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$\alpha q\varepsilon$.

During a period of 21 months I have tabulated a list of 248 cases. These were all examples of Acute Lobar Pneumonia, from which Broncho-pneumonia and hypostatic consolidations were carefully excluded. Not all however were uncomplicated by preceding disease. Some followed upon Phthisis, others upon Cardiac and Neural diseases, one was associated with irritant poisoning and complicated with Gonorrhoea, Cystitis and Nephritis. But the immediate if not the sole cause of death was in all cases Acute Lobas Pneumonia.

A number of these had a short term of residence in hospital, no fewer than 22 dying within one day of admission while 9 died on the second day and 12 on the third. These 43 cases were beyond the reach of treatment from the first and will be again observed in considering the influence of treatment.

The generally accepted fact that age increases the mortality of Acute Lobar Pneumonia is well borne out by my statistics. Under the age of 20, were 27 cases, and of these only one died, a weakly, neglected, and houseless lad of 18, moribund on admission. Between the age of 20 and 30, 57 cases occurred and 20% of these died. Between 30 and 40 there were 59, the highest number, of cases, and of these the mortality was over 35%. Between 40 and 50

are tabulated 52 cases with a mortality of 42%, while between 50 and 60 of 37 patients 57% died; and over 60, 50% of 22 cases proved fatal. Appended is a complete list of these cases arranged in periods of five years.

Years of age	Males			Females			Totals		Total Cases	Percentage Mortality
	Recovered	Died	Total	Recovered	Died	Total	Recovered	Died		
1 to 5	1	-	1	-	-	-	1	-	1	-
6-10	1	-	1	3	-	3	4	-	4	-
11-15	3	-	3	1	-	1	4	-	4	-
16-20	14	1	15	3	-	3	17	1	18	6.5%
21-25	16	4	20	2	1	3	18	5	23	21.7%
26-30	19	3	22	4	2	6	23	6	28	17.8%
31-35	10	9	19	3	1	4	13	10	23	43.4%
36-40	20	6	26	5	5	10	25	11	36	30.5%
41-45	8	12	20	5	0	5	13	12	25	48%
46-50	15	8	23	2	2	4	17	10	27	37%
51-55	10	11	21	1	2	3	11	13	24	54.1%
56-60	5	7	12	0	1	1	5	8	13	61.5%
61-65	6	7	13	1	0	1	7	7	14	50%
66-70	3	3	6	1	0	1	4	3	7	42.8%
71-75	-	1	1	-	-	-	-	1	1	-
Total	131	92	203	31	14	45	162	86	248	34.6%

Owing to the small number of cases in early period this table is of much less value than the synopsis in decades given above. Examples of true lobar pneumonia under the age of 15 are very few, (9 in all,) though bronchitis and bronchopneumonia are very common and very fatal in the case of children admitted. That this is due to a less liability on their part I have had several opportunities of

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verifying; where a mother and child had having been subjected to the same conditions of privation and exposure, the child was bronchitic, the mother suffering from Lobar Pneumonia. From the aspect of Prognosis these cases support the universally acknowledged fact, - extreme youth and pre-existing disease excluded, - that a happy termination may in almost every instance be expected in Acute Lobar Pneumonia under the age of twenty. Of 27 such only one died and he had been under no treatment. For the most part the cycle of their lives had been narrowed down to poverty, neglect, rags, exposure and neglect, alternating for a space with the dingy day light of Poorhouse relief. Above the age of twenty a sudden and startling rush to death begins. Agreement with published statistics is seen only in the steadily increasing mortality up to the age of Sixty; but, for the sudden increase of numbers attacked, and the large percentage of deaths, other factors than age are responsible. In the aged these causes have ceased actively to operate, and the proportion of deaths is here again within customary limits.

It is mentioned by most authorities the liability of the sexes to Pneumonia is in the ratio of two males to one female, instead of 4.5 males to 1 female as the above table indicates. But that sex per se has any influence in generating

Intemperance.

the disease there are two rational grounds for holding. In both sexes the largest number of cases occurred between the ages of 30 and 40 (36 males and 10 females), and mortality in both is nearly equal but slightly greater in males, (35.4% : 31.1%). Upon this point there is slight variation of opinion, but the mortality in the sexes may in the main be held as approximately equal, and sex to be by itself to be of no importance in estimating the probable issue.

That alcohol not only predisposes to Pneumonia but increases its mortality all authorities agree. In the large number of my cases, and in the high and sustained death rate over the age of 20, rests ample confirmation of these propositions; *viz.*, do I think that I overstate the truth when I say that 90% of those over 20 years led lives of alcoholic dissipation whenever opportunity offered. It is often a matter of extreme difficulty to elicit confessions of intemperance. If any were moribund on admission and in very few of these cases could facts of previous history be gleaned. Further the statements of poorhouse habitues are most unreliable; suggestions of intemperance they will often indignantly deny with lips redolent of alcohol. To give two instances: — E. B. aet., 47, a man of robust proportions admitted on March 9th 1894 with face, neck, hands and feet congested, anxious expression,

eyes bright and glistening and restless. He mutters to himself at intervals, and delirium is obviously impending. His occupation has not exposed him to weather or hardship, he has caught a cold he cannot tell how, and he has not tasted alcohol in any form for years. Pneumonia is left apical, the whole lung rapidly becomes solid, and violent delirium arises. Two days later a patient in the ward informs me that E. B. has been known to him as an inveterate drinker for years; that once the proprietor of a steady business he had drunk himself adrift from home and friends, and was now reduced to live in Model Lodging Houses, picking up a day's work now and again; that he got drunk whenever he had money, and in his cups was suspicious, passionate, and actively belligerent on the slightest provocation. On the 14th he died and his relatives who are prosperous and respectable confirmed in sooth this story.

P. E. age 37 admitted 4th April 1894, was a man of powerful physique with complete consolidation of the left lung from apex to base. Here the fatal event was presaged on the man's face. His expression was dull staring and stupid, and he died within forty hours of admission, all the usual methods of treatment, bleeding included,

being but drops of water to a raging furnace. His statement was that he followed the occupation of fireman; that he was subjected to alternate heats and colds; that he earned good wages and preferred keeping his money in his pocket, and taking quiet constitutional walks into the country, to spending his means on liquor, as many of his companions did. His landlady on the other hand asserted that he had not worked for months at his trade; that he had been constantly drunk for three weeks, during which time he had taken scarcely any food, and had lain out in the streets at night, one one occasion being drugged, robbed and left lying in an exposed courtyard all night. To this she ascribed his illness.

Taking 62 cases of all ages, and unselected but for the fullness of the reports, alcoholic propensities were admitted or were found for certain to have existed in 47. 7 were doubtful and in 8 the habitue did not exist. The latter were all young and for this reason alone we would expect a small percentage of deaths. Yet two of these died; viz., - A. P. aet. 20 a young man of fair development, a labourer, with history of injury to chest and subsequent exposure to wet. Death occurred two days later, and I found the right lung in a state of red hepatization from

Apex to base. The visceral layer of the pleura was thickened but non-adherent, and all the other organs were healthy.

Family and personal history were both good, and no adequate explanation of the severity of the attack was applicable.

The other W. C. aet. 21 had been much neglected from infancy. He was a weakly starved lad, his mother had been long dead, and his father was a confirmed Sot. Dying 7 days after admittance the Post Mortem Examination showed consolidation of the lower three-fourths of the right lung with pleural surfaces thickened and firmly adherent. Solidification was of the grey variety and on the lateral aspect formed a rhind about half an inch thick internal to which the great bulk of the lung had broken down into a pus cavity.

Of the seven doubtful cases 5 recovered (4 under 30 and one 38 years of age), while 2 died aged 36 and 47 years respectively.

F. H. aet 47 was a man of average height and moderate strength. He had suffered from bronchitis for thirty years and had no fixed place of residence for two months before admission. He had been going about the city in quest of work, doing odd jobs, suffering often from utter desititution and frequently not earning enough to pay for a bed, on which occasions he lay on stairs -

landings during the night. He had acute Lobar Pneumonia of 10 days standing, and he died on the morning following his admission. He affirmed that he never drank but Post mortem the liver and kidneys were cirrhotic.

The other a female E. G. at 38 was a deaf mute and had been an inmate of the infirm wards of the poorhouse for the past four years. She had an attack of acute Pneumonia (left basal) about a year before the second, which terminated with death on the 10th day. The aortic valve was incompetent, the liver slightly cirrhotic, the common bile duct blocked and the gall bladder full of minute concretions of solid bile products. The right lung was in a transitional stage between red and grey hepatization in its upper two thirds, and the left lung had marked engorgement off its base.

Passing on now to those 47 cases in which Alcoholic habitance in varying degree was proved, we find that 30 recovered and 17 died, a percentage death rate of 36.2 nearly. Yet this is lower than the 42.8% mortality assigned to the intemperate by the collective investigations returns, or of the 41% of Poolidge and Townsend.

Of the fatal cases two were under the age of 30; viz., -

1. J. D. age 29 a moderate drinker in the sense that he only got drunk at intervals, - a labourer having very fatiguing work to do, and constantly exposed to cold and wet. On Novr 26th 1892 he was carrying coals and got his clothes soaked. Thereafter he got drunk and slept in an exposed situation during the night. He was admitted to Hospital on the 30th (4th day of illness) with consolidation of the right lung, extreme apex excepted, and died in 36 hours.

2. C. D. age 25, a prostitute with a history of habitual drunkenness exposure and destitution. She lay for 5 days neglected and unheeded in a dirty hotel. On admission she had a temperature of 104.6°, P.R = 100:40, and the right lung was solid from 2^d dorsal spine to base. Striking was diffused all over this lung, and there was no expectoration. She lived 3 days.

Two were between 31 & 35.

1. H. O'D age 33 a stout-built man with a record of periodic drinking bouts, exposure, heavy work, and a previous attack three years before. He was admitted on the 2^d day of illness already exhausted by the severity of the attack, and died on the 5th day with consolidation of the lower lobe of the left lung, hepatization being (post mortem) found in a transitional stage between red and grey.

2. J. H. G. age 32. Admitted Oct. 3^d 1893, a man of robust habit, a heavy drinker,

who had fallen from a height 7 days previously on the left side and shoulder. Pneumonia seemed to have developed directly after the injury, and implicated the left lung from the base to within 2 inches of the apex. He died on the 7th day of disease and 4 days after admission.

Two were between the ages of 36 and 40.

1. M. M. act 35, a female, admitted Nov. 16th 1893. Of robust habit, she had always enjoyed good health, had been exposed with bare feet on a cold wet day, and admitted to having consumed a good deal of whisky in her time.

The section revealed advanced ^{red} hepatisation of the lower lobe of the right lung, acute pericarditis and pleurisy, fatty liver, and dermoid cyst of the right ovary. Death occurred 2 days after admission, on the 8th of disease.

2. J. Y. act 38 admitted Feb. 3^d 1894, well nourished but of spare habit, a carter to trade, constantly exposed, with no previous illness, and addicted to frequent alcoholic excess. Illness dated from the day he sought admittance and lasted 11 days. The left lung, generally adherent, showed paroxysmal degeneration of the upper and advanced red hepatisation of the lower lobe.

Three were between

41 and 45; viz, -

1. J. G. act 43 with long continued bronchitis

he had been a constant drinker. No crisis occurred and he died 16 days after admission, with the whole of the right lung in a state of tissue destruction and purulent infiltration.

2. O. H. age. 42. ^{31/12/93.} A stout labouring man, who was drunk whenever he had money. Admitted to hospital on the 2^d day of illness, he lived 6 days longer. The left lower lobe and a small portion of the right base were solidified, but no P. hy. could be got.

3. J. F. age 45 admitted Decr. 19th 1893 with right basal pneumonia. He admitted to incessant drinking for four days, taking no food during that time, and sleeping out of doors at night. Recent pleuritic adhesions were general over both lungs, the right lower lobe was in a condition of red hepatization, & the rest of the pulmonary tissue showed general engorgement. Death followed within 24 hours of admission.

Two were between

46 and 50.

1. M. H. age 48. admitted in a condition of cyanosis and collapse on Decr. 2^d 1893, with consolidation of the left upper lobe. This was on the 4th day of illness, and she died on the 8th. A woman of spare habit and dissipated appearance, she was obstinately silent with regard to her past, but her sister informed the nurse of the ward, that the patient

who did general house cleaning, had been drinking heavily for many months, and had often starved herself and pawned her clothes for drink.

2. E. B. at 47 admitted Mar. 9th 1894. Consolidation of the left upper lobe developed in three days, and two later he died. An inebriate drunkard, already referred to.

Three were between 51 and 56; viz., - 1. W. H. admitted Oct. 14th 1893. A man who earned his livelihood by carrying advertising boards, well nourished, healthy in the past, and a constant soaker in alcohol. Dying in 2 days, the P. hy. examination exhibited red hepatization of almost the whole of the right lung. With a feeling of malaise for a full week before he came to the poorhouse, he had continued going about.

2. H. H. at 53, a female, admitted Mar. 16th 1894. Her occupation was that of house cleaner. She was stout and very fat, and suffered from right basal pneumonia. She had been out late on the 10th of the month, and coming in wet found her bed occupied by her landlady who was drunk. Having to go about and lie in her wet clothes all night, she had several shivering fits next day, but managed to keep her feet for

three days longer. She had not drunk heavily as a rule but had consumed a good deal of whisky during the past month. She died on the 8th day of illness, no P. sy. was attainable.

3. H. H. K. aet 54. admitted Jan. 15th. 1894. She had suffered from Chronic Bronchitis for 4 years, and was weak and debilitated, destitute and drunken. Death occurred on the following day with red hepatization of the left lower lobe, and general bronchitis, pulmonary engorgement and edema.

There were 60 years of age or more; viz, -

1. J. B. aet 62 was admitted on Oct. 18th. 1893. He was thin and starved, had no fixed occupation, no fixed residence, and had been intemperate for many years. First under treatment on the 4th day from onset, he succumbed on the 11th day, with grey hepatization of the upper and red hepatization of lower two lobes of the right lung. The aorta was atherosomatous, and the mitral and aortic valves were thickened.

2. J. A. aet 61 with date of admission Oct. 30th 1893. An old cotton spinner, stout but somewhat flabby, he had been reduced to labouring work for the past ten years. He had always enjoyed good health, but had been

a heavy drinker, and his wife would not live with him on this account.

He died at midnight of Decr 31st, 4th day of disease, and post mortem body findings showed general hyperaemia with red hepatization of the lower lobe of the right.

3: P.P. age 60. very weak and broken down entered the poorhouse on Feb^r 22^d 1894. A blind beggar, he earned his livelihood by singing hymns and ballads, led about by a dog. He had been a soldier and had served his country in the Indian uprising. He had been ailing since the New Year as when since he had been drinking heavily, and sleeping in draughty low dens full of people. He had suffered from cough and tough expectoration yellow or brown and sometimes tinged with red blood, as people told him, ever since. Resolution occurred but with no distinct crisis; râles appeared over the right upper lobe (the part primarily affected); and he gradually emaciated and died 16 days after admission.

I could extend

this record of degradation, misfortune, and vice, these woeful tales of human wretchedness, the only relaxation for which is a revelling in the forgetfulness of intoxication; I could present the vista of poverty, hardship and beggary

to much greater length, but enough has been said to demonstrate the awful fatality of Pneumonia to the drunkard.

Yet 30 cases out of 47 recovered, though the general tenor of their lives is but a repetition of the above.

The following table summarises the ~~mortality~~ of Pneumonia among habitual drinkers.

Habitual Alcoholic Indulgence - 47 cases.

Age.	Recovered	Died	Total	Deaths per cent.
20 and under	9	2	11	18.18%
31 to 40	8	4	12	33.33%
41-60	8	5	13	38.46%
61-80	3	4	7	57.14%
Over 60	2	2	4	50%
Totals	30	17	47	36.2%

From cases of my own I am unable to institute any comparison with the death rate in temperate subjects; for these are practically unknown in parochial practice over the age of 25. Every under 20 alcoholic indulgence is frequently admitted, but is to be reckoned of small moment in contrast with the lowered vital resistance accruing from a habitual and confirmed vice of many years standing. In total abstainers the collective investigation returns shew a mortality of 10.4%, in the temperate of 17.4%. But, as Coombs and Sturgis say, "obviously such statistics are vitiated by the inclusion of children under the first head, as well as by

Hysteria, Insomnia, Delirium &c.

Prostration of
Strength.

The elasticity of the terms employed to denote drinking habits". In conclusion, alcoholic indulgence is a serious element in the combination of ^{the agent in} Acute Lobar Pneumonia; and the more confined is the vice, the longer it is protracted, and the more it loses potency it becomes to lead its army of addicton and constantly increasing sufferings, by so much the more does it endanger the chances of recovery. Old age however is a leveller, for the tissues of temperate and intemperate are alike degenerated, and to both are insured a shelter from cold and hardship.

Although the consideration of sleep, delirium, and mental state is taken now somewhat prematurely, yet ~~forsoom~~ it is warranted by the close relation of these to previous alcoholic habits. And first, to begin with Prostration of Strength. According to Hale "prostration of strength as a rule occurs from the first and is so positive and so marked that the fact may be made available in diagnosis. The exceptions are very rare". That rhus, malaise, muscular weakness, headache, short cough, pleuritic stasis, PTC are rarely absent as initial symptoms, there can be no difference of opinion; but to the dictum that prostration is positive and marked

from the first there are in my cases exceptions by no means rare.

Among 69 cases of all ages in which the history of onset was obtained, the following table indicates the period of illness on admitting to Hospital.

Admitted on 1st day of illness 16.

"	2 nd	"	"	10.
"	3 rd	"	"	4.
"	4 th	"	"	12.
"	5 th	"	"	9.
"	6 th	"	"	4.
"	7 th	"	"	7.
"	8 th	"	"	2.
"	9 th	"	"	1.
"	10 th	"	"	1.
"	11 th	"	"	1.
Doubtful				<u>2.</u>
				<u>69</u>

Although many of these were confined to bed before admitting, their symptoms generally were not so urgent as to excite alarm; and, save those in whom an advanced stage of the disease ^{was reached} ~~extorted~~, the great majority came to the poorhouse on foot.

One patient P. H. G. age 22 (peculiar) had symptoms of bronchitis for 6 days, and continued at work for that time after exposure.

W. H. C. 63. (died) was unable to work for 7 days; but he walked about all that time and died 2 days after admission with almost complete

Consolidation of the right lung.

J. A. 61. (dead) had short cough and general muscular pains but went about for 3 days before he was forced to take to bed. 5 days after he died with pericarditis, slight peritonitis and hepatic action of the right lower lobe.
J. D. 19. (recovered) had shivering, headache, diarrhoea and pleurodynia but continued his work for a week thereafter.
E. B. 54 (recovered) had cough and pain in left side, rusty expectoration, and malaise, but went about during almost the whole duration of illness; viz., - 6 days. Crisis occurred on the night of admission.

The case of P. W. age 40, who recovered, was also of very extraordinary. On admission he was weak and feverish, bronchitic, with fine moist râles at both bases behind. Symptoms receded, and he was allowed out of bed. In my absence he was permitted at his own desire to leave the Institution, feeling as he said quite well.

A warden, observing him toiling painfully along, brought him to me when extensive consolidation involving about $\frac{3}{4}$ of the right lung was discovered.

A good crisis occurred 3 days later.

J. P. 50. (recovered) had shivering, headache, slight jaundice, and pleuritic pain, but continued going about in search of work for a whole week. On

admission with extensive solidification of the right lung, crisis occurred; and this was accompanied by great prostration and cardiac enfeeblement. He was in a very precarious condition, and energetic stimulation alone saved him.

Drowsiness or restlessness and insomnia appear to be the most common nervous symptoms at the onset of acute pneumonia. In a few instances total unconsciousness for a limited period (in one case for 8 days, in another for 4,) occurred, and in several delirium ushered in the attack. Extreme prostration from an early period is an alarming occurrence, and more to be dreaded than delirium; for it indicates a feeble power of vital resistance, and is often seen in the aged or associated with a serpiginous type of solidification. In many such cases subsequent degeneration of the lung tissue and the formation of pulmonary abscess are liable to follow: - E.g., W.C. ad. 21. died 7 days after admission. Extreme prostration was succeeded by low muttering delirium alternating with periods of drowsiness and partial stupor which deepened into coma and death. Post mortem examination showed large pulmonary abscess.

J. Sart-48. and J.C. ad. 26. were profoundly prostrated since admission, with inability to rise and assist themselves in any way, and with low muttering delirium alternating with stupor. In both cases the affected lobes

Telurium

ultimately underwent purulent infiltration and excavation.

I. F. 43. was drowsy with periodic muttering, feeble and sunk down, on the first day of disease. He lived 16 days and the autopsy revealed purulent infiltration of the right lung with cavities full of thick pus.

Of the same 69 cases delirium was a conspicuous element in 27 instances, and 15 of these terminated in death. By the other hand 6 died without at any time showing this symptom.

In general terms, "delirium is a grave feature as regards prognosis"; (Powell.) but its weight in establishing the gravity of a case depends much on the age of the patient, his habits of temperance, of the day of its first appearance, its character and its concomitants.

As Compland and Slurges remark, "In the young early delirium may indicate little, and after ushering in the disease, may quieten down and pneumoniae of ordinary severity follow". In young persons of susceptible nervous organization or in the subjects of hereditary mental instability such neuropathic disturbance is no more than would be expected; but in those who are old and where the type of delirium resembles that of acute alcoholism a more serious outlook is impending.

Of the 9 cases with delirium which ended happily I. F. B. act 40 was delirious

from admission to crisis, (3 days); but, under the influence of appropriate narcotics he slept fairly well at night.

2. J. H. suffered from insomnia from the period of onset. By the night following the 2^d day of pyrexia he was restless and wakeful; but he slept for the greater part of the 3^d day, becoming delirious towards evening, with fitful and broken sleep during the night. These events repeated themselves on the 4th & 5th days, and on the day of crisis he was actively delirious, remaining in this condition for one day longer.

3. C. C. at 22, was only delirious for one night when after a false crisis the temperature rose rapidly to 104.6.

4. W. H. T. at 53, was delirious for one day preceding crisis but after the appearance of perspiration he became calm and collected, drowsy and inclined to sleep.

5. J. H. T. at 50, had for several days broken sleep with great pleuritic pain culminating in transient delirium on the afternoon preceding crisis.

6. J. S. at 48, a weakly broken down man who afterwards died of Phthisis had a peculiar startled and anxious expression with periodic incoherence of speech and ideation since admission. This alternated with short intervals of normal consciousness. Even after crisis, 6 days later, he spoke in a melancholic tone, and shewed great disinclination for speech or physical exertion.

R. W. H. age 32, had been drinking heavily for four days, and eating no food. For four days subsequent, he was violently delirious. This passed off; signs of pneumonia developed; and though weakly and deliriated there were no further symptoms of a serious nature until the day of crisis, when, with falling temperature and profuse perspiration he was again for a few hours delirious.

E. J. D. at 19, had maniacal delirium for one night before crisis.

G. P. H. S. at 38, who had been drinking heavily had delirium tremens with loss of consciousness for 8 days. On admission he had signs of commencing consolidation followed next day by erysipelas Capitis. Delirium of a low type persisted for six days longer by which time erysipelas had faded and pleuritic effusion had developed. Thereafter he had broken sleep until free drainage of Empyema but no more delirium.

Of the fatal cases, 24 in all, 6 were never delirious at any period. Insomnia however was common, sleep was fitful and broken in most, and in all drowsiness and stupor were speedily followed by exhausting, total unconsciousness, and death.

In 5' delirium occurred only on the day of death with such precedents and constants as, - insomnia, staring, restlessness, struggling to get out of bed, shouting aloud or rambling incoherence, anxious pain excited or stupid expression,

Sustultus, Caphology, conjestion and lividity, profuse perspirations or cold and clammy skin with coldness of extremities, harassing cough, involuntary passage of urine and faeces, contracted pupils, rapid and laboured respirations with mucus collecting in throat, weak thready and irregular pulse, dry brown tongue and sores on teeth, foul breath, refusal of nourishment, unconsciousness, death.

8 were delirious for 2 days before death. In one case where delirium was violent, muscular twitchings and general convulsions appeared. In 2 cases maniacal delirium with strong muscular efforts produced sudden collapse and death in an hour or two. In all a prior stage of sleeplessness and restlessness occurred, in most from the beginning of the attack.

2 were delirious for 3 days before death, one being of a low muttering asthenic type; the other maniacal at first, but followed by collapse tossing the arms about, moaning, muttering and stupor.

One was violently delirious for 5 days before death. J. F. at 38 had delirium for 6 days before his decease. Wild maniacal excitement in sudden outbursts alternated with intervals of prostration and stupor for 4 days. He then became violent and utterly unmanageable for 36 hours when sudden collapse terminated the scene.

W. C. at 21 had low muttering delirium

drowsiness, stupor, and great prostration from admission to death 7 days later.

To sum up its position in prognosis, delirium is a symptom which almost invariably affords some ground for anxiety. But the outlook is not serious if it occurs at the onset only, and that more especially if the patient be young and well nourished, or at least temperate and not old. The same may be said of delirium associated with falling temperature and critical signs, of transient delirium after a false crisis, or of slight mental confusion following crisis. But the issue is more doubtful if delirium be maniacal in type, or if protracted or recurring; still, if there are alternations with periods of rest and sleep, hope may not be altogether abandoned. But if violent, maniacal, and either sustained or relapsing into intervals of prostration, or if restlessness and insomnia obstinately persist, and are accompanied by great anxiety of countenance and signs of embarrassed circulation, grave doubts of recovery must be admitted. Indeed, violent delirium late in the disease is in my experience invariably followed by collapse and death. Delirium-dreams is always a formidable complication, but it may pass off and the subsequent pneumonia have no alarming symptoms.

Environment.

Inevitably fatal also is a low muttering and protracted delirium, such as we associate with purulent infiltration.

It is commonly asserted that delirium is most commonly found in connection with apical consolidations, but the above 27 Examples rather tend to the conclusion that its presence is much less due to site than to extent, & degree of constitutional disturbance. In these cases the affected areas were,-

Right Apex 5	7	Total
Left " 4		9.

Right Base 7	7	10.
Left Base 3	3	

Whole of Right Lung 5	5	8
" Left " 3	3	<hr/> 27

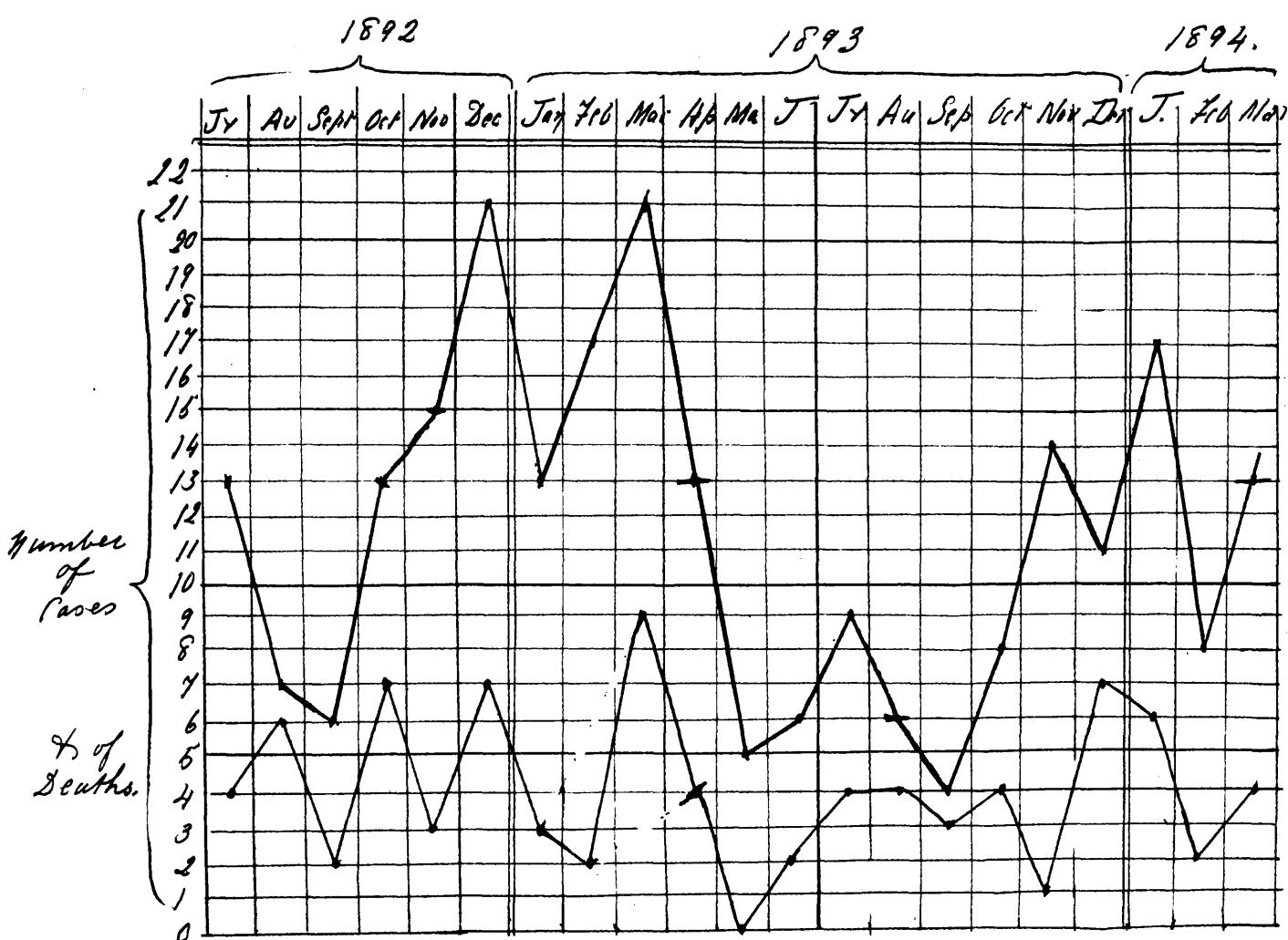
The environment of the individual not only predisposes to Pneumonia or more directly excites its action, but modifies its course and exerts a strong influence on its mortality. Severe weather conditions, exposure, chill, bad hygienic living, alcoholism, starvation, bodily and mental fatigue, and in some instances shock following bodily injury, are all important as aetiological factors in this disease; and, that these in toto or in great part form the environment of the lowest classes of our population,

Weather Conditions.

Gives a satisfactory explanation of its wide prevalence in Parochial Hospital practice.

As it goes beyond the scope of the present inquiry to discuss questions of aetiology, I shall merely allude to these conditions in their connection with prognosis.

Only aimed is a table giving the number of cases in each of 21 months, and the number of deaths in each.



This exemplifies the general conclusions of the Collective Investigation Committee that pneumonia is least prevalent towards the end of Summer, and that sudden variations in temperature, a rapid falling of the thermometer accompanied by high

winds from the north and north-east favour its development. But while Decr. Feb., and March of 1892-93 and Nov., Jan., and March of 1893 yield the greatest number of cases, and the former season more than the latter, the relative mortality is largest in the summer months, most strikingly so in Aug.^t 1892 and Sept.^r 1893.

In Aug.^t 1892 however the seven cases noted all exhibited an alcoholic habitus, with one exception they were all over 40 years of age, and three of them died within 48 hrs. of admission. Again in the summer ^{& autumn} of 1893, May had no deaths; in August three out of four died within one day of admission and one was over 70 years of age; while in Sept.^r, all were young but one of the three was moribund on admission, another having been drinking and lying out at night, while in the third, who lived for three days, previous neglect and lack of treatment were outstanding facts. No value therefore can be attached to the relative high mortality in these months; besides, the number of cases is much too small on which to initiate any questioning of established data. In order to form an independent opinion with regard to the "Epidemic Constitution", and to the tendencies to death at different times and seasons, a large number of cases taken over an extensive period of years is demanded.

Occupation & Habits.

and further the problem becomes much more complex when dealing with the utterly destitute, than when investigations are conducted from cases of a more respectable class.

As a rule my patients have no fixed occupation. Some pick up a living by tramping about the streets, begging, portering, carrying advertising boards, hawking small goods about the city, labouring, laying drains, working in wet underground excavations, filling coal carts, carrying Coals, &c. A few have fixed occupations, as carters, furnacemen, Shoemakers.

Amongst the money cleaning, scrubbing, Sewing, prostitution, are the common modes of earning a livelihood.

Many persons of both sexes have no employment of any kind, and how they continue to live in the intervals between their numerous terms of residence in the Poorhouse, is a mystery over which most are reticent. Many of the males have frequently to ~~pass~~ ^{spend the} their nights out of doors, sleeping in doorways, on stairs, in all manner of pools and crannies that might escape the vigilance of the policeman; or, if fortunate enough to secure the heedful coppers they seek repose in common lodging houses reeking with filth, foul breaths, and strong whisky.

Very few indeed are sufficiently clad to withstand the rigours of our climate. Vermin, incrustations of dirt, and sores

* I have not a single example
of true *infectious pneumonia*
in my list.

are but a poor defence. Yet with all
very few are actually starving, food being
seen by within reach of the lowest.
But the temptation to spend their scanty
earnings on alcohol gains the mastery
over the cravings of natural appetite ;
and in consequence, unwholesome,
insufficient and irregular dieting adds
more fuel to the pyre of degraded nature.
Such a mode of living spread over
months and years ; and, in ^{& around} the individual,
are aggregated and amass'd the whole
aetiological elements of Pneumonia ; —
vital resistance lowered, the organism
vulnerable at all points. A draught,
a sudden chill, sleeping in wet clothes,
lying drunk in the open air, sudden
passage from a hot stuffy room to the
streets, shock from a fall, a severe
trailing and kicking, or after a cold
got no one knows how, are
sufficient to kindle into deadly conflict
the flames of Pneumonia and the
sapped and withered roots of life. It
would be useless labour to isolate
and tabulate here, or to attempt to
relate to each separate item its value
in prognosis. Generally speaking mortality
is in direct proportion to the sum of
these precipitating and predisposing
elements ; but very here exceptions are
by no means rare, as when a
man past middle life, a moral
and physical wreck is attacked by Pneumonia.

Habit of Body.

¶

Previous attacks.

which apply all prove to be of only moderate severity, and with an early and happy crisis he weathers the storm. Let such a result always come upon one as an agreeable surprise, an exception worth noting, a freak of nature.

"A plethora state of body," says Powell, "seems to favour the occurrence of pneumonia and to add much to the severity of attack." It is a common dogma that pneumonia attacks preferentially the strong and robust; but to a statement so bald must be appended the inseparable ~~item~~ ^{association} of lowered vitality. In 53 cases where special mention is made of the bodily habit, I find that 29 were stout and well nourished, but some of these were fat and flabby, others exhausted with dissipation, nearly all bore the stamp of the fallen Lucifer. 15 recovered and 14 died. 16 were of spare physique and only 3 died, while 10 were emaciated and a like number died. It would seem from this, so far as it goes, that the chances of recovery in descending progression are in favour of (1) those of spare habit, (2) the weak and emaciated, (3) the stout and robust.

A former attack is alleged to favour recurrence; yet, in 69 cases, I find only 12 for certain who had previous attacks, one with three, three with two, and six with one. Only two of these died. The former attacks varied in time between 17 years and 11 months before the present, attacks 2 or 3 years being the most common.

Pre-existing Diseases.

But from the loose statements and general ignorance of my informants little positive information, few facts that are reliable, can be drawn. I therefore attach little value to the above figures.

The ^{consideration} ~~importance~~ of preexisting disease in the individual, or of former illness, must not be omitted in weighing the gravity of a case.

Out of 56 cases in which such inquiries received no equivocal replies, 21 had been previously weak and ailing, while 35 stated that they had always enjoyed good health. Of the latter 16 died or nearly one half, and of the former 7 died or only one third.

Chronic Bronchitis or Emphysema or both existed in 8 cases, and of these 4 died. Rheumatism, Cardiac valvular disease, cardiac hypertrophy, Malaria, Enteric fever, Delirium Tremens, dysentery, or sunstroke occurred in the past history of 9 cases, all of which recovered. Symptoms of Phthisis preceded pneumonia in two, one fatal; and acute rheumatism with valvular impairment was followed by pneumonia in two, both dying.

Returning to the old dogma that pneumonia has a predilection for the strong and robust, it is easily apparent how this is so, excluding of course all the continued fevers and exhausting diseases which it complicates. The Constitutional tear and wear, physical and mental, which gradually moulds the organism into suitable shape for its reception, is only compatible with a certain amount of previous good health.

With a multiplication of causes a slight occasion suffices to raise the Pneumonic process into activity. But these causes do not so accumulate in the physically or even in the mentally weak. In my Lunatic practice I have much often seen pleuritic effusion or Empyema than pneumonia; further, pulmonary disease as is well known ~~use~~ is unusually great prevalent in the shape of Phthisis in Asylums. And so with the physically weak, they are debarred from the enjoyments of nervous excess as they escape its results. Conservation of what little energy they possess insures for them protection.

The subjects of Chronic Bronchitis seldom develop pneumonia; but when they do and especially when complicated with Emphysema, Prognosis is markedly affected: - e.g. of the 4 fatal cases with a history of chronic bronchitis 3 in which post mortem examinations were made had Emphysema in both lungs. But when, with a history of alcoholism and exposure, acute bronchitis arises, and from this with only moderate temperature comes dullness with fine crepitation & consolidation, the case is almost invariably hopeless unless in young subjects. Early congestion, lividity, and coldness of the extremities appear; the heart fails; and warmth and stimulation of all kinds are of little avail.

I have no record of cases of established Chronic Bright's disease in which Acute Pneumonia occurred. In these also the disease is

grave but fatal. In 51% reports however I find increase of weight of the kidneys, cystic disease, thinning of the cortex, ~~or~~ cirrhosis.

The previous condition of the heart is a matter of prime importance in pneumonia. But though of chronic valvular disease of the heart hypostasis and slowly developing splenization or induration are common, the acute process is not often seen. The gravity of such cases depends much upon the adequacy of compensation. It will be of interest here to observe the frequency of cardiac or pericardial disease in post mortem examinations. The following are notes on this point taken from 10 examination.

1. J. A. 61. Pericardium is firmly adherent to heart, more particularly on the right side. The heart muscle is normal in appearance, with valves competent, and along with pericardium the weight is 23 oz.

2. J. F. act 38. Fibrous material extends from the left lung all over the pericardial sac: about 2/3 of serous fluid containing flocculi of fibrin in sac. Heart pale with hypertrophy of muscular walls of left ventricle. All cavities full of post mortem clot. Slight atherosoma over aortic valve and thickening of mitral flaps; all competent. Weight 12 oz.

3. E. G. act. 35. Heart $10\frac{1}{2}$ oz, pale and flabby, normal on right. The mitral valve has warty thickenings along its edges. The aortic valve shows coalescence of two cusps by a cartilaginous nodule, and is incompetent.

4. F. M. 47. Heart weighs $10\frac{1}{2}$ oz., and is normal. Fibrous clot in all cavities. No pericarditis.
5. P. E. act. 37. Pericardium covered with thick layer of plastic fibrin. Endocardium, and endothelium of great vessels are deeply stained. Valves normal. Weight $14\frac{1}{2}$ oz.
6. M. H. act 38. Parietal pericardium covered with loose yellowish white fibrous membrane which extends over right lung. Dilatation of both ventricles, especially the right. The aorta did not admit two fingers unless forcibly stretched; the pulmonary artery admitted three fingers. Valves competent and healthy. Weight 14 oz.
7. F. B. act 36. Mediastinal glands enlarged and softened. No pericarditis. Great hypertrophy of left ventricle, and thickening of edges of mitral valve. Aortic valve competent and normal. Atheromatous patches numerous in aorta, some with calcareous plates and one with ulcerated surface; others show commencing ulceration. Weight $18\frac{1}{2}$ oz.
8. W. H. C. act 53. Heart muscle soft and friable. Aorta atheromatous; one patch on inner aspect of aortic valve. Weight $11\frac{1}{2}$ oz.
9. A. P. act 20. Heart normal. Weight $10\frac{1}{2}$ oz. No pericarditis.
10. H. O. D. act 33. Heart very soft and flabby, right ventricle dilated and walls very thin. Left abs thinner than normal. No pericarditis. Weight 11 oz.

In only three out of ten examples were the heart and pericardium healthy and uninvolved. Indeed, it is exceptional

in post mortem examinations of cases of Acute Pneumonia to find the important vital organs all sound.

Acute Pneumonia may attack the subjects of Phthisis and run an ordinary course, though with lungs already seriously damaged there is little hope of recovery. On the other hand a man previously weak and complaining of long persistent cough, bright expector or haemoptysis, is seized with acute consolidation & pyrexia, followed by crisis or typhus and partial reabsorption of the hepaticized area; and after a few weeks of comparative health the lung excavates, hectic fever develops, and he dies. These acute consolidations, often extensive, seem after resolution to weaken the resistive power of the pulmonary tissue which rapidly becomes a prey to tubercular destruction. From experience of such cases I am inclined to believe that the gravity of pneumonia in phthisical subjects is rather understated by authorities.

Meningitis is said to be very fatal as a complication of pneumonia. Of this I have only one example, and the discovery was ^{first} made in the post mortem room.

To summarise, - pre-existing diseases of vital organs all affect prognosis unfavourably; and cognizance of their presence, with notification of the amount of damage already inflicted by their agency, is all important in

mode of onset.

Summing up the case. In doubtful cases these weights to the scale may determine the ultimate fall of the balance.

It is generally impossible to prophesy from the primary symptoms. But in the presence of no infectious agents in a crimsonous onset is by no means to be regarded as menacing or dangerous. Such a state of affairs is not at all likely to last, indeed can scarcely possibly continue beyond a very limited period, and death before the fifth day of illness is seldom if ever seen. A more violent explosion with early lassitude, dyspnoea, delirium, epistaxis and melanæra is more serious though except exceptional, while a complication of acute bronchitis is as already stated very grave in all but young subjects. Again the primary symptoms may be so trifling that the patient may not be congnized until near the time of crisis. Here the onset would indicate a benign course were rest and means of treatment available from the first; but these cases raise the mortality of Parochial Hospitals to a very appreciable extent, and more than any others shew the danger of neglect in the early stages of the disease. In others, the termination is more fortunate, e.g. - a man had a "cold" of six days duration which did not seem to cause him much concern.

Observing however that his breathing was rapid I examined him and found the whole of the left lower lobe in a state of hepatisation. He was consigned to hospital against his will; and next day pleuritic pain was gone, respiration appeared over the whole part affected, the temperature dropped from 101.2° to 98° , and in response to my inquiries he laughingly assured me that he was quite well, and demanded to be allowed up out of bed. He no doubt considered me a fool for my pains. In another instance where rest in hospital was insisted upon, and where the left upper lobe was perfectly solid, the patient, a returned convict, of surly and savage disposition, seemed to regard our attentions as superfluous and insulting. The nurse of the ward persisting in holding a drinking cup to his lips, he dashed it out of her hand, and threw her violently onto the middle of the floor. But although informed that his life was at stake he insisted on being discharged, and I never heard of him again.

Such experiences are enough to raise the question, ^{are} there cases of pneumonia so slight as never to reach the stage of prostration at all?

In striking contrast are those examples where an old man has had a cough for a week or so. He is feverish and would like a rest. Next day he finds rest for ever

and the P. h. examination reveals red hepatization of the right lower and splenization of the left lower lobe.

A woman, age 56, stout plethoric and flabby has a series of sharp epileptic fits, and lies for two days prostrate and semiconscious. Respirations are observed to be quickened, pulse rapid, temperature 101.2. Percussion is dull over the left lung behind up to the level of the 8th ribs. Fine friction sounds and whipping tubularity are heard over patches of the left lung behind, over the right base, and over an area of 2 ms diameter at the right apex in the supra-scapular region of the scapula. Treatment is of no avail and she dies in three days. This leads up to a large class of cases, for the most part met with in the old where prostration is the prominent feature from the commencement. There is often neither cough nor expectoration; temperature is never high; there is little or no pain; the intellect may be clear, but drowsiness is common; the face retains its natural expression; the pulse not much quickened; appetite is impaired; and, but for the last, and the fact that respirations are quickened, no physical examination would be made. Double basal, ~~or single~~ unequal on the two sides, or single basal consolidation reaching as high as the scapular angle behind,

Area of Consolidation.

is discovered, and very seldom does recovery follow. A similar insidious onset is sometimes seen in younger subjects, though for the most part confined to the weakly and thin ^{persons.} Here prognosis though grave is not necessarily fatal. Delirium as an initial symptom of prognostic importance has already been considered.

Synopsis. In a large majority of cases the onset gives no indication of the probable termination. Many cases otherwise slight, may, from being neglected, terminate fatally. In some instances the initial and subsequent symptoms are so trifling that diagnosis may not be reached. Sudden development of consolidation from prior bronchitis symptoms is serious, especially so in the old. An insidious origin must always give concern. In the aged it is almost invariably fatal.

In 66 cases in which the distribution of hepatisation, its mode and direction of spreading, and its ultimate limits were all carefully noted, I find that the apex or upper portion of the lung was primarily involved in 21 instances, the base or lower portion in 40, 5 remaining doubtful. Of those beginning at the apex 7 died or $\frac{1}{3}$, while 14, or almost the same fraction of the basal

basal cases proved fatal. The five double pneumonia had complete consolidation of one lung (with partial implication of the other in 3), and all died. The right lung was singly or primarily affected in 46 cases with 15 deaths, the left lung in 21 cases with 11 deaths.

Site of Hepatization in 66 cases.

Single Pneumonia	Basal	Right Lung.	19	with 7 deaths.
		Left "	8	" 3 "
	Apical	Right ..	13	" 2 "
		Left ..	6	" 3 "
	whole of	Right ..	8	" 6 "
		Left ..	3	" 2 "
	Double Pneumonia	Right Lung most.	5	" 0 "
		Left Lung most.	5	" 3 "
			<u>66</u>	<u>26</u>

Statistics vary slightly in regard to the relative fatality of apical and basal consolidations. Walde says that "the side affected does not seem to exercise any positive influence, but that inflammation commencing with the upper appears on the whole, though evidence is somewhat contradictory, to be more dangerous than that first implicating the lower lobe."

Powell again affirms "that apex pneumonia excluding cachectic (especially tubercular and alcoholic) cases is not more dangerous than basic pneumonia, and recovery may be as complete in the

one as in the other". Coopland and Sturges assert "that their own experience suffices to shew that while no distinction whatever can be made as between the left side and the right, apex pneumonia is not so fatal as basic".

Any prognostic significance in this respect is at the most trivial, so many associated circumstances of far higher value demand scrutiny in individual cases.

Of more importance is the extent of solidification. The statement of the Collective Investigation Committee "that the rate of mortality when both lungs are involved is more than double that where only one side is affected, the proportion being 7 to 3.3", is not at all borne out by my cases.

The number of Bilateral cases was very small, only 10 out of 66; and this to some extent renders the difference minimised. The importance of any divergence from established data. Of the three fatalities, one had consolidation of a single lobe on one side with a small patch at the corresponding part of the other lung. In the other two one lung was consolidated completely before the other was attacked, and none were over 47 years of age. Of those who recovered one had a small patch at both bases to begin with, but consolidation spread throughout

one lung before any extension occurred in the other. There were cases of advancing basal pneumonia, signs of consolidation of the opposite side appearing near the time of crisis. One was apical appearing two lobes before a small area became solid in the other lung.

The meaning of the term 'Bilateral Pneumonia' is not precisely ^{enough} stated in statistical returns. If it emphasizes a condition where hepatication is considerable in both lungs, this is seldom seen until the period of expected crisis is near, or until that has passed, and extension is still taking place. An extensive determination of blood, with the physical signs of congestion & adema, to a lung previously clear is no doubt very alarming, but this does not justify the appellation of Double Pneumonia. Judging from post mortem appearances which alone are infallible, complete bilateral hepatication is a rare occurrence; engorgement or splenization secondary to hepatication on one side is more usual. But small patches in the lung other than that solidified may appear at any time during the course of the disease. As a rule these foci remain entirely subordinate, or they quickly disappear altogether. Their early presence gives no indication whatever of prognostic import, and if late their importance is much less than the degree and extent of accompanying engorgement and adema. In this connection I may mention that

a fallacious appearance of bilateral hepatisation
is often given by conduction of harsh metallic
tubal breathing to the sound lung. Finally
percussion, friction, and more particularly the
relative difference in vocal resonance resolves
the doubt.

In my cases by far the greatest mortality
occurred with complete solidification of one
lung entire lung. The anterior border however
frequently escaped, yielding Stethac resonance
at some stage during life, and being
found empty sometimes after death.

8 out of 11 such cases died, and of those
which recovered the respective ages were
15, 16 and 30.

Of 27 basal cases 10 died. Of these, two
had consolidation of more than half of
one lung, and in two extension spread
passed almost to the apex. By the
remaining 6 only one lobe was consolidated;
but 3 exhibited general pulmonary exophthalmos;
one was dying on admission; and two
had general bronchitis as well.

Of the 17 recoveries signs of hepatisation
reached the spine of the scapula, the
upper limit of the axilla, and in front the
3rd rib or thereabouts, in 5 cases. Two lobes
were invaded in 4 instances, and only one
lobe in 8.

Of 18 apical cases 5 died. In four
extension reached the base, in the fifth
only one lobe was affected. Two of these
died not of pneumonia but of Phthisis.
Consolidation resolved to some extent, to be

followed by excavation late.

In the 13 examples of recovery, hepatisation reached the base in front in 3 instances, - in one case with three false crises followed by renewed extension, - while 10 were confined for the most part to the upper lobe.

In instances of simple pneumonia, apical and basal, the whole lung for nearly the whole ^{lung} was solidified in 14 with a proportion of recoveries to deaths in the ratio 8:6. Consolidation affected one or at most two lobes in 31 cases with Recovery to death as 22:9.

The deduction to be drawn from these statistics is, that the greater the amount of lung involved the more serious is the case. With complete hepatisation of a whole lung death is more frequent than recovery, but in young subjects, *celeris partus*, a favourable prognosis may be admissible. In Bilateral Pneumonia the extent of solidified lung is the proper fundamental consideration, unless in old subjects or otherwise debilitated subjects. Evanescent patches with signs of moderate consolidation demand careful observation, but per se need cause little anxiety.

Unilateral hepatisations affecting one lobe are in the main associated with recovery, but nevertheless may end in disaster; - age, habits, the previous condition of the pulmonary tissue, of the heart and other organs, determining the issue.

Little or no importance is

attachable to the direction of pneumonia extension. More valuable information can in some instances be derived from its mode of development. A lung, which on one day shews slight basal dullness, friction and inspiratory crepitus, may in 24 hours be hepatised to a considerable extent.

A slowly developing consolidation is always suspicious of a tubercular basis, especially when it only partially affects one lobe, and creeps slowly over part of those adjacent.

The day of disease on which consolidation first appeared in 69 cases varied between the 2nd and 6th, but as the great majority had already been ill for more than four days before admission my information on this point is precise in only 27 cases. I am not in a position to dogmatise on the significance of its early or late appearance. In some with early signs death occurred, in others long delayed the end was the same. In many cases the central or peripheral position of the solidifying nucleus must be the cause of this discrepancy; and, the character of the expectoration, the pyrexia and hurried respirations may warrant diagnosis for some days in advance of auscultatory phenomena. It is commonly stated that these may never appear, and, that on the other symptoms and signs diagnosis may rest. Excluding Epileptical and aborted attacks, I cannot recall an instance of 'Latent Pneumonia'. It is so infrequent experience to find auscultatory

The Expectation.

Symptoms arising in the course of a few hours, and as rapidly becoming obscure again; due to overflowing of fibrinous exudation into the tubules, or it may be to the shallowness of inspiration arising from pleuritic pain. Thus the stage of perfect tubular breathing may be altogether missed, and it may never reappear until after the next expectoration.

Or again superficial turbidity and bronchopulmonary may be limited to a small area, ^{often} in the axillary line or external to the body of the scapula; parts which in a cursory examination are apt to be overlooked. With careful and systematic daily examination, I am of opinion that cases of latent pneumonia would become few indeed.

The following is a summary of the appearance of the sputa in 25 fatal cases

- (1) Fluid and dingy brown.
- (2) Copious mucopurulent expectoration, frothy and at parts tinged with brownish yellow (developed out of bronchitis). It gradually became more scanty, like lemon-jelly, and lastly greenish yellow, opaque, and containing leucocytes.
- (3) No expectoration from admission to death. (3 days).
- (4) " " (4 days).
- (5) " " (2 days).
- (6) Scant, gelatinous, apricot yellow in tint with streaks of frothy mucus.
- (7) Free expectoration, tough, adherent, and streaked with pure red blood.

- (8). Typically rusty on 3^d and 4th days of pyrexia. Then cleared altogether. Death on 7th day.
- (9) No expectoration for 7 days. Scanty, tough, light yellow, for one day. Cleared for one day. Death.
- (10) creamy yellow and fluid, gradually becoming more blood stained.
- Day of Disease = 3^d. 4th. 5th 6th 7th 8th
- Amount of Expectoration - 3X. 3XVI. 3X. 3XII. 3VIII Stopped & (11) On admission scant and rusty but death.
Thinner than usual. Next day more abundant (3XVIII), watery and brownish red.
On 3^d day from admission, typically prune juice coloured. On 4th day more scanty, dirty greyish brown, full of leucocytes (3X). Next day it cleared - death.
- (12) Rusty, scanty and tough, - 3IV in 24 hrs. Entirely suppressed for two days. Just before death it reappeared, dirty whitish brown, opaque and of syrupy consistency.
- (13) Gelatinous and rusty, 4th day, - 3X.
Lemon colored 5th day, - 3XII.
Stopped - death - on 6th day.
- (14) Scanty frothy mucus for 2 days. Entirely disappeared for 6 days when death occurred.
- (15) No expectoration for 3 days. Scanty and prune juice with frothy top for 2 days, and death.
- (16) Scanty, tough and stringy, dark rust colour with abundant froth. Stopped 8 hrs before death.
- (17) Rusty for 2 days. They innumerable blood stained lumps floating in a frothy matrix, for 3 days. Suppression for 2 days, - death.

- (18) Typically rusty for 3 days. Suppression for 2 days, and death.
- (19) Young mucous expectoration sustained throughout.
- (20) None before 4th day.
Greyish white and frothy on 4th & 5th days - very scanty.
Young frothy mucus on 6th day, - 3 X.
" " " but slightly yellow on 7th, - 3 VI.
" " " " " on 8th, - 3 V.
Darker more fluid with frothy top on 9th, - 3 VI.
Prune juice but lighter and containing leucocytes on 10th. - 3 VI.
Suppressed, - death on 11th day.
- (21) Typically rusty for a week, then mucopurulent.
Finally almost pure pus and death on 16th day.
- (22) No cough or expectoration throughout, - death on 10th day.
- (23) Rusty and only 3 III from admission to death (12 hours).
- (24) Mucopurulent and not tenacious throughout.
(complicated with bronchitis). Death on 5th day.
- (25) Turbid, frothy, non-coherent, rust colored, containing micrococcii singly and in groups, rod shaped bacilli, diplococci, also Friedländer's bacillus with halo round it in abundance.

All the common varieties of Pneumonic spata are included in this list. Death may terminate a case in which the expectoration has been purely bronchitic, or gelatinous and only slightly tinged with yellow, throughout. But this is very uncommon.
Total absence, barring young children,

Seldom occurs in cases which end happily. Pure juice expectoration though of serious augury is not always followed by death; but if it becomes opaque and lighter in colour with pus cells seen under the microscope, death in my experience is certain. A gradual thinning, darkening, and lessening of the amount, and complete suppression for two, three, or more days, of an expectoration previously typical usually harbingers a fatal event. True haemoptysis, says Walde, indicates a tubercular origin. This I have not seen in pneumonia.

Sputa which become lighter in colour and not less in amount as the case progresses, afford a satisfactory indication.

In reviewing the cases which ended favourably, I find that in several the expectoration became syrupy and prune-juice-like just before crisis. In only one out of 43 recoveries did the expectoration cease during pyrexia, and in another there was neither cough nor expectoration throughout.

With regard to the daily amount in favourable cases there were all variations between 3 V and 3 XII . 3 XII was a common average.

As to microscopic characters the presence of numerous leucocytes is only found in very grave cases, while the character of the microorganisms affords no index of the result. I only make mention of these only in one fatal case

Temperature

where their number and variety were greater than usual.

The onset of pneumonia is usually allied to a rapid elevation of temperature accompanied by rigor. By the words of Wunderlich "The temperature in a few hours rises above 102.2° , and still continues to rise till it attains a height of about 104° , or in severe cases even 105.5° or more."

No further course is usually a continuous one, but it may be remittent or relapsing. These three types present various modifications and may merge into one another. I have made an analysis of 69 charts in which the temperature was taken every four hours, and the following are the results.

First, in those cases which terminated fatally, I find that in one case of continuous pyrexia death occurred suddenly by collapse, and in two a gradual lowering took place before death. In five instances false crises occurred (in one the temperature remaining abnormal for two days), followed by a slight elevation before death. Gradually ascending temperature, in most with short descents followed each time by sharp ascents to a higher level than formerly, was seen in five fatal cases. A similar temperature succeeded by one, two, or more false crises had three examples, and in two

a temperature, regular and only moderately high, suddenly ascended, and after oscillating for two days, death occurred. Another beginning high gradually descended to reach 100° on eleven days, then with sharp oscillations it reached 104° on the 4th day thereafter, when the patient succumbed. One was typically intermittent throughout, early morning temperature being normal or subnormal, and at death 99.6° . One was continuously low (between 101° and 102°) for four days after admission, and another for sixteen days; and lastly a true crisis appeared on a typical chart, to be followed by prostration oscillations to 101.4 for two days, and death.

In this brief summary are included most of the types found in pneumonia, but after a careful study of charts I find that as regards early prognosis my results are absolutely nil. A temperature which day by day reaches a great elevation is doubtless in itself sufficient to create anxiety, but crisis suddenly appears and our fears are allayed.

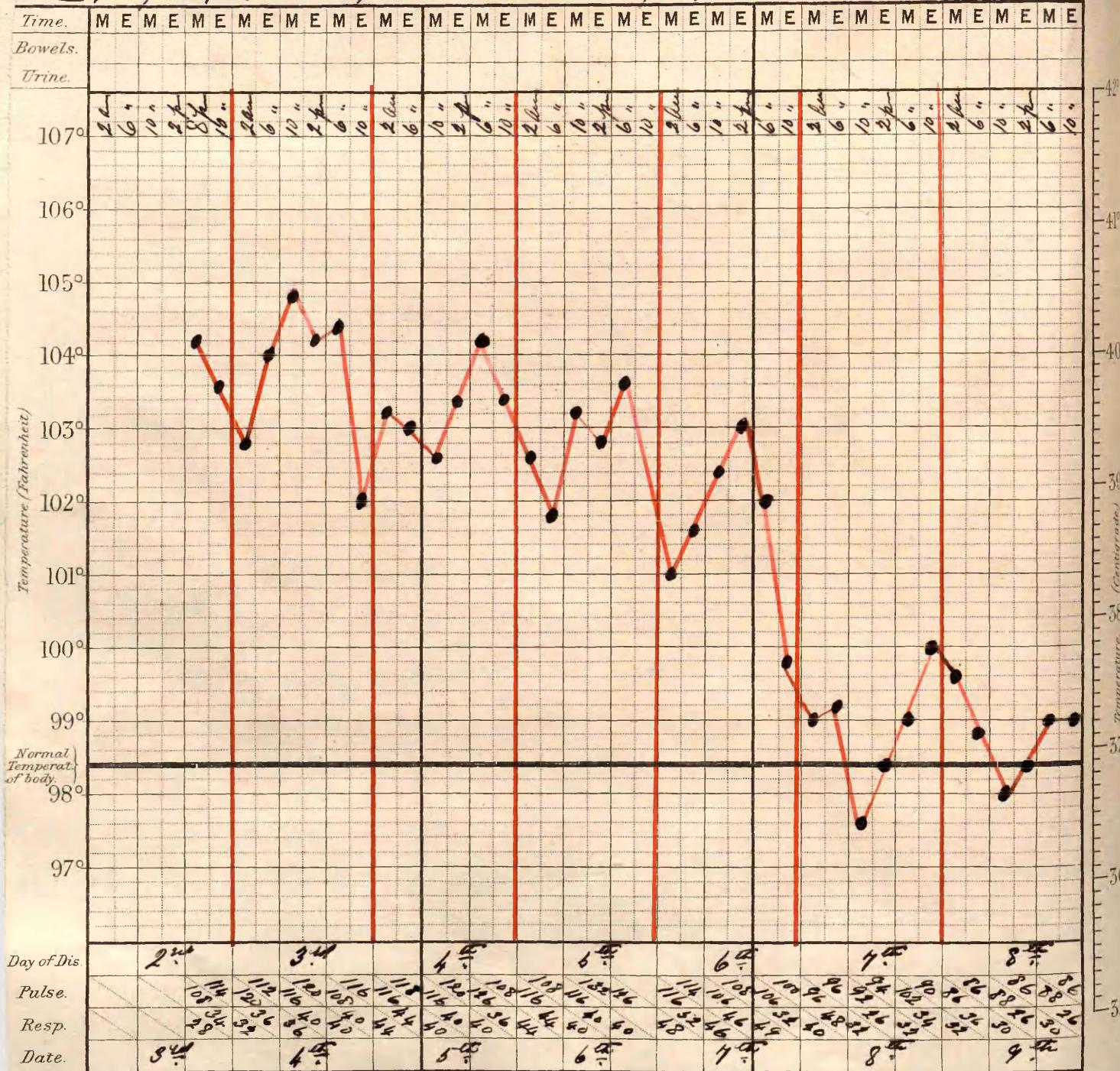
Again the chart presents a smiling face for 5 or 6 days, and a rapid elevation either blights our premature hopes or raises needless alarm. It is a race between death and crisis and a few hours decides may suffice to decide the event. More disappointing still

are those cases where true crisis with its usual symptoms and signs has occurred, but shock thereby sustained in an already previously exhausted frame dooms it to extinction.

A sudden fall of temperature is, as Wunderlich points out, common at any period of the fastigium; but to be deemed by such is almost an impossibility when the general features of the case are brought under review. Has crepitus redux appeared? Has the usual time of crisis come? Are the pulse and respirations diminished? Is the skin bathed in perspiration? Does the patient look well and feel well? By the absence of such, false crisis is our dictum; but false crisis is often the precursor of true defervescence, and by the absence of mimical symptoms and signs may be the first satisfactory indication of a speedy change for the better.

There may be several false crises, and the fastigium may run on into the second week; and the longer its course thereafter the more serious is the outlook. Yet here also the general condition and a correct appreciation of the state of the lungs and heart have prior claims to our notice. Is hepatisation creeping onwards from lobe to lobe and from lung to lung; or is there still a sufficiency of clear lung and

T. John Harris, 29. admitted 3/10/93. Recovered.

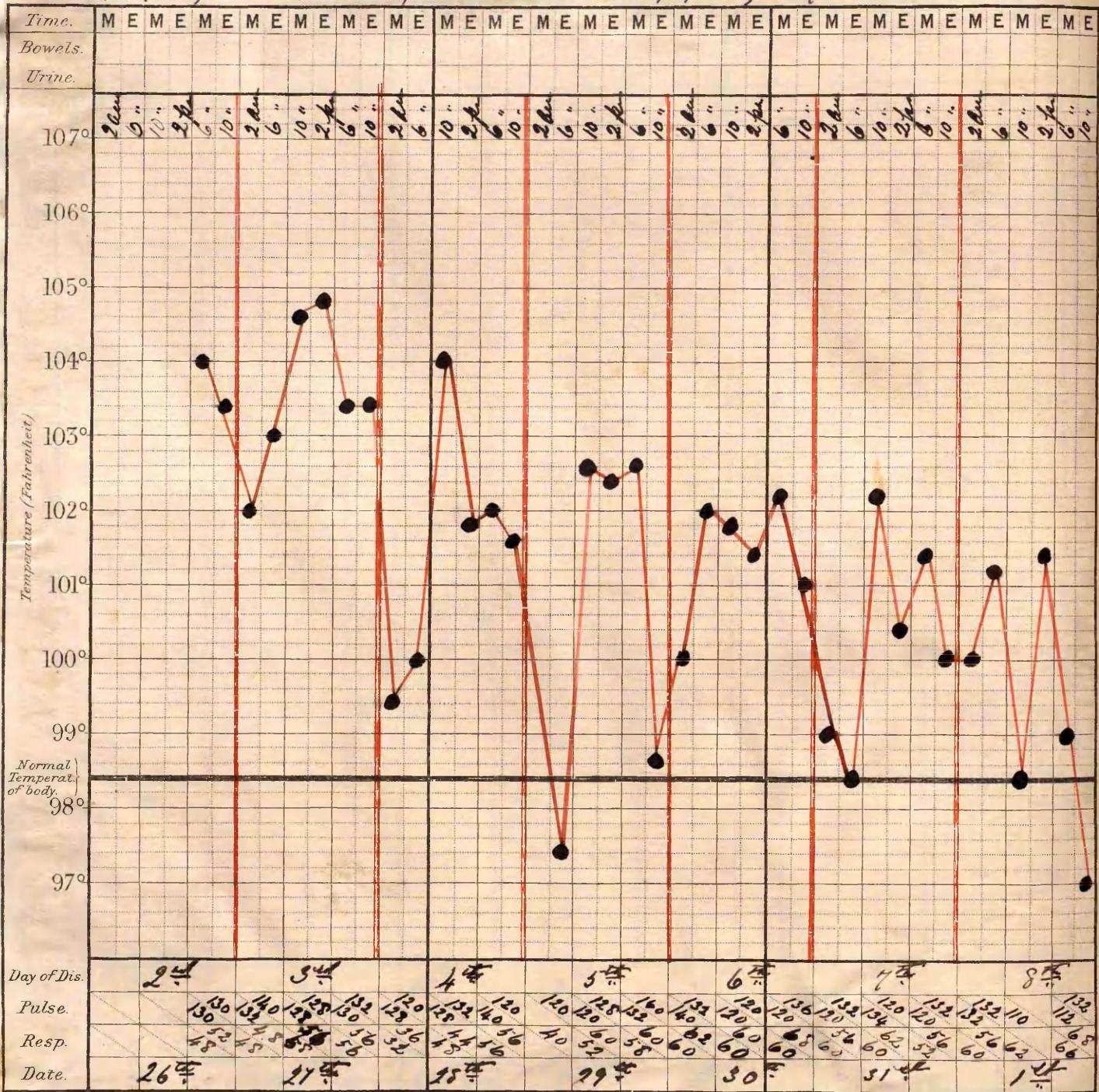


normal respiratory murmur? Is prostration extreme with low muttering delirium and semi-consciousness; or is the intellect clear, the pulse respiration ratio approximate to that of healthy, expectoration clear and sleep attainable? If the former case renewal of pyrexia is ominous, in the latter of trifling importance.

Finally, the thermometry registers low temperatures throughout, - the patient old and weakly or much debilitated - though consolidation may be wide-spread, prostration extreme, & the P. R. ratio highly perverted. The result if not death is a protracted and often incomplete convalescence.

Here are three examples of a continuous fastigium. By hos I. & II. the temperature reached its highest point on the third day of the disease, and both cases recovered. By hos III. this was not attained until the 6th day, but afterwards temperature follows a descending course with pulse rate and respirations steadily increasing, and death on the 8th day.

II. Jane Robertson 27. admitted 26/3/94, bedridden.

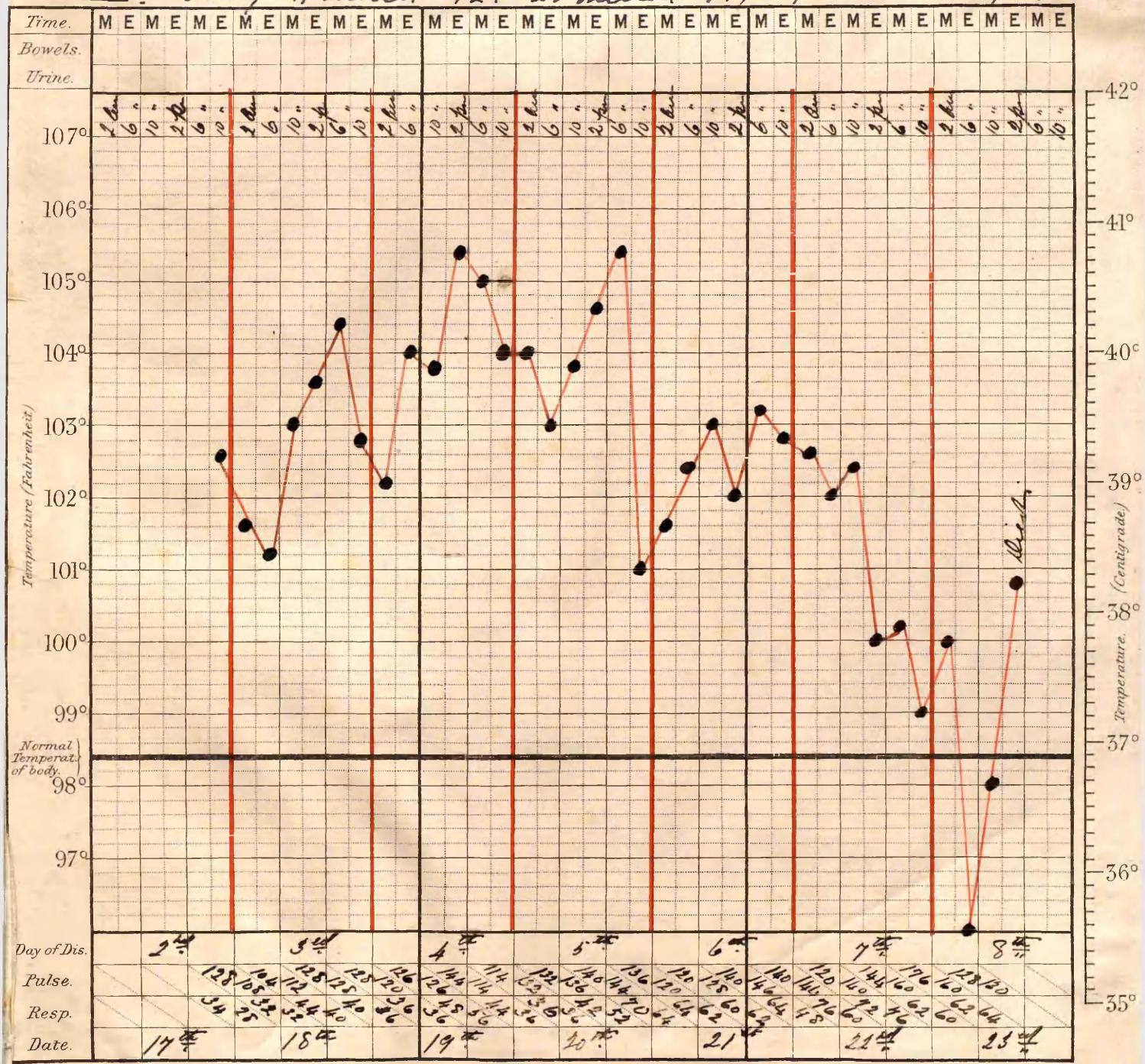


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III. Owen Kennett 42. admitted 17/10/93 died 23/10/93.



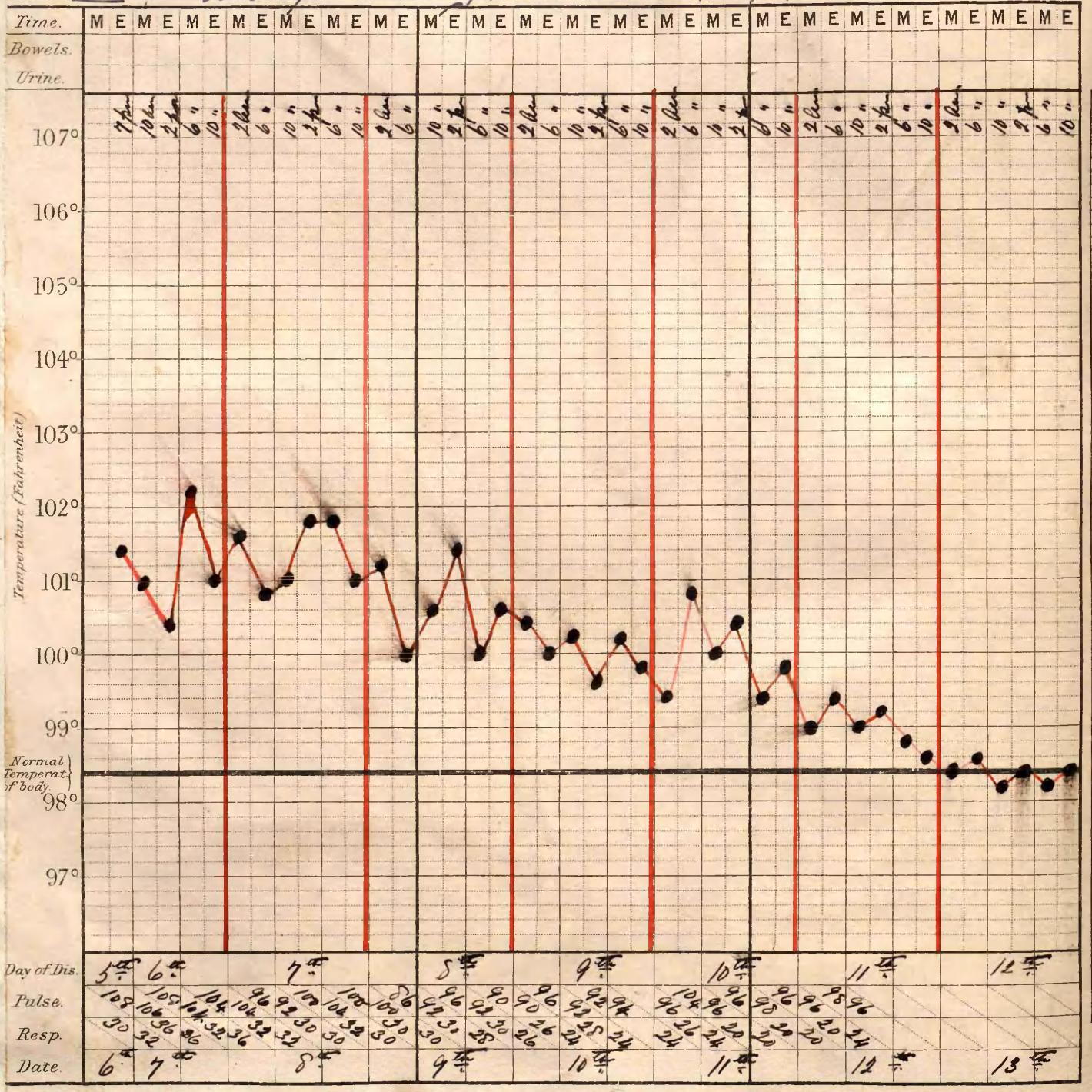
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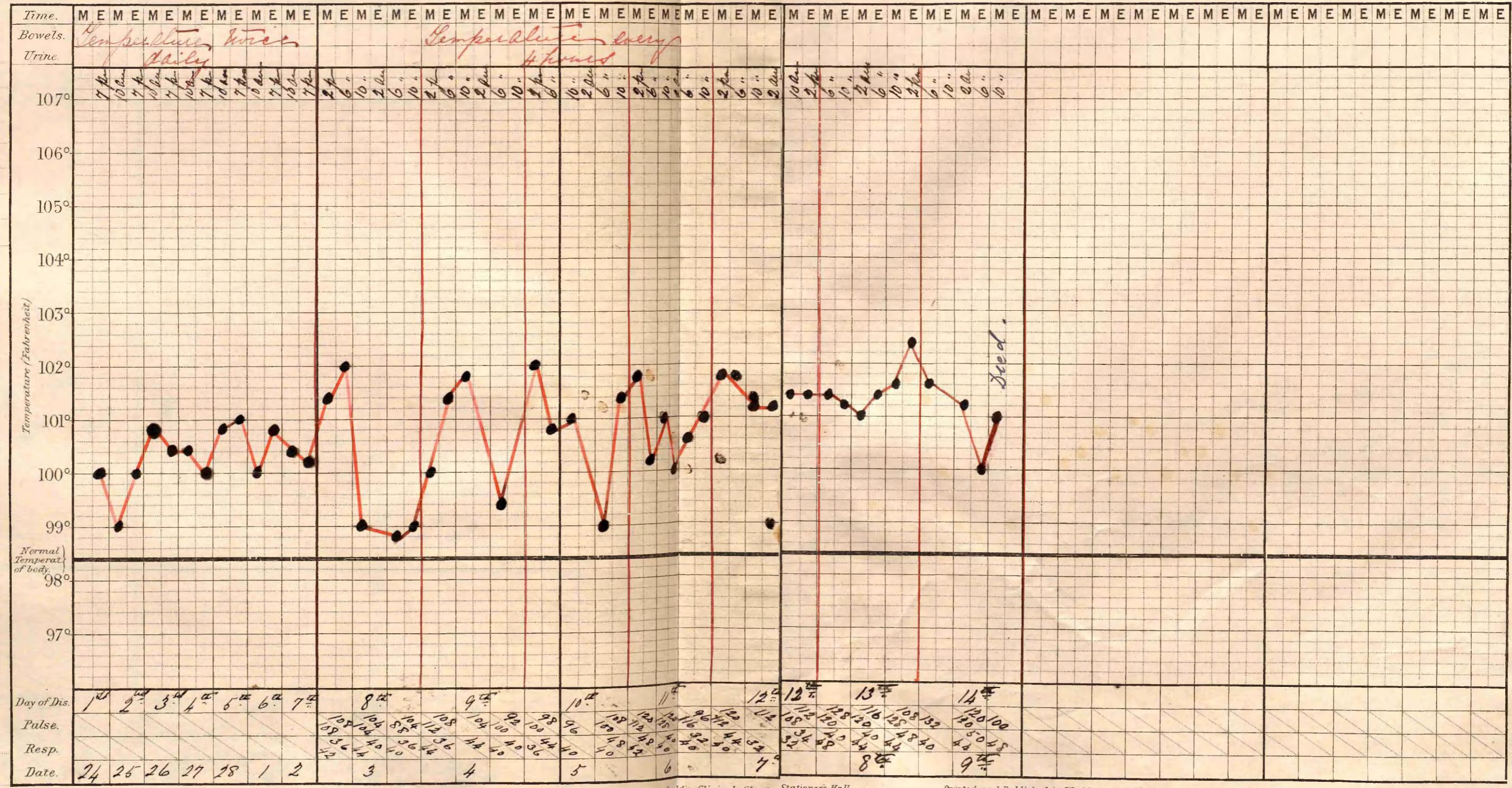
Gould's Clinical Chart.

nos. IV and V are examples of low temperature fevers as commonly met with in the old. Both are protracted, one descending, with no crisis and incomplete resolution, the other ascending becoming more sustained and terminating in death.

IV. Allan Shand. 61. admitted 6/11/93. Recovered.



DISEASE.



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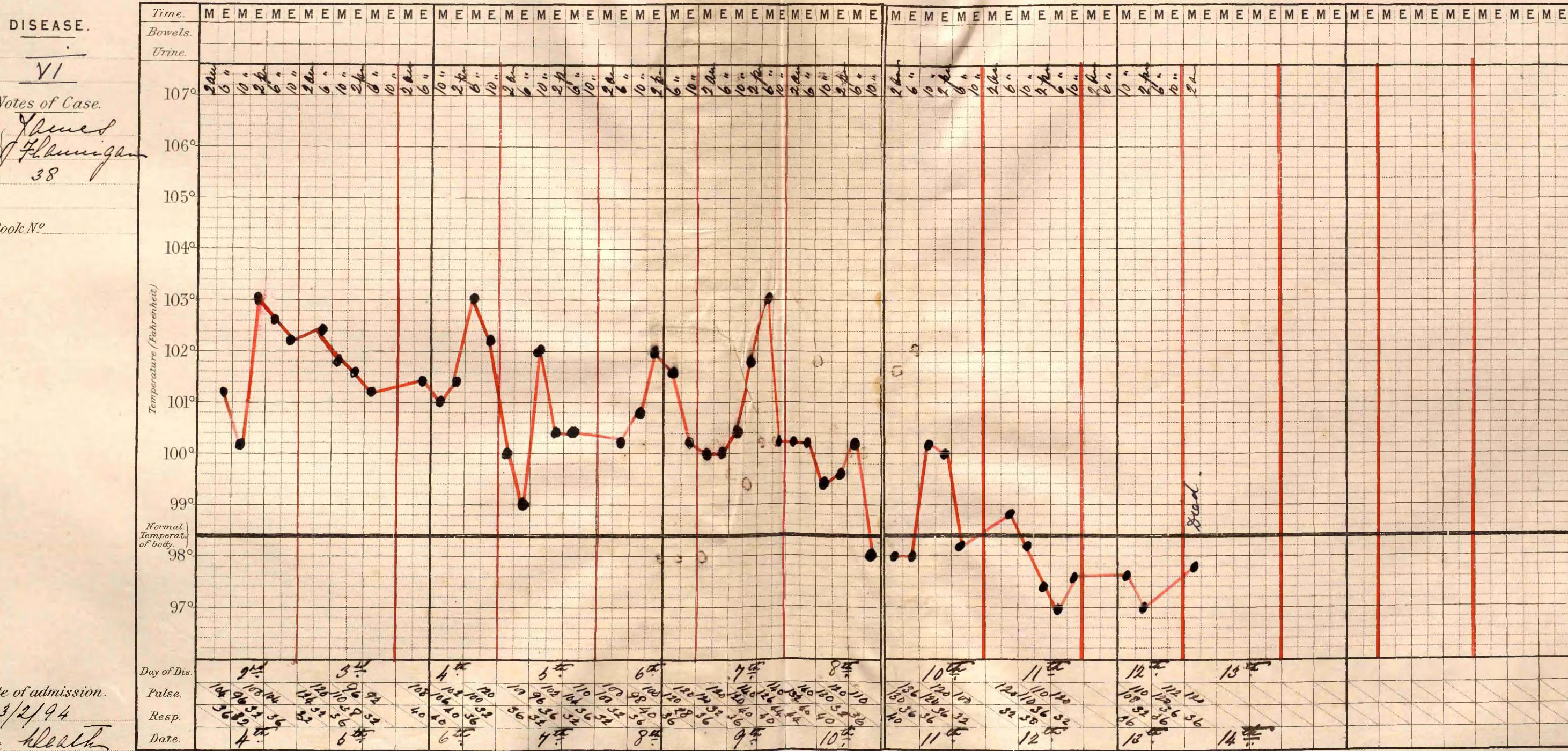
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hos VI & VII show pyrexia prolonged to 10th day.
 Both are of the continuous descending type, but VII
 is higher throughout than VI and pulse comes more
 marked. 'VI.' died, 'VII.' recovered



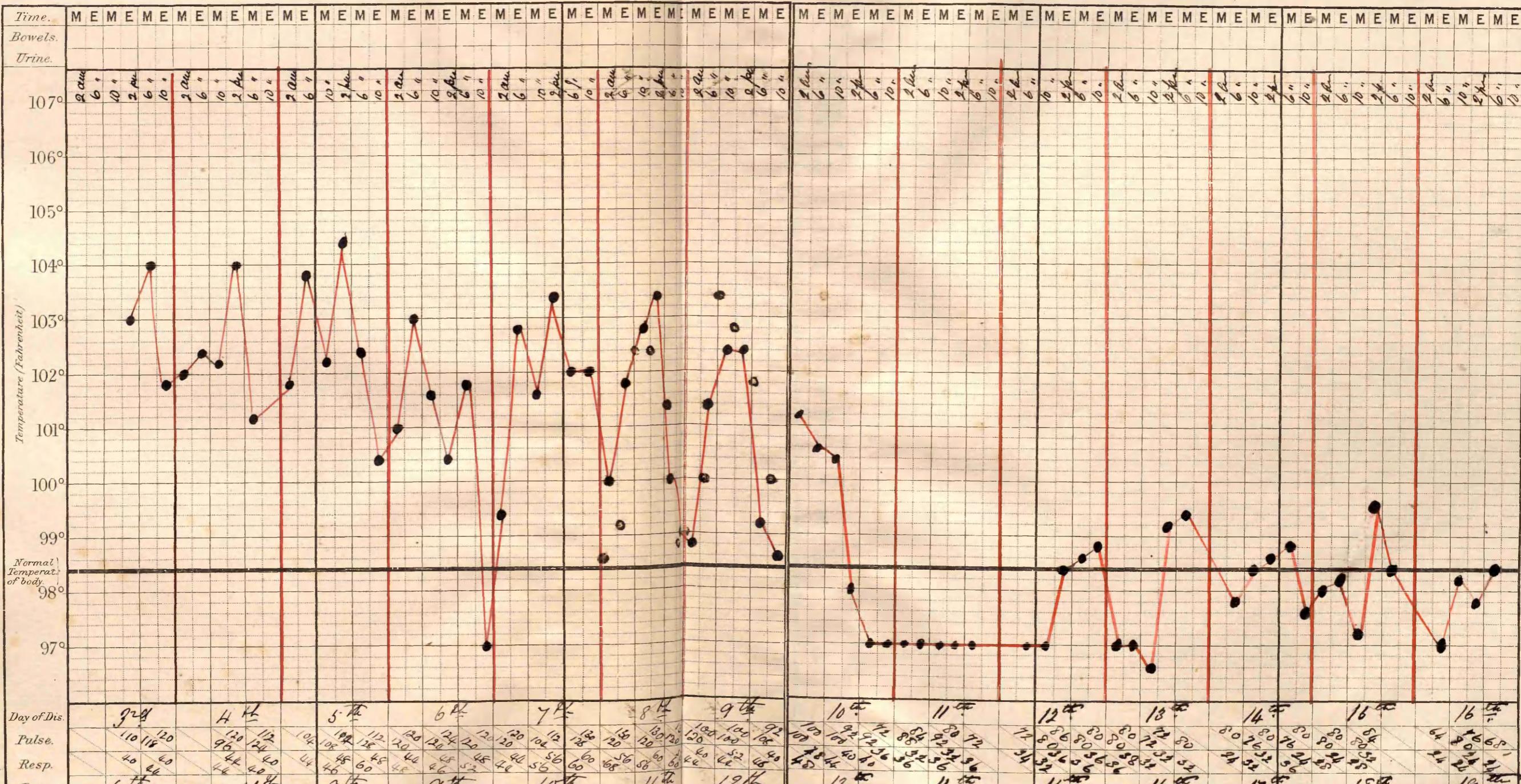
DISEASE.

VII

Notes of Case.

Hillman
Golson
age 18

Case Book No.



Date of admission.

6/3/94

Recovery

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DISEASE.

VIII

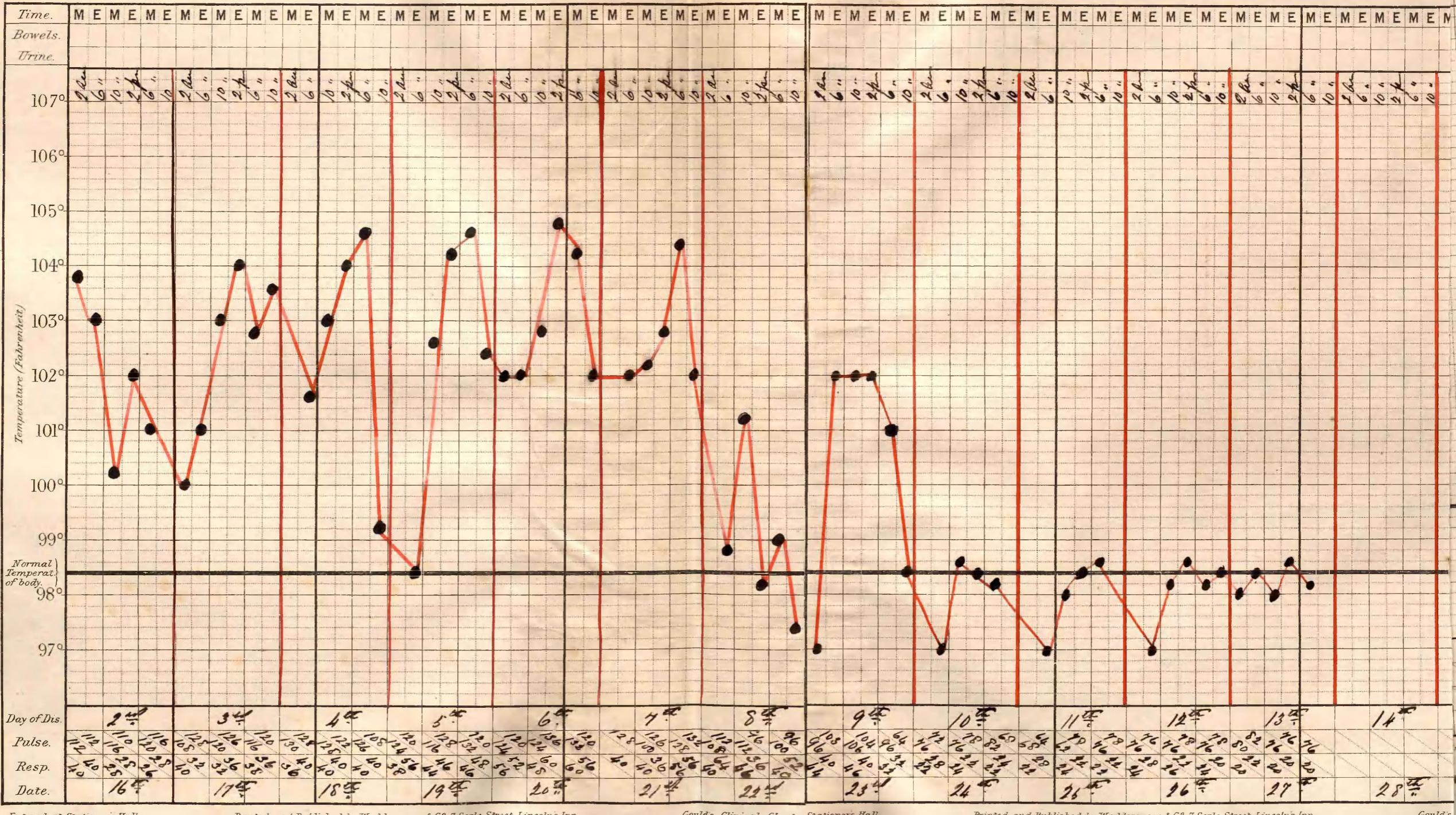
Notes of Case.

*Lohules
Chawford
22*

e Book No.

Date of admission.

15/11/93
ult *Pneumonia*



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Gould's

by VIII and IX temperature is ascending and continuous.
 The one presents false crisis beginning on 4th day
 followed by recrudescence. True crisis beginning on 7th day.
 The other shews a diminution of pyrexia for 12 hrs
 following blood letting on the 3rd day, with recrudescence
 and death on the 5th day.

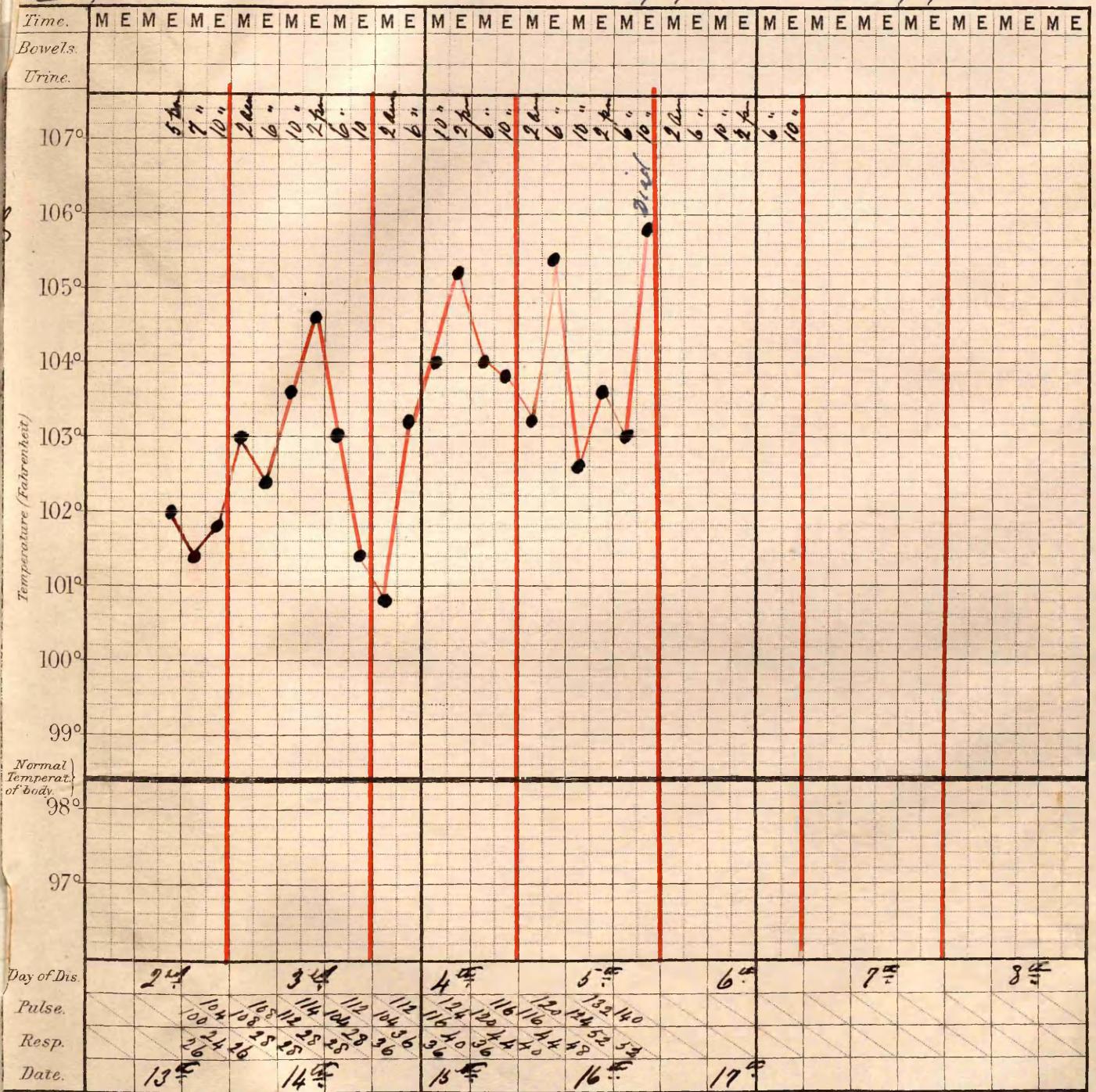
DISEA

VIII
sites of
cold
blad
22

No No

admis
11/9
Dec

IX. James O'Donnell. 33. admitted 13/9/93. Died 16/9/93.

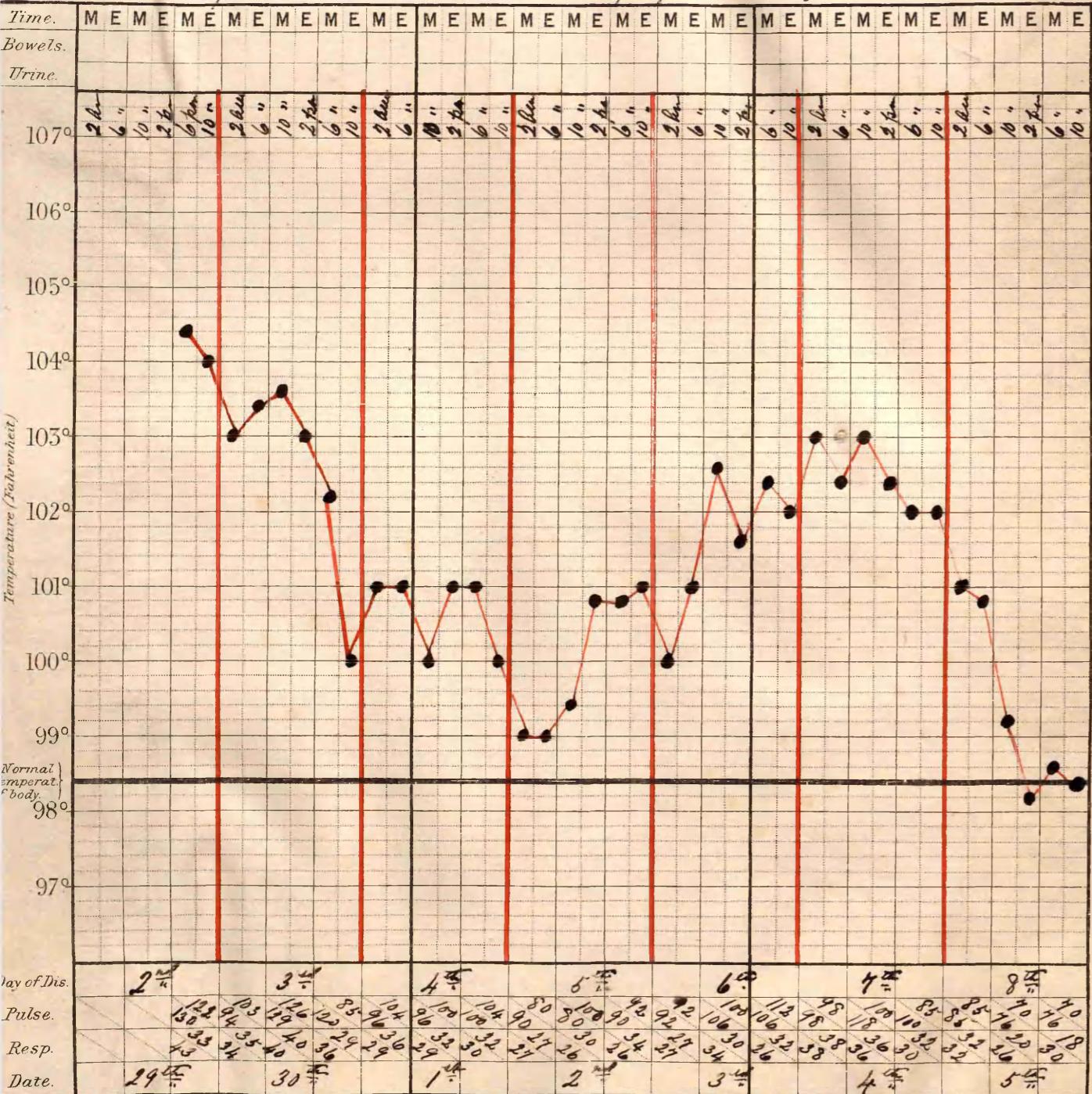


hos. X & XI are examples of relapsing Pneumonia.

In the X there is lowering of temperature on 4th & 5th days, in the XI on 5th and 6th. In both a crisis-like fall occurs on the 8th day, - in one a true crisis, in the other with no alleviation of symptoms.

The latter (XI) dies with rising temperature on the 9th day.

X. Pat^t Mr. Ernie 22, admitted 29/11/92. Recovered.

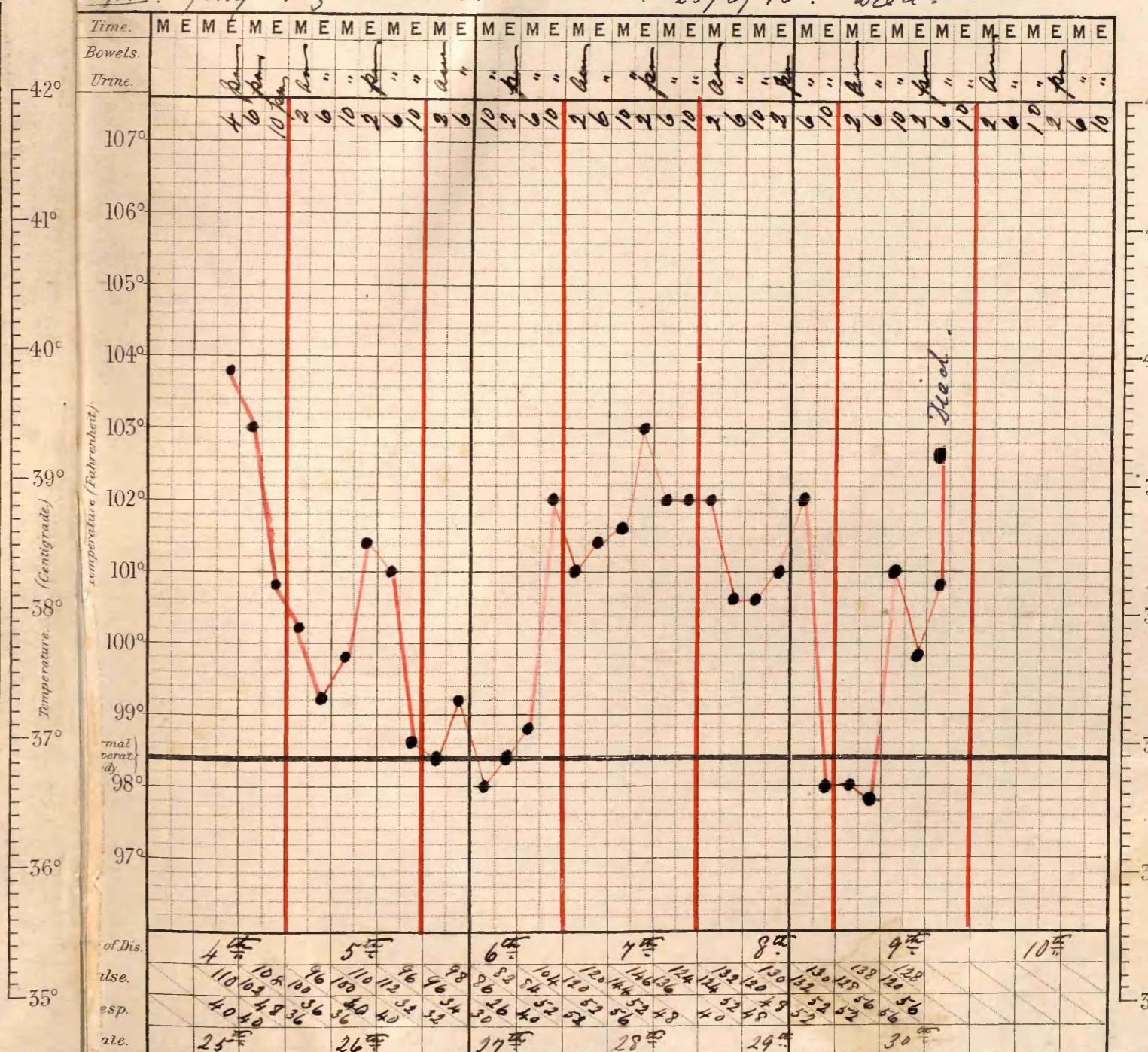


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XI. Poly Théobald Charles. 33. admitted 25/6/93. Died.



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Gould's Clinical Chart

Day of Crisis.

Authorities are for the most part agreed that defervescence usually takes place from the 5th to the 8th day of disease, on the 7th, 6th, and 8th in descending order of frequency.

of 42 cases in which a sharp and complete crisis occurred, 11 were on the 6th day,

9	"	-	-	7 th day.
9	"	-	-	8 th day.
6	"	-	-	5 th day.
3	"	-	-	10 th day.
2	"	-	-	9 th day.
2	"	-	-	4 th day.
1	"	-	-	11 th day.

I exclude from this list all cases where critical signs were prolonged over 48 hours.

The time of the critical fall of temperature was 8 hours in 13 cases.

12 hrs in 10 cases.

16 hrs in 6 cases.

20 hrs in 4 cases.

4 hrs in 2 cases.

over 20 hrs & under 48 hrs in 7 cases.

In 23 fatal cases, in which the day of disease was known (with two exceptions) for certain, 5 died on the 8th day.

4 " " " 9th day.

4 " " " 5th day.

2 " " " 7th day.

2 " " " (?)

1 each died on 6th, 10th, 11th, 12th, 13th, 16th ^{exp.}

It is on the 6th or 5th days that symptoms and signs are first available for purposes of prognosis, when a general review of

Pulse and Respiration.

of physical condition, habits, and past surroundings P.T.C. will enable us to foresee what the next few days may bring forth, no less than that actual condition of the patient at the moment.

The events of the next four days are pregnant with fate. The thermometer gives no indication of the amount of lung implicated, or of the probable duration of the case, but the pulse-respiration ratio and the general appearance of the patient may then serve to yield us a glimpse of the future.

The pulse-respiration ratio taken along with the state of the heart are now of most value. And this leads to the enquiry of what ratio is compatible with recovery and what prophetic of death.

From a list of 69 cases of the highest pulse-respiration ratio found at any period of pyrexia, the following are the results.

(A) In cases which ended in death. The four highest ratios were. Age of patient.
 $R:P = 68:108$ 28 years.
 $R:P = 64:108$ 40 ".
 $R:P = 76:120$ 42 ".
 $R:P = 60:100$ 48 ".
all nearly = 1:1.6.

(B) of the recoveries the four highest ratios were

	Age of patient
$R:P = 60:112 = 1:1.8$	22 years.
$R:P = 42:92 = 1:2.1$	46. "
$R:P = 50:110 = 1:2.2$	19. "
$R:P = 44:106 = 1:2.4$	39. "

(V) The four lowest ratios found in fatal cases were.

	Age of patient
$R:P = 48:132 = 1:2.7$	54 years.
$R:P = 44:120 = 1:2.7$	61. "
$R:P = 50:140 = 1:2.8$	56. "
$R:P = 48:128 = 1:2.6$	32. "

It is stated on the authority of Walde and Guigensay that the respirations may exceed the pulse early in the course of pneumonia, and to this Guigensay adds that such a degree of perspiration is seen in old people with slowly acting hearts and attenuated vessels: that a like phenomenon may occur in the very young.

Leaving aside these, of which I have not met any examples, my statistics shew that in persons young or middle aged, with a ratio of $1:1.6$ appearing at any time of the fastifying, invariably died: that ratios as high as $1:1.8$ to $1:2.4$ were the highest compatible with recovery; but that ratios as low as $1:2.6$ and $1:2.8$ ~~sometimes~~ are seen in cases which terminate fatally, for the most part in persons who have passed middle life.

The number of respirations

is important. As a rule they gradually increase in frequency while consolidation is spreading, reaching their highest limit with the mature development of the local lesion. But in some cases breathing may be most rapid at the onset; or rather most hurried, on account of pleuritic pain being very acute. Wakshe says that "the frequency of breathing does not depend on any particular seat of the pneumonia nor even on its extent, - at least necessarily. He has known double pneumonia attended with a less number of respirations than inflammation of a limited portion of one lung! This is true, but nevertheless the number of respirations may give some hint of the severity of the case. The following table shows the highest number of respirations per minute recorded at any period in 69 cases, with the result in each instance.

Respirations per min. Recoveries. Deaths. Total cases.

26 to 30	2	-	2.
31 to 40	16	2	18
41 to 50	17	9	26
51 to 60	7	6	13
61 to 70	3	4	7
71 to 80	-	3	3

No more than 60 respirations per minute were reached in the great bulk of cases, and the more frequent the

breathing the more ~~grave~~ grave the sequel.
A moderate rate (30 to 40 per min.) may
end in death in the old; or, in slow
creeping pneumonia, as in the two
such ~~in the above table~~ noted above.

A very rapid rate (61 to 70 per min.) may
be attended with no consciousness of
dry expectoration, and if in young subjects
or only temporary, recovery is
possible. More serious is the
outlook with a gradual increase
to the 5th day and beyond when
the number reaches 70 or more.

In only 3 did the frequency of
respirations exceed 70 per minute.

Of these the respective ages were
37, 42 and 60, and all died.

The pulse rate as a rule attains its
maximum on the 4th and 5th days,
about 120 per minute being the
average rate in those who recover.

When this rate is exceeded, and
 pari passu with age, grounds for
anxiety exist: and, when 130
beats are registered more than half
prove fatal. According to Walshe
"pneumonia may run its course
both in old persons and in young
adults with a pulse never exceeding
60; but in these individuals the
healthy standard has on recovery
proved very still lower". I have
met with no examples of this kind.

Subjoined is a list of the highest pulse rates recorded in the same 69 cases.

Pulse Rate per min. Recoveries Deaths. Total.

90 to 100.	4	0	4
101 - 110.	11	0	11
111 - 120.	14	3	17
121 - 130.	2	3	5
131 - 140.	11	6	17
141 - 150.	2	9	11
160 - 170	1	2	3
176.	0	1.	1

In four instances the pulse reached its maximum rate on the day of death.

Sometimes the highest pulse-respiration ratio is then attained; and more frequently the respirations have on that day increased beyond any previous number observed.

On completion of crisis the pulse rate was usually between 80 and 90, with a respiration rate somewhere between 20 and 30. But they are subject to great variations either way; e.g. - in two extremes the pulse in the one was 50 with respirations 24, in the other 120 with respirations 56.

From the numerical height of pulse and breathing little fore-knowledge is attainable unless synthetically added to the total grouping; for the personal equation of the individual and the character of the pneumonia may cause great fluctuations in these rates, so that a low respiration may be serious

Character of Pulse and Condition
of the Heart.

In one case while a high regular
strikes no warning note in another.

At the beginning of pyrexia the pulse
quicken's and as the case progresses
becomes softer and more collapsible,
bounding or dicrotic, or partaking of
the character of Corvisans pulse.

When it begins to miss an occasional
beat, or when it becomes irregular,
being at the same time large, soft
and rapid, grave danger is imminent.

A small weak pulse, not necessarily
frequent, may attend serious signs,
and with the addition of arrhythmia is
often the preface of impending death.

In one case S. R. act. 20 a soft
blowing V. S. mitral murmur appeared
on the 4th day, and two days later
the pulse, previously soft and
rapid, became irregular. Death
seemed imminent when crisis abruptly
changed the scene.

In another J. H. act 50, the pulse
was regular, but the heart sounds became
faint, and throbbing was apparent
in the epigastrium one day before
crisis.

A third patient, J. Y. act 50, was prostrate
and semi-conscious on admission
with a pulse as collapsible as that of
a well-marked case of aortic regurgitation.
The heart sounds were very feeble,
the apical impulse lost, and

Trembling pulsation was seen in the epigastrium. Crisis occurred during the night, and next morning the pulse was slow and full and the cardiac sounds feeble but much improved.

J. P. at 27 presented the usual type of pulse until the 6th day when it became very small, weak and rapid (135 per min), but remained regular up to the 8th day when crisis occurred. Pulse rate continued high for 8 days longer, and weak for nearly two months, during which time oedema of the legs appeared whenever she remained for any time out of bed.

These were all cases where the result was doubtful, the symptoms alarming, and the heart on the point of yielding, but for the timely intervention of crisis.

Turning now to the fatal cases, —

J. F. 33, died on the 9th day of illness.

On 5th day pulse was rapid & full. - 100 per min.

7th day " rapid, irregular, & compressible - 144 per min.

8th day " " stronger, fuller, more regular & slower; - 120 to 130 per min.

9th day pulse of collapse almost imperceptible; - 130 to 135 per min.

This temporary improvement was due to oxygen inhalations and powerful stimulation.

My. 8'D. act 33. died 5th day. Pulse very large, soft
and diastolic from 3rd day of disease, becoming
more empty with greater amplitude of wave
(140 per min) by the 5th day. Sudden failure
of the heart occurred, the sounds were
almost inaudible, and before death the pulse
was too small and rapid to be counted.

The post mortem examination showed a weak
flabby heart with dilated ventricles, esp. the right.

A. P. 22 died on 7th day. He was admitted on
the 5th day with pulse rapid (105 per min)
but small, with every 4th or 5th beat lost.
Marked improvement for one day followed
stimulation and oxygen inhalation with
digitalis. But suddenly collapse followed on
the next. Post mortem examination
showed a healthy heart with decolored clots.

T.B. 56 died on 11th day. Admitted on 6th day
with fairly good pulse, it gradually quickened
until it reached 144 per min, but it remained
fairly full and steady up to a few hours of
before death. Heart sounds were weak
and toneless. Apex beat under 7th rib, very
faint, and about 1 inch external to line
of nipple.

Post mortem examination of heart (already
referred to) showed great hypertrophy of left
ventricle with extensive ulcerous
degeneration.

My. My. 38 died on 8th day. Admitted on 6th day
with pulse soft, bounding, regular and
compressible, - R: P = 56: 120. Heart
very intermittent (125 per min), rapid & small,-
before death almost imperceptible.

1st sound of heart was short, high pitched, and clicking on admission. Next day cardiac action was tumultuous, the sounds becoming gradually more faint. Post mortem the heart was dilated, and the great vessels narrower in calibre than normal. The lungs showed fatty infiltration. J. A. 61 died on 5th day. The pulse remained regular and good up to day of death when it was irregular and small (120 per min). The heart sounds throughout were dull and muffled with hearing of the right ventricle on day of death. Post mortem, adherent pericardium, and by autopsy of whole heart. Afterward, J. F. at 38 died on 13th day. On the 5th day the pulse was full and regular but the heart sounds were both sharp, short and high pitched. Two days later the 2nd sound was reduplicated, and the pulse missed once in about 20 beats. Thereafter it became gradually weaker, more irregular, and more rapid, reaching 140 in the 9th day. On the 10th day it became somewhat slower (120 per min) but smaller and weaker. By this time the temperature had become normal. From the 11th day up to the point of death the pulse was so rapid, small, and irregular that its rate could not be ascertained with any degree of certainty, and the temperature varied between 97° and 97.8° at death. The left ventricle was found P.M. to be hyperplastic.

Increasing impatience and rapidity of pulse
is often the first indication that the
potential energy of the heart is on the
point of giving out. When ^{in addition} regularity
of rhythm and force has become established
death usually follows within three days;
but timely crisis may intervene like a
as an efficient brake in the downward
path. A large bounding pulse which
suddenly becomes small points to collapse
and death in a few hours. The
changes in the cardiac sounds indicating
of progressive enfeeblement are
muffling of the sounds, a sharp clicking
1st sound, reduplication of the 2^d sound,
loss of apex beat, a circumscribed 3. S.
muted murmur, throbbing of the
right ventricle, simultaneous action,
trembling in epigastrium, irregularity,
sounds becoming almost inaudible with
venous distension and lividity of face
and extremities. Every why such a
condition is reached recovery sometimes
follows; but, if signs of cardiac
failure appear as early as the 3^d day
there is probably an organic basis underlying
a thus early derangement of function,
and death in my cases always followed.

In contradistinction
to the foregoing argument that "it
is to the heart we must chiefly look
for indications of the probable outcome
of the case" (Lupton & Sturges), are
those cases occasionally met with

Aspect and general Condition.

where crisis has passed but has left the patient enfeebled and sunk down, like a ruin with its interior destroyed by fire, his down fixed on his face. He may linger for four or five days, with pulse and respirations still above normal frequency but not to the same extent as before.

Indeed the heart may continue to display a force and steadiness utterly surprising in the circumstances, nevertheless death must inevitably comes, and post mortem the lung affected lung is found in a state of purulent degeneration.

In all cases of Pneumonia no signs are more reliable in prognosis than the aspect and general condition of the patient. A troubled anxious look may at the onset be due to pleuritic pain, and may pass off when the inconvenience is removed. Early dyspnoea followed by perspirations are ominous, but some cases end indefinitely where the skin has been hot and dry without any appearance of sweating throughout. An expression of great suffering persisting in spite of treatment must be regarded as serious, though in some instances deep-seated pleurisy and subjacent local peritonitis has been found post mortem to account for this. Hacking eyes, a startled look, an excited appearance, restlessness, and

Digestive organs.

Late Commissum Pt C.

Herpes.

insomnia passing into delirium have been already commented upon.

Cyanosis, dyspnoea and coldness of the extremities may in exceptional cases be precursors of favourable crisis.

Toul breath, sores on teeth and lips, a dry brown tongue, great prostration, moaning respirations, involuntary evacuations, harassing and ineffectual cough without expectoration, early collapse are all serious indications.

The state of the digestive organs is sometimes of value in prognosis. Anorexia, persistent vomiting, slight jaundice, and diarrhoea are often present for the first 2 or 3 days, if of longer duration they add to the gravity of the case. Prognosis is very bad when food is refused, but it is surprising how often the appetite remains good almost to the point of death.

Convulsions, stupor, coma, late in the disease are usually precursors of death.

In much fewer cases than the proportion usually stated was herpes seen; viz.; only in 8 cases out of 69. The vesicles occupied the usual site except in one instance where ^{herpes} ~~they~~ took the form of a diffuse pustular eruption over the lower ribs laterally and in front on the left side. In two cases death occurred, both after crisis,

Albuminuria .

and in both herpes persisted, its vesicles filling with pus and enlarging.

As a sign of favourable auspices herpes is said to be of some value. On Geissler's authority, 9.3% with herpes die as compared with 29.3% among those with no herpes. ~~by our~~ Cases are too few in number to prove anything here, but it is not worth noting in how few herpes ^{did} appear.

Albuminuria was present in varying degree in 21 cases out of 40, and in 16 fatalities albuminuria complicated the case in 11 instances. This leaves 24 recoveries with albuminuria in 10.

In most the amount of albumen was small and its appearance transient. In two only was it considerable in amount and both died. In none of the favourable cases did it persist after crisis was passed, but I can recall two instances, both patients being boys under 13, in which it remained for a month after defervescence.

As regards prognosis, albuminuria is found in the great majority of cases which end in death, and in nearly $\frac{1}{2}$ of those which terminate happily. If slight, it indicates little. If considerable and symptomatic of chronic nephritis, it greatly aggravates ~~the~~ ~~seen~~ tendency to death.

Chlorides in Urine.

So Resolution.

gives a serious bias to our views on the case. In young persons albuminuria may continue for a month or more after crisis without apparent damage to the renal structure.

With regard to the quantity of Chlorides present I find all variety. Measurements were taken usually daily after the precipitate had stood for 6 hours in test tubes.

In some cases Chlorides were almost nil in the early days of pyrexia, but steadily increased afterwards.

In some they were uniformly diminished throughout. In very few were they entirely absent. In some daily variations from $\frac{1}{6}$ to $\frac{1}{2}$ occurred. In others diminution was seen after crisis. At death also the amount varied from $\frac{1}{2}$ or $\frac{1}{4}$ to almost total suppression.

From such confusion I can only extract one positive fact; viz., that those cases in which chlorides were almost entirely absent from the urine ~~all died~~. Throughout the disease all died.

For a day or two before the time of expected crisis the affected area of lung may shew signs of impending resolution, ~~which~~ thus be of assistance in prognosis. One would hesitate to accept the mere presence of Crepitus Redux as indicating of resolution

in its commencement, apart from the total grouping of all features in the case.

It is commonly affirmed that resolution begins in the part last involved, and progressively extends backwards to the primary seat. This statement seems to demand considerable modification in the light of daily examinations made for days and sometimes for weeks after crisis.

For example, J. W. act 15. Crisis on 7th day. Crepitus Redux was heard on the 6th day at the left base behind, the original site of hepatisation. Above this the lower lobe speedily lost its tubular quality of respiration without crepitation. On the 8th day the upper lobe still exhibited harsh metallic tubular breathing as high as the spine of the scapula. Then P. ty and V. R. both became diminished at the base with dullness only left; and redux crepitation did not appear over the upper lobe until 5 days after the crisis, by which time the lower lobe had nearly returned nearly to a normal state.

C. C. act 22. Crisis on 10th day. A case of right basal pneumonia, solidification passing upwards over the entire lung. On the day of defervescence crepitus was generally diffused over the back on the right side, but was less so

was coarser and more abundant over the lower lobe. The extreme apex behind and the front of the upper lobe showed pure tubular breathing, and bronchop honk for two days longer; after which all signs faded in the upper region without any crepitation being heard. The whole lung was normal but for dulness and diminished R. H., in 4 days.

W.C. at 10. Crisis on 11th day. The right apex, where hepatisation first appeared, lost its intense tubular breathing on the day preceding crisis, and C.R. was generally diffused over it. Almost complete resolution of the upper lobe had occurred taken place, while lower down posteriorly and in the axilla there were still typical signs of hepatisation. A week later the lung had entirely cleared.

In 13 out of 31 cases I found that Redux Crepitation appeared first at the site of the primary nucleus of solidification. These were in persons of all ages, and the 6th was the earliest day of crisis. In most resolution appeared to be complete in a week; but, where areas of tubular breathing and bronchop honk remained, it is worth noting, that these were usually in the situations of the first observed consolidated parts.

my conclusion therefore is, that in many cases the part longest solidified begins first to resolve; but that it requires more time than the more recently hepatalized areas to complete its resolution.

It is only in widely spread consolidations that such phenomena can be observed with any degree of accuracy. In ephemeral engagements and in complete or very recent hepatalizations resolution takes place very early and very completely. When one lobe only has been solidified C.R. is generally diffused on its first appearance. This I found in 6 cases.

In only 2 was C.R. entirely missed. In 2 C.R. first appeared over the whole periphery of a circular hepatalized area, and in the central part resolution was delayed. In one case signs of resolution appeared in one lung while in the other hepatalization was spreading.

In 2 instances only did C.R. appear first in the situations of most recent extension, and in both resolution was unusually rapid.

In two cases resolution was imperfect, dullness and weak tubular breathing remaining for three weeks or so, then coarse mucous rale attacked the whole area, and excavation of the lung succeeded. Both primary consolidations

were of the type of Acute Pneumonia. Finally in two examples no crepitus was heard but coarse moist rales appeared immediately after crisis and resolution was soon complete.

One case previously complicated with *Erysipelas Capitis* culminated in Empyema, for which I resected about 2 inches of the 9th rib. The patient made a good recovery, but R. h. continued weak all over, except at the front of the upper lobe which escaped consolidation.

I can recall two instances, one of 6' and one of 2 years standing, in which consolidation was permanent. In both, a slight catarrh or bronchitis caused elevation of temperature, and increased harshness of tubular breathing in the chronically solid lobes, so that they have been frequently readmitted to hospital. By that of 2 years duration, haemoptysis has now occurred several times.

After crisis there a speedy and complete resolution is probable. That after this, a predisposition to future attacks remains, does not accord with my experience, though that is not extensive enough to warrant more than a reservation of opinion. In some cases resolution is not complete for weeks or months, in a few it is

Treatment.

tong imperfect, in very exceptional instances it never occurs, and it occasionally happens that crisis is followed by death. Sequelae are uncommon, but Phthisis, Abscess, Gangrene, Emphysema may follow attacks of Acute Lobar Pneumonia.

In conclusion, Prognosis is influenced by the presence or absence of treatment. Of my total 248 cases, 86 died, and of these one half had received no treatment till within 3 days ^{at most} of the fatal event. Nay, in most had the disease had been neglected before their admission. It is impossible to know how many of them would have recovered had they been placed under favourable conditions from the first; but even if these are entirely disregarded, the fact that the total mortality was 34.6%, signifies remains to signify that ~~by~~ through the exercise of judicious treatment some improvement is possible in the death rate of Pneumonia in the drunken and debilitated.

I have strictly followed modern methods, and have no new curative measures of miraculous efficacy to announce. For the past two years I have given an extended trial to oxygen inhalations, and have not neglected venesection in cases which seemed to warrant it. I am convinced that in presence of

a failing heart oxygen gas can accomplish pro tempore as much in the way of improvement as blood-letting, and that without lessening a tax on the strength of the patient. Continuous inhalations over two days is too expensive a measure for my class of practice, but repetitions at short intervals often seem to serve.

In several instances at the critical period when signs and symptoms were so many omens of evil, such inhalations seemed to bring about a happier though unexpected issue.

By far the greater proportion require no treatment beyond fresh air, cleanliness, a comfortable bed, and a sufficiency of wholesome easily digested food. Deprivation of these must affect the mortality in the pauper class to some extent. But on the other hand many show a downward tendency from the beginning, and all treatment is powerless to avert the decree of fate.