to the scapular angle, with the subsequent development of a scapular consolidation two inches in depth, whilst the basal region continued as above.

In other patients , this Bronchiolar combination of signs are associated with the presence of a consolidation on admission. Thus a Scapular consolidation is present with diminished movement, R. M. and sibilant rhonchi in the other parts of the same lung (cases 2 and 10) and coincidently

with a basal consolidation these signs are observed at the apical region (case 5). When a consolidation is present in a certain portion of lung tissue, and if the remaining portion is not extended into, the general ~~tendency and~~ experience is to find that such a consolidation leads to increased respiratory movements and puerile R.M. in those parts. On can readily understand how an apical consolidation will lead to diminished movement of the apex and a basal consolidation to effect the base in like manner, but if an apical or basal consolidation co-exists with diminished movement, diminished R.M. and distant dry wheezing sounds in the rest of the lung, it is not at all probable for such to be the direct cause. It is here, I think, that the presumption of the presence of a bronchiolar congestive condition becomes highly probable . That there is some obstruction to the free admission of air is also evidenced in ordinary cases by the invariable diminution in the respiratory volume, where large areas of pneumonic crepitation are examined.

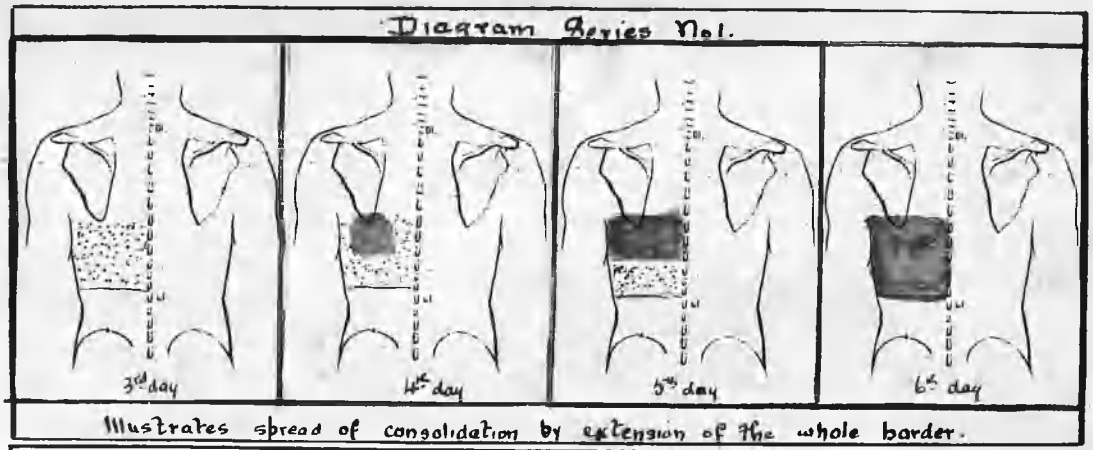
The significant features of these Bronchiolar signs to remember are (1) They appear to be of dry origin and (2) are confined to the affected lung and strictly unilateral in their distribution.

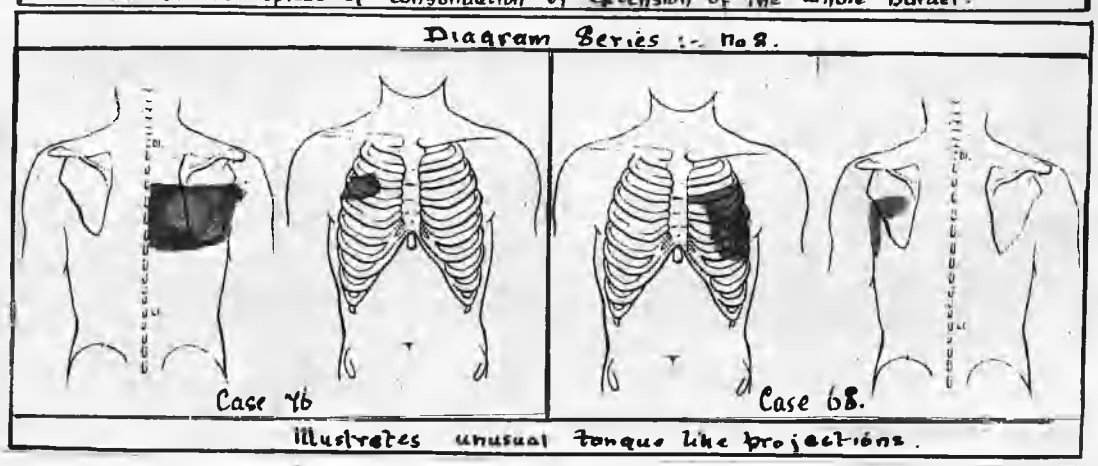
When a solidification sets in, it may appear at any part of the lung and the area first observed may vary greatly in size, But in all cases which come under observation early enough a " Centre of Solidification" for the consolidated area can generally be detected from which the process extends so that ultimately the whole lung may become solid. A lung never becomes completely solidified from top to bottom at once, but invariably is by extension from one or possibly two centres, but what determines the localizing of such a centre I am unable to state. A lung consolidation may go on spreading and only reach its maximum on the day of crisis. On the other hand its maximum extent may be reached on the third day and persist in statu quo until death or recovery ensues. The area of maximum extension moreover is a very

variable one and may be anything from two inches square to a complete solidification of the whole lung.

When a consolidation appears at either the anterior or posterior basal region the most general experience is to find it confining itself to the corresponding aspect of the chest and it is the rule in posterior basal consolidations to find the anterior or outer border coinciding with the posterior axillary border and the upper margin on a line with the lower scapular angle.

In many cases however, the outer border extends to the midaxillary line or even the anterior axillary border and the upper border may be at the midscapular region or even on a level with the scapular spine. The favourite border lines for the anterior basal consolidations are the anterior axillary border and the fourth rib. It is not the rule to find a posterior basal solidification extending round to the anterior base, but in the apical region, however, the consolidation is usually detected both in front and behind, and these generally remain strictly apical in distribution throughout the illness. Apical consolidations are generally first observed either at the scapular region behind or about the third or fourth rib in front, and spread upwards. When a strictly posterior median consolidation is present it is difficult to foretell with accuracy how it will spread and in a very few instances indeed does it remain strictly median. The majority extend, either upwards to the apex or downwards towards the base. When a solidification is extending the borders generally spread throughout their whole extent and it is exceptional to find a tongue like process shooting out into the still unaffected lung tissue as shown in series 2. Page 27. Series No 1 page 29.

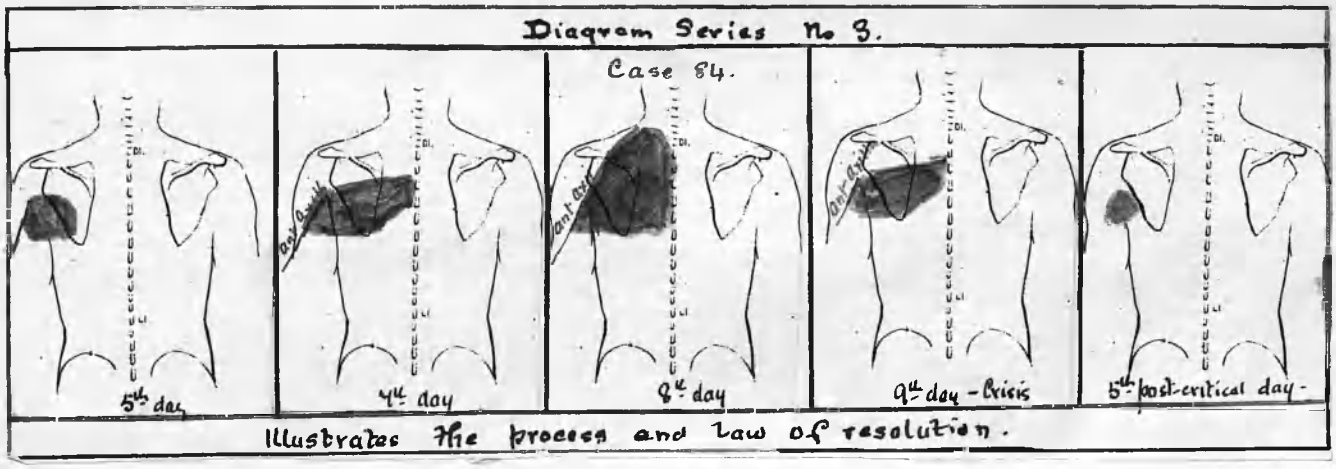




About the critical period or very soon after the consolidation begins to disappear; the lung undergoes resolution. Crepitus redux is most frequently the first sign observed. This is soon followed by an alteration or disappearance of the tubular breathing and bronchophony. Indeed tubular breathing may disappear completely from an extensive area within twenty four hours. (Case 63) (~~Case 62~~). Percussion dulness next lessens in intensity, or disappearsaltogether in the course of a few days. It may however, take weeks and in a few cases it never quite clears away whilst under observation. In some cases the consolidation signs (T.B. and Br.) rapidly disappear leaving only moist rales which may persist for a considerable period : from ten days to two months. In other instances again the lung undergoes partial resolution, but a small consolidated area persists and may exist unchanged for eight to ten days, then suddenly clear away within twenty four hours without any crepitus redux ever having been heard. To follow such is a very striking experience but is exceptional. The most

common course is for the crepitus redux to creep slowly into and through this area : leading ultimately to complete resolution.

During this disappearance a law of the process of resolution can almost be asserted, namely, that " That portion of the lung first affected is the last to clear away".

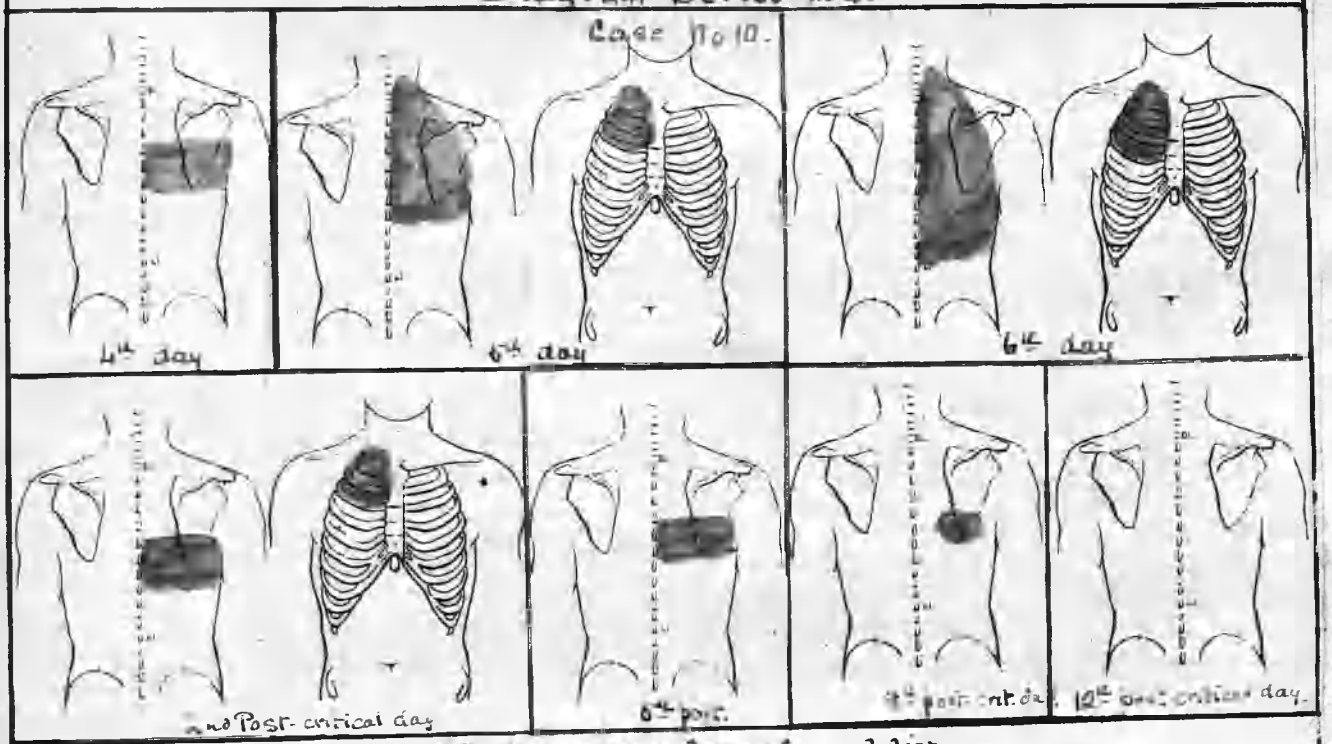


In diagram No. 3 (case 84) patient was admitted on the 5th. day with an axillary consolidation. In two days this has spread inwards according to diagram to the vertebrae. Next day the consolidation had reached the apex. This, the maximum extent was attained only on the day preceding the crisis. After the crisis the consolidation cleared away almost in the inverse order of its spread as shown in the series. Series No.4 page 29. (case 10) is also a good illustration of this law of resolution.

Thus, patient was admitted on the fourth day with a scapular consolidation. Next day this had extended slightly downwards below scapular angle, and upwards over the apex down to level of 3rd. rib. On the 6th. day the consolidation made its final extension down to posterior base. The inverse order in the clearing away of this extensive solidification is also observed but the ultimate persistent scapular existed for 12 days before it finally disappeared.

Diagram Series No 4.

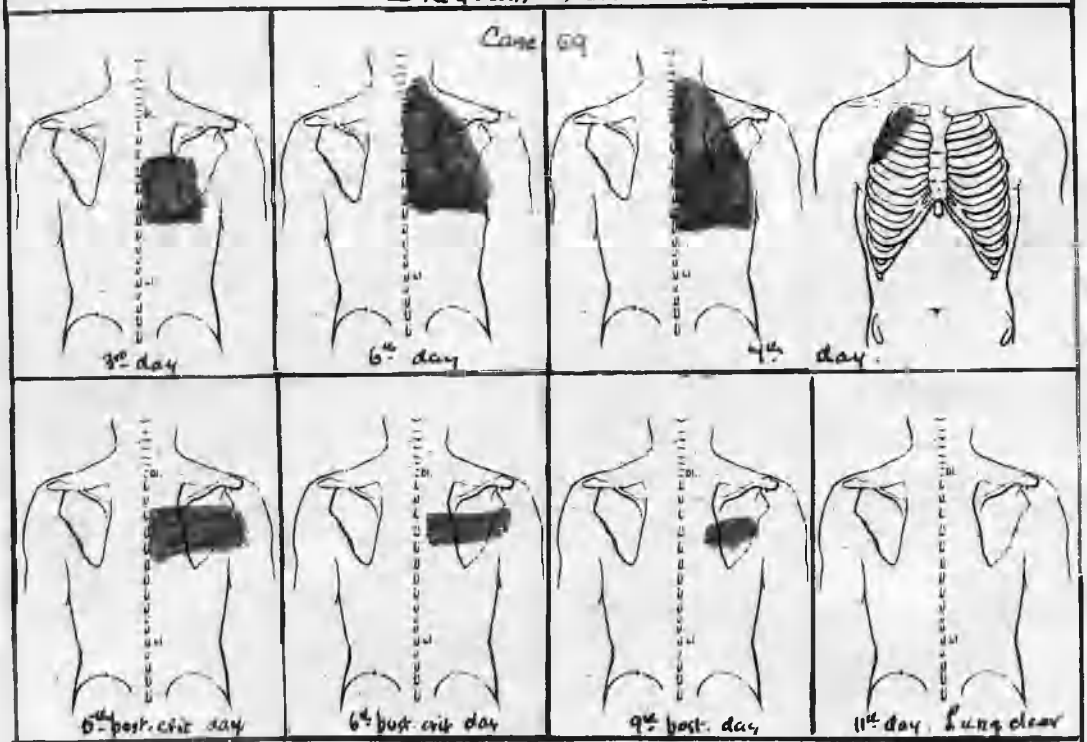
Case No 10.



Illustrates the process and law of resolution.

Diagram Series No 5

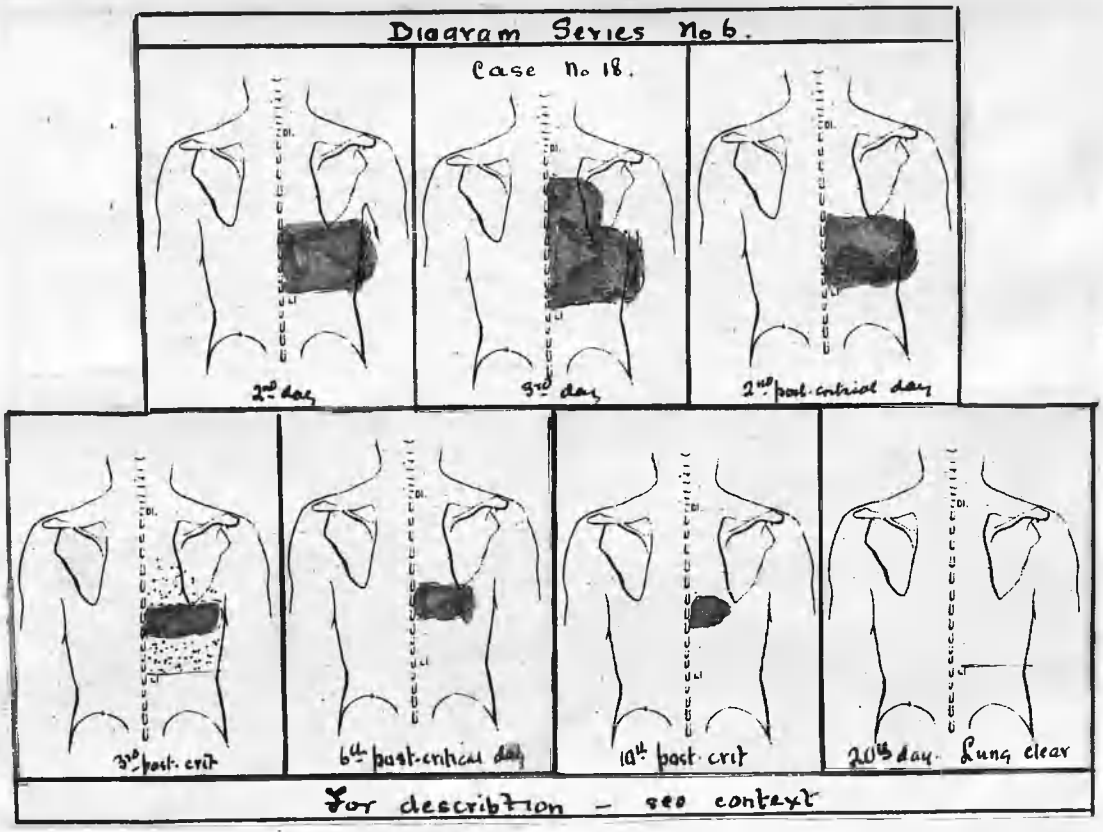
Case 69



Illustrates the process and Law of resolution

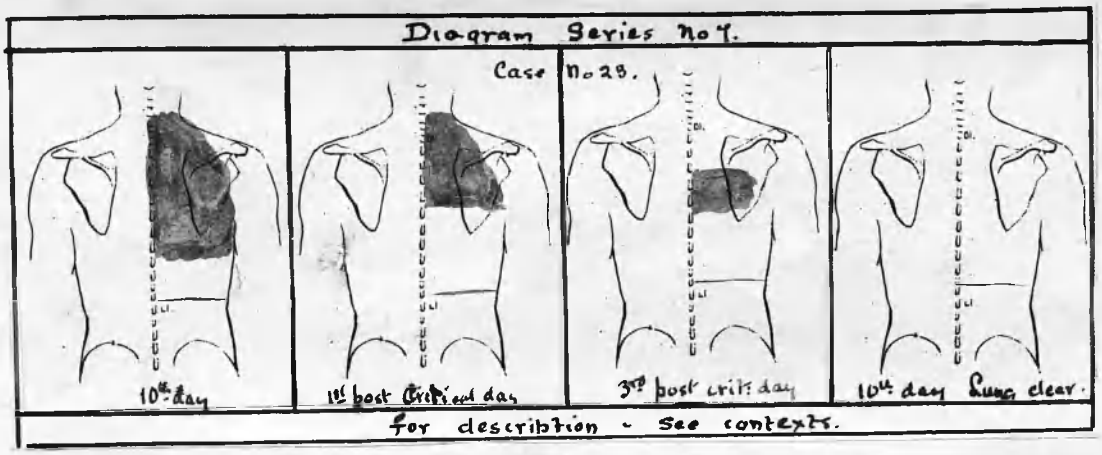
Case No. 5 (see series 5) page 29 is one where a scapular consolidation was first observed on the 3rd. day, but where the ultimate maximum extension remained apical. The first extension was upwards to supra- clavicular region and outwards to axilla posterior border. On the 9th. day it had spread about 1" below scapular angle and also forwards into the extreme apex and outer border of apical region in front. The method of clearance here, although not mathematically exact, illustrates the law of resolution remarkably well.

After a little thought this sequence in the process of resolution is not so remarkable as it would at first sight appear, because, the part first affected, is the part, the structures of which have been subjected to the pressure of the pathological products for the longest period and consequently has had to live on an unduly restricted blood supply for the longest time. Thus presumably its recuperative power would be most impaired. Moreover, being generally situated towards the centre of a solidified area, it is the part most remote from the active capillary and lymphatic circulations which during resolution are carrying away the broken down debris from the lung alveoli. In a few cases the consolidation disappears so rapidly that this sequence is not observed, but in a decided proportion of instances where the course of the disease has been carefully followed, it is very strikingly illustrated. So frequently indeed is this phenomenon observed that in those cases which have been admitted with a large area of consolidation already existent, it is not unreasonable to infer its seat of origin and centre of solidification from the manner of its disappearance . Thus in case 18 (see diagram series No. 6) page 31 patient was admitted with a right posterior basal consolidation with its outer margin in a line with the anterior axillary border. By the next day this consolidation extended three inches higher to the scapular spine. After this the crisis set in and the first part to clear up, was, what personal observation showed to be the latest addition.



But the question that here arises is , " Supposing one had followed the lung condition from the very first would such observation have shown that this consolidation began at the scapular region as we knew that it there ended? The same question also arises as to whether we are justified in making the same deduction as to the probable centre of origin of the affected area in those cases in which, on admission, the whole apical region is consolidated , and which when resolving, reveals the persistence and ultimate disappearance of a consolidation in the scapular region. "Did this apical solidification begin in the scapular area and spread from this" ? is the quastion that here arises. (see series No. 7 page 32) This question although of subsidiary scientific interest,is of no clinical importance.

In the event of there being two centres of solidification in one lung, their future development may be two fold. I have only seen three cases altogether that might belong to this category. In two of these

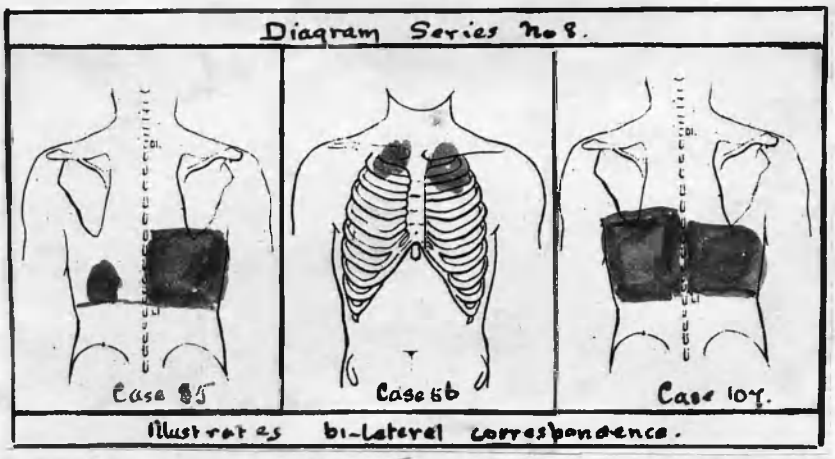


two distinct and separate areas were detected where the classical signs of consolidation were observed, but in one of these the post mortem showed a uniform gray hepatization of the right basal lobe, so that here it was possible, that only one solidified area really existed, the characteristic signs being obliterated in the intervening area by some accidental cause (89). In The other case, a small solidified area was observed between 3rd. and 4th. ribs in front, and next day a small consolidation was observed in the interscapular space of the same side, but here the post mortem revealed only one uniform gray hepatization of the middle and lower part of the upper lobe, so that here what appeared to be two areas was ~~in reality~~ probably due to some accidental obliterating cause, leading to the suppression of the recognized and unequivocal signs in the intervening region. In the last case (No. 88) the first consolidation was first recorded at the left apex, but subsequently towards the end of the illness a second one was observed at the left axillary base to the 4th. rib, whilst the apical region during this interval had resolved. Here again , at the post mortem, gray hepatization of the anterior portion of the left basal lobe was found, but the apex was only found in an oedematous condition, nor did the microscope show distinct evidence of fibrinous and lencocytal deposition in the alveoli. in the apical region.

In 12 cases (= 10%) the consolidation appeared in both lungs. This

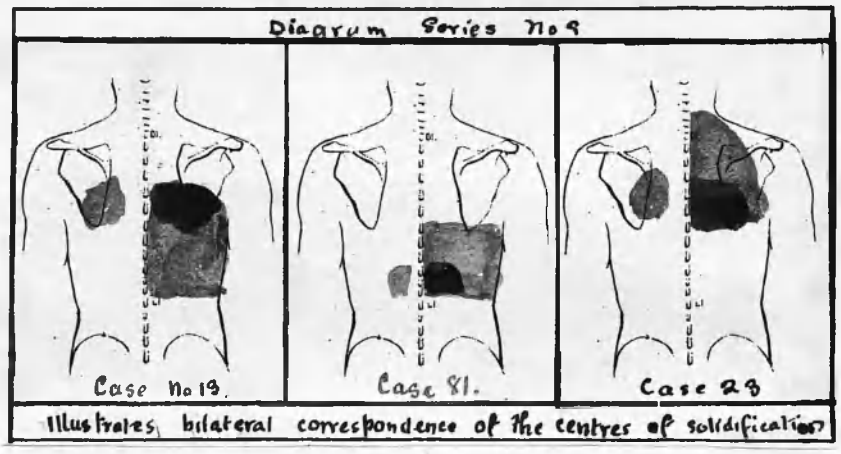
number is too small for any deductions therefrom to be of much value and any conclusions that may, here, be more or less obscurely indicated, are of value mainly that they impart an interest to future cases for their verification or otherwise.

If a double consolidation is present the main force of the disease seems to be expended on the lung initially attacked, which, in these cases, with only one exception, was always the lung most extensively affected. Further, this second consolidation practically always sets in after the first one has already been in existence for some time : the area affected is less extensive and its duration is always shorter : it always disappears before the lung first implicated has resolved. Moreover when this second solidification appears there would seem to be a tendency for it to commence at that portion of the lung approximately corresponding to the area affected in the first lung. In other words if a basal consolidation appears at one base you expect to find



the other at the opposite base, a median to be followed by a median and apical by an apical consolidation. To go further, the second consolidation frequently appears at the area approximately corresponding to the centre of solidification for the first lung, and in only two of these twelve cases was this indubitably negatived. In one, however, the diagnosis lay equivocally between Lobar and Broncho-pneumonia. In the other an anterior and anterior left basal consolidation extended over the apex and ultimately to the extreme posterior

base with the later appearance of a second consolidation at the right scapular region. In the others, however, this probability seems indicated with some reason. Thus in cases 13 and 106 (see series No. 9 where the centres of solidification are indicated by a darker shading) a scapular consolidation appeared in the right lung with ultimate extension to the posterior base and subsequently the second consolidation developed at the left scapular region.



This exact sequence was also observed in one case I afterwards saw in private practice. In case No. 23 (Series No 9.) a consolidation appeared in right side with extension to apex, but still this was followed by a left scapular consolidation. In case No. 56 (Series No 8 page 33.) a strictly apical consolidation first detected in the left lung was followed by a consolidation in the right lung strictly apical in its distribution. In case 35 and 81 an extensive right basal consolidation was followed by corresponding consolidations at the left base and lastly in case No. 107 a post mortem showed the ixistence of a solidification of the right base not detected during life, ccinciding with a known left basal consolidation.

As already stated these observations and ideas are based on too few records to be dogmatically asserted.

A comparison of the clinical signs with the post mortem records of the lung also brings out some interesting and unexpected features. Certain facts and experiences would appear to indicate that where the signs of consolidation are

best marked (pure hissing , T.B. etc.) post mortem reveals the presence of red hepatization, and also that these signs are apt to become obscured when presumably gray hepatization has set in. Few experiences are more startling than to find, at the post mortem, consolidation in cases where it was least expected, especially when no classical signs were observed to denote its ante-mortem existence. In many instances its existence must only be inferred from the presence of percussion dulness and diminished respiratory murmur. Herein lies a possible source of error in diagnosis, for these signs by themselves quite commonly persist after an attack of acute pneumonia and are usually attributed to pleural adhesion and thickening, but post-mortem experience shows that behind these indefinite physical signs a gray hepatized consolidation may lie concealed.

A certain patient was admitted, whose sole complaint was of difficulty in swallowing and regurgitation through the nose when he atempted to swallow fluid. This was verified by personal examination, but a laryngoscopic examination and the passage of oesophagead bongies revealed no gross lesion. He, however , suffered from Chronic Bronchitis and slight dulness was observed at the left base. There was no cough and at the post-mortem the left base was found in a state of gray hepatization.

Another patient admitted for operation stated that two and a half months ago he suffered from Acute Pneumonia. At the routine physical examination previous to the operation, slight dulness and diminished B.M. was detected at the left Base and attributed to pleural adhesion. Here again post-mortem examination revealed the existence of gray hepatization although since his last illness the patient had gained both in strength and weight.

PLEURISY. The relationship of Pleurisy to Acute pneumonia would seem to be threefold. (1) In some instances it apparently exists as a primary idiopathic condition with the secondary development of Acute pneumonia as a complication (cases 33 and 55). (2) Pleurisy itself would appear to set in as a complication after resolution has set in and be the cause of death (case 41) (3) It may coincide with and be an actual part of a pneumonic attack. In many cases of pneumonia the illness sets in with an acute pain in the side, but it is decidedly exceptional in such to detect pleuritic friction over this area.

In this series in 22 instances pleuritic friction was recorded. This seems a small percentage (18.5%) considering that practically in all post mortems of such cases, fibrinous exudation is found on the pleural membranes. In many patients, however, fine crepitant sounds, difficult to discriminate accurately between true crepitus and fine friction, are heard and in fact the diagnosis remains purely a matter of election on the Physician's part. In these cases such atypical instances were reported as crepitus, and so may account for the above small percentage.

Undoubted pleuritic friction is seldom , if ever, heard over a solidified area, but generally is present just beyond the consolidation margin (cases 12 and 79). Sometimes it is found a considerable distance from the affected region, as basal in an apical pneumonia (cases 3 and 50). When heard at the beginning of the illness pleuritic friction may be locally co-existent with crepitus but disappears or becomes displaced when solidification sets in.

" POST-MORTEM PULMONARY CONDITIONS".

The following is based on the record of 20 examinations. Within certain limits the post-mortem appearances vary considerably. On opening the chest, pleural adhesions, old or recent, may be seen, and in

some instances there may be a considerable fibrinous pleural deposition even to half an inch in thickness. In others again a serous or milky fluid effusion is observed.

On inspection and palpation it is perfectly obvious that a certain portion of the lung has become solid, and that it greatly outweighs its neighbour. Indeed its weight may increase from thirty five up to seventy four ounces (cases 11 and 72 both weighed seventy four ounces). Taking twenty five ounces as the normal weight of the lung, this indicates that in these two patients these has been a transference of fifty ounces, or a little over three pounds of material to the lung tissue. The consolidation , however, may vary greatly in weight and size, and on section is seen to vary in colour somewhat in different cases, being mostly , however, red or gray : a few cases being reddish or pinkish gray in colour suggest—— a transition between these two extremes. Sections also show that the consolidation margin is always sharply defined from the unaffected lung, and does not merge gradually into normal tissue. The borders may coincide with and be limited by the natural lobar divisions of the lung, but more frequently it extends through or beyond these to the neighbouring lobes, but the extending border still maintains its decided character. Moreover any pleural deposition present, generally extends beyond the consolidation edge.

On reviewing and contrasting the post mortem conditions with the ante-mortem clinical records some instructive and startling results are obtained.

(1) Although pleuritic deposition of lymph is the rule, in only one of these twenty cases was pleuritic friction recorded.

(2) When a consolidation shows both red and gray hepatization in different parts or in different lobes but fairly well differentiated, it is found that the red hepatized area corresponds

to the most recent extension.

(3) It is difficult to state with accuracy, at what period, gray hepatization may be expected to be present, at least in fatal cases, as some observers doubt if gray hepatization ever appears in patients who recover.

In 8 instances only was distinct red hepatization alone present. In these death occurred on the third and fourth days, and in another case where death took place on the ninth day the red portion coincided with a recent extension. In the remaining cases, death occurred only once as early as the fifth day, the others varying between the sixth and the ninth day, but the seventh was the most common. In all of these gray hepatization alone was present. It would thus appear, in fatal cases at least, that this condition is characteristic of the later stages. It is generally, four days at least after the first detection of a lung solidification ~~before~~ gray hepatization is present, but in one case (no. 16) it was present on the third day. Thus the longer a case has been going on and the older that a consolidation is, the greater is the certainty of its having reached the stage of gray hepatization.

(4) The post-mortem lesion is frequently much more extensive than is indicated by the classical consolidation signs (Tubular breathing, Bronchophomy etc.) . Thus clinically a scapular consolidation may be all that is indubitably revealed and yet post-mortem gray hepatization of the whole upper lobe is found (cases 1 and 16) : the obscure signs at the anterior apical region being attributed to collateral congestion and also to the close proximity of the posterior solidification. In others an extensive gray hepatization of the base was present, not revealed by positive clinical signs (cases 83 and 107). After reviewing such types of cases one fact stands out prominently , viz, the the consolidations which

thus escape detection are found post-mortem to be in the stage of gray hepatization. It would appear that when this stage is reached it is apt to be associated with the suppression of the classical physical signs of consolidation. It is therefore unsafe to exclude the existence of solidification even if dulness and moist rales are alone detected. A positive examination only is trustworthy, as a negative observation does not exclude the existence of this pathological pulmonary condition.

--------oOo----------

MICROSCOPIC APPEARANCES.

Under the microscope the alveoli are seen to be filled by cellular and fibrinous contents. The amount of fibrin present varies greatly even in different alveoli of the same specimen, and in other specimens the contents appear to consist of nothing but a mass of red blood corpuscles. The alveoli suffer very little indeed considering the grossness of the lesion. In some instances very little cellular infiltration is present ; in others again it is obviously crowded and distended, even in the same section. Epithelial cells are most frequently detected when a specimen of lung tissue is examined, which ~~microscopically~~ is only congested. I am, however, not qualified to go into any great detail regarding the microscopic appearance thus seen.

THE EXPECTORATION.

The expectoration when viscid scanty and rusty in colour is very typical if not pathognomonic of Acute Lobar Pneumonia. Although generally scanty (2 to 3 oz. in 24 hours) it not infrequently increases up to 15 or 20 oz. In such instances as its viscidity is always greatly lessened ; this is really not due to the augmentation of the true pneumonic sputum but to the appearance and admixture of a mucous and watery element.

This rusty expectoration may be present before any lung signs are observed. Occasionally at this early stage , the sputum is scanty clear and very viscid, but still not rusty. This type is, however, when present equally characteristic. Not infrequently a few streaks of blood appear independently of the part or extent of lung involved. Sometimes the expectoration disappears altogether for a day or two to reappear before the crisis. In two cases, was the prune juice expectoration recorded and in both it at first consisted almost totally of a dirty froth which ultimately ran together to form a watery prune juice expectoration. After the crisis the expectoration not uncommonly ceases altogether even although extensive areas of solidification are resolving, but not infrequently it increases in amount becoming clear, watery, and very frothy. On the other hand it may continue rusty tinged for five days.

Finally, practically in all cases for twelve to twenty four hours before death all expectoration ceases suddenly and completely.

In all the cases (24) where the sputum was examined systematically the pneumonic diplococcus, in very varying numbers, however, was always detected either singly, in rows or in groups and in a few instances were present either in or on the leucocytal or epithelial cells. A few of the latter appeared croweded with these germs, but in the vast majority they are found principally scattered throughout the mucus. They may be detected before consolidation is present and after it has disappeared (case 66), and in one patient they were observed in the scanty mucous expectoration twenty days after the crisis (cases 55 and 69.).

--------oOo----------

PART 2

The diagnosis of Acute Pneumonia is practically always made from the signs already remarked on, viz : the personal appearance, the temperature, pulse and respiration, the lung condition and lastly the expectoration. These, especially the last two, constitute the essential elements of diagnosis, but when the course of this disease is followed many comcomitant phenomena are evident, and although perhaps of little or no diagnostic importance, are equally interesting, and help to construct a more complete composite picture of the life history of this disease. Amongst the first of these may be mentioned the Cough. This may or it may not be present and is very variable. Sometimes it is scarcely noticeable; at others times it is a source of great trouble and discomfort to the patient. and is also restrained if the pain is very severe, but at best is a variable and unreliable sign.

THE NERVOUS CONDITION.

In some patients a peculiar nervousness of manner is seen : a suppressed nervousness with an anxious watchful expression with either flushing or pallor of the cheeks . The patient, however, is quite rational and answers questions sensibly. The condition is well described by the word "Heady."

A pneumonic patient is practically always troubled with sleeplessness and often with delirium.

DELIRIUM. Most constantly but by no means invariably, delirium , in the absence of supervening cardiac failure, is concurrent with a high temperature range (103° or 104°), and very frequently it portends oncoming heart failure. It may then coincide with a low or falling temperature. Delirium may appear at any stage of the illness, either before or after the appearance of consolidation and its intensity is in no way proportionate to the extent of the lung mischief. Indeed, sometimes delirium only sets in after the consolidation has reached its maximum, and in odd cases it is only

observed during the critical fall of temperature. Many patients, however, die without delirium ever being noted.

In pneumonia when a patient is delirious and talkative, his thoughts, words and actions are mostly in relation to his daily occupation. When cases are brought in at first suffering from Acute alcholism, the delirium is then associated with all the horrors and hallucinations of Delirium Tremens but when the pneumonic condition has become fully established, this type becomes submerged by that characteristic of Acute Pneumonia, viz, hallucinations pertaining to his daily occupation.

Post Mortem examinations reveal no gross lesions to account for the existence of delirium. In some, serous ventricular effusion is present; but violent delirium exists where little or no effusion is subsequently found. In one autopsy the intraventricular pressure was so great that a jet of serum spouted out on opening into this cavity, yet no delirium was recorded. Delirium would appear, therefore, not to be due to either the presence or absence of intraventricular pressure. In pneumonic post-mortems it is the rule to find the veins and venules distended especially over the cerebral lobes. This is probably of hypostatic origin, but is not more marked in those who had been delirious than in non- delirious patients.

This is perhaps the most appropriate place to deal with the few records in which KNEE JERKS were either diminished or gone altogether, but my attention was not drawn to the existence of this condition until my opportunity for further observation was nearing its close.

In thirteen out of perhaps twenty five or thirty cases the Knee Jerks were reported either diminished or gone, and this apparently independent of the height of pyrexia or extent of lung mischief. In a few cases it was at first reported diminished in one leg and completely absent in the other but ultimately its absence was recorded in both. The absence of

Knee Jerks was not observed during the early days of the illness and seldom before fifth or sixth day. In six cases with recovery the knee jerks returned either on day of crisis or very soon after, and in the seven fatal cases they could be elicited until one or two days before death, but in others they were always obtained, even on the day of death.

In order to ascertain any possible cause for this phemomenon, in every subsequent case of pyrexia , either continous or intermittent, from whatever cause (Phthisis, Acute Rheumatism, Puerperal Fever, etc.) the knee jerks were systematically examined, but only twice (both Enteric Fever) was its absence noted. This therefore is rather against pyrexia per se being the prime factor in the causation. The rapid post- critical recovery of function is against any structural defect in the nerves being the cause. It may, however, be due to temporary paralysis or paresis of nerve influence from auto intoxication, but on the other hand it may well only be a reflex of muscular exhaustion and prostration, as it only appears late in the disease in cases which recover, and only a day or two before death in fatal cases.

HERPES. being possibly of nervous origin may also appropriately come under the present heading. This was noted in 18 cases (15%) with only two deaths (11%). It may appear at any stage of the disease, before advanced pulmonary lesions are present, and then spreads and matures throughout the course of the illness (cases 56 and 62), but it may not come out until the day before crisis, in which case it matures during the post- critical period. It generally is deteced about the upper and lower lips or the angles of the mouth. It is also found on the alae nasi and even on the lobes of the ear (case 98) and in one instance Herpes Zoster was noted on right side (case 70). Its presence does not seem to discommode the patient to any extent and in no way delays convalescenee. With the exception of sudamina vesicles, herpes

is almost the only skin eruption which appears during the pneumonic attack.

Another interesting if not recognized feature found in many cases of pneumonia is the odour of the breath. Smells and odours are notoriously difficult of verbal description and this "PNEUMONIC ODOUR" is no exception. This is ~~in~~ not foetid or disagreeable but is of a peculiar heavy aromatic quality. It was only after considerable experience that the existence of this phenomenon became impressed on one's consciousness and recognized, but a careful look out for its appearance in subsequent cases seems to render the fact of its existence undoubted. Indeed sometimes the diagnosis of Acute pneumonia was first suspected by the recognition of this odour in the breath even when consolidation had not yet appeared (cases 4, 51, 65, 82, etc.) and in one instance it did not appear until three days afterwards (case 26).

It is noted when the tongue is clean and moist (case 24) or white and furred but still moist (case 4), and lastly when it is dry, brown, and cracked (cases 71 and 76). Thus it is not likely that the appearance of this odour can be attributed to the local condition of the mouth. Sometimes the pneumonic odour is only detected after consolidation has set in and in those cases where it appeared in the pre-consolidation stage, it becomes more distinct with the appearance of solidification. In a few instances it is not recognized until a day or two before death. It is possible that in some way this pneumonic odour is related to the presence of consolidation, as it becomes more evident with the appearance or extension of lung mischief. When one remembers the grossness of the lung lesion and also its close proximity with the breath, the idea of there being an odour, more or less characteristic is perfecty feasible, nor is the treatment given a likely cause as it is recognized in patients on admission, nor is it stopped by altering the routine treatment. The following synopsis of four cases are

are inserted as illustrative types.

Case 4 :- Male aged 35. Admitted on third day. On 4th. day pneumonic odour detected; tongue clean and moist, crepitus only between third and fourth right ribs. Crisis 9th. day.

Case 15:- Male aged 35. Admitted on sixth day, with left basal consolidation. Pneumonic odour detected on ninth day and well marked: tongue dry and brown. Death 10th. day.

Case 26 :- Male aged 26. Admitted on second day with well marked pneumonic odour : "Brionchiolar" signs only in right lung : tongue moist and fairly clean. Consolidation appeared on fifth day. Death on 8th. day. During all this period this odour persisted and was very distinctive and decided.

Case 65 :- Male aged 40. Admitted on fifth day. Pneumonic odour present but faint : crepitus only right base: tongue moist and furred. Next day consolidation detected and this odour was recognized and persisted until death on seventh day.

GASTRO-INTESTINAL SYMPTOMS. :- There are no alimentary symptoms peculiar to this disease and any that appear seem to be referable to the pyrexia. The Tongue may at first be moist and slightly furred and may continue thus throughout the whole course of the illness, but in others it becomes dry, brown, cracked, and tremulous.

The gastric symptoms are generally trivial and patients can usually take their nourishment well. Vomiting may, however, be observed and flatulence is not an infrequent symptom.

Diarrhoea is the most frequent intestinal complaint and sometimes is very severe. In this series it was often present, and at intervals the character of the stools would arouse the suspicion of Enteric Fever.

Nothing characteristic is detected clinically about either the Liver

or the Spleen. The Liver may extend below the costal margin and in odd cases it is painful and tender on palpation, but these could generally be attributed to probable alcoholic congestion. The Spleen I have never seen enlarged clinically. Post mortem examinations also corroborate these clinical experiences. The spleen is seen to be very soft, pulpy and sticky in consistency and varies greatly in weight ($\frac{8}{2}$ to 12 oz.). The Liver weight varies considerably (60 to 90 oz.) but nothing characteristic is observed on section, except perhaps occasional suspicion of a fatty appearance, also probably of alcoholic origin.

TEMPORARY CARDIAC CHANGES.

During the course of An Acute Pneumonic attack, certain changes from the normal physical conditions of the heart are of frequent observance, and they appear to be as much a reflex of the changed physical conditions in the circulatory system, viz.the greatly increased tension in the pulmonary circulation, as of the changes attributable to the pyrexia.

Perhaps the first and most constant of these superadded physical conditions , and indeed one almost invariably present, is an Accentuation of the second pulmonic sound. This sets in early and continues throughout the illness until the crisis, and may persist for a week or two longer, especially if there is a small consolidation showing ~~and~~ tardy resolution. Somewhat frequently the difference of tension between the pulmonic and systemic circulations is so great as to lead to well marked reduplication of the second sound at the base. This rapidly disappears after defervescence is completed, but may persist for four days longer (case 83). In only four cases did a V.S. Pulmonic murmur appear. In one this disappeared completely on the day of crisis (case 66) and in the other on the second post-critical day (case 109). The remaining two are somewhat curious, inasmuch as this murmur did not appear until after the crisis. In the first of these the V.S. pulmonic set in on the fourth post critical day and disappeared on the 27th. day (case 75), and in the other case it appeared on the second post-critical day and persisted for two and a half months.

The increased tension of the pulmonic circulation may be reflected still further backwards, leading to the presence and observation of epigastric pulsation and increased loudness of the cardiac sounds in this area from augmented action and dilatation of the right ventricle. Indeed this may be so marked that the impulse may extend along the abdominal wall below the umbilicus. In a few instances cardiac pulsation is seen in the intercostal spaces and also in the vessels of the neck.

In eleven patients the appearance, and in eight who recovered, the disappearance, of a mitral systolic murmur was recorded. As the arterial tension, as shown by the dicrotism of pulse tracings, is greatly lowered, and as it is probable that the left side of the heart is not receiving its full complement of blood, this complication can hardly be ascribed to the same causes that led to the appearance of the afore mentioned adventitious signs of the pulmonic artery and right ventricle, viz increased circulatory tension. Indeed the fact of there being this difference in the tension between the right and left side is occasionally revealed by the appearance of a reduplication of the first sound. As this mitral murmur is of late appearance it most probably is due to the effects of the concurrent pyrexia on the heart leading to weakness and dilatation of the left ventricle. When such a murmur has occurred it may disappear on day after crisis or persist for four or five days and in one case (no. 103) it was detected for twenty one days after crisis.

THE CHANGES IN THE BLOOD IN PNEUMONIA.

In the use of the haemacytometer the sources of fallacy are many and obvious. At first the error may be slight and inappreciable but when it is multiplied by one hundred thousand to make up for the dilution required by the process, the final results obtained are occasionally startling and obviously fallacious. The final calculation may be so contradictory as to merit rejection. Two or even three subsequent examinations may be attempted until what appears a more probable result is obtained and accepted. This after all may not truly express the real state of matters.

In 30 cases I attempted to record the condition of the blood and note what changes , if any, would appear to result from the disease ; the differentiation of the white blood corpuscles not being attempted. The results are however, somewhat disappointing and not too reliable. The method adopted was to examine the blood on admission, or at least once prior to, and then immediately after the crisis, presuming that the difference , if any, between the two observations would to some extent indicate the blood changes attributable to the pneumonic process. Ten patients died before consecutive records could be obtained, but on looking over these isolated results one facts stands out clearly in them all, viz the existence of Leucocytosis.

Leucocytosis is a condition which would appear to be constantly present in Acute pneumonia, although in very varying degrees. By the third day it may be very decided. If at the initial observation, the proportion of the red to the white blood corpuscles is nearly normal, it is found that as the disease advances leucocytosis sets in and becomes more and more marked so that the proportion may be reduced from 1 to 300 or 400 to 1 to 91 (cases 56, 67, 76) or even as low as 1 to 61 (case 24) . This disproportion begins to lessen after the crisis but the return to normal is a gradual process, and leucocytosis may still persist and be very decided as late as the

eleventh post critical day (cases 56, 66, 70, 77 and 81). Table No. 5 is constructed from 12 selected cases to show the progress of this leucocytosis. On the left side are the records indicating the changes during the precritical days and on the right hand the post-critical results, indicating the progress towards recovery. It is seldom that a patient can be persuaded to stay longer thay a week after crisis, and consequently in no instance have I had the opportunity of tracing the progress towards complete recovery. The figures recorded in black indicate the number of white blood corpuscles per cubic millimetre, and those in red, the proportion of the white to the red corpuscles.

Selected Records on Leucocytosis. Table No 5.

	Pre-critical Period.				Post-critical Period.			
Case Number	Day of Illness	Result of Examination	Day of Illness	Result of Examination	Day of Illness	Result of Examination	Day of Illness	Result of Examination
26	3rd	24.300 (1-184)	6th	36.200 (1-200)		Death		
82	"	27.200 (1-154)	"	37.600 (1-117)	7th	25.800 (1-193)		
56	"	37.700 (1-90)	"		1st	12.100 (1-288)	11th	24.300 1-152.
24	4th	12.400 (1-321)	"	14.700 (1-61)	6th	20.000 (1-239)		
66	"	16.000 (1-268)	7th	25.700 (1-143)			11th	11.100 (1-360)
67	"	47.300 (1-93)	"	48.500 (1-99)		Death	7th	day
76	"	34.300 (1-134)	"	42.400 (1-99)		do	8th	do.
77	"	25.700 (1-132)	"		3rd	22.700 (1-197)	11th	16.600 (1-262)
118	"	24.800 (1-398)	"	30.000 (1-134)				
71	5th	24.800 (1-198)	8th	25.300 (1-195)		Death	8th	day.
80	"	50.600 (1-90)			4th	12.200 (1-345)	8th	16.600 (1-200)
81	6th	16.400 (1-339)	9th	44.600 (1-103)	5th	11.600 (1-343)	11th	20.800 (1-92)
70	9th	30.000 (1-160)	11th	41.000 (1-107)	8th	32.800 (1-141)	12th	35000 (1-134)

From this table it will be observed that the white corpuscles may increase from 12000 to 74000 per c.m.m. during the illness and that by the sixth post critical day be reduced to 20000 (case 24). The same sequence can

be seen in the other illustrative cases although not to so extreme a degree.

The Red blood corpuscles do not seem to be affected to any great extent by the disease. In this series, these averaged from 3,500,000 to 4,500,000 per c.m.m. and subsequenh examinations , either pre-or post-critical, did not reveal any great alteration of these numbers from the first observation.

The Haemoglobin like the red corpuscles seems to be little, if at all, affected by the pneumonic process. For these patients the adopted standard of the haemoglobinometer (100) would appear to be too high. It was also impossible in those patients who came under observation, to accurately ascertain the normal blood condition for any given patient when in health. Only on one occasion was 100% of Haemoglobin registered . In 45% of these cases the maximum averaged only between 70 and 80 and between 80 and 90 in 30%. Moreover when using the haemoglobinometer one cannot depend on the accuracy of the result within 5%. When what appears to indicate the percentage of dilution has been obtained, it will be found that this can be diluted further without any appreciable difference.

Selected Records on Haemoglobin & Red Corpuscles Table No.6

	Pre-critical Period				Post-critical Period.			
Case Number	Day of Illness	Result of Examination	Day of Illness	Result of Examination	Day of Illness	Result of Examination	Day of Illness	Result of Examination
26	3rd	4560.000 82%	6th	4.480.000 70%		Death.		
41	"	4.800.000 100%	6th	4960000 84%		Death		
56	"	3.400 000 62%	8th	3480000 62%			11th	3.800000 66%
76	4th	4200 000 62%	7th	4200000 64%		Death.		
24	"	4000 000 76%	8th	4560000 74%	6th	4800 000 70%		
67	"	4400000 92%	4th	4800000 88%		Death		
70	9th	4800000 82%		4.400000 76%	8th	4660000 92%	12th	4720000 90%

Table No. 6 is constructed from seven selected examples to illustrate the progress of the red corpuscles and the haemoglobin. In

contrasting these records it will be seen that the general tendency is for the haemoglobin to persist in statu quo (see red figuring) and that in a minority a tendency towards diminution is observed during the fastigium, but in only three cases was this decidedly marked. In cases 26 and 71 there was a loss of 12% and 16% respectively and of 14% in case 70. These were characterized by a high temperature , a high urea excretion and almost complete absence of chlorides.

Case No. 70 also shows this diminution of haemoglobin may result when th the red corpuscles are little affected, and in this case the loss was rapidly made good after the crisis.

These observations may be summed up thus 1st. In all cases of Pneumonia some degree of leucocytosis is always present, and its recovery towards normal proportion would appear to be gradual. 2nd. The red corpuscles are little affected but in a few cases a decided destruction of haemoglobin is noted, which, however, is rapidly regained after the crisis.

THE URINARY CHANGES.

In Acute pneumonia some very interesting changes are observed in the urine. It is nearly always deepened in colour ; of varying shades of amber. THE SPECIFIC GRAVITY varies somewhat and may be anything between 1010 and 1030. It is not necessarily high even during the height of the illness. Reaction :- In the vast majority of cases the urine is acid in reaction, the intensity , however, varying between high and very slight acidity, but in Eighteen instances at some period, an alkaline reaction was observed and in this series such specimens were always ammoniacal. An investigation into these reveals some very curious and some inexplicable vagaries in the behaviour of the reaction. Sometimes the urine was consistently alkaline throughout the illness, to become acid again within two or three days after the crisis. In others the acidity and alkalinity alternated at intervals of one or two days during the fastigium. A very striking type is where during the illness the urine has continued acid, but where during the post-critical epoch the reaction becomes alkaline to continue thus for three or four days when it finally becomes normal. When phosphates were deposited the microscope invariably revealed the presence of triple phosphates, Urate of Soda and Bacterium termo, even in those instances where the alkalinity was evanescent and alternate. What the exact significance of these changes, is, it is difficult to state. Its existence does not inconvenience the patient. There is no urethral or bladder pain and undue frequency of micturition was not observed, nor does the presence of this alkaline and ammoniacal urine seem to retard recovery. Neither would it appear to render the prognosis more grave as only three of these eighteen cases died (= 17%). The quantity of urine passed in the twenty four hours is seen to vary very considerably. The tendency is for it to be diminished in quantity (16 to 35 or 40 oz. in 24 hrs.) during the fastigium. This is best marked in those cases which died. This although generaaly speaking

true is by no means invariable as cases are observed where the amount excreted was little below 60 or 70 oz. per day. After the crisis also the amount passed becomes only slightly augmented. Indeed it may continue diminished for two or three days before this slight increase is observed. One does not see that the small quantity passed daily during the illness becomes suddenly augmented immediately after the crisis.

During an attack of Acute Pneumonia there are two urinary conditions which become strikingly manifest, viz Albuminuria and diminished chlorides. The more characteristic of these is DIMINISHED EXCRETION OF THE CHLORIDES. This diminution is of very constant observance. In the absence of proper Scientific instruments and reagents for the accurate quantitative estimation of chlorides, after a little thought the following method was evolved and adopted. The urine to be examined is poured into a test tube until a column an inch in depth was obtained. To this the requisite reagents (Nitric Acid and Silver nitrate solution) were added. The precipitate which now appeared was allowed to settle for twenty four hours when the depth of the deposit was read off in terms of an eight of an inch;- the adopted unit equivalent. After testing several normal urines it was found that the chlorides thus precipited formed a column about four eights or five-eights of an inch indepth. This half-inch deposit was adopted as the Standard of Health for comparison with the pneumonic condition. The quantity of urine excreted in the twenty four hours had also to be taken into consideration. This method being adopted, it was found that in pneumonic urine, the chlorides were generally diminished to an extent varying from 25 to 75% of the normal. In other words when the column of chlorides precipitated was read off, instead of being four-eights of an inch in depth it was generally only two or even one-eight of an inch deep. In a few instances , however, the amount of deposit was so slight that its measurement by such clumsy means was impossible. It is exceptional to find the chlorides so diminished that no visible deposit

is observable and in no case have I ever seen the chlorides completely absent.

Taking 50 oz. to be the average daily excretion of urine in twenty four hours and adopting a deposit of four-eights of an inch as the average chloride excretion in health we thus obtain the total daily excretion of chlorides. When calculated out we find this to be 200 eighths of an inch ; an eight of an inch as before stated being arbitrarily adopted as the unit equivalent. This number 200 is therefore adopted for comparative purposes.

In 20 cases the urine was collected and measured daily, as far as possible, to enable this estimation of the chlorides and also of the urea to be made. From this it became apparent that there was a great variation in the amount of chlorides excreted daily during the illness. Thus in Case 24 on the 4th. day 250 units were excreted, an increase of 25% above the normal of health. Next day 136 units only were recorded equal to a diminution of 32% below normal. On the 6th. day as there was no measurable deposit the chlorides could not be estimated. On the 7th. , 160 units (- 20% diminution) and on the eight it again jumped up to 250 units (- 25% above normal) and finally on the day of crisis only 68 units were registered thus indicating that the chlorides had agin undergone a marked diminution to the extent of 66% below normal average. This case is fairly illustrative of the average type . When a crisis has taken place the diminished chlorides begin to increase in quantity as shewn by the deposit increasing, either from being visible only or from one or two-eights of an inch to four or five eights, but here again the chlorides may continue diminished for days after a crisis. Thus in case 24 on the 7th. post-critical day the chlorides were fully 50% below normal. In case 66 on the 4th. post-critical day they were still 50% below normal whilst in case No. 70 they were 76% below the average. In the latter the chloride excretion mounted to 156 units on 7th. day after crisis still, however, indicating a diminution of 22%. This post-critical continuan-ce

of diminished chloride excretion was observed in 9 cases out of 12 who recovered (75%). It would thus appear that although during the fastigium the chlorides are diminished they do not suddenly reappear or suddenly increase after a crisis, but that several days elapse before the normal is reached.

The method by which the above data were obtained is a very crude and rough one, and although the data are not scientifically accurate or expressed in grains per oz, they still possess some value for comparative purposes, as they give some indication, however dim or obscure, of the curious variations and changes of chloride excretion during a pneumonic attack.

Albuminuria.

According to various observers, this condition is present in a very varying proportion of cases: from 0% (Metzger) up to 50% (Griesinger). But in 103 patients of this series where I was able to test the urine, albuminuria at some period or other was observed in 93 (=90.3%.) Its appearance and disappearance, and also the amount vary somewhat. Sometimes it appears only on the day of crisis (case 31), or it may only be present on one day in the early stage of the disease, or again it may persist through out the illness. Finally, in only a few cases, however, no albuminuria was ever detected during the fastigium. In a few odd instances albuminuria only appeared after the crisis and persisted for two or three days. In no instance was albumen absent in a fatal case. By the heat test the amount of albumen present is indicated by the terms, a haze, a slight haze, a distinct cloud, etc., and in the majority of instances no deposit of albumen was observed, but in 20 cases, a distinct and measureable deposit was reported varying from one eleventh of a column to $\frac{1}{6}$th , and in one patient a varying degree of albuminuria, two days before death, became so suddenly augmented that the urine was almost solid

after boiling. Of these 20 cases 14 died , a death rate equal to 70%. Indeed if a deposit of albumen is observed for two or three days the patient is very liable to succumb, but still this is not invariable. Albuminuria may be observed when the temperature is by no means high and its appearance would seem also to be independent of the extent of the lung mischief.

After the crisis albuminuria practically always disappears altogether. Of 33 cases, selected because of the urinary condition having been recorded for several days after the crisis, albuminuria disappeared on the first post-critical day in eight cases (= 24.2%) a similar number on the second day and seven in the third (= 21.2%); or roughly, in 70% of these cases the albuminuria had disappeared by the third day, and only in one patient who continued under observation for twelve days did this condition fail to disappear altogether. This would indicate that this change was due to conditions resultant from the disease itself and not to any pre-existent Kidney disease, and in the post-mortems of the fatal cases , the Kidneys were only slightly affected in three or four, ~~in which was present~~ Only twice during life were tube casts detected.

UREA EXCRETION:- The amount of urea excreted per day varies greatly within very wide limits. 500 grs. per day is generally accepted as the average, but in these cases the daily excretion varied enormously. It cannot be said that it is the rule for the excretion of urea to become increased during the illness, and that after the crisis this increase suddenly falls to normal. Case No. 70 is the only instance where this sequence was closely observed. Here during the illness a daily excretion varying between 700 and 800 grs. fell to 500 grs. after the crisis, but this is exceptional. In fatal cases there is a tendency for each succeeding day towards the end to be associated with an increasing amount of urea. Thus in case 74 on the 4th. day 500 grs. were passed, 667 grs. on the 5th. and 918 grs. on the 6th. with death on the 7th. Again in case No. 76 the daily excretion of urea

mounted up from 380 to 528 grs. during the four days preceeding death. This is in marked contrast to case 79 where during the last three days the amount of urea passed was 42 grs. 75 grs. and 290 grs. respectively. The amount of urea per day during the height may vary from 180 grs. on one day to 4,5, or 600 the next day. It is difficult to state what is the daily average, perhaps 300 or 400 grs. would be near the truth. Frequently before the urea excretion attains its normal on the second, third or fourth post-critical day there is a sudden increase in the amount passed. It may amount up to 700 grains from 300 or 400 and after this becomes normal.

About the critical period also, a deposition of urates frequently is seen. This may be observed a day or two before the crisis or only on the day of crisis, or it may be delayed until the second or third post-critical day. Occasionally during the illness , especially if there has been a false crisis this deposition is also noted.

THE DIAGNOSIS.

In many cases this is perfectly obvious from the appearance of the patient, his flushed countenance and dusky expression, the pyrexia, rapid breathing and perhaps dilating alae nasi and also the nervous "heady" manner; and the detection of a distinct rusty expectoration or the development of consolidation, at once clinches the diagnosis.

But there are two classes of cases met with where the diagnosis cannot be made at once and with such ease. In the first of these may be placed those instances where distinct consolidation never develops, while all the concomitant signs and symptoms point to the condition being pneumonic . Indeed no pulmonary physical signs may be observed, or crepitus, either with or without suspicious impairment of the percussion note, may be all that is detected, and yet these signs, although obscure, disappear after the crisis. Lemon tinting of the expectoration may be noted and deplococci may be numer-

ous (cases 80, 82 and 118). Albuminuria and diminished chlorides may even be present ; also herpes (cases 82 and 99) and in one case the pneumonic odour was recorded (case 82). In fact all the concurrent signs habitually met with in undoubted cases may be observed in these equivocal cases. In this series there were thirteen patients of this type. Unfortunately the detection of pneumococcei in the sputum does not settle the diagnosis as they may be numerous and very typical in cases which undoubtedly are not pneumonic, as Enteric Fever, Appendicitis, Phthisis etc. Indeed the most numerous and typical capsulated diplococcei I have ever seen, were obtained from a patient in whom the diagnosis lay between Enteric Fever and Acute Miliary Tuberculosis. Any obscure or early pulmonary signs that may be detected are possibly due to the early active congestive stage which, however, becomes aborted at this period. Although this is very probable there is no way of proving it absolutely and conclusively, but in spite of this draw back, the probability that these are really cases of Acute but aborted pneumonia must be admitted.

The second type of case differs very materially from the above. A few patients, obviously not in good physical condition, give a history of Acute chest pain as the beginning of the illness, but otherwise the history both of their previous health and present illness is very indefinite and unsatisfactory. On examination consolidation and râles are observed. At first the diagnosis may be uncertain, but two or three days observation soon reveals the phthisical nature of the complaint. In three such cases, the pneumococcei were detected in the sputum. A diffidence in making a decided diagnosis at once in such cases does not cast any reflection on a physician's ability. Thus supposing a Poorhouse patient is suffering from Phthisis, but who is unaware of its existence and who has never felt specially out of sorts, probably because he may never in his experience have felt in-sorts, comes under observation with a

history of Acute pain in his chest perhaps coincident with a pyrexial attack. If on auscultation a consolidation is present- it need not be apical, as in two cases I have seen it basal, one posterior and the other axillary; in which a temporary error of diagnosis might quite excusably be made. It is quite a common experience for Phthisical patients, where the presence of consolidation is known, and where there is little or no pyrexia and who are able to go about, to be seized with sudden pain and pyrexia. Now, supposing such a patient at this stage , especially if unintelligent, were to come under observation with a history of sudden and acute pain of one two or three days duration and on examination a distinct consolidation is found, the diagnosis of Acute pneumonia would almost be justified. A few days continuous observation however, soon clears up any doubt in such instances.

Lastly in one or two anomalous cases, the diagnosis of Acute pneumonia is made because it is difficult to think what else it can be. Case 110 was admitted with a history of two weeks illness which commenced with sudden pain of right side just above the liver. As the result of the physical examination a diagnosis of right pleural effusion was made, and 42 oz. was drawn off. After this on auscultation a consolidation of the right posterior base extending upwards to the scapular angle was noted. During the next two weeks the temperature was intermittent ranging between 102, 103 and 104 and normal or subnormal. The expectoration was mucous but not rusty and on three occasions the deplococci were observed. Eleven days after the waves the temperature had become and continued normal, and twenty three days after the first observation the consolidation had cleared away. Was this condition pneumonic? This diagnosis seems to be almost the only allowable one in spite of the anomalous and irregular course of the disease.

PROGNOSIS.

In giving a prognosis of any diseased condition, the observer forms an idea of the probability of either death or recovery ensuing. This presupposes a knowledge of the life history of the disease and the influence of various conditions which may be pre-existent or may arise during the course of the illness. One naturally, therefore, desires to know what is the average death rate and how it is influenced by age, habit, complications, etc. Of these 120 cases there were 49 deaths equal to about 41%. This high death rate may possibly be due to the great prevalence of the alcoholic habit. These figures when arranged according to the day of death show that comparatively few patients died before the sixth day. Only one case died on the 3rd. day; and each succeeding day up to the 8th. is accompanied with an increasing proportion of deaths. (see Table 7).

Table No 7

Table of deaths according to Day of Death

Day of Death	No. of Deaths	Percentage	do.
3rd	1	2%	
4th	1	2%	
5th	3	6%	18%
6th	4	8%	
7th	13	26%	
8th	11	22%	66%
9th	8	16%	
10th	3	6%	
11th	4	8%	16%
12th	1	2%	
	49	= 41%	= 41%.

Thus in only 9 cases = 18% did those causes that make for death result in a fatal ending by the sixth day. But it would appear that the 7th. 8th.

and 9th. days of the disease are very fatal days. On the seventh day thirteen died, eleven on the eigth, and eight on the ninth day, roughly about 66% of all death. There is a peculiar hardship about this fact as one cannot help feeling that these patients died just on the thres-hold of the crisis, and that could they have been tided over another twelve hours or twenty four hours all might have been well. Again it is observed that death may be delayed even as late as the twelfth, and 16% of all deaths did not result until after the 9th. day.

These figures, again when classified according to age show that the more advanced the age the greater is the natural tendency towards death. Thus in the decades 30-39 and 40-49 although the numbers are about equal (29 to 31 cases) the death rate of the latter (50%) is higher than the former or lower decade (41.5%). The younger the patient the greater are his chances

Table No 8.

Showing liability to death as affected by AGE.

Age in Decades	Total Number Cases	Number of deaths	Percentage
0-9	6	0	0%
10-19	9	2	22%
20-29	16	5	31%
30-39	29	12	41%
40-49	31	16	50%
50-59	16	7	43%
60-69	11	7	63%
70-79	2	1	50%
	120	49	= 41%

of recovery, and that even in the Work-house, no person below ten years of age should die of Acute Lobar Pneumonia. Also when these records are classified according to the part of lung diseased it would seem that the part of the lung affected has little or no special bearing on the death rate. There were 31 right apical pneumonias with 14 deaths equivalent to a death rate of 45%. Of the 37 right basal pneumonias 16 died (43.2%) and of

34 left basals 14 died equal to about 41%. Thus according to these figures it would appear that the mortality is little influenced by the part of lung consolidated.

In pneumonia it is very unsafe to give a prognosis of either recovery or death. Statistical arrangement of cases that have been, show some interesting conclusions but unfortunately the conclusions that can be drawn are at such variance with what one sees, that they are of little use when brought to bear on a case under observation. In the absence of heart failure all one can say is that the patient is doing well so far, but one never knows when this complication will supervene. Many cases are seen where the patient seems all right, the consolidation is small and is not spreading. It exists in statu quo for a few days then with little warning heart failure sets in and death ensues. Again many cases recover which appeared almost hopeless. So many are these surprises and so many apparently contradictory conditions are present therewith that in the absence of actual heart failure almost no scientific prognosis of any real value can be made. Until the crisis the only course is to watch and wait.

One would naturally expect that the more extensively the lung is diseased the greater would be the certainty of death. Yet the only three cases that I have seen where the whole lung was solidified recovered and of the twelve double pneumonias only 4 died equal to 33% only, as against 41%, the average for the total series. Thus a classification and assortment of these records do not bear out one's first natural impressions. Under the heading " Temporary Cardiac Conditions" there were eleven patients in whom a mitral murmur developed, and four in whom a pulmonic murmur appeared. One would naturally expect that as the result of the strain that was put on the heart, the heart had dilated somewhat, giving rise to the physical conditions favouring the appearance of a murmur, and consequently would be in a more weakened and dangerous condition than if no such murmur were present, and

to that extent would be nearer the line that divides cardiac incompetence from competence. Moreover one would reasonably expect that they were justified in giving a more grave prognosis, yet of these only three died (= 26%) .

Again there were 18 patients in whom the urine became alkaline. Here one would be justified in attributing this to weakened inhibitory action of the bladder mucous membrane on bacterial growth, and consequently a general weakness of the tissues and system generally. Of this type, however, only three died a death rate of 17% as against the total average of 41%. Here again a rational proconception is not proven by experience.

There were 20 patients in whom a deposit of albumen in the urine was recorded with 14 deaths, equal to 70%. This clearly shows the great gravity of this complication. Still its appearance does not render the prognosis necessarily hopeless as the remaining 30% recovered. In cases 18 and 23 there were distinct deposits of albumen for two andthree days before the crisis. In the former the urine was normal on the seventh postcritical day and in the latter on the 5th.

In 13 instances the patients were admitted with the existence of cardiac valvular disease. Of these seven died (= 54%). Here again the existence of cardiac valvular disease does not render prognosis absolutely hopeless as the remaining 46% recovered, but here it is worthy of note that where recovery ensued, the cardiac lesion was a mitral systolic one.

In fact so many surprises does one see in the course of pneumonia, that very little reliance can be placed on the value of a prognosis, so long as cardiac failure is not actually present. Short of this, there is almost no extreme from which a patient cannot recover : high temperature, high pulse or respiration rates, extensive or double consolidations, temporary and permanent cardiac lesions, marked albuminuria etc. etc.

When however, Cardiac failure sets in, the case becomes quite hopeless and

the patients sinks surely and steadily towards death. It is recognized by extreme rapidity and weakness of heart and pulse, and perhaps irregularity may be present. The face becomes pale and livid and the whole body covered with a cold perspiration. Delirium may set in , if not already present, and the patient sinks down into the bed, the breathing which becomes more and more rapid becomes associated with loud mucous tracheal rattling He or she generally remains conscious and although is unobservant may still answer questions in an intelligent but breathless and exhausted manner. Sometimes he may become completely unconscious for a few minutes, bt finally death closes the struggle and all is still.

But when a crisis is once fully established the prognosis is almost absolutely perfect . That is to say one expects that almost everyone then will recover. Only once have I seen a complicating sequela ensue, namely in one case where gangrene of the lung set in. In none was there anything to lead to a suspicion of a tubercular element being fanned into flame by a pneumonic attack. Any relationship that there may be between pneumonia and Phthisis must be a very loose one. This is also seen from an investigation into this point , from the other side, viz by noting the past histories of patients who were the victims of phthisis, and observing the time relationship in those, in whom a history of a previous atack of pneumonia was recorded. One hundred such were questioned. Of these 19 had had Acute pneumonia previously, but in 12 this was from 10 to 25 years of ago. Of the remaining 7, one had acute pneumonia a year ago, four two years , and the remaining two, three years ago. There is thus a possible causal relationship between pneumonia and Phthisis, but it is very slight. Practically, therefore, in all cases after the crisis the recovery is perfect and complete.

Treatment:- There is nothing special to record, as these were practically treated all alike; and complications, as they arose. The routine treat-

ment was stimulant in character, digitalis, brandy, etc. as indicated.
Of the complications of pneumonia I am in a position to say little as I have seen very few with the exception perhaps of Cardiac Valvular lesions.

Joseph Adam Clarke.

"Declaration"

I hereby declare that the foregoing Thesis has been prepared entirely by my own efforts from clinical material, collected personally, except in so far as I was indebted to the nursing staff for the registration of the temperature, pulse and respirations; also for recording the initial onset of either delirium, diarrhoea or vomiting &c.

Signed

Joseph Adam Clarke

MB ChB