The Bronchio-pneumonia of Influenza as it occurs in the adult subject

by

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"That Omnipotence which has called the world
" with all its living creatures into one animated being,
" especially reveals Himself in the destruction of
" great pestilences. The powers of creation come into
" violent collision; the sultry dryness of the atmosphere;
" the subterranean thunders; the mist of overflowing
" waters, are the harbingers of destruction; Nature
" is not satisfied with the ordinary alternations of
" life and death, and the destroying angel waves
" over man and beast his flaming sword."

"The Black Death" by J.F.C. Hecker.

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Nomenclature

The term 'Bronchopneumonia' was first applied by Seifert in 1838, to such cases as I am about to describe. Other terms have been used such as "Peripneumonie peripneumonia notha", "latent pneumonia", "catarrhe suffocant" (Laennec), "lobular pneumonia", (Burnet and de la Berge,) catarrhal pneumonia, etc. Such names as latent pneumonia, suffocative catarrh, and catarrhal pneumonia are too vague. They refer either to special predominating symptoms, or to certain pathological features, and do not indicate the essential nature of the disease.
Lobular pneumonia, again, is misleading, as one may have a lobular pneumonia without a true bronchopneumonia.
The term "Bronchiopneumonie" which was first applied by

Henri Roger[1] is by far the most appropriate name which can be given to this disease. As he points out, the prefix Broncho - (Gael Bronchos) refers to the large tubes of the respiratory tree, whereas Broncho (Gael Bronchia) applies to the smaller bronchial tubes — to the parts affected in the disease under consideration.
The inflammation, which tends to occur in a patchy manner, invades the smaller bronchioles as well as the lobules in their continuity, and the bronchial and lobular systems with their lining walls, connective tissue, blood-vessels, lymphatics, &c., suffer more or less in their entirety during the course of the affection, and markedly so in this the Influenza form of the disease.
To the term Bronchio-pneumonia, therefore, I shall adhere in this paper.

Definition of the Bronchiopneumonia of Influenza.

During the prevalence of an epidemic of Influenza, or during the period which follows in which the disease no longer assumes an epidemic form, and when occasional cases are still observed, respiratory affections of all forms are very frequent, and are one of the chief factors in the concurrent elevation of the general death rate.
Bronchiopneumonia, which is generally a rare affection

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1. Dictionnaire Encyclopé

in the adult, is very frequent, and is almost as fatal in its nature among these as when it attacks children in its ordinary forms.

It may arise in an acute or subacute fashion, more frequently the latter. In either case, resolution is slow and convalescence protracted, while in many cases, resolution never occurs. In the latter case, the disease, which may, to begin with, have been limited to a special lobe or lobes, spreads throughout both lungs. Soon the lung tissue begins to break down, and a condition hardly to be distinguished from galloping consumption supervenes, and ends fatally in a few weeks.

In more chronic cases, however, a portion of one lung may have suffered, and incomplete resolution takes place, and the patient rises from bed and resumes his customary duties. He is in a disabled condition, his strength is impaired, he still coughs and sweats, and soon a fresh chill forces him back to bed. One or many more relapses may occur to bring about a chronic bronchio-pneumonia with fibroid thickening and contraction of the affected lobe or lung, and death finally results from asphyxia, or from haemoptysis, gradual implication of the other lung, acute bronchitis or pneumonia, or finally from complications arising in other organs.

Post-mortem examination in the acute or subacute forms, reveals the presence of grave bronchio-pneumonia with marked tendency to suppuration and liquefaction of the peri-bronchial nodules, and even to the formation of large abscess cavities or to gangrene. Bronchial dilatation of variable extent is frequent, and pleural affections are almost always present.

In the chronic forms, grey induration and marked pleural thickening are present in intensity proportionate to the chronicity of the case. Caseous degeneration of the products of exudation is prone to occur. Emphysema or pneumo-thorax may develope, and pulmonary collapse, though not nearly so frequent as in children, may be noticed. Inflammatory affections especially tending to the formation of pus are frequent in other organs. Signs of tubercle, as well as the tubercle bacilli are usually absent, whereas the Streptococcus pyogenes is everywhere abundant in the early stages of the disease, not only in the secretions, excretions and inflammatory exudations during life, but in the organs themselves when examined after death.

Etiology.

Bronchopneumonia as a primary disease is very frequent in children, and is one of the most fatal of all the maladies of early childhood. All conditions of debility, want of proper nourishment, bad hygienic surroundings of all sorts, the presence of certain morbid conditions such as chronic diarrhoea, rickets, a tendency to the formation of tubercle, all tend towards its production. As a sequela or as a complication, it may arise from a severe bronchitis in debilitated children, and it is a most frequent cause for anxiety in the course of the various infectious fevers, of diphtheria, of roma, and often after severe burns of the skin.

In the adult, an acute bronchitis may become aggravated into a bronchio-pneumonia in individuals whose general health has been already impaired by the presence of various chronic maladies, such as Emphysema, Chronic Bronchitis, Chronic Bright's disease and Chronic alcoholism. In such cases too, an acute lobar pneumonia may become complicated with a bronchio-pneumonia in the other lung. Fox says that "Pneumonia arising from heart disease presents in many cases all the features which are most characteristic of catarrhal or bronchio-pneumonia" 1

As in children, so in adults, the infectious fevers are active agents in the causation of bronchio-pneumonia, although the percentage of such cases is only much lower than in childhood. Bassot reports in fifteen women who died from the effects of childbed, ten cases of lobular pneumonia. 2 Bronchio-pneumonia, too, rather than fluid acute lobar pneumonia is the form in which pulmonary lesions tend to arise in the various forms of cachexia.

Secondary forms of a more localised and more chronic nature are always present to a greater or less degree around tubercular nodules, syphilitic gummata, various tumours, abscess or gangrene of the lung; it follows upon penetrating wounds of the chest; the introduction of foreign bodies within the bronchi causes it, and it invariably arises when the secretions which collect in dilated bronchi tend to become fœtid. The bronchi suffer in conjunction with

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1 Reynolds's System of Medicine. 1871. Vol. III. p. 709.

2. Bassot. "Pneumonie lobulaire chez l'adulte"
Thèse de doctorat de Paris 1872.

the other tissues of the lungs in the various forms of Pneumonia - Koniosis such as Anthracosis, Siderosis and Silicosis.
It is not however with any of these that we have now to deal but with the various forms of bronchopneumonia which affect the adult specially, during an epidemic of Influenza.

End of Chapter I.

Chapter II

History of previous epidemics and of the late epidemics of 1889-90 and 1891.

The literature upon the subject of Epidemic Catarrh — the Influenza or La Grippe, is of such vast proportions that it will be quite impossible for me to give more than a very brief resumé of some of the principal epidemics in which the presence of pulmonary complications has been more particularly signalised.

One of the first epidemics described in which pulmonary lesions were frequent, occurred in 1557. Of it, Vallesiola writes - "Towards the seventh or fourteenth day of the disease, "the expectoration of very viscid matters took place which were "very difficult to get up". Cardan also writes of the same

outbreall :— " On opening the bodies, the lungs were seen to be full of a bloody matter." Similar reports were made by Baillou in Paris in 1570 and in 1571, by Forestus in Delft in 1580, by Willis in London in 1659, by Pew in Paris, and by Stwiller in Germany in 1675. Sydenham wrote from London in 1676 :— " The autumn " of 1676 was so beautiful and so fine as far as the end of " October, that one would have thought that it was summer ; " but, the weather having changed suddenly to become cold and " damp, there arose an epidemic cough so violent, that " almost no one was exempt. Entire families were attacked " simultaneously, and it was not without danger to the " patients, because along with the cough, there was fever, " with all the symptoms of a pleurisy such as acute pain " in the side, bloody expectoration, etc. The disease always " arose with headache and with pains in the back and " extremities — the ordinary symptoms of the reigning " constitutional fever. The only difference was that the " morbific matter excited itself more particularly upon " the respiratory system, favored by the cough which " irritated its membranes. Although the symptoms " seemed to indicate a real pleurisy, the disease never- " theless always demanded the same treatment as that " employed for the constitutional fever, and in fact, the " usual treatment for primary pleurisy was very hurtful " in these cases. Generally, primary pleurisy ~~is~~ only " occurs in spring. Thus we must regard this autum- " nal form as only a symptom of the reigning malady " and produced by the action of the cough upon the " respiratory apparatus " With regard to treatment Sydenham also wrote :— " Narcotics, anodynes, and

" spirituous liquors were harmful and tended to increase
" the density of the humours; and further, in acting upon
" the circulation, to bring about a peripneumonia or a
" pleurisy. The quantity of remedies and the great frequency
" of bleedings employed by several physicians had the
" most dire effects, for almost all the patients, thus treated,
" succumbed."

Later epidemics were described in 1691 by Welsfer in Germany, in 1709 at Rome by Lancisi, in 1730 by Schenkzer[1], and by Beccaria at Bologna, in 1732 at Edinburgh and in 1733 in Italy by Cirnelli.

In England, the grisp raged in 1733, 1737, and 1743. In reporting these epidemics, Stuyham observes with regard to the "catarrhal pneumonias" that "of all the matters which
" are expectorated, there are none worse than that which is
" livid, corrupted and bloody, often like the lees of red wine,
" sometimes bluish and even foetid."

Grave pulmonary complications also marked these epidemics which were reported by Le Dussieu in 1733, by Henderns in 1734, by Philip Vidauté in Saxony in 1742 and in the same year in London by Baller and in France by Razoux. The years 1775, 1776, 1780, 1781, 1782, 1800, 1822, 1831, 1833, and 1837 were also distinguished by outbreaks of contagious catarrh. Referring to the 1837 epoch, Landau, Piory and Gouraud declared that the great frequency of pulmonary complications was more than a coincidence, and ought to be regarded as a double manifestation of the same morbid condition. Pidagnel[2] observes that instead of the pneumonias

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1 Les actes des Curieux de la Nature par Jacques Sheenkzer.

2. Gazette Méd. de Paris 1837.

having a fault inflammatory character, they presented the features of a mixture of pulmonary catarrh, pneumonia and pleurisy. Sandras [1], however, signalises the presence of a double epidemic — the grip benign in people whose lungs are not already weakened or diseased, and the pneumonia, eminently malignant.

Piorry describes these pneumonias, thus: — "The pneumonia "of these times has differed essentially from the ordinary "pneumonia. It has succeeded upon a bronchitis or upon a bron- "chorrhoea. The point of departure in the one is in the bronchi, in "the other in the blood vessels; its invasion is latent and gradual. "Its physical signs are at first weakness, then absence of respiration; "one finds then neither crepitant râle nor dulness, and the res- "piration soon becomes tubular. The expectoration is clear with a "light rust tint; later on it becomes opaque and rounded like "the sputum of certain phthisical subjects. Breathlessness "rapidly supervenes by reason of the obliteration of so many "bronchial tubules. The site of the pneumonia has been almost "always towards the base. The autopsy shows, independent of the "ordinary lesions of pneumonia, obliteration of numerous bronchial "tubes by plugs of mucus, clear and liquid, or thick and opaque, and "moulded to the lumen of the bronchial tube. The mortality has "been eight in sixteen in place of one in eight in ordinary "pneumonia. As for treatment, one succeeds very little by bleeding "or by Antimony." 2.

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1. Bulletin de Thérap. Paris. 1837. and Sandras et Landouzy in the Journal des Connaissances Méd-Chir. 1837. and also Sandras "Nature et traité de la pneumo-bronchite épidémique qui accompagne la grippe." Journal des Connaiss. Méd. Chir. 1837.
2. Piorry — Gaz. Méd. de Paris. 1837.

Landau [1] insists upon the obscurity of the symptoms, upon the frequent absence of stitch in the side, upon the absence of the characteristic expectoration, the general prostration, the small slow pulse and the intense dyspnoea out of all proportion to the extent of the lesions.

Mona [2] also describes the same epidemic at great length and details similar experiences. He as well as Magendie describes as a very frequent occurrence the presence of false membranes in the bronchi of the hepatised lobes.

At the same period, there raged in England a similar epidemic accounts of which were given by Thomson [3] and others. Graves describes the pulmonary lesions in Influenza during epidemics which raged in Ireland during 1837, 1847 and 1848 and Peacock [4] describes the same complications in London in 1847.

Gairdner [5], in 1858 described an epidemic in Edinburgh with pneumonias, broncho-pneumonias, and pleurisies, as frequent complications.

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1. Landau. Archiv. Gen. de Méd. 1837.
2. Mona. Archiv. " " " "
3. Annals of Influenza or Epidemic Catarrhal Fever in Great Britain from 1510 to 1837. J Thompson and also in Medical Review vol V.
4. Peacock "On the Influenza of 1847-48.
5. Gairdner - Edinburgh Medical Journal 1858.

In 1889, Mennetrier published an account of the "Grippe et Pneumonie" as these occurred and were related the one to the other in epidemic form during the previous year. He maintains "that in this case, the grip and the pneumonia are quite two affections distinct and independent the one from the other, although presenting great affinities. The one seems to predispose to the other and both appear to be favoured by the same causes" 1

This brief account brings us up now to the two latest epidemics – those of 1889-90 and of 1891.

The two recent epidemics –

The earliest records of the epidemics of 1889-90 were reported from Bokhara in Central Asia in May 1889 and in the same month though rather later from Greenland and from British North America. It spread to Europe early in November, appearing in St. Petersburg about the first of the month, in Paris about the 14th and at Vienna and Berlin towards the end of the month. A few cases were noted in London in the middle of December but the real epidemic did not develope until the last days of the year, and very soon, the entire kingdom felt the presence of the disease.

As to the proportion of the population which suffered, Franklin Parsons 2 says with regard to the epidemic in London: — "It is impossible to say with any

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1 Mennetrier "Grippe et Pneumonie"
Thèse de doctorat de Paris. 1889.

2. Franklin Parsons MD. "The Influenza Epidemic of 1889-90 and 1891 and their distribution in England and Wales. Brit. Med. J. Aug. 1891.

" approach to exactness what proportion of the general population
" of this country suffered from influenza in the epidemic
" of 1889-90. Using the figures of certain public services and
" large establishments as the basis of a rough guess, we may
" estimate the proportion of persons in and near London,
" disabled of influenza as about 25% or 1 in 4 among
" those employed in large offices, and about 12.5% or 1 in 8
" among those employed out of doors. In some elementary
" day schools the proportion of children absent from school
" through the influenza epidemic was as high as 50%."

The Spring of 1890 saw the disappearance of the disease in an epidemic form although it may be said to have smouldered on throughout the year, showing itself here as an isolated case, there as a limited epidemic. Towards the beginning of March 1891, however, it reappeared in epidemic form in Hull and in North Yorkshire rather earlier. Thence it attacked in succession Sheffield, Leeds, Bradford, Nottingham and Birmingham and finally London became cognisant of its reappearance six weeks after its rise in Yorkshire. By the time that London became affected, the disease had spread throughout the Kingdom. In the Lancet for May 9th 1891, we read:—
" the outbreak of influenza which first made its reappearance
" in this country about a month ago, and was for a few
" weeks confined within a comparatively limited area, has
" now spread over the whole community" .....
"..... " Dr. Whitwell, the medical officer of health to
"St. Olave's district, in a report presented to the Board,
"stated that, so far as his experience went, the nervous type
"of the disease, complicated with severe lung symptoms was
" more frequent than last year. This is borne out by the
" exceptional rise in the mortality from Bronchitis and

" pneumonia in the towns which have been most affected.
" Sheffield still holds an unenviable preeminence in
" this respect, its mortality rate having last week advanced
" to the extraordinary figure of 70 per 1000; but we are
" glad to learn that the virulence of the epidemic is on
"the decline in that town." ..... "So far then, as
" compared with the epidemic of 1889-90, its spread has not
" been so rapid, but it has been none the less sure; and
" there has been considerable variation in the severity of
" the type of the disease, some districts which furnish many
" sufferers having a much lower mortality than others.
" On the whole, however, we fear that there can be
" little doubt as to its virulence being greater than last
" year's epidemic, which, contrasted with previous out-
" breaks was comparatively mild, and was not responsible,
" either directly or indirectly, for so great a mortality as
" has occurred in this country during the past few
" weeks, especially from the acute diseases of the
" respiratory system."

Early in March, the disease appeared in the United States and assumed serious proportions there during the month of April. About the same time too, it was present in epidemic form in Southern Russia and here and there in Portugal. Towards the end of April, the southern parts of England and Kent in particular became the seat of the epidemic, while in Norway and Sweden, it was making great havoc. Towards the end of May, Russian Poland had become affected, but the rest of Russia showed a remarkable immunity compared with the previous year's experiences of the disease.

In its essential features, the disease, in these two

epidemics presented characters similar to those of previous outbreaks such as I have already noted.

There are three different forms described — the nervous, — the respiratory (or catarrhal) and the abdominal form.

The nervous form includes the severe headache, pains in the eyeballs, in the head and limbs, sleeplessness, vertigo, even delirium, and often a very high temperature. Under this heading also are the many symptoms referrible to implication of the vagus nerves, to some which I shall touch later on.

When the respiratory symptoms are most evident, nasal, pharyngeal, tracheal or bronchial catarrh may be complained of; and in a few cases though certainly not in such a large percentage of cases as was expected, there was watering of the eyes along with nasal catarrh, and occasionally severe bronchitis and even grave pulmonary lesions supervened during the height of the primary affection.

The abdominal symptoms were, as a rule, sickness, vomiting and gastric pain frequently accompanied with diarrhoea, but in some cases these symptoms were so severe as to simulate peritonitis or even perforation, so acute was the pain and so intense the depression*, while at other times, one would have thought that he had a veritable case of typhoid fever to combat. Generally, however, no one class of symptoms was present without the others showing themselves in some degree, and it was only when one class of symptoms predominated that one was justified in applying such terms as the Nervous Form, the Gastric Form or

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* "Cases of Influenza with severe abdominal pain and Collapse": by Robert M. Simon M.D. British Med. J. June 13. 1891.

the respiratory form.

The Mortality in uncomplicated cases was very low. Parsons states that "the deaths in London attributed primarily to In-
" fluenza in the thirteen weeks ending July 11th 1891 has been
" 2·015 equal to an annual rate of 1·9 per 1000 inhabitants."[1]
" In the first quarter of 1890 the deaths from influenza were
558 or 0·5 per 1000 inhabitants, showing that the second
epidemic though not so widespread, was yet more virulent in
its nature.

Had Influenza ended here, without giving rise to that train of sequelae and complications, the traces of whose fatal trail are still for too evident around us, the main harm done would simply have been the temporary paralysis of commerce produced by the fact of such a large proportion of workers being rendered "hors-de-combat"! Perhaps no known disease presents such bizarre anomalies. Though essentially benign as an initial affection, even so much so as to kill but one in a thousand inhabitants, it has; nevertheless, through its complications, laid its fatal hand in a very heavy manner upon the general death rate. In this sinistre aspect one may well endorse the words of Shetler with which I have commenced this paper.

To give an account of all the complications which have been reported up to this date, would be foreign to the matter in hand. Their name is legion. No organ of the body, we may say, has been respected; each has at one time or another, in one case or another, the same tale to tell, and as a general

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1 Parsons - loc. cit.

* "L'Enquête Médicale sur l'épidémie de grippe"
par Ch. Talamon. Journal de Méd. 1er Mai 1890.

rules, the morbid tendency has been no light one. Meningitis, otitis, peritonitis, metritis, pulmonary lesions and affections of the kidney, spleen, eye and cardio-vascular system have all been recorded. It is with the respiratory complications, however, that we have to deal with especially. All observers are unanimous in stating that these pulmonary lesions have proved to be the most frequent, and at the same time the most fatal of all the principal complications. In 600 cases of grippe, Ch. Dalamon notes 300 times Bronchitis, 140 times Bronchio pneumonia, 30 pleuro pneumonia, 12 pleurisy and 315 times otitis.*

In Paris during the first week of 1890, 2683 deaths were registered in place of 155 during the same week in 1889.

* Dr. J. Bertillon, comparing these two weeks, gives a number of interesting figures. These show that children escape much more readily than adults. Here are his figures

Number of deaths	during 1st week of 1889	and 1st week of 1890.
from 0 to 1 year	149	203
" 1 to 4 years	117	171
" 4 " 19 "	51	91
" 20 " 39	179	570
" 40 " 59	214	743
" 60	260	905
	970	2683

The inflammatory diseases of the respiratory organs formed the principal cause for the augmentation of the normal ratio of mortality. The total of these for 1890 was 972 compared with 155 for the same week in 1889. Bronchio-pneumonia accounts for 154 of these compared with 32 in the previous year, and when this figure is taken in connection with the fact that children have comparatively escaped from these outward complications, we can more readily appreciate the greater frequency in

the adult of bronchio-pneumonia, which is usually at that time of life a rare disease. Further, in these figures, the number of men was double that of the women for adults, while for the aged, the figures were practically equal. *

In Madrid too, the death rate amounted to 2,559 from December 21st 1889 to Jan 10th 1890 inclusive, while during the similar period of 1888-89, the figures only reached 960. Here again we find the same story:— "The complications have generally been bronchio-pulmonary". † As in Paris, so in Madrid, the greatest mortality was in individuals from 30 to 50 years.

In London, too, the general death rate was exceptionally high. As I have already shown, the death rate attributable primarily to influenza, so far as could be calculated with any degree of exactitude was 0.5 per 1000 per annum in the 1889-90 epidemic and 1.9 per 1000 during the outbreak of 1891. Taking the figures for the rate of mortality due to all diseases, we find according to Parsons that "in the four weeks ending January 25th. 1890, the deaths
" registered in London above the average were equal to a rate of 6.6
" per 1000 inhabitants. In the eight weeks ending June 20th.
" 1891, 45584 deaths above the average have been registered in
" London, equal to an annual rate of 9.0 per 1000 inhabitants." §
The same observer also draws attention to the fact already quoted from Bertillon — that the fatality incurs most heavily upon the middle periods of life. "A circumstance which
" seems to point to a difference between the epidemic influenza
" and what goes by the name of 'influenza' in non-epidemic
" years, is the difference in the incidence of the mortality on

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* These figures are taken from a paper by M. Bertillon, entitled "De la mortalité a Paris" published in the Bulletin Méd. 8th Jan. 1890

† La Grippe à Madrid. M. Del Valle. Médicine Moderne. Feb. 6. 1890.

§ Parsons — loc. cit.

" persons of different ages: the deaths ascribed in ordinary years to
" "Influenza" being chiefly those of young infants and of elderly
" persons, whereas during epidemics, the deaths are more numerous
" in proportion to the whole at the middle periods of life. Different
" epidemics, however, have differed as regards age mortality, the
" epidemic of 1891 agreeing with that of 1847-48 in showing the
" highest proportion of deaths among persons of 60-to 80, whereas in
" 1890 the highest proportion was at ages 40 to 60.
" The principal causes", he continues further on, " contributing to
" the excess of deaths in the four weeks ending Jan. 25. 1890
" besides Influenza, were whooping cough, Phthisis, bronchitis,
" pneumonia, other diseases of the respiratory organs, diseases of the
" circulating system, and alcoholism. The greatest excess of deaths
" from Bronchitis and diseases of the circulating organs occurred
" in the week ending Jan 11th.; that from influenza, Phthisis and
" pneumonia a week later — namely, the week ending Jan 18th.
" The deaths from Phthisis continued somewhat above the average
" until the middle of March, but those from Bronchitis and
" Pneumonia fell below the average in February". 1

In a lecture upon Influenza and its complications, W. Mitchell Bruce says," Bronchopneumonia is an exceedingly rare
" disease in the adult; so rare that some of our best authorities
" decline to allow that the disease is ever seen post-mortem
" except in children. At the present time in connection with
" Influenza, there is no question whatever of its existence." 2

Kahler and Kundrat at Vienna, Reynaud and Gauchet at Paris, and Soltaloff at St. Petersburg, all report the

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1. See Parsons paper for full tables of figures.

2. "A clinical lecture upon Influenza and its complications - Lancet. May 30. 1891. also a paper by T. Nicholson, M.D. in the British Med. J. "The Complications and sequelae of Influenza."

frequency of bronchio-pneumonia and dwell upon the great tendency to the formation of foci of suppuration or even of gangrene of patches of the affected lung. They also signalise the markedly fatal nature of the disease, its slow defervescence and convalescence, and its very frequent termination either in a chronic condition or in an actual condition of tuberculosis.

End of Chapter II.

Chapter III.

The Pathology of the bronchio-pneumonia of influenza: pathology of influenza and of "traumatic pneumonia": traumatic pneumonia compared with the bronchio-pneumonia of influenza: bacteriology of influenza and of bronchio-pneumonia.

Before going on to discuss the pathology of the various forms of bronchio-pneumonia and more especially of those which are associated with the grip, let us just glance at the clinical condition and state of affairs into which the system has been brought by the primary attack of influenza.

As a general rule, there is extreme debility — extreme, I repeat, out of all proportion to the subjective feelings of the patient. He may have felt giddy and sick, may have complained of various pains in the head, bowels or limbs, but if he has gone to bed, he feels that he has not been really ill, until he attempts to rise again. Then, he is more than astonished to find that his legs can scarcely bear his own weight, and I have known of many cases where the patient, upon first assuming the erect posture, staggered and fainted right away. Occasionally the cardiac weakness has been one of the first symptoms to show the serious nature of the complaint. There is, as it were, in all these cases, a bulbar lesion — a condition which seems to have varied not only in its nature, but also in its tendency to preponderate over particular areas of that portion of the central nervous system. Such symptoms have been reported as rapid weakening of the heart muscle and even syncope, slow and often irregular pulse, or it may be a very rapid pulse, Angina Pectoris, slow respiration, or

rapid short respirations with great dyspnoea, Cheyne-Stokes respiration, pallor, dilated pupils, cyanosis, even sudden and fatal heart failure, and finally in some cases a very high temperature. * Such conditions point, some to irritation, some to paresis and even to paralysis of the vagi, others to

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* See the following papers: —

Alison - Mémoire sur les symptômes et les complications de la Grippe. Arch. Gén. de Méd. Av. et Mai 1890. "Slow pulse" cité.

V. H. de Brun "Manifestations Nerveuses de la Grippe". Méd. Moderne 30th Oct. 1890.

Soltoloff. On the Grip. Russian Medical Soc. St Petersburg. Jan. 1890.

Willis - Heart manifestations in Grip. Lancet. May 3. 1890.

Marriot. Abt. Angina Pectoris in Influenza. Lancet. March 29. 1890.

Caragorgides. John. Ly. M.D. Uncomplicated Angina pectoris after Influenza. British Med. Journal June 21. 1890.

Bourneville - Gazette Hebdomadaire de Médecine et Chirurgie. Jan. 25. 1890.

Drasche. Royal and Imperial Society of Vienna. Discussion upon Influenza. 7th March. 1890.

Nicholson. The complications and sequelae of Influenza. British Med. Journ. June 13. 1891. Page 1273.

Lyon. "La Grippe à la clinique de l'Hôtel-Dieu de Paris. La Médecine Moderne. Jan. 25. 1890. on dilatation of the left ventricle cité.

Hanchard Société Médicale des Hôpitaux. Jan 24. 1890 and La Médecine Moderne 30 Jan. 1890.

irritation or paresis of the vaso-motor centres, and others, still graver, in an inhibition of the higher centres as in those cases where Cheyne-Stokes respiration has been noticed.

The heart muscle stops and the ventricles, especially the left one, undergo dilatation, the lungs tend to become congested, and the bronchi and bronchioles may lose their natural tonicity. The gastro-intestinal filaments too, of the vagi, have very frequently shown signs of implication.

In proof of the argument that these lesions are due primarily to implication of the vagus nerves, let us glance at the conditions to be found in that artificially induced form of pulmonary affection called "Traumatic Pneumonia", and which occurs in all animals except birds when both pneumogastric nerves have been divided.

Since the days of Valsalva, who was the first author to give any definite information upon the subject, this condition of the respiratory system has been carefully studied by many competent and most able observers.

The changes which most frequently take place when both vagi are divided above the origin of the recurrent laryngeal nerves (the state most analagous to our cases of paralysis) are as follows: —

(1.) There is paralysis of the muscles of the larynx, trachea and bronchi as well as loss of sensibility of the larynx, trachea, bronchi and lungs. The reflex act of coughing is abolished, and foreign bodies may now pass readily into the air passages owing to the paralysis of the glottis. As the reflex act of coughing is abolished as well, these substances pass on into the bronchioles and alveoli, there decompose and set up a chemical as well as a mechanical state of irritation.

(2). The respiratory movements become slower, and the

inspiratory phase more expansive. Practically a form of Cheyne-Stokes respiration developes. The movements may be so disturbed as to result in interstitial haemorrhages from tearing of the pulmonary tissue. The atmospheric pressure within the lungs, during these long inspirations falls to a minimum, and there rapidly ensues a severe pulmonary congestion. The vessels are overfilled, serous exudation occurs through the walls of the distended capillaries, a condition of oedema of the lungs follows and there may even be a true haemorrhagic exudation as well. It is uncertain to what cause this congestion owes its production, whether to the abnormal respiratory movements and especially the prolonged inspirations, or whether to an actual vaso-motor paralysis of the pulmonary blood-vessels, or finally to certain alterations in the cardiac rhythm, which I shall now detail.

(3). Section of both vagi paralyses the inhibitory nerves of the heart,— the nerves which constantly moderate the cardiac pulsations. There follows, therefore, a notable acceleration of the pulse rate, which increases in a marked degree, the general blood pressure.

(4). Although there is as yet no definite proof, the general belief is that the pneumogastric nerves supply trophic as well as motor and sensory fibres to the respiratory tree. When the connection of these with the pulmonary cells is severed, trophic changes as well as those other alterations above mentioned, are said to follow.

(5). Finally, an exhaustion of the respiratory centre now takes place and if the animal has survived the operation long enough, this will bring about the fatal result. This is in all probability the cause of death in birds which do not suffer from traumatic pneumonia.

According to Traube and others[1], the real active agent in the excitation of the bronchio-pneumonia, is the inclusion of all kinds of foreign bodies (saliva, food, liquids, dust, irrespirable gases, etc.) into the air passages and the irritation therein set up. One cannot however neglect these other various alterations in the normal functions of the lungs and heart. Although they may not of themselves lead to inflammatory conditions, they yet supply a most suitable basis for further changes.

It is the presence to a more or less marked degree, in many cases of pulp, of such functional changes that I insist upon as a most important predisposing cause of the disease under consideration.

In most cases, the initial stage has been irritation of the vagus centre as well as that of the fifth pair of cranial nerves. According to Althaus, this state of irritation is sufficient to give rise to all the symptoms of influenza including catarrh of the mucous membranes supplied by the affected nerves. In an address to the Medical Society of London upon the Pathology of Influenza with special reference to its zymotic character, he remarked:—

"It seems to me evident that this lesion — whether congestive or inflammatory — must be very high up, as the "symptoms habitually implicate the whole extent of the "area which is under the influence of these nerves. "Moreover, their simultaneous affection, which seems

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1. Traube. "Die Ursachen und die Beschaffenheit derjenigen Veränderungen, welche das Lungenparenchym nach Durchschneidung des Nervus Vagi erleidet. Berlin 1845. also Gesammelte Beiträge I vol. pl. 1871.

" in the catarrhal form of grip would lead us to assume a locality
" where they are lying close together. The two pairs of nerves are
" in close contact, however, in the uppermost portion of the spinal
" cord and the bulb, where they originate with two nuclei, a smaller
" motor, and a larger sensitive one, lying respectively on the top
" of the anterior and posterior grey comua of the spinal cord.
" Irritation of this portion of the bulb is therefore shown to account
" for the symptoms observed in the catarrhal form of grip, just
" as irritation of certain other centres in the bulb has been seen
to account for the symptoms of the nervous variety of the grip." 1

But, I would go further than Althaus, in holding that
'irritation' alone cannot account for all the symptoms reported.

It is evident in considering the various clinical records of the affection that a state of exhaustion — of paresis of these centres is soon brought about, when probably a larger dose of the influenza poison is circulating in the blood. Then it is that the analogy which I have drawn becomes more evident.

In the irritant form we find a somewhat general, though in most cases, not a severe catarrh of the respiratory tree even in its entirety, with rapid breathing, high temperature and in very many cases (if it be not the actual rule) a slow pulse.
I have frequently found a pulse of 80 to 90 with respirations as frequent as 28 to 32. According to Jurgensen, this unusual temperature and pulse ratio rapidly changes, and may be gone before twenty four hours. He says: — " After the first
" twenty-four hours, the pulse temperature ratio varies.

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1 Althaus. Julius, M.D. An address on the Pathology of Influenza with special reference to its nervous character". read before the Medical Society of London, Nov 2nd 1891 and published in the Lancet for Nov 14 and 21. 1891.

" Both may be normal, or both raised; but in uncomplicated cases the pulse often remains at or below 80." 1

Soon however, paralytic symptoms from exhaustion of these nerve centres, begin to show themselves, although there are great variations in the rapidity of onset and in the intensity of their nature.

In comparing the two forms of paralytic symptoms, of which one set is produced artificially — the traumatic lesions of the vagi, the other through the circulation of a poison in the blood and affecting the nerves at their origin — the grippal affection, we find representatives in the latter of most of the lesions described in the experimental form.

Thus for example, to take the paralysis of the larynx, trachea, and bronchi, we have a condition described by Hanchard, who concludes some notes upon influenza by remarking:— " finally, I would note a sort of bronchial paralysis already indicated by Graves, and without bronchitis; one might almost say that the patient had both his pneumogastric nerves cut." 2.

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1. Jarbstein. "Note upon the temperature and pulse in Influenza. Lancet March. 1890 p. 1273.

also Nicholson in a paper referred to upon page 21 of this thesis, says:— " Occasionally, at no period is the temperature raised, but as " a rule, the mercury rises quickly to 101° or to 102° or higher. The pulse however, often remains quite slow throughout."

2. Hanchard. Société Médicale des Hôpitaux. 24th. Jan. 1890. reported in La Médicine Moderne. 30th. Jan. 1890. Paris.

Dr. Wm. Ord tho, in writing of Bronchio-pneumonia in Influ states :— " In some of these cases, I have also observed " a phenomenon indicating (as it appears to me), serious affection " of the central nervous system. A patient is blue and livid, " violently with tinged cheeks; he is breathing fast and with a distinct " rattle, audible at some distance. On auscultation there are " signs of the presence of large quantities of secretion in his " bronchial tubes. Yet there is no expectoration, and no sign of the " swallowing of secretion brought up into the fauces. It reminds " one of what is called the " death rattle" wherein paralysis of the " muscular walls of the bronchial tubes doubtless occurs". 1

In a case of Influ which occurred under my care, and in which the bulbar symptoms (especially of an irritative type) were well marked, attacks of asthma with a similar rattle were very frequent. They arose some few days after the onset of the illness and lasted for over an hour at first, but gradually diminished in intensity and duration as convalescence became established.

Recently, the slowing of respiration, the dyspnoea and even Cheyne-Stokes respiration, have each a counterpart in Influ.

Slow respirations, Quick short respirations, Cheyne-Stokes respirations have all been noted by Dr. H. de Brun, and others. 2

Ferrand noted a peculiar state of the lungs which seemed to precede for some days, the bronchio-pulmonary complications of Influenza; there was a notable diminution of respiration upon one side of the chest with increase of percussion resonance, without râle or rhonchus. The pain this several

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1. Dr. W. Ord, " on the influenza epidemic of 1892. British M.J. 1892. p 1143.
2. Dr. H. de Brun " Manifestations Nerveuses de la Grippe.
La Méd. Moderne. 30th. Oct. 1890 et seq.

times and in individuals at different ages, and forty-eight hours afterwards, the pneumonic signs appeared. He considered that it was due to modifications in the permeability of the pulmonary tissue. 1

Monsieur Hanchard, already quoted, says, "As for respiratory complications, I have seen pneumonia and broncho-pneumonia, but "besides — very peculiar congestions, sorts of states of passive "congestion with rales only in big and deep inspirations. At other "times, I have seen active congestions with ~~complications~~ haemoptysis."

Oedema, with a tendency to blood in the sputum has been a frequent occurrence. In two cases, I saw this to a marked extent. The expectoration was in the form of a watery mucus, somewhat frothy in colour from the presence of blood evenly diffused through it, and holding in suspension masses of firmer mucus. In one case, the fluid was highly aerated, in the other there were but few air bubbles. In the latter, the daily amount expectorated, at first amounted to 200 grammes, and at both bases there was slight dulness to percussion with sub-crepitant râles.

Dr. C. N. Gwynne of Sheffield reports a remarkable series of severe pulmonary cases ending fatally after a few days' illness. In these, besides consolidation, there seemed to be oedema as well, and in all, the sputa contained blood, showing a grave state of the pulmonary circulation. In all of these cases too, exhaustion of the respiratory centre seemed to be the cause of death. As to the heart conditions, still ~~portion~~ referring to Dr. Gwynne's paper, he describes in all his fatal cases towards death, a very rapid pulse. In one case, it rose from 130 to 150 before death although the respirations remained at forty,

while

1. Ferrand. Société Médicale des Hôpitaux de Paris.
June 10th 1890.

while the temperature was 104.6. 1.
Further heart implication is shown as has been already indicated in dilatation, irregularity in rhythm, angina, etc.

Such interference with the functions of these important nerves, whether from irritation or from actual paresis, would of itself lead to disastrous complications in any case of Grip in which they happened to be well marked, but even in milder attacks of this disease, we have other grave conditions to relate.

Although no definite unassailable evidence is forthcoming with regard to the discovery of a specific microbe, the symptoms, course, and infectious nature of the disease, all point by analogy to its origin from a specific germ, which though of a short life history, has yet time to produce highly poisonous products or ptomaines.
These ptomaines, circulating in the blood, not only cause the disagreeable symptoms such as headache and other pains and the

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1. C.N. Symme. "Notes on the recent epidemic of pneumonia in Sheffield". Lancet. Dec 27. 1890.
In connection with this paralytic form of pulmonary congestion, an interesting case was reported to the Société Médicale des Hôpitaux de Paris, May 22nd 1891 by M. Rendu. She described a form of phrenic and pneumogastric paralysis consecutive to sore-throat. The diaphragm was paralysed and at the bases of the lungs there were detected rales and tubular breathing indicating pulmonary congestion. ~~The diaphragm~~ The diagnosis was — abscess of the circumoesophageal cellular tissue, leading to a neuritis of the trunks of these two nerves. The symptoms referible to the vagus were mainly the pulmonary congestion, cardiac failure and excessive vomiting. Recovery occurred with marked general emaciation.

various forms of catarrh seen during the height of the attack, but also they exert their baneful influence still further, in hampering and even in destroying the phagocytic action of the amoeboid corpuscles. 1.

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1 As this paper was almost completed five months before the publication of the discovery of the Influenza Bacillus, I had not the benefit of the papers which appeared in the British Medical Journal for Jan 16. 1892 on "The Influenza Bacillus". These papers are: —

1. "Preliminary Communication on the exciting causes of Influenza. by Dr. R. Pfeiffer. Chief of the Scientific section.
2. "On the Influenza Bacillus and the mode of cultivating it." by Dr. S. Kitasato.
3. "On a microorganism in the blood of influenza patients. by Dr. P. Canon. Assistant Physician, Berlin.

It may be briefly stated that the Influenza bacillus appears as a tiny rod-shaped body, about the thickness of the bacillus of mouse septicaemia, but only half the length of that organism. On staining with dilute Ziel's solution, and with hot Loeffler's methylene blue, it is seen that the two ends of the bacilli stain most deeply so that forms are produced which are remarkably similar to diplococci or streptococci — a resemblance increased by the fact, that these may be seen in chains of three or four. Gram's method does not show them. They are found exclusively in cases of Influenza in the purulent bronchial secretion and often in the protoplasm of the pus cells. Pfeiffer says that "the presence " of bacilli kept equal pace with the course of the disease; with the cessation " of the purulent bronchial secretion the bacilli began to disappear. Only in " apes and rabbits could positive results be obtained from inoculation " experiments. In view of these results I consider myself justified in " pronouncing the bacilli just described to be the exciting causes of " influenza. Dr. Kitasato has succeeding in cultivating the influenza " bacilli to the fifth general on glycerine agar."

taking all these facts into consideration — i.e. the weakened state of the heart and lungs, and the altered chemical and physical properties of the blood such as I have described

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Note upon Influenza Bacillus (Continued) —

Canon reports " During the last few weeks, I have under the direction
" of Dr. Guttmann, examined the blood of twenty influenza patients in
" stained preparations, and in almost all cases I have found in the
" blood one and the same microorganism." ..... " As on the basis of my
" researches, I am of opinion that this microorganism occurs in the blood
" of all persons suffering from influenza (at least in those of those who have
" fever), and as it is not found in the blood of other persons, and as it is a
" micro-organism hitherto unknown, I believe that it stands in direct relation
" to influenza".

Klein in a paper entitled "Some remarks upon the Influenza Bacillus" in the British M. J. for Jan. 23. 1892, ~~refers~~ gives his experiences of the microbe from experiments conducted during the end of December 1891 and the beginning of Jan. 1891 during the early presence of the epidemic in London. He found in the blood and bronchial sputum bacilli, of which he remarks " as regards
" thickness, length and colour staining, they compare completely with those described
" by Pfeiffer and Canon, but some of the bacilli were quite as long as those of the
" mouse septicaemia with which Pfeiffer compares them. Cover glass speci-
" mens made of the fluffy masses from the broth cultures and of similar fluffy masses in the
" condensation water of the agar cultures showed the growth to be entirely made up of strings and
" filaments; each filament is composed of short thin rods some staining uniformly, the great
" majority however, showing a granule at each end; in this respect the filaments look exactly like
" streptococcus chains composed of dumbbell cocci but the presence amongst them of
" uniform bacillary elements without polar staining proves the organism a leptothrix of
" minute bacilli. ..... From this it is seen that what is here stated of the bacillus in
" the sputum in broth and agar cultures, completely coincides with and confirms what is described by
" Pfeiffer and Kitasato. One appearance which is not mentioned by these in their preliminary account, and
" which seems to be a very striking appearance, is that the growth in broth is entirely made up
" of filaments " —

elsewhere [1], we can readily see how easy the incursion into and growth of the microbes in the blood might come about.
All the other forms of lesions, such as grave bronchopulmonary affections and all the different kinds of suppurative conditions, which have been reported, ought rather to be considered as secondary lesions in influenza —— secondary in respect to the initial attack produced by a specific germ, for we shall find that they are not always accompanied by the same microbe.

The microorganism most frequently found in these secondary affections has been the Streptococcus Pyogenes, although others were frequent such as the pneumococcus lanceolatus of Friedländer, the lanceolate microbe of Talamon-Fränkel, the staphylococcus albus and the staphylococcus aureus.

Leyden says: "I have observed catarrhal as well as acute fatal pulmonias. In the latter the pneumococcus of Fränkel has been constantly found. In two fatal cases, only the diplococci and streptococci were found." [2]

Rilobat found five times in eight autopsies upon patients dead from influenza, the same streptococcus, — which he likens to the streptococcus pyogenes or erysipelatus and which he rather inclines to believe as playing a very important rôle in the disease, if it is not even the specific microorganism itself. [3].

Bouchard describes different microbes according to the organ affected; in the vesicles of herpes labialis, it is the streptococcus pyogenes aureus, with which one meets; in the pneumonias, it is the pneumococcus lanceolatus; and in the bronchial secretions

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1. Case of symetrical Gangrene following upon an attack of Influenza by H. Highet - M.B. Ch. British Med. Journal. July 18. 1891.
2. Soc. Med. Interne. Berlin. Jan 6. 1890. Discussion upon Influenza.
3. Rilobat. Microbiological researches in Influenza (Deutsche Med. Wochenschrift) Nov 15. 1891.

it is the streptococcus or staphylococcus.(1).
Weichselbaum confirms in almost all details these experiences of Bouchard. Heiler of Vienna, described acute pneumonias, of which, some were due to a diplococcus, others to the bacillus of Friedländer and others to the streptococcus pyogenes.(2)
Viellard with the assistance of Vincent made a careful study of the subject in Paris, and in all cases found the same streptococcus. In a case complicated by bronchio-pneumonia and phlebitis of the popliteal vein, he found swarms of streptococci in the blood of the affected vein, as well as in that taken from the spleen two hours after death. In the sputum, as well as in the various forms of effusion such as pleural, etc. especially when these were purulent, the same streptococcus swarmed.(3) At the same meeting of the Société Médicale des Hopitaux, Netter remarked that the streptococcus was a normal inhabitant of the mouths of some persons.

Burlureaux(4) describes how the anaemia of convalescence was always very marked as if the pathogenic agent of the Grippe, had upon the red blood corpuscles, an action similar to that which we know belongs to the pathogenic agent of the malaria. In one of his cases, that of a young and healthy cavalry officer death was apparently due to a septicaemia without special local signs, and without appreciable gate of entry for the poison. He had a septic endocarditis, degenerated

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1. Bouchard. Académie de Médicine de Paris. 26th Jan. 1890.

2. Heiler. Imperial and Royal Society of Vienna. Feb. 21. 1890. Med. Moderne 27th Feb. 1890.

3. Viellard. Société Méd. des Hop. Paris. 24th Jan. 1890.

4. Burlureaux. Quelques réflexions à propos de l'épidemie actuelle de la grippe. Gazette Hebdomadaire de Médicine et de Chirurgie. Jan 25. 1890.

liver, congested kidneys and an enormous spleen, recalling the appearance of a recently delivered woman dead from septicaemia. All the organs swarmed with streptococci.
Similar accounts come from Talamon[1], Reichmer[2], Babes[3], Klebs,[4] Kowalski[5], Heligiannis[6] and Prin[7] with regard to the frequency of the streptococcus.

To recapitulate then :—

(1) The bronchio-pneumonia of influenza is generally a secondary affection.

(2) It is most frequently noticed in males between the ages of

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1. Talamon. La grippe et les microbes. Méd. Moderne Nov. 1890.
2. Reichmer. Researches upon Influenza — Centralblat f. Bakt. u. parasitenkunde No 12 1890.
3. Babes — An introductory note upon the bacteria found in Influenza. Centralblat f. Bakt. u. parasit. No 15. 1890.
4. Kowalski researches upon the microbes of influenza. Wiener Klin. Wochenschrift. No 13. and 14. 1890.
5. Heligiannis — Bacteria of the grip. Galenos Nos. 1890.
6. Prin. Researches upon the microbes and complications of the grip. München. Med. Wochenschrift. No 13-15. 1890.

twenty and sixty.

(3) It is not necessarily induced by a chill[1]; in fact, very often it is quite otherwise, and it may arise before the patient has recovered from the primary attack and has risen from bed.

(4) The toxic agents, secreted during the primary attack, and now circulating in the blood, diminish the resisting power of the organism as a whole, besides hampering, if not destroying, for the time being, the phagocytic power of the wandering leucocytes.

(5) Through the poisonous influence of these same ptomaines, the central nervous system, the blood, the circulatory and respiratory systems, etc. all are more or less interfered with and their normal functional activity is thrown out of gear.

(6) As a result of such deterioration of the system, the facilities with which microbes may enter and develope within, are greatly enhanced.

In health, we are not free from the presence of many kinds of microorganisms in our secretions, in our various cavities and free passages, and even in the circulating blood itself, but as long as our resisting power remains capable of dealing with these, as long as the phagocytes can absorb them, so long will these germs remain innocuous to the general health. Once they are permitted, however, to increase in numbers beyond the pitch up to which the system can resist them, then their multiplication is unlimited. Their products of secretion tend further to diminish the resisting power of the phagocytes, although in the end, the ptomaines prove poisons to the very germs to which they owe their origin. In the present case, as we have seen, it is the Streptococcus pyogenes which has run riot in the system, and has been the active agent in the pathology of the bronchio-pneumonia. The general gravity of this form of lung affection is in this way accounted for, and it is only in patients of a very robust constitution and in whom the treatment can be carried

out in the least possible manner, that one can hope for a perfect or even a good recovery. Too often the streptococcus leads to extensive suppurations, to hectic exhaustion and even to local gangrenes, and the system undergoes a process of poisoning either from the ptomaines themselves, or from the products of decomposition of the various pathological lesions to which they have given rise.

As for the other forms of Broncho-pneumonia which develope in the course of specific fevers, etc. the same reasoning to a large extent, holds good. The pulmonary exudation arises secondarily in a system already weakened by another disease and poisoned by the products of the germs of that disease. The active pathogenic agent is always a streptococcus, which may enter the system from without — a heteroinfection, or be absorbed from the various cavities and passages of the respiratory tract etc. auto-infection. Mosny of Paris in a recent monograph upon 'Broncho-pneumonia' says" that whatever be the clinical aspect of the lobular pneumonia, whether it be primitive or secondary to measles, to diphtheria or to any other disease its pathological agent is always the same, and that is a streptococcus — the streptococcus pyogenes". "whatever be the causes of the infection" he continues" the paths of propagation are always the same; the agents of the broncho-pneumonia in all cases proceed by way of the respiratory tracts, through which they pass directly to the pulmonary alveoli. Never, has any one found them primitively in the blood-vessels, nor in the foci of atelectasis or of emphysema. It is in all cases by way of the lymphatics that the infection becomes general, and [illegible] lymphangitis marks the commencement of this generalisation" He concludes by asserting that "broncho-pneumonia whether it be primitive or secondary, and in the latter, whatever be the affection which has preceded it, is contagious and epidemic." [1]

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[1] Mosny. Étude sur les lésions histologiques et les causes bactériennes de la broncho-pneumonie. Thèse inaug. Paris. 1891.

In cases I. IV. V and VII Streptococci were found in abundance - The methods used in staining these were those employed by Mossy. The cover glass preparations were placed for 10 minutes in a hydro alcoholic solution of gentian violet (10 drops of a saturated solution in 5 cmc water). These were then removed from the staining fluid, thoroughly washed and slightly decolorized in a One per cent solution of acetic acid. Where the tubercle bacillus was suspected he employed the following method (after Mossy) which colours the bacillus of tubercle red and all the other bacilli blue. For ten minutes the cover glass lies in a solution of Fuchsin (Erlich's), then after washing must thoroughly, leave them for 2 & 3 minutes in this solution (Fraenkel):

Acid. nitric fume 20
Distilled water 30
Spt. Vect. 50
Methylene blue q.s. ad sat.

The following results were obtained -

Case I. Streptococci swarms : alone and in chains.

Case IV. Streptococci swarms -

Case V The same result found in the bronchial secretions after death and in the pulmonary cavities.

Case VII. The same result found in the Sputum.

In all there was an absence of the tubercle bacillus.

1876

Case I. N 023/91 -
Gentian Violet. Acetic Acid.

Chapter IV

Pathological anatomy and histology.

Historical: —

Before going on to the actual description of the lesions in bronchiopneumonia it may be interesting to glance at the history of the various steps which have been made in the separation of this disease from bronchitis on the one hand and pneumonia on the other, and its evolution into a real entity in the nosology of diseases.

It may be said that the disease "Bronchopneumonia" was born in 1823. It was in this year that Léger described a condition in children which differed from the pneumonia of adults in that the lesions were bilateral, often symetrical and of such an appearance as to resemble the spleen. For this appearance, he coined the term "Splenisation" a name which has remained a favourite with his fellow-countrymen. The distinctly lobular character of the lesions was pointed out by Bertin in 1828 by Burnet in 1833 and by de la Berge* in 1834. Rilliet and Barthez in 1838 drew attention to the very frequent fact of Bronchopneumonia in children arising as a secondary affection. Gaussel described the lobular abscesses — "grains jaunes" in 1840 and held that they were of purely mechanical origin and due to the penetration of pus into the bronchi from the bronchi and bronchioles. In 1844 Lemoine, Legendre and Bailly differentiated the lesions and described two independent series of changes. The primary and the sole characteristic lesion according to them is a capillary bronchitis. This may lead to the other or secondary lesions such as pulmonary atelectasis (l'état fœtal), general congestion of the lobule, catarrhal pneumonia, mostly by mechanical means, or at times a partial lobular pneumonia by inflam—

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* Consult Bibliography at end of paper.

auld page 110

making extension. The latter, however, they regarded as always a secondary affair and not necessarily an entity of the disease. Bartels in 1860 and Ziemssen in 1867 concurred in the main with Lenguedoc and Bailly. In 1878, Balzer reproduced the theories of Charcot with regard to the connection between the various lesions and described that authors theory of the "nodule peribronchique". Balzer holds that the capillary bronchitis is the primary lesion but he does not go so far as Lenguedoc and Bailly and their followers in considering all the other lesions as of a purely secondary and mechanical origin. Some such, as bronchial obstruction, are mechanical certainly, but others are assuredly the result of extension of the bronchial inflammation. This extension may result secondarily therein in two ways. Superficially, it extends along the bronchial epithelium and causes the changes in the alveolar epithelium —— the catarrhal pneumonia & splenisation. Deeply, the inflammatory changes extend along the bronchial walls and the surrounding tissue and by their further extension, result in hepatisation of the neighbouring alveoli and thus form Charcot's "nodule peribronchique". In Balzer's mind, therefore, bronchio-pneumonia is a disease of the interlobular and intralobular connective tissue in distinction to lobar pneumonia (pneumonie franche) which attacks the pulmonary parenchyma. Some recent workers, however, such as Eberth, Grawitz, and Vulpian lay great stress upon the alveolar lesions and consider them equally of an inflammatory character. Damaschino (1867) also admits these views with regard to the inflammatory nature of both sets of lesions but considers the alveolar changes as catarrhal although occasionally they may be accompanied by fibrinous exudation as Virchow and Rindfleisch had already observed. Roger and Köster (1877) described still further the analogy between acute pneumonia and bronchopneumonia. As for Rautenberg, he described

the two diseases as identical in all except that lobar pneumonia was a primary disease and bronchopneumonia a secondary - Niemeyer's opinions agreed in the main with those of Damaschino. He considered the atelectasis and splenisation, however, as of mechanical origin and the resulting dilatation of the bronchioles by pus. Germain Sée (1885) distinguished as essential inflammatory lesions the bronchitis, the splenisation and the hepatisation, and as mechanical effects, emphysema, atelectasis and congestion. Among English observers, Fox in 1871 in Reynold's System of Medicine, and Hamilton in the Practitioner for 1880-81 deal with the subject at great length. Like Legendre, Bailly and others, most English observers have been especially impressed with the bronchitic element in the disease. It is generally said to arise out of a capillary bronchitis by extension of the inflammatory processes. Hamilton in a series of papers upon "Catarrhal Pneumonia and Tubercle in the Human Lung" insists upon the essential difference between catarrhal pneumonia and lobar pneumonia. He says:—

"The two diseases croupous and catarrhal pneumonia, in their
"acute stages are, therefore, totally different in nature; for, while the
"former is characterised by the exudation of the solids of the blood
"into the air vesicles, the latter is essentially an epithelial proli-
feration." I shall refer further to these very able papers later on. Explosion now is the ancient theory to which Eustace Smith still adheres in his manual upon Diseases of Children, that bronchio-pneumonia may arise secondarily to collapse of the lung. Cause and effect changed places to evidence such an erroneous theory. Auld, in a recent monograph upon the subject, differentiates between Catarrhal Pneumonia and Bronchopneumonia. "Some
"authors" he says, "are inclined to discard the old term "Catarrhal
"pneumonia", but it is difficult to see for what reason. Irritants
"giving rise to this form of pneumonia do not all enter the lungs

" by the bronchi. Some enter from the pleural cavity and others by the
" pulmonary artery. Hence, there is a bronchopneumonia and a
" catarrhal pneumonia, which may and usually do possess similar
" anatomical features — features which though presenting more
" or less variation according to the nature of the irritant, ~~never~~
" nevertheless are at all times sufficiently well demarcated from
" those which characterise acute lobar or fibrinous pneumonia." *

Morny ϕ then sums up what he considers to be our present knowledge of the disease:— "We have then, examined a
" certain number of bronchopneumonias without regard to their
" clinical signs, and if our study leads us to affirm the almost
" absolute identity of the histological lesions of confluent pneumonia
" and of lobular pneumonia, it shows us on the other hand: —
"1. that there is a profound topographical difference in the lesions of
" confluent pneumonia and of bronchopneumonia, in the latter of
" which, we distinguish two principal types: —
" (α) a lobular type which constitutes bronchopneumonia
" properly so-called, and
" (β) a pseudo-lobar type which ought to be attached to
" the confluent variety of pneumonia.
"2. that the difference in the bacteriological origin of these two
" anatomical types, of which the latter approaches confluent
" pneumonia, shows us that the lobular or bronchopneumonic
" type is due to the action of the streptococcus pyogenes, while the
" pseudo-lobar form is due to that of the pneumococcus
" lanceolatus of Talamon-Fraenkel."

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* Auld — The Pathological ~~Anatomy~~ Histology of Bronchial Affections, Pneumonia and Fibroid Pneumonia — 1891.

ϕ Morny — loc. cit.

Continued in Vol. II.

Volume II.

The Bronchopneumonia of Influenza.
(Continued).

by Hugh Stiglet MB. Cm.

Contents of Volume II.

Chapter IV (Continued)

Pathological Anatomy and Histology.

The Lungs:-

Following upon the original plan of Hamilton, I shall divide the pathological description into three stages:—

the first – the acute or subacute stage,
the second – that of caseation, and
the third – that of phthisis or destruction of the lung.

The First Stage.

On making a post-mortem examination at this stage we find the following appearances in the lungs:—

There is generally an absence of acute pleurisy and if inflammatory changes of the pleura are present, they are usually limited to the visceral layer and to the portions of this layer which cover the areas of hepatisation of the pulmonary parenchyma.

"The organ" Hamilton says, "when removed from the chest,
"feels vesicular throughout, often more so than a normal lung,
"from the difficulty which the air experiences in leaving it."

At the base and the posterior portions of the lungs, the tissue has a deep red colour and here the consistency is somewhat denser than normal. This condition persists as a rule while further changes may go on in other reaches of the organ. Such was the case in Cases III. IV V and VI and to some extent in Case VII. The reason for this will be discussed at a later period. Scattered over the surface of the lung and more especially at the base and the posterior border, are noticed frequent dark purple, somewhat angular patches which may project slightly above the surrounding surface. This projection however is not constant and is not to be depended upon

H Higher del. et pinx.

Naked eye appearances of portion of lung from case IV, magnified two-times. a. alveoli filled with exudation products which have undergone fatty degeneration and are rapidly caseating. a' ditto. b. dilated bronchiole with grape-like arrangement of affected lobules e. emphysema around and between the nodules. p. thickened pleura. s. thickened interlobular septa.

The first stage has just merged into the commencement of the second stage.

as a distinction from patches of atelectasis. In most cases, the surrounding pulmonary tissue is considerably distended by emphysema, so that these dark patches of hepatization seem rather to be small beneath the general pulmonary surface which is paler in colour than normal. In the adult, patches of atelectasis are very rare. When present, they appear somewhat like the above described areas of condensation but their site is contradistinction to these is upon the lateral or anterior borders or even at the summit of the organ. They are depressed beneath the surface, they do not crepitate, and to the touch they are firmer than the areas of hepatization. When entire, they float in water, while patches of consolidation sink at once. Almost all the remaining portions of the lungs seem to be emphysematous and frequently this is enormously exaggerated. The apices and anterior borders show this well, and everywhere throughout the organ between the areas of condensation and collapse, the alveoli seem to have undergone a compensating distension. On cutting into the lung, it will be noticed that the patches of consolidation already noticed from without, are most frequent towards the surface, that they are usually pyramidal in outline, and that their base lies against the pleura. On section, they are rather smooth and if granular, only slightly so. In the early stages they are not sharply defined from the surrounding tissue, and their colour like it is deep purple. Later on, they become lighter red and even greyish in colour with fairly sharp borders. (see water colour drawing No I.) They are dry, somewhat raised, and give one the feeling of tiny bags of grains as I described in Case III. The portions of the bronchioles are quite evident, are usually somewhat dilated, and often show a drop of pus.

Upon squeezing the lung, pus and pultry mucus wells from these lobules & lobelets. When these points of pus are well marked, they form the peribronchial abscesses.
The intervening tissue is markedly congested, while immediately surrounding these patches of consolidation, the air cells seem dilated and actually emphysematous. (See Watercolour Sketch No I.)
Upon squeezing the lung, blood in considerable quantity oozes from the general congestive pulmonary tissue and the large bronchi as well as the above mentioned lobules exude considerable quantities of a pultry purulent mucus derived mainly from the peribronchial abscesses, although their mucous membrane may be somewhat congested as well.
The areas of atelectasis, upon section, are deep violet in colour dry and uniform in surface and on squeezing them, only bloody mucus exudes, never pus. They float in water and can be insufflated so as to make them return to their normal dimensions.

Anatomical Forms of the disease.

Taking the distribution of the peribronchial nodules as a basis for classification, many different anatomical forms have been described. Thus we have the lobular disseminated or lobular discrete form of Bouchut, where the lobules are totally distinct and separate from each other. There they lie along the posterior borders at the corners & edges as a general rule and hence this form has often been called Marginal or Cortical Pneumonia.
Again, the foci may become confluent, involve almost a whole lobe, and yet each remain distinct from the others. This in its second stage gives the appearance of pneumonia and is called the generalised lobular form (Lobaire confluente of Bouchut).

In the pseudo-lobar form of Barrier, the separate foci are no longer distinct. In place of an irregular surface, we have a flat, smooth, non-granular surface which is somewhat friable upon section. This form can, however, always be distinguished from ordinary pneumonia by the inequalities in color and the non-granular surface. There there are concentric zones of varying tint, associated with bronchial lesions, while in other regions of the same lung, we may see the typical disseminated form with emphysema, and it may be, patches of atelectasis. Microscopical examination, too, confirms our diagnosis at once.

Histological appearances during the first stage. Examine a single lobule from one of these patches and notice in turn the various elements. In the centre of the lobule a towards the pole according the site of section, the large rounded tube of a bronchiole is seen, distended, as a rule, with cellular products. In its neighborhood lies the accompanying branch of the pulmonary artery and grouped around the bronchiole, the air vesicles are seen loosely packed with cellular products of the catarrhal inflammation. The bronchiole shows signs of capillary bronchitis. The epithelial cells are swollen and are seen to be proliferating and forming embryonic cells towards the basement membrane. The bronchial walls are congested and soon show infiltration of leucocytes, an infiltration which ever longer ends in destruction of the muscular fibres, and then the section shows a distended ring of fibro-elastic tissue, lined in leucocytes and lined of the remains of the original epithelium in the form of irregular a flattened cells lying against the swollen basement membrane.

As for the alveoli, their contents consist of cells and

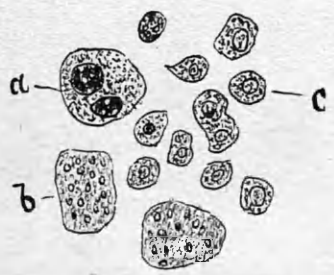
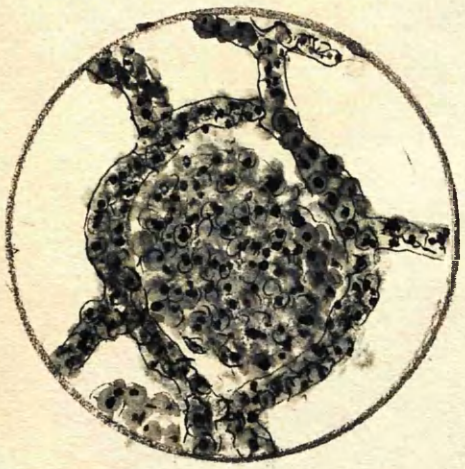


Fig 7. Catarrhal Cells. Acute catarrhal pneumonia. × 450 diameters. a. large catarrhal cell with two nuclei; b, an epithelial plate becoming fatty; c, germinating catarrhal cells. (Copied from Hamilton's paper in the Practitioner Vol XXIV. p179. 1880).

a mucous fluid. According to Hamilton, fibrin is rare, but later observers insist upon its presence especially in the central alveoli. Auld holds that "the alveolar cavity will be "found usually to contain a fibrinous exudation, which in "the majority of cases cannot be distinguished from that of "acute pneumonia — that is to say, the alveolar cavity is "filled with fibrin, which adheres closely to the alveolar walls and "contains a variable quantity of epithelial cells, leucocytes and red blood corpuscles in its meshes." The amount of fibrin depends upon the acuteness of the inflammatory condition. The same fact influences the cellular nature of the exudation. This consists of three kinds. According to Hamilton, "the first "most commonly observed is represented at a. It is a large "flat body with a finely granular peri-plast and usually two "or more nuclei, and within each nucleus there is commonly "a protoplasmic plexus. There is evidence in the occasional "dumb-bell shape of the nucleus that division and multi-"plication have been going on. The difference in size which the "nuclei often frequently show. (2) supports this idea. Certain "of these cells however, do not show any nucleus, but in the "cytoplasm exhibit undoubted evidence of fatty degeneration (β). "Oil globules are visible in them, at first few in number, but "subsequently converting the whole cell into a com-"pound granular corpuscle. (Fig. 10 d). Other cells of "smaller size (c) are also abundantly found, each having a "large nucleus with a delicately granular peri-plast. These also "show clear evidence of dividing — the nucleus first, the peri-"plast afterwards. The first bodies of round shape, evidently blood "leucocytes are sometimes met with, but not often and they do "not form an essential element of the catarrhal secretion".
"The largest cells which are flat and devoid of nuclei are the



Case III. a solitary alveolus filled with catarrhal products. (High Power).

Case III. Alveoli at the end of the first stage: caseation commencing.

"desquamated epithelial plates, the smaller nucleated bodies are
"the embryonal progenitors of the same, while the compound granular
"corpuscles are either of these in a state of fatty degeneration."
Hamilton has also clearly demonstrated the mode of origin
of these cells. In place of groups of germinal epithelial cells
scattered upon the alveolar wall, the whole wall is now germinal
in character. The nucleus divides until many of the cells contain
three or four nuclei. In their rapid growth, the cells arise above the
surface of the alveolar wall, assume a pedunculated form and
soon drop off into the cavity of the alveolus. Such a general
and rapid cell formation soon crowds the alveoli, and mixed
with the mucus and also fibrin in many cases, these constitute
the catarrhal pneumonic effusion. These cells cannot maintain
an independent existence for more than a very few days, but
rapidly undergo retrograde changes. Fatty degeneration soon
transforms these into compound granular corpuscles. In
acute cases, where the embryonic cells are very abundant
and where fibrin is present amongst them, the resemblance to croupous
pneumonia is very evident. Examine, however, the interlobular
septa and more especially the interalveolar septa. The vessels are
distended and stretched and they may be seen projecting in loops
into the lumen of the alveolus. The fixed connective tissue cells
amongst them are germinating rapidly, and herein lies the
main distinguishing feature of the disease. As Auld
remarks, "what then is the marked distinction between acute
"lobular pneumonia and acute lobar pneumonia? As has
"been seen, there normally exists in the former a fibrinous
"exudation which is indistinguishable from that so characteristic
"of the latter. But in the case of acute lobular pneumonia, the
"fixed connective tissue cells of the alveolar walls invariably ger-
"minate and give rise to a great crowd of young cells which pass
"

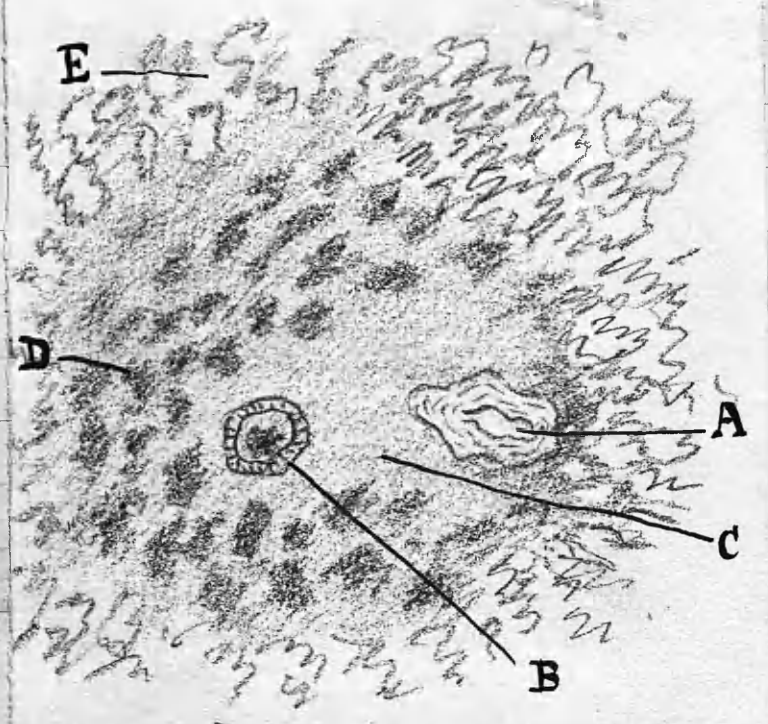


Figure 34 from Balzer.

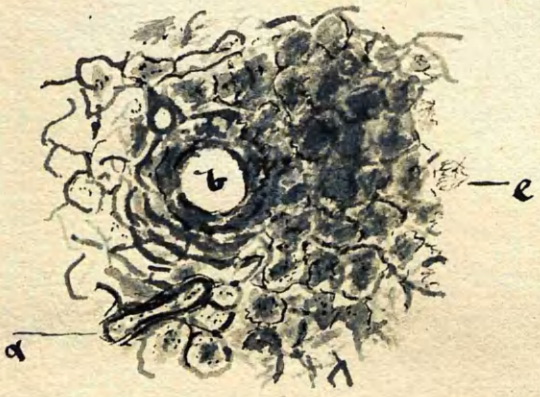
A artery B bronchus with epithelium in place and containing pus. C peribronchial connective tissue thickened. D zone of hepatisation (alveoles filled with fibrin & pus). E. Splenisation.

H. Higlet del.

" fill up the alveolar cavities. No such germination occurs in the past
" majority of cases of ordinary pneumonia; when it does occur it is ex-
" ceptional and gives rise to grave consequences. It is not the swelling
" and infiltration of the alveolar walls nor the presence of fibrin, nor of
" desquamated epithelium; all of which in either of these diseases may be
" less or more found — it is but these to which any diagnostic
" value can be ascribed, they are but extrinsic ~~fact~~ features;
" the sole anatomical distinction consists in the germination of the
" connective tissue corpuscles in the one and not in the other. This opinion
" I have formed after prolonged examination of a large number of examples
" of both affections, and am convinced that no other view respecting the
" anatomical distinction will be able to hold its ground".

Briefly then, the appearances in a lobule are these:— in the centre is the round tube of the bronchus, generally deprived of its epithelium and distended with catarrhal products of which most of the cells have undergone fatty degeneration. In rare cases such as Balzer figures (figure given) the columnar epithelium prevails although its organic connection with the basement membrane has been severed owing to the dense proliferation of cellular elements from the bronchial walls collecting below it. Surrounding the bronchiole, there is a zone of alveoli distended with acute catarrhal products. Here fibrin is most constant, and embryonic cells and blood corpuscles are most numerous. The shape of the alveoli is considerably altered. They are often much flattened by reason of the bronchial dilatation. Further out, the alveoli, which maintain their normal shape, contain only the cellular elements, and still further out in early cases, the pri-
mary congestion of the capillaries with commencing cell proliferation may be seen. Balzer was the first to draw special attention to these zones hence the name Balzer's Zones. (see figure). In the interlobular septa surrounding these patches, great activity of the cellular elements is also seen. There is generally a cellular infiltration resulting in the production of elements quite similar to endothelial cells, and in fact often derived from the endothelium lining the lymph channels.
(see figure).

Case VII. A series of peri-bronchial nodules, at end of first stage. (Low Power.)



Case VII Peribronchial nodule. a, artery. b, bronchus. c, almost blocked with exudation. Products at the side of the bronchus away from the artery.

In other cases (though rarely) it is purulent. The walls of the pulmonary blood vessels also show thickening from a similar cell proliferation. The lymphatic channels and spaces are distended with variously shaped cells derived from these different sources (see figure).

These then are the normal appearances in the first stage of the disease when the irritation has not been of a very severe nature. If the case goes no further and if the products of exudation undergo a moist fatty degeneration, resolution may occur from absorption or evacuation by expectoration of the debris. In these cases due to the grip however, such a happy result was somewhat uncommon. Death frequently occurred as a result of implication of both lungs in the greater area of their surface. In these cases, haemorrhagic exudation was very frequent and the expectoration consisted of almost pure blood.

The most frequent course was in suppuration. Foci of suppuration arose in the midst of the affected lobules and gave the appearance of multiple abscesses. In very virulent types, the lobular & surrounding alveolar walls rapidly broke down and abscess cavities of varying size soon formed. Death frequently results when this occurs but in adults life is often prolonged into one or both of the subsequent stages.

The Second Stage
a Stage of Caseation.

In the affected lobules, the catarrhal cells accumulate and block up the alveoli. The blood vessels are pressed upon, the tissues are gradually rendered anaemic and dry, and fatty degeneration ensues. As Hamilton has pointed out, the further evolution of these products of fatty degeneration varies according to the amount of fluid present in the tissues. If the attack has been ~~an~~ acute ~~one~~, the fatty degeneration has been a moist one, and the debris is readily got rid of in this state. But if the onset has been of a subacute nature as is so frequent in the influenza forms, the

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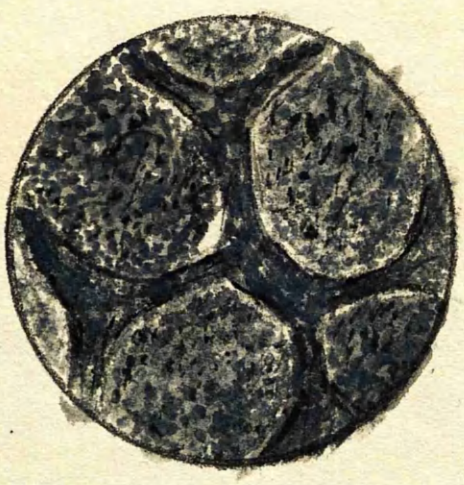
Naked eye appearances of portion of lung from Case III. Second stage well advanced. at p. a portion of thickened pleura is seen rough upon the surface where the adhesions were torn. The general pulmonary surface is highly anaemic; scarcely any trace of red colouring remains even around and between the nodules. The caseous and now fairly firm nodules are seen especially towards the pleural surface. Many have coalesced. Considerable quantities of pigment are distributed around and within (in some cases) the nodules. Two dilated bronchioles are seen at b. b. (Magnified twice.).

anaemia is brought about gradually, there is considerably less fluid effusion, the degeneration is a dry one and the debris dries in the alveoli and undergoes a further change — a caseous metamorphosis. The gross eye appearances of the lungs in this stage were well exemplified in ~~this stage~~ Cases II III IV and V although in these, evidences of the other two stages were also present.

In all cases pleural adhesions were discovered upon opening the thorax and in the region in which pain had been complained of during the second stages of the clinical history. In Case I. the adhesions were slight as the duration of the case was so short, but in the other three cases, they were so extensive as to almost obliterate the pleural cavity. The adhesions are fibrous but at this stage they are readily broken down. In Case II. I note "The lungs are adherent to the chest wall by recent and very general adhesions of a thin fibrous nature which are easily broken up except at the diaphragmatic surface of the right lung. The interlobar surfaces are also glued together but there is no fluid in either of the pleural cavities." "The lungs look thoroughly anaemic and pale especially at the edges and corners of the lobes where emphysematous bullae are remarked. On handling the lungs they do not give one the usual feeling, they seem ~~more~~ less crepitant than ~~usual~~ usual, to be heavy and here and there to give quite a solid impression to the grasp. The pleural surface of the lungs is irregular, and scattered over it are nodules in great numbers which vary in size from a split pea to a hazel nut. They are of a greyish yellow colour, their borders are sharply defined and the intervening tissue is considerably pigmented." On cutting into the lung, I note in case II. "Through the entire surface of the pulmonary section, there are thickly distributed yellowish white foci, apparently of the nature of areas of consolidation. They are especially evident in the basal and portion of the organ on account of the contrast in colour. To the touch, they feel like half boiled sago-grains and are in size, similar to these, though here and there several have run together and coalesced to form large masses of consolidation, perhaps as large



Case III.

Caseation of alveolar exudation and commencement of same in alveolar walls. Amorphous condition of tissues almost general throughout portion seen.

as a large unit. These nodules, especially the large ones, have a caseous look but in very many cases they are just undergoing liquefaction. Both lungs in fact seem to have remained in their upper two thirds in a state of grey hepatisation, while the lower portion had only, so far, got the length of red hepatisation or here carnisation as the French writers call it, but in addition, there were these yellowish grey nodules. They are largest and most frequent beneath the pleura where they tend mainly to a pyramidal form with the base lying upon the surface. In many cases a bronchiole is seen in their centre and these bronchioles seem so large for their situation that they are evidently dilated in a funicular manner. Here and there where the nodules are small and still separate from each other, they are seen to be scattered along either side of a bronchiole and its branches "like grapes upon a stalk". (See Sketch No I).
The nodules are often most numerous at the apices as in Case IV.
The lung tissue as a whole is firm and dry except where the caseous tissue has broken down and formed purulent cavities.
To such caseous nodules as these, Laennec gave the name "Crude Tubercle", but space does not admit of me dealing any further with such a very important and classical theme as the unity or duality of Phthisis.

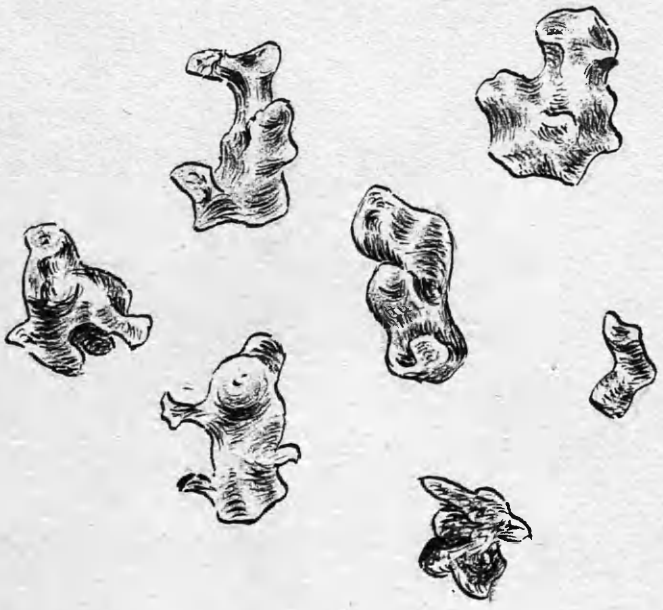
The pleura shows signs of commencing thickening — a process which is already evident in the interlobular septa as well. (see Plate I.)

On examining one of these nodules with the microscope it will be seen that each is made up of a group of air cells filled with solid constituents. The surrounding air cells may be quite healthy in appearance although they are generally considerably dilated and emphysematous, but in many cases an acute catarrh has arisen around the caseous centre. In the latter case the contents are loosely packed, are similar to those described in the first stage and may be mixed up with mucus and often with fibrin if the inflammation has been at all acute. Towards the centre of the lobule, the alveoli become more densely packed and the cells are

markedly fatty, and many of them have completely broken down. Fatty and albuminoid particles are mixed up with these cells, and give a highly granular appearance to the alveolar contents. "The "perivious blood vessels within this area are very much less numerous "than in the area more removed" * Farther in, the structural characters of cells and of alveolar walls begin to disappear. The alveolar contents are granular and amorphous, they completely fill up the cavity of the air cells and the adjacent walls are seen to be undergoing the same obscure changes as the cellular elements. The centre of the whole area becomes an amorphous dusky patch in which no trace of structure can be made out. This central portion refuses to take on staining agents at all or only very slightly, hence stained specimens give a good walled eye demonstration of these facts.

There is nothing special in this caseation. Some authorities affirm that it is always tubercular in nature but it is by no means so. It arises as Hamilton says "in any part of the body where the blood supply is gradually cut off from the part. In subacute catarrhal pneumonia, the conditions are eminently favourable for the termination of the products of exudation in a state of caseation. As we have already seen that the alveolar epithelium proliferates steadily and persistently until the cavity is filled up and distended; the capillaries are gradually obliterated by pressure from without, and a slow form of cell necrosis called caseation invades the alveolar contents in the first place and thereafter extends to the alveolar walls and capillaries. Hamilton aptly likens this process to the changes which go on in the "ripening of cheese". The same authority lays some stress upon another factor which he considers as playing an important role in the production of caseation. This is the irritable and virulent emph which is supposed to act in a dynamic manner. "In the first stage of the disease", he says "a considerable amount

* Hamilton - The Practitioner. loc. cit.



BRONCHO-LITHS.

FROM

CASE V.

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of fluid is contained within the catarrhal pneumonic secretion, which undoubtedly will be pressed out of the air vesicles when the patient coughs, while the solids will be left within the air vesicles in a more or less dry condition".

Such a dynamic process — such a compression, must have been the principal factor in the formation of the

Broncho-liths in Case V.

In this case upon post-mortem examination the lungs were found to have advanced to the third stage — that of disintegration in the upper portions and especially at the apices, while the middle & basal regions showed the first and second stages. In clearing out the gruinous contents of one of these cavities, I found three hard cretaceous particles lying loose in the cavity and on washing them, they were all seen to present similar characters. (See Sketches). The largest was scarcely 1/8 inch long. The colour was yellowish white and the shape was as I have attempted to picture. That is, each was knobbed and slightly branched. Each was a cast of the bronchiole and the immediate portions of its branches in which it had been formed. On clearing out the other cavities, one or more similar calculi were found in each. On washing sections of the tissues still in the second stage, several calculi were found in situ. Each lay in the lumen of a dilated bronchus which again occupied the centre of a caseous mass. One or two were still tightly grasped by the bronchial walls, and on picking them out, the little branches were seen corresponding with the knobs upon the calculi. In other places, the first stage in the formation of a cavity had commenced. The bronchial walls had broken down around the calculus as well as the surrounding caseous tissue to form a soft pulpy little mass. These cavities will be described later on, but here I may simply mention that they were formed by this breaking down of the degenerated tissues around a calculus, and never yet dilated bronchi.

These bronchial calculi have been rendered even more interesting

some months later,
to me by the fact of the publication of a monograph, entitled "Les pierres du poumon" [1]. In this thesis, Monsieur Poulalion divides all solid productions found in the lungs into three divisions according to their histological analysis: — I. Cartilaginous and pseudo-cartilaginous. II. Osseous. III. Calcareous; and again, he distinguishes them according to their site of production.

A. productions and metamorphoses formed in the tissues themselves of the bronchio-pleuro-pulmonary system and constituting what he calls parenchymatous concretions (pneumolithes). These may be cartilaginous, osseous or calcareous.

B. productions and metamorphoses found in the interior of normal or accidental cavities of the respiratory system, which are always of a calcareous nature and according to their texture, constitute homogeneous stony concretions or else calculi properly so-called. From his conclusions we may quote the following: —

"The caseous tubercle of bacillary origin (Koch) occupies the first place among all pulmonary lesions capable of undergoing a calcareous transformation. Most authors, even, only admit this form of calcareous concretion. But other non-tubercular pulmonary lesions can also undergo this change: such non-tubercular lesions as infarctions, bronchopneumonic nodules, miliary abscesses, etc.; the pseudo-tubercles of pneumonogenesis, cavitations and cysts, etc.; finally cysts and tumours of the lungs."

"The bronchiolithiasis may be accompanied by pulmonary tuberculosis, or exist with a complete absence of all tubercular lesions in the lung. In the latter case, it may adopt a chronic course, and give rise to a clinical picture, in all points like phthisis. We have given to these manifestations the name of pseudo-pulmonary phthisis of calculous origin (pseudo-phthisie pulmonaire d'origine calculeuse".

"In an anatomical point of view, this disease is characterised by the existence in the bronchio-pulmonary system, of irritative or ulcerative inflammatory lesions, dependent more or less directly upon the action of a calculus or of a cartilaginous, osseous or calcareous concretion,

set in the parenchyma or pure often free in the air passages; further, these different lesions exist in the absence of all other tubercular alteration of a bacillary nature."

" In the clinical point of view, the disease is characterised by functional and general symptoms and by physical signs simulating in all points the manifestations of tubercular phthisis properly so called, but differing in the absence in the sputa of the bacillus of Koch and in the existence of the bronchiolithic expectoration - the expulsion of calculi or concretions."

In the case under consideration, the calculi were either produced in a cavity, or in a bronchus. As we have seen, however, a few calculi were actually found in situ in a bronchiole and thus there could be no doubt about the mode of origin of these. Those found free in the cavities might develope in a different fashion. They may have been originally the result of infiltration of the pulmonary tissue with mineral salts - a condition favoured by the presence of a caseous exudation, and have become free owing to their setting up an ulceration of the surrounding tissue ending in cavity.
Or again, a cavity being formed in some other way, they may have been produced as the result of infiltration of a particle of inspissated mucus or pus with subsequent deposition of layer upon layer of mineral salts such as are seen so often in urinary calculi. Such a formation is frequent when we have pus and mucus lying, say, in a cavity communicating with a very small bronchus and cases of this nature have been reported by Andral[1], Rogée[2], Ball and Vie[3], Gubler[4], Pallalin[5], &c.

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1. Andral. Clinique Médicale vol IV. p139.
2. Rogée. Clinique Médicale, also "Essai sur la curabilité de la phthisie pulmonaire, ou recherches anatomo-pathologiques sur la transformation des tubercles et la cicatrisation des excavations tuberculeuses des poumons". Arch Génér. de médecine. 3rd. series. vol. V. 1834. [illegible].
3. B. Ball et A. Vie. "Concretions calcaires multiples, trouvées dans une excavation pulmonaire chez un tuberculeux." Soc de biologie. p135. 1858-59.
4. Gubler. In Charcot's Thèse d'agrég. Paris 1860. & Soc. méd. d. hôp. vol II. 2nd series. 1866.
5. Pallalin. loco citato. p. 161.

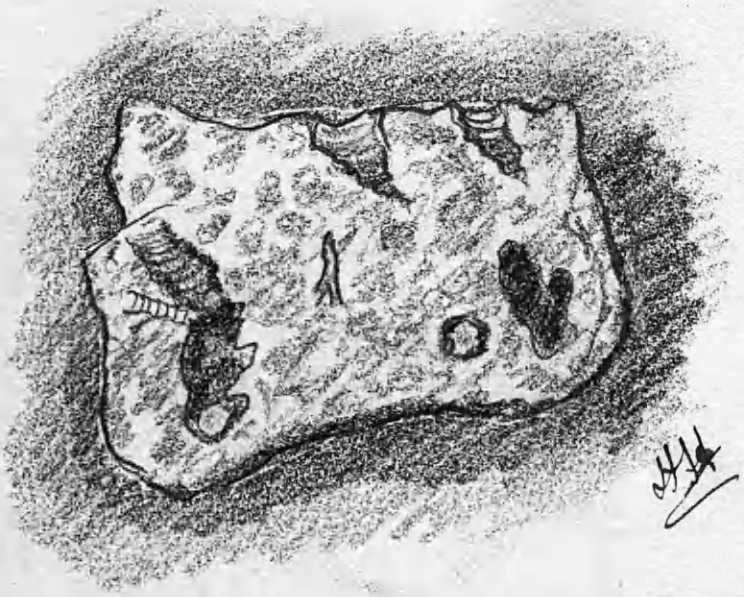

Figure 2. Topographie macroscopique des cavernules, des bronches dilatées et des calculs intra-cavernuleux. (Obs. A.M. Poulalion.) 1/1.

In a note descriptive of his sketch which I have copied, Pollalin writes:— "Just [illegible] from the cavity in the right lung, one sees another "concretion, but this is set in the tissues which surround the excavation (above described). This is a calcareous parenchymatous nodule. One understands that if the progress of the ulcerative destruction which forms these cavities, this concretion would have finished by projecting it into the cavity and there would easily have been set at liberty. It is this that we must explain the expectoration of parenchymatous concretions." 1

Analysis showed that these calculi consisted largely of phosphate and carbonate of lime with traces of organic matter. The analysis of similar calculi by Ball & Vie gives Phosphate of lime 72.75 %, Carbonate of lime 5.86, fatty matters and Cholesterine 4.18, organic matters 9.83 and the remainder of phosphate of soda, magnesia, etc.

In our own case — Case V. there can be but little doubt that they were entirely bronchial in their nature. These, and these alone, merit the name Broncholiths while to the nodules of parenchymatous origin the term Pneumonoliths more appropriately applies. They have been known to the profession since the days of Galen and since his time, Dodonée, Morgagni, Félix Plater, Salmuth, Dufour, Laennec, Hamilton, Guibourt, Vidal, Leclerc, Muscato, Jean de Wier, Fabrice de Hilden and others have reported cases of pulmonary calculi expectorated from the bronchi. 2

Bronchial calculi may be found in large bronchi, in the smaller tubules or in their dilated terminations in the infundibula. They are usually few in number as in Case V, although occasionally hundreds have been expectorated in a single case. Their shape is characteristic as they almost always are moulded according to the cavity which has served as their matrix. They are often branched like coral, but they may be more irregular in shape, rounded, or twisted with rounded knobs

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1. "Les Pierres du Poumon, de la Plèvre et des bronches et la Pseudo-phthisie Pulmonaire d'origine calculeuse." par S.A. Maurice Pollalin.
Thèse de doctorat de Paris. 1891.

2. See next page.

irregularly distributed over their surface (see Sketch). The surface may be smooth but often there are rough areas as well, or the whole calculus is rough and irregular. They are small and vary from the size of a millet seed to that of a pea, bean or even a filbert. They are usually denser than ordinary pulmonary or parenchymatous concretions, which are somewhat porous as a rule. The reverse may be the case however, in texture, they may be homogeneous as in our own case. But most commonly bronchial calculi have a nucleus of mucus, fibrine, blood corpuscles and around this nucleus, the mineral salts lie in concentric layers like a peritoneal urinary calculus. The structure of the nucleus may be made out with the microscope, upon dissolving away the mineral salts with weak acid. Chemical analysis agrees pretty much with that obtained from pulmonary concretions. Phosphate of lime predominates but carbonates and other salts are also present in variable proportions. In our own case, M. Hogg of Paris, who very kindly made the analysis for me, reports: — "I have examined "the concretions you found in a patient's bronchia after death. They "are principally formed of Cholesterine and contain a small pro- "portion of phosphate, carbonate and sulphate of lime."
Bronchial concretions of urate of soda in crystals have been reported

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1. Galeni opera. De locis affectis. Lib. IV Cap. 9.
2. Dodonée - Observ. Medicin. Cap 23. Apud Schenckium. De calculis pulmonum.
3. J. B. Morgagni. Epist. cit. Epist. XV § 19.
4. Félix Platter. Sepulchr. I. in Schol. Bonet. apud Morgagni.
5. Laennec. Traité de l'auscultation médiate, de la Ed. Vol II. p 311. 317 in note.
6. Dalmas - Journal hebdomadaire 1829. No 29. 18th April - p 65.
7. Dufour. Bull. Soc. anat. Paris. 1852. Vol 27. p 109.
8. Hamilton - Pulmonary concretion. Dublin Q. J. of Med. Sc. Vol XVII. 1854. p 446.
9. Guibourt. Bull. et mém. Soc. méd. des hôp. 2nd Series Vol II. 1865. p 66 seq.
10. Vidal. Bull. Soc. anat. Vol XXVIII 1853. p 88. Bull. Soc. med. hôp. 1866. VII.
11. Mazzati. Un raro caso di calcolosi bronchiale. Gazz. d'osp. Milano. 1884. Vol I. p. 1474 - 1482.
12. Jean de Wier. De praestigiis daemonum. lib. IV. cap 2. p. 353.

by Bence Jones[1] and also by Bernheim[2] in gouty subjects. These bronchial calculi which we have described at length, are always secondary to a pathological condition of the bronchial tube and its secretions. According to Laveran,[3] Hanot[4], Darenberg[5] and others, the expectoration in various bronchial catarrhs contains a large amount of phosphates, carbonates and even chlorides. Given such a composition of the sputum then, given in addition the presence of the purulent exudation with which it is mixed and which has undergone fatty and even caseous degeneration, and allow these to stagnate in dilated bronchioles and infundibula such as we have already demonstrated to be present in this disease, then what more suitable grounds could we have for the formation of bronchial calculi and especially as in our own case of calculi largely formed of cholesterine and organic matters. Particles of fibrine, of blood clot, degenerated cells of all sorts, are all ready to serve as nuclei for such broncholiths and we do not require the presence of such imported foreign bodies as cherry stones, grains of maize, wheat etc., which have been reported by various authors as the starting point of bronchial calculi.[6]

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1. Bence Jones. Pathol. Soc. Trans. & The Lancet 1856 p 98. also see Charcot's note to Gerrod's work upon Gout. French translation. Ollivier 1867 p. 275.
2. Bernheim. Soc. med. Strasbourg. 6th. June 1872.
3. Laveran. Académie de Méd. de Paris. 1863.
4. Hanot. Vide Article Phthisie. Dict. de méd. et de Chir. Pract. V & VII. 1874.
5. Darenberg. (G.) De l'expectoration dans la Phthisie Pulmonaire. Thèse de Paris 1876.
6. A.M. Eggedes. Ephemerid. med. phys. curios. decem. Bnn. X 1692 p 123.
   Stoethmann in Bull. Soc. med. Gand. 1862.

*. Cruveilhier (J.) Traité d'anat. pathol. général. 1852. Vol II. p 2143.
Mastichmat-Lagénard. Bull. soc. anat. Paris. XIV. 1839. p 303.
Masers. (Thèse Lede) Ann. Soc. méd. Gand. 1862.
All these except the case reported by Cruveilhier, which had a grain of maize for a nucleus, had a cherry stone as nucleus and all gave rise to symptoms closely simulating or identical to pulmonary phthisis.

The further history of these calculi depends upon many various conditions. That they may remain perfectly latent throughout life is a well known fact ~~among~~ those accustomed to perform many autopsies. This was apparently the fact in our own case.

Sometimes the calculus gives rise to a moderate amount of irritation around it, the connective tissue proliferates and finally the stone becomes encysted in the same manner in which aseptic foreign bodies undergo a similar process of inclusion in other parts of the body. This in fact may be designated as one of the methods of cure for while it thus lies dormant, it gives rise to no trouble.

On the other hand, the result may not be such a happy one, especially in calculi within cavities. The calculus may lie without giving rise to any trouble but no cyst wall forms around it. Some day, however, a septic infection occurs in and around this area of danger. A more or less acute and destructive inflammation arises around the calculus and according to the site and direction of progression of this disintegration of tissue will the concretion travel. If it pass into a bronchus, it will be expectorated giving rise to ~~purulent~~ fits of coughing and to severe pain as a rule — Bronchial colic of Pulmonalis, before it is finally got rid of. ##

It may travel through the pulmonary tissues and in its course it may perforate large vessels, giving rise to severe and even fatal haemorrhage, or it may perforate the pleura and set up a pneumo- or a hydropneumothorax.

Finally, its local irritant action it may ~~set up~~ produce large pulmonary abscesses or cavities as in our case V and remain at or about its original site. This is especially the case with pure parenchymatous concretions. These abscesses may burst into a bronchus however, and leaving giving rise to a sudden expectoration of a large quantity of purulent debris, may by its incursion into neighbouring air passages, ~~give~~ produce ## fresh tunnels pulmonary

Further cases of cure after expectoration of broncholiths.

21. Quantin, de Solnges. Soc. méd. des hôp. ~~P~~ March 28. 1890. Vol VII p 313.
22. Pieresne. Progrès médicale 1874 p 159. 174.
23. Montané. New York Med. Record. 1887. Vol 32. No 5. July 30 p. 146.

mischief.

In our case – Case I. these calculi remained absolutely latent so far as symptoms of bronchial colic were present during life. Their destructive effects were only seen post mortem.

When happily there have been only one or two such broncholiths, and when these have been evacuated by expectoration, a perfect cure has been the result in many cases. Such for example, are the cases reported by the under-noted authors.

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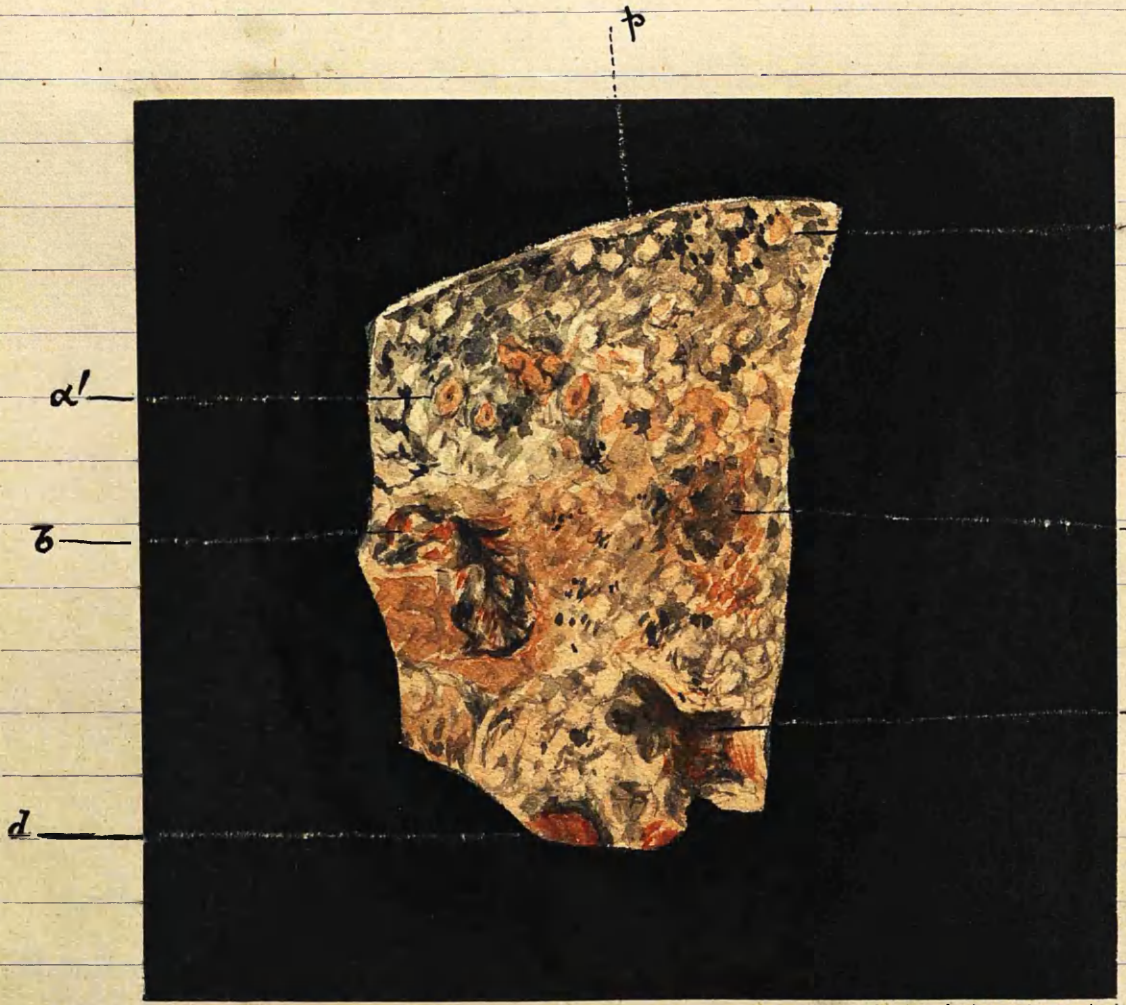
1. Alexander Trallianus. Lib. V. cap 4. apud Schenck.
2. Jean de Wier. loc. cit.
3. J. Schenck de Grafenberg. Obsev. med. Frankfurt 1600 Vol I. p 357. De calculis in pulmonibus.
4. Morton (Richard) Phthisiologia London 1689. De phthisin calculo in pulmonibus generatis.
5. Fuchs (Jacob). Miscell. D. I. Ann II Jena 1671.
6. Peechlin. John Nicol. Obsev. phys. med. Libri tres Hamburg. 1691. in Acta eruditorum. Lipsiae p 233.
7. Boerhaave (Hermann) Praelectiones acad. Edit. Haller Gottingae 1744 Vol. VI. §835 p 221.
8. Cullen. Opera.
9. Magagni. opuscul.
10. Coste. J.E. de Salins (Jura). La Clin. d. Hôp. et d. l. ville. Paris 1827.
11. W. H. Judd. loc. cit. 1838.
12. Duhamel. Gaz. des hôpit. 1844.
13. Forget. Union méd. 1854. Vol VIII p 97.
14. Laballery. Gaz. hôsp. Paris. 1862. Vol XXXV p 142-574.
15. Barth. Bull. Soc. méd. des hôp. 1865. Vol II. 2nd series. p. 9.
16. Guthrie. H. article Broncholiths in Dict. Encycl. 1866. Vol V p 658.
17. Schmit. Bull. soc. d. sc. méd. d. Grand-duchy of Luxemburg. 1867 p 121-23.
18. Vargas (A.M.) of Madrid. Communication to the first Congress upon Tuberculosis Case. 1. p 1461-62. 1888.
19. Fraenkel (A), Berlin. Klin. Wochenschrift. 11th March. 1889. p 192.
20. Moscato. Gazz. d. Osp. Milano. Vol I. p 473. 482.

such there is the history of most cases of pulmonary calculi. In our case, the broncholiths were secondary to a caseous bronchio-pneumonia and were probably instrumental in the production of the villous-walled vomicae present in the case. That they are not necessary for the production of every cavity in a lung is shown by a study of the other cases. In none of these were calculi found. Some special tendency in the system must have favored the deposition of these mineral salts, and as the man was a Cockney and was never a tea-totaller, one might readily imagine a gouty diathesis. There were no other signs of gout, however, in the case.

The Third Stage,
that of Phthisis or destruction of the lung.

At death, in all our fatal cases, the third stage had been arrived at, and in fact in adults in general, the disease seldom proves fatal in the stage of caseation. In children however, post mortem examination often reveals a pulmonary condition in which death has occurred without showing any signs of the destructive changes characteristic of the third stage. In adults, we generally find all three stages represented in the same lung. This was the rule in our cases and the order in rotation from above downwards was generally third stage at the apex, second in the middle and first at the base. To the naked eye, the appearance of a lung at this stage is somewhat as follows:—

The pleural adhesions which were easily separated in the former stage, are now firm and tough. Visceral and costal surface are indistinguishably united together and the whole pleural tissues are greatly thickened, especially over the area of a cavity. In case V, I noted that "the pleura



H. Hiphet. del. et pinxit.

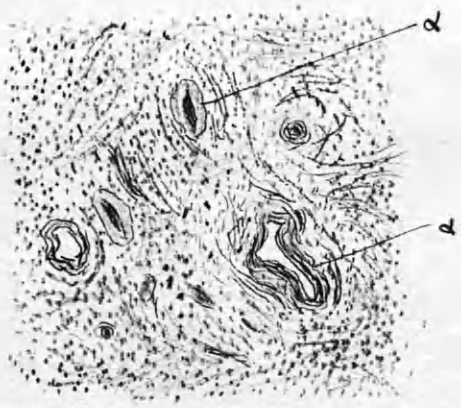
Water colour drawing of portion of lung in the second and third stages from Case V: twice the natural size.

a. Caseation of alveolar contents and walls, etc with considerable interlobular pigmentation. a' and a'' alveoli with intra-lobular pigmentation highly developed. b. dilated bronchus and cavity. c. cavity. d, area of healthy lung separated from the neighbouring tissue by a layer of pigmented fibrous tissue. p, thickened pleura, rough upon the surface where the adhesions have been torn asunder.

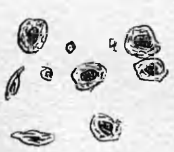
over the apices of both lungs is very much thickened. The visceral and parietal layers are firmly adherent and a tough, fibrinous capsule is the result - a capsule, which is glistening white upon the outer surface and which cuts with difficulty though it has not yet assumed the characters of "cartilaginous" such as has been described by other observers. This condition is confined to the upper lobe in each lung and does not extend beyond it." When the pleura is non-adherent, we may notice beneath its surface, numbers of small, grey, glistening nodules. These are tubercles which have appeared as secondary lesions in this stage. They are absent in the former stages.

The bronchial tubes when emptied of the large quantities of muco-purulent debris, show signs of bronchial catarrh of a somewhat chronic nature. Tubercles are rarely present in their mucous membrane.

"The apex of the lung as is known", says Hamilton, "is the situation in which softening and destruction of the lung tissue usually commences." This sentence is well exemplified in Cases III, IV, V and VI, in all of which cavities were present at the apices and absent at the bases and even midregions of the lungs. In case VII a large basal cavity was present in the left lung. At the apices in this case, the second stage had not been passed. As I note in Case IV. "These cavities are more frequent at the apex and at the anterior part of the upper lobe, especially at its anterior edge." In Case V the report states that "the apex presents two large cavities, each 1½ inch long, freely communicating with one another, but not with any large bronchial tube. The pleura over has almost penetrated the lung tissue. Its outer and posterior wall is formed of a thin layer of thickened fibrinous, dark, greyish lung, less than ¼ inch thick, and firmly adherent to the thickened pleura. This cavity is rather over 1½ inch long in its greatest diameter — an obliquely horizontal one. The walls are fairly smooth, but seem to be formed of caseous lung tissue, undergoing a process of disintegration. The contents are a grumous purulent, brownish-yellow mass of debris. There are other small cavities in this lobe



Case V Thickened inter-lobular septum. a. arteries with thickened intima. The highly cellular nature of the fibrous tissue is well seen. (low power.



Epithelioid Cells in above.
High Power.

as well, which are remarkable in that they contain not only gummous pyogenic debris, but generally one or more hard cretaceous masses. In a cavity one inch long, four of these were found. Between these cavities and the greyish yellow areas of caseation the lung presents a pale pink tint, but the green and grey tints predominate and there is also a considerable quantity of pigment scattered about. Passing from the pleural surface in the upper lobes, there are frequent bands of fibrous tissue of a greyish-white, translucent appearance upon section. They cut firmly to the knife although one could not say that they crumble upon section or that they are quite of a cartilaginous consistency yet. They pass between nodules and cavities towards the root of the lung. Many of them are fully ½ inch thick. At their origin from the thickened pleura, there is neither depression nor cicatricial puckering. They are present even low down in the lobe where the pleura seems almost normal." According to Hamilton, it is these which go often from the trabeculae follow even traversing cavities, and not the vessels of the lung tissue dissected out of the dissolution of the surrounding lung parenchyma."

My own microscopical experience confirms this view.

"When they are microscopically examined their structure is seen to "be that of dense masses of fibrous tissue, with a few blood-vessels of "ordinary size contained within them, and when their course and "distribution are studied, there cannot be any difficulty in under- "standing what they are. They are simply the interlobular septa "of the organ, now much thickened, which, of having more power "of resisting the process of destruction going on around, have "been left in an isolated condition when the infiltrated lung- "tissue became ~~dead~~ disintegrated. They are abundantly supplied "with vessels, and hence, probably, it is that they are less easily "destroyed than the infiltrated lung-parenchyma, in which "the blood supply has been gradually although completely cut off."[1]

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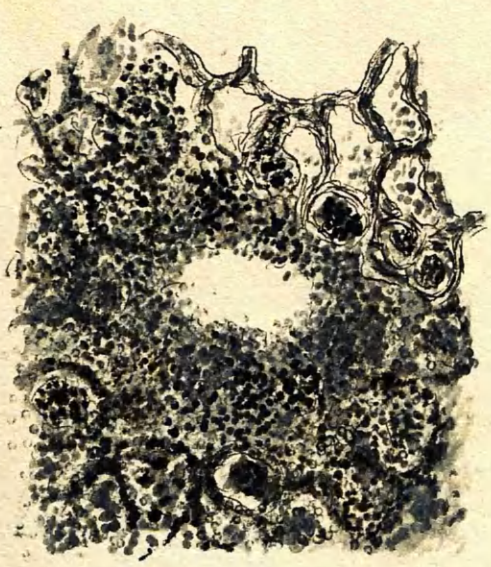
[1] Hamilton. loc. cit. Vol XXV. 1888 p. 341.



Case VI.

Caseous centre with cavity.

The bronchi are not markedly dilated but the small bronchioles show very frequent fusiform enlargements. Herein lies the difference between chronic lobar and chronic lobular pneumonia. In the former, large bronchial dilatations with bronchiectatic cavities may be seen but the bronchioles are normal.

"The bronchial glands are enlarged, adherent to one another and very deeply pigmented." (Case V). Abscesses are often present in these glands but microscopical examination shows a total absence of tubercle.

How do these cavities form?

In the centre of these caseous masses in the course of time, a series of chemical changes take place and end in liquefaction of the dead tissue. This spreads throughout the affected area and neighbouring cavities may coalesce through the breaking down of intervening areas of tissue. In some cases, this destructive process is very rapid, as in Cases III, V and VI, and is then due or greatly encouraged by the associated obliteration of the blood-vessels. An endarteritis obliterans effectually blocks the lumen of the tubes. Occasionally, this destructive process is hopefully limited by those thick bands of fibrous tissue which have been already described. In these cases if the discharge is removed of an opening into a bronchial tube, the walls of the cavity may become smooth and fibrous, closely simulating a true bronchiectatic cavity, and further mischief is greatly limited. When this has taken place largely throughout a lobe, the walled up appearances show considerable cicatricial contraction of this lobe. In Cases III, IV and V cases, with the comparatively short duration that they had, (III and IV lasted but 60 days from the onset of the pain), I note that the lung is often of a pale slaty grey colour. In Case IV, especially, "the upper lobe is of a slate grey colour, and is marked off from the lower by a layer of thickened pleura of a deep greyish pink tint, which glues the two lobes firmly together. The upper lobe is honeycombed by six large cavities full of sero-pus and lung debris." In Case VI, again, I note:— "The lung tissue proper of each upper lobe presents a remarkable appearance. It does not

partake of the nature of lung tissue at all; all elasticity seems to have gone, the tissue is firm and solid to the touch, sinks in water, and does not contain much moisture beyond the purulent juice which flows into the cavities and bronchial tubes."

In conclusion, I cannot better sum up than by giving the following points in Hamilton's synopsis:—

" 1. Croupous pneumonia and catarrhal pneumonia are two totally " different diseases, and ought not to have a common designation. Croupous " pneumonia is merely an exudation of blood constituents due to suddenly " increased blood-pressure, while catarrhal pneumonia essentially " consists in the over-stimulation and proliferation of the epithelial " cells lining the alveolar walls.

" 2. Catarrhal pneumonia runs through three distinct stages—

" (a) the acute or putaceé

" (b) the caseous

" (c) the excavating.

" The disease usually passes through all three in adults, but in " children frequently proves fatal in the first or second.

" 3. In the acute stage the alveolar epithelium proliferates, " and the cellular products derived from it accumulate in the " air-vesicles.

" 4. In the second stage these dry and become caseous, " thereby loosing their characteristic form.

" 5. In the excavating or third stage the necrotic caseous " material softens, and this softening is a purely chemical process, " corresponding to the "ripening" of cheese.

" 13. There is a form of catarrhal pneumonia in which the " nodules are of small size, and are widely disseminated throughout the organ. It closely resembles tubercle in its pulled-eye " characters, but it is totally different from it in its actual " structure and mode of development." [1]

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1 Hamilton. loc. cit.

Condition of the other organs.

Heart. The muscle is usually soft and pale and the left ventricle slightly dilated as in Cases III. IV + VI. In all, large white thrombi were found in the left ventricle and the right ventricle, great veins and pulmonary arteries all contained large quantities of venous clot. In Case V, the endocardium of the left heart was considerably mottled. In Case VII, the right ventricle was somewhat dilated as is frequently the fact in such long continued cases. In all, the valves were competent and perfectly free from disease. weight in Case V 345 grams.

Blood vessels. In the lung, the thickening of the intima has been already noted. Considerable and general venous engorgement was present in all these cases and more especially so in the subacute forms.

In Case IV. thrombosis of the left iliac and femoral vein occurred toward the end of the case and was probably of a marasmatic origin. It was a simple clotting without disease being apparent in the vessel walls.

Liver. In all these cases, this organ was enlarged and deeply congested so that the "nutmeg" appearance was always well marked. The microscope shows the condition very well.

Average normal weight = 1 Kilo - 500 grammes.

In case IV = 1 Kilo 710 grammes. In Case V = 1 Kilo 980 gram.

Case VII = 1 Kilo 825. These weights in conditions of advanced anaemia and emaciation, are quite convincing.

Spleen. Enlarged, soft, diffluent with great venous engorgement.

Normal Weight 112 to 280 grammes.

Case IV = 200 gram. Case VII 1160 grammes: Others not weighed.

Kidneys - Venous engorgement well marked as a rule.

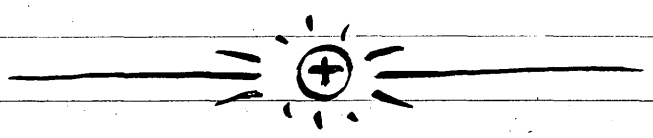
Peritoneum. In case III, the whole abdominal organs were glued together by thin, membranous, recent adhesions, which were easily torn up.

Bowels. Same as well as in the peritoneum, the general venous engorgement was very evident.

Brain: venous engorgement here and slight basal meningitis

in case III. In some of these cases, were fine abscess such as has been described by other observers as a fairly frequent complication.

The usual complications of all these organs are dealt with in a future chapter.



Chapter V.

The disease: the acute and subacute forms: the acute suffocative type.

The Disease.

The Bronchiopneumonia of influenza may arise either in an acute or in a subacute manner. In its course, it may prove acutely fatal. Few cases recover very rapidly. The general course is a subacute one, while in a fairly large number of cases, the disease passes into a chronic condition and ends very slowly in recovery, or as is more often the case, in death.

The Acute Form.

Before going on to describe the true inflammatory form of Acute Bronchiopneumonia which is purely a complication and not an actual entity of the Grip, a few remarks may be made upon that form of the disease which I regard as primary, and which occurs during the height of the attack and which is probably of a neurotic origin. The neurotic nature of the primary changes

in the lungs has been already discussed in the chapter upon the Pathology of the disease. In some cases, no further changes result and a rapid recovery follows: in these, however, inflammatory conditions supervene which, in proportion to their intensity, area of distribution and duration, lead to various ultimate forms of the disease.

This nervous type which we might call the Primary or Concurrent form, is generally of a very flitting character. Owing to the fact that inflammatory changes are absent or are only very slight, a return to the normal seems in a truly astonishing fashion.

Upon physical examination, one or a few limited patches are found upon the posterior surface of the lungs as a rule and especially towards the bases, and these present a crepitant or sub-crepitant rale, which, as the respiratory murmur is usually weakened as well is heard only to best advantage upon prolonged deep respirations. These rales are followed in a few hours by somewhat diminished percussion resonance though usually not so intense as to amount to actual dulness. In these cases, the temperature may rise to 101°F, rarely higher, the respirations are often quickened out of all proportion to the pulse rate. This was noticed in many cases and markedly so in one of my own patients — a man, aged 38 who had a pulse of 120 and a respiration rate of 22. The cough is often excessively troublesome and may be accompanied with only a very slight mucous expectoration. The nature of the cough quite bears out its irritative character. In a case already referred to at page 27 viz a young woman of 24 years, besides a very irritable and persistent laryngeal cough, frequent attacks of asthma were a troublesome feature which commenced at an early period of the disease. In the young and healthy adult, these conditions disappear rapidly with energetic treatment, and in fortyeight hours or so, the lung may be free from all abnormal signs. The cough and the asthma disappear more gradually however. In the aged, a favourable issue seems more slowly and in some cases not at all. In unfavourable cases, the neurotic irritation passes

on into a state of paralysis and leads to a condition of congestive oedema of the lungs which rapidly terminates in suffocation or in cardiac failure. This mode of termination will be referred to further on in association with the inflammatory forms of the affection.

The Acute Suffocative Type.

This form is traceable to a distinct chill during the course of the grip or during the immediate state of convalescence. After the patient has been very warm, for example, and is perspiring freely (a state of matters seen so very frequently after influenza, and probably due to vaso-motor disturbances) he is chilled as in Case II, when the patient sat down in a railway carriage opposite an open window when in a state of great warmth.

Soon he begins to feel cold, shivers, and may even suffer from typical rigors. In a few hours, a feeling of tightness and oppression arises in the chest — a sensation most complained of beneath the sternum, where pain of a peculiar heavy nature is also experienced in many cases. Difficulty in breathing soon makes itself manifest, and ere long this becomes much exaggerated, so much so in many cases that it develops into orthopnoea. The patient cannot sleep on going to bed, but has to sit up with the shoulders thrown forwards, and perhaps even with the hands grasping the knees in the struggle for breath. The voice becomes hoarse, the throat irritable and a very hacking cough becomes a most troublesome feature in the case. The cough is especially troublesome when the patient tries to assume the recumbent posture. The sufferer feels a difficulty in speaking aloud not only on account of the dyspnoea and the laryngeal hoarseness, but also because of the strange feeling of constriction in the chest. In this respect, I may be pardoned for giving my own personal experience of such an attack though rather a milder one that what has just been described.

I was unable to sleep. There was severe frontal and occipital

headache of a continuous throbbing nature. The pharynx, fauces and palette were red and inflamed, the tonsils were of a deep purple and were swollen somewhat, the nose felt stopped, and altogether, I had the feeling as if the mucous membranes of the nose, pharynx, larynx, trachea and bronchi were swollen and dry. The feeling of constriction was general throughout these parts. The chest felt tight as if held in a vice and there was a peculiar substernal feeling of oppression and of dull pain. The vocal sounds seemed to be thrill and of a higher pitch than usual. The normal sensation of chest resonance of which one is conscious when speaking, was gone, the sounds seemed to be thrown back upon one, and one could hear them but indistinctly. The swelling of the mucous membrane seemed to have extended to the Eustachian tubes, and for a fortnight after convalescence had been established, I still experienced a peculiar feeling of obstruction in the ears with slight deafness and tinnitus aurium, and occasional giddiness even. Like the cases above described, I also suffered from severe abdominal pain, located to the hypogastric and right iliac region, accompanied with ~~but~~ tympanitis though without diarrhoea.

The cough is generally dry and hacking especially upon lying down flat in bed. This dry at first and it is only after the first 24 or 48 hours have been passed that a slight mucous sputum begins to appear. Soon, however, this becomes more abundant, changes its characters, becomes more fluid, contains pus and often streaks of blood. Occasionally there may ensue, a regular bronchorrhoea when the sputum consists of a thin watery mucous slightly tinged with blood, and containing floating about upon its somewhat frothy surface, large rounded masses of mucopus. In other cases the expectoration remains very thick and viscid and clings to the side of the receptacle. The quantity of blood present, varies from only a slight streak here and there to an actual expectoration of pure blood as in cases cited by Bouchut and who also mentions a case in which pure - juice sputum was present from the first few

days of the disease until death. The presence of such prune juice sputa is always a sign of great gravity, an opinion also expressed in many of the previous epidemics, and already quoted in the historical portion of this paper. The stitch in the side, so general a symptom in acute pneumonia, fails as a rule in this affection. In nine of my cases was it present until late on in the subacute or chronic stage, although it has been reported by some observers to have been occasionally of an unusually acute nature. Frequent and profuse perspirations are complained of upon the slightest exertion, often when quite at rest in bed. The pulse rate is accelerated ~~even~~ but not in direct proportion to that of the respirations. A pneumonic ratio results and figures such as 1 to 2·3, 1 to 2·5, 1 to 3·2 etc., are obtained. The temperature rises to 101, or 102 or even as high as 102·8 in adults as it did in my case. The acme is not accomplished until a few days are passed while in very unfavourable cases, a temperature of 104 or 105 may be registered before the crisis or just preceding death. The urine is muddy from the presence of urates, and there may be a trace of albumen. When diarrhoea is absent, the bowels tend to be bound.

On physical examination of the chest

the percussion note may be quite unchanged. In the majority of cases, however, I have found slight dulness at the bases, or a dull tympanitic note, either of which may extend to the upper portions of the lungs both and front when the pulmonary implication is widespread. In one case (Case II) the whole lung back and front gave a dull tympanitic note to the finger. Marked dulness is absent at this early stage of the disease. The respiratory murmur tends to be harsh at the upper portions of the lungs. Lower down it becomes weaker and is generally accompanied, ~~by~~, if not masked, by various adventitious sounds or rales. Sibilous, sonorous rales of a dry character are evident in front and back above, while towards the bases, they become weaker, minuter and scarcer. Here crepitant sounds of various kinds are heard. A frequent occurrence is to have a sibilous fairly dry rale in inspiration with a subcrepitant rale towards the end of expiration.

The crepitant rale is distinctly minor as a rule than the typical pneumonic rale. It is more of the nature of the crepitus redux although one often hears it in both phases of respiration. If it happens to be present during inspiration, a fairly frequent occurrence, it is preceded by a piping rale, and only develops towards the end of inspiration to become more frequent and pronounced in the expiratory portion of the respiratory murmur.

The voice sounds are as a rule, but little altered. At the base, they are heard well and resonantly with quite an egophonic distinctness, but generally there is no difference upon the two sides at this stage.

Such is the state of affairs during the first few days of this form of the disease. The further evolution may present any of the following variations.

Death may occur rapidly from collapse, from suffocation, or from an actual condition of septicaemia the cause of the Bronchio-pneumonia as has been described by Burlureaux among other observers. M. le Prof. Peter [illegible] lecturing upon the pulmonary complications of grippe, continues thus with regard to these grave forms of Bronchio-pneumonia: — "A form which I consider to be still "graver than Pneumonia, and which I deem inevitable, is general "pulmonary congestion. It arises in individuals whose organism has "been already in an altered state. This the suffocative catarrh of the "ancients and the patients truly suffocate. One observes in the upper "parts of the chest sonorous and sibilant rales, and in the lower parts, "subcrepitant rales. The pulse is small, frequent and poor, but the "temperature is not generally altered. The expectoration takes on the "aspect of a bloody serum. It is a sort of salivation of the bronchi "mixed with blood which escapes on account of the enormous congestion "of the lungs. It is especially dangerous in the emphysematous, the "diabetic, alcoholic etc."

Peter. Pulmonary Complications of Grippe. Clin. Méd. de l'Hôp. Necker. published in "La France Médicale" Jan.-Mar. 1890. p. 52.

DISEASE.

Notes of Case.

Name Case I

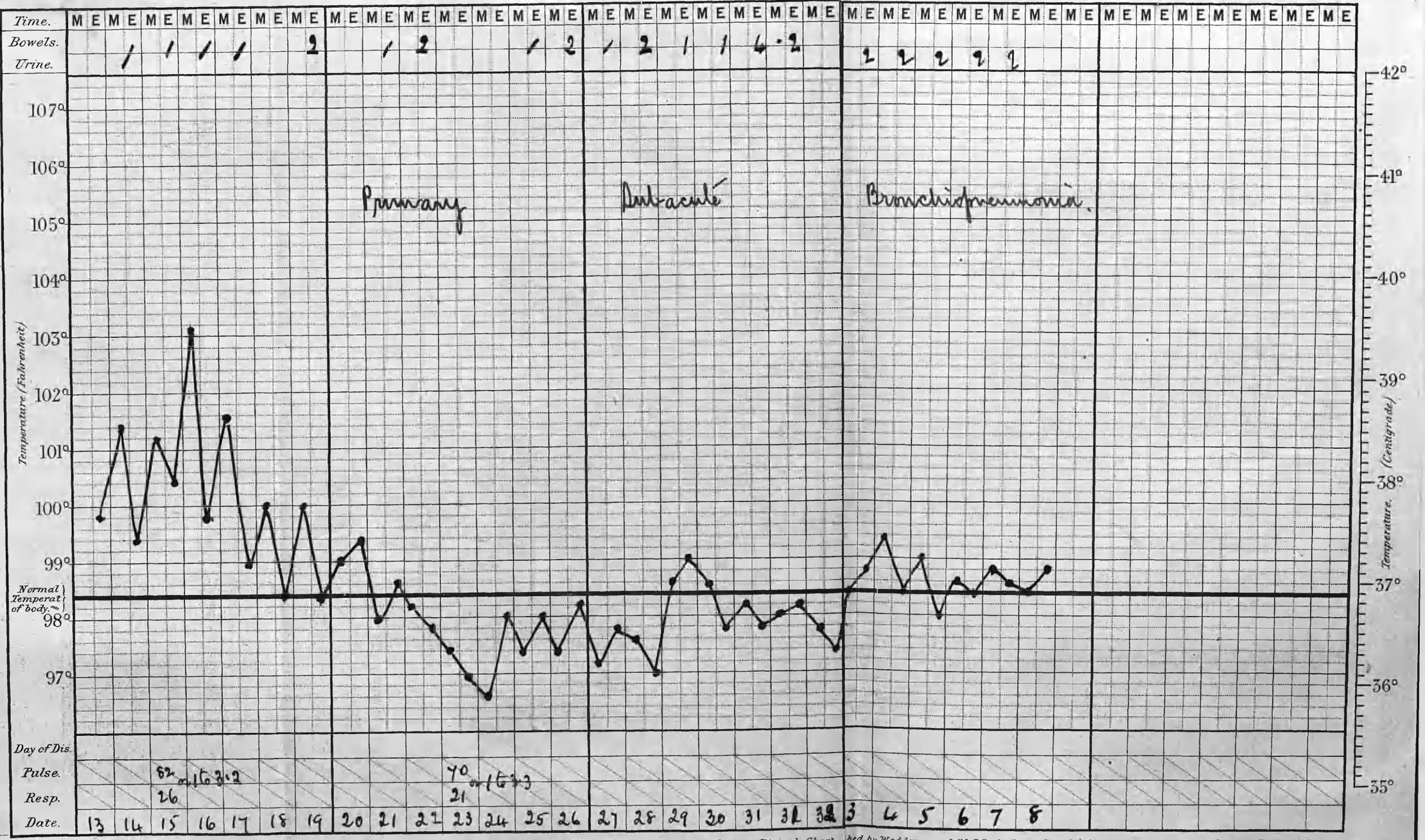
Age

Diet

Case Book No.

Date of admission.
October 13

Result



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hed by Wodderspoon & Co. 7 Serle Street, Lincoln's Inn. Gould's Clinical Chart.

Resolution may now commence, and the case ends favourably in fifteen to twenty days or so, leaving the patient, however, in a state of great general debility. The fall of temperature takes place by a gradual lysis and seems to be almost hectic in type for a few days, becoming accompanied by perspirations and often by diarrhoea.

In other cases, however, complications may arise during the course of the disease, such as acute pneumonia. Case I illustrates this point.

History of Case I.

The patient, a coachman aged 32 was admitted to the Hertford British Hospital, Paris, suffering from acute suffocative Catarrh following upon influenza. A fortnight before admission, after riding very hard and perspiring freely, he received a chill when working in the stables. The same evening, he complained of a great feeling of oppression in the chest especially beneath the sternum and was very breathless especially upon exerting himself. There was no shivering although he felt cold, nor did he suffer from any gastric symptoms with the exception of loss of appetite. He soon began to cough, and the cough was of a severe hacking nature unaccompanied by any ~~sputum~~ expectoration for the first few days when it appeared thick and yellow with slight streaks of blood in it. Two days after the onset of the cough, he began to perspire and has done so profusely ever since. He never complained of any stitch in the side.
On admission the stethoscope revealed abundant dry sonorous fremitus rales accompanied by large bubbling sounds above, and by sub-crepitant rales at both bases. Percussion was dull tympanitic all over and there was no actual dulness anywhere. The R. Lung was hard in front and there was evidently considerable emphysema. The temperature was 99·8 upon admission.

Everything seemed to be going on well until one night the temperature began to rise and upon the following morning – the 18th Feb, four days after admission, the thermometer registered 103·2. There was marked otherpnoea, the respirations were 26 and the pulse 82, giving a ratio of 1 to 3·2. At the right base, percussion gave a dull note, the

voice sounds were exaggerated and there were abundant crepitant rales of a somewhat moist character, but without any tubularity of respiration. The sputum which formerly had been of a muco-purulent liquid character, was now viscid, frothy and slightly rusty. A fly blister was applied to the base and a mixture containing Ipecacuanha, Iodide of Potash and the Tr. Jin of the Acetate of Ammonium was given freely. The same evening the thermometer registered 99.8. The consolidation disappeared steadily day by day, with marked morning and evening remissions (see chart), and he left the hospital well, three weeks after the onset of the pneumonic complication.

Similar cases are reported by Dr. Mitchell Bruce and others. Burlureaux lays great stress upon the fact of the absence of the "tubular" murmur from the areas of consolidation, thus showing their distinction from ordinary pneumonia.

Finally, many cases pass into the subacute or chronic class. This is especially the case in debilitated patients, in those who have not taken sufficient care of the initial symptoms, or in those whose lungs are already disabled by Chronic Bronchitis, Emphysema, heart disease or tuberculosis.

Both Cases I and II, were sufferers from Chronic Bronchitis and Emphysema. In both, did the same disease develope and in the same manner, but the difference in the course of the Bronchio-pneumonia may have depended upon the fact that Case I was in a man, and that he remained in bed from the onset of his illness whereas Case II, a woman, went about at her usual work of teaching, travelling about Paris and its environs and exposed to all the changes of the very variable Parisian climate.

The history of Case II

is as follows:— Annie H. aged 31. Governess. was admitted to the Hertford British Hospital, Paris, upon the 12th Oct. 1891. The patient states that a fortnight before admission, she suffered from influenza when she complained chiefly of general pains throughout the body, of giddiness, headache, sickness and diarrhoea with griping

pains in the abdomen. Pain, chiefly confined to the top of the head was excessive and was of a very oppressive character. She continued to follow her usual occupation — that of a teacher, and a week after the onset of the first symptoms, she received a chill after sitting down in a railway train, after in a state of great warmth and perspiration. The same night, she complained of a feeling of cold and of shivering. There was also a sensation of stiffness and of oppression in the chest and of dyspnoea to such an extent as to force her to sit up in bed all night. The voice became hoarse in a few hours and a dry barking cough of a persistent hacking nature set in. In a day or two she began to spit — mucous at first and soon purulent matter, but never any blood. There never was any stitch in the side but a feeling of soreness and oppression beneath the sternum was a constant symptom. On the least exertion, cold perspirations broke out over her body.

She has suffered from attacks of bronchitis every winter for a number of years, otherwise her past history is good.

On admission she complained of cough, spit, breathlessness and great weakness. The physiognomy was quite similar to that described later on in other cases. She had the pinco earthy pale of the complexion, a look of anxiety, a slight condition of cyanosis, an appearance of dyspnoea while drops of perspiration thickly decorated her face and forehead although she owned that she did not feel "sweaty". Physical Examination revealed a dull tympanitic note all over the chest, head and front except at the bases where the dulness became more distinct although not to any marked degree.

The R. Ax. is marked by abundant subcrepitant rales heard during both phases of respiration and taking on a loud bubbling character in the region of the large bronchi. The Voice sounds are heard equally well and sharply all over the chest. The heart sounds are very weak. Spleen and liver seem normal. The urine contains a trace of albumen. The sputum consists of rounded bloody purulent bodies floating in an aerated mucous juice which is stained pink from admixture with blood. The cough is continuous and hacking and

DISEASE..

Notes of Case.

Name Case II

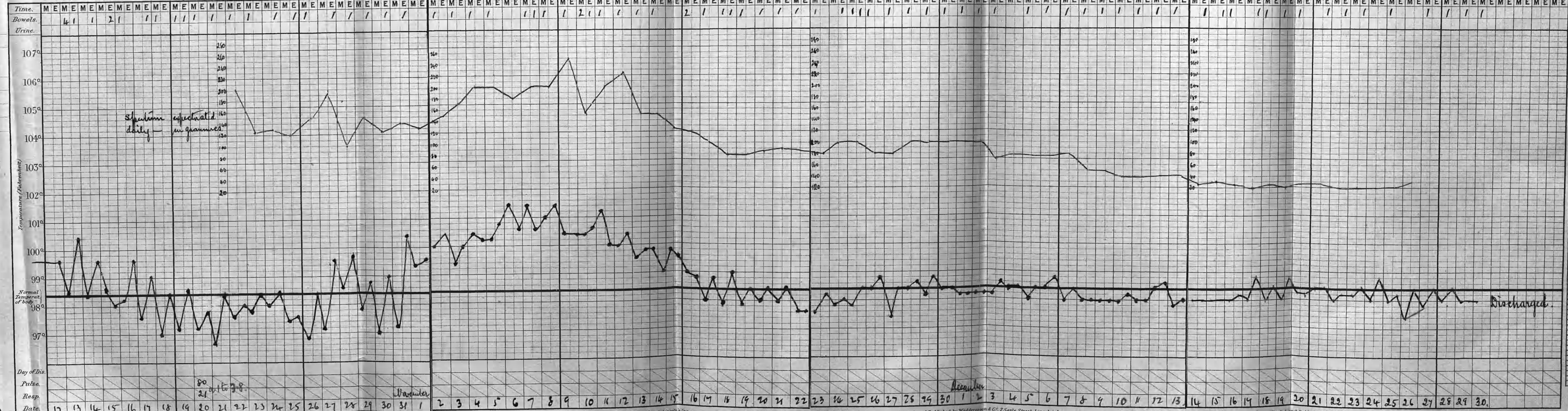
Age

Diet

Case Book N°

Date of admission. October 12

Result



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gives rise to great substernal pain. temp. 99.6 on evening of admission.

October 21. The temperature which has been gradually falling since admission, presents daily remissions of at least one degree. The chest conditions remain much about the same however.

Nov. 24th. At the right base and at the middle third of the right lung, the rales are moister than before and a similar condition is present at the middle third of the left side. Elsewhere, the loud rales have almost disappeared, although the R. In. remains harsh. The percussion note ~~was~~ now partakes of the true emphysematous character throughout.

Dec. 1st. At the bases, the rales are much drier now. In inspiration, there is a frequent fibroy rale with a dry crackle at the end of the respiration, while in expiration the bronchial sounds tend to be sonorous. At the mid region upon the right side there is slight pleural friction. At the scapular region upon both sides, the percussion note has almost a cracked-pot sound, and here, bronchophony and whispering pectoriloquy are quite evident upon either side. There are no rales however but the R. In. is blowing, tubular and almost amphoric. At the base, the voice sounds are loud and clear. At the upper portions of the lungs behind, there are a few dry bronchial rales and here as well as in front, the R. In. is loud and rather exaggerated, though free from rales in front. The sputum was noted upon Nov. 24th. to be composed of a watery mucus, very slightly tinged with blood and containing irregularly shaped masses of pus. Today it is more tenacious and more frothy and there is scarcely any tinge of blood. As will be seen from the chart, the quantities have fallen from 200 grammes daily upon the 29th Oct. when the first measurement was made until now it amounts to 75 grammes.

Dec. 21. There are no rales of any sort today. The R. In. is tubular and almost cavernous at both interscapular regions where also bronchophony and whispering pectoriloquy are still very evident. Patient still complains of very slight substernal pain upon coughing. The sputum is mainly composed of ~~very slight~~ a frothy mucous slightly tinged

with pus and mne slightly still with blood.
Dec. 30. Today patient left the hospital feeling able for work. For the last seventeen days the sputum has varied between 20 and 25 grammes per day. (See Chart)

The Subacute Form.

This arises in a subacute, less often in a chronic manner and is the form in the great majority of cases in which the Bronchio-pneumonia of Influenza manifests itself. In most cases, it arises upon a pre-existing Bronchitis and may therefore be truly called — a Secondary Type, but that is not always its mode of origin. I have however shown by case VI. - here, there were no pulmonary signs or symptoms on admission to the hospital, but these developed under observation in a subacute — may almost in an acute manner.

The following is the history of a typical case of the subacute form. The patient is suffering from Bronchitis, which may or may not be giving cause for anxiety, when, after, or even without any exposure to cold, an exaggeration of the respiratory symptoms supervenes and the physical signs undergo great alteration.
In only a few cases are there marked rigors, but the temperature is found to have risen, it may be as high as 104°F in the evening, although as a rule, the morning reading is one or nine degrees lower. This variation between the morning and evening temperature occurs from the onset, and lasts throughout the course of the disease, and is an important factor in the diagnosis as now also remarked in describing the acute type. Symptoms of constitutional disturbance vary in intensity with the individual and in proportion to the gravity of the case. Headache, malaise, sickness, want of appetite, vague pains throughout

the bowels constipation or even slight diarrhoea, ~~usually~~ precede the pulmonary trouble. Symptoms of respiratory affection may form a slight increase of cough and expectoration, ~~to~~ mottled cyanosis and lividity, with rapid laboured breathing. Pleuritic stitch, so frequent in acute classical pneumonia, usually fails here. It is rarely well marked, and in a few cases where stitch in the side has been severe, one is led to regard it, from its character, as a real pleurodynia and not as a result of pleurisy. The pulse rate rises rapidly to as high as 140 or 160 and the respirations to 30 or 40 and in very bad cases to even 60 or 80. The ratio becomes strictly pneumonic, and such figures as the following, quoted from W. Mitchell Bruce's paper, are obtained:—

$\frac{96}{32}$ or $\frac{2 \cdot 75}{1}$, $\frac{150}{54}$ or $\frac{2 \cdot 7}{1}$, $\frac{128}{48}$ or $\frac{2 \cdot 6}{1}$, $\frac{112}{48}$ or $\frac{2 \cdot 3}{1}$ -

Cough becomes very troublesome, and is of a severe hacking nature, in many cases giving rise to great substernal pain. Sensations of tightness and oppression in the chest, similar to those described under the acute suffocative type, are also frequently complained of by sufferers from this form of the disease.

Upon Physical Examination of the chest at this early stage, the percussion note is often unchanged. In many cases however, there is already a tendency to dulness at both bases, more frequently than at one only. The dulness does not usually extend above the angle of the scapula, and diminishes in a quadrant manner from base towards summit. There is no appreciable limit between dull and clear area such as one meets with in acute pneumonia and blending with effusion. The vocal vibrations depart very little from the normal, although in some cases they are slightly increased, and occasionally I have found the sounds to present an almost bronchophonic character in the absence even of distinct dulness. In place of a dull note, the

Mitchell Bruce. Influenza & its Complications. Lancet May 30. 1891. p 1143.

initial stage may give a heightened note of a dull tympanitic character present at all portions of the chest. Over the upper two-thirds of both lungs bronchial rales are present and these may run a fine or sometimes dry, hissing to organ pipe sounds of a moist character accompanied by loud bubbling rales. At the bases the respiratory murmur tends to be weak, & is rarely tubular and is accompanied by fine crepitant or sub-crepitant rales, heard during both phases of respiration. The expectoration varies in character. It may be at the commencement a viscid mucous, may vary in tenacity and age from the presence of blood, or there may be blood in varying amount from a slight occasional streak to a typical rusty tint, and in very dangerous cases, it often becomes quite frank pus.
It may in other cases, present the characters of a purely mucous which rapidly becomes purulent, such as we found in Case II where the report states that "the mass in the vessel is composed of rounded blood stained purulent bodies, floating in an acutely mucous fluid which presents a fairly uniform pinkish tint due to the presence of blood pigment." This is a most frequent appearance of the sputum. The further evolution of the disease may now proceed along one of ~~the~~ four courses.

(i) Resolution may now take place and the case ends favorably in a manner quite similar to that in which case II recovered. The striking features in such cases are the gradual resolution, the fall of the temperature by a lytic lysis, the slow disappearance of physical signs and the continued prostration and subsequent prolonged anaemia from which these patients suffer.

(II) The scattered areas of pulmonary consolidation may multiply, run together and give the appearance of a massive pneumonia, and death rapidly follows. These cases two, where a fatal termination develops so rapidly, present marked oedema of the lungs and conditions such as have been described by Symmers of

1. loc. citato.

Sheffield, by Prof. Peter of Paris[1], and by others.
In Mr. Gueneau's cases the pneumonic consolidation did not bear the characters of a true classical pneumonia, even of a very dangerous and potent type. As I have attempted to show in the Chapter upon the pathology of the affection, they presented rather the characters of an acute "traumatic pneumonia", the result of Vagus paralysis. This as we know occurs in the form of a bronchio-pneumonia. The cases terminated in death in a few days from exhaustion of the respiratory centre. The pulse became very rapid, as high as 150, the respirations 40 and the temperature rose as high as 104·6 despite the quinine. The sputum consists of a fluid mucus deeply tinged with blood.
Monsieur le Professor Peter, in lecturing upon the pulmonary complications of phthisis, continues thus with regard to these grave forms of Bronchio-pneumonia. "A form which I consider to be still graver "than Pneumonia, and which I deem inevitable, is general pulmon- "ary congestion. It arises in individuals whose organisation has been "already in an altered state. It is the suffocative catarrh of the ancients "and the patients truly suffocate. One observes in the upper parts of the "chest sonorous and sibilant rales and in the lower parts subcrepitant "rales. The pulse is small frequent and poor, but the temperature "is not greatly altered. The expectoration takes on the aspect of a blood "cream. It is a sort of salivation of the bronchia mixed with blood which "escapes on account of the enormous congestion of the lungs. This "especially dangerous in the emphysematous, diabetic, alcoholic etc."
In even more severe cases, as in those quoted at page 27 from Dr. Ord, where the paralytic symptoms are still more prominent, the bronchial secretions collect in the lungs, and are not evacuated. Then as Rendrat says of similar cases, "The patients are, so to speak,

1. Pulmonary complications in phthisis by Messrs. les Prof. Peter in Clinique Medical de l'Hôp. Necker. also in La France Medicale Janv. 9th. 1880. p. 52.

drowned in their own mucus" [1].

III Resolution may be incomplete, or may not take place. The pulmonary conditions frequently remain stationary for a short time, varying from days to weeks, but soon the progressive change in the most acute, and a fatal termination is the most frequent ending of this — the true form of tuberculo Bronchio-pneumonia.

Taking a chronological survey of this type, beginning in its origin in the primary attack of the grip, and ending with the final stages through which we are about to follow a case, we may for the convenience of a continuous description divide it into the following stages.

I. The Initial in which we have the primary attack of Influenza and those most interesting forms of pulmonary engorgement of a purely neurotic character.

II. The pre-mucal stage or the first stage of pulmonary inflammation: and we have the various forms of Bronchitis and more especially those forms which affect the tubes of lesser calibre. In many cases, alveolar engorgement and oedema accompany these changes.

III. The second stage of pulmonary inflammation in which the catarrhal changes have spread to the alveoli from the bronchioles. Now the alveolar changes predominate and signs of consolidation become manifest upon physical examination.

IV. The period of softening: — the alveolar contents begin to liquefy and the expectoration may in this way be removed by expectoration as well as by absorption and a cure results. In many cases, however, the expectoration have undergone caseous degeneration and when liquefaction sets in, as it is very prone to do in such a case, the process of disintegration extends and the fifth stage is soon reached.

1 Montreal Medicine Moderne. Feb 20. 1892. p 178.

V. This is the formation of cavity. The softening and liquefaction of the exudative products does not usually confine itself to the contents of the alveoli, and finer ramifications of the bronchial tree, but as we shall see later on, the pulmonary parenchyma in the midst of these areas of caseation has also undergone a similar degeneration. Softening begins in the central portions of these areas in the same way as it does in tubercular changes in the lungs, the products of degeneration usually find their way into a bronchiole and are expectorated. In this way a cavity or vomica is formed.

Contemporaneous with the last three of these stages we find three pathological changes which mark out clearly the distinction between true pulmonary consolidation and true lobar pneumonia. These are (1) pulmonary emphysema apparently of a compensatory type
(2) interstitial inflammation resulting in the formation of fibrous tissue and in subsequent cicatricial contractions
and (3) dilatation of the smaller bronchi and of the bronchioles.

The emphysema manifests itself early in the course of the disease by the physical signs peculiar to it but it is only in cases approaching to a chronic course that the other two changes manifest themselves at all distinctly during life.

As the first two of these stages, i.e., the Delirk and the Prodromata have been already described, I shall pass on at once to the third stage which is the

III. Second Stage of Pulmonary Inflammation.

For a considerable time the general symptoms as well as the physical signs may remain stationary. The cough continues to a slight degree and the expectoration if there be any is of a distinctly bronchitic nature. The general health is impaired, there is malaise, want of appetite and breathlessness with fatigue upon exertion. Night sweats too are occasionally complained of by the patient. The temperature tends to rise a degree or more above the normal at night.

The physical signs are those of a persistent bronchitis upon both sides of the chest.

Some day however, it may be the result of exposure to cold, an aggravation of the general symptoms takes place. There is a feeling of chill, and even in some cases repeated slight shiverings may be noticed but a rigor is rare. Headache, nausea, thirst and want of sleep arise. The tongue is dry, furred, red at the tip and edges and constipation is marked. In other cases diarrhoea and griping pains in the abdomen, frequently accompanied with epistaxis, complete a clinical picture which, with the addition of the rise of temperature to 102° or even as high as 104°7 forms a close resemblance to the onset of Enteric Fever.

The pulmonary conditions also undergo a change. The cough usually becomes very troublesome and seems in fits and the expectoration becomes mucoid and glairy but it rarely takes on the rusty tint of pneumonia. The respirations quicken and may even be as frequent as 30 or 40 while the pulse rate rises to 100 or 120. The characteristic pneumonic ratio is not the rule, the usual ratio being about 1 to 3, or 1 to 3·5. At some portion of the lung, the rales have become finer and dryer. They are subcrepitant and even crepitant. The expiratory portion of the R.M. becomes prolonged and may present the interruptions in its rhythm known as the cog-wheel respiration. Tubular breathing at this stage, as has been already noticed, is rare. The percussion note becomes progressively dull, although it is some time before the additional classical signs of consolidation, such as increase of vocal resonance and fremitus are well marked. Before these can be distinct, the separate areas of lobular consolidation must have coalesced in considerable amount. The initial site of the consolidation is not always the base as many observers would have it to be. It is generally unilateral and in very many cases basal. In two cases IV and V the apex was found to be dull as well as the base when the first examination was made after admission to the hospital. In Cases III and VII it was

distinctly basal at first and also in the two cases I and II already described in a former chapter. In Cases V and VI the apices seemed to have been first affected and in all these four cases, III, IV, V and VI, the apices showed the most advanced signs of pulmonary ~~consolidation~~ destruction when the post-mortem examination came to be made. Absence of pleuritic stitch at this period is the rule and in none of my cases was there signs of the tiger-present, another of the well known signs of acute classical pneumonia.

The pulmonary consolidation rapidly increases in density from coalescence of adjacent lobules and a pseudo-lobar form is that which is most frequently seen in the more chronic cases. The R.M. becomes more markedly tubular but soon diminishes in intensity and occasionally to such a degree that, as in Cases III and VII the respiratory sounds may be only heard during expiration when forced breathing is done the sound is a tubular one. The lobular condensation spreads in a short time to the adjacent lobe or lobes of the same lung and soon extends to the upper or lower lobes of the other lung.

The general symptoms become more evident. The anaemia which is so marked in these forms of the disease which follow upon influenza increases at an alarming rate and in association with evident lividity gives a striking physiognomy to the disease. The face is of a pallor earthy pallor and rarely presents even a livid flush. The tips of the ears, the lips and the fingers and toes have all a cyanosed appearance and all are cold to the touch. Beads of perspiration stand upon the forehead and cheeks and the patient prefers to sit up in bed not only on account of the cough which becomes more troublesome on assuming the recumbent posture but also because of the dyspnoea which is growing steadily more distressing.

Continued in Volume III,
and concluded therein.

Volume III.

The Bronchopneumonia of Influenza

(Concluded)

by Hugh Hight MB. Cm.

Contents of Volume III.

Chapter V (Continued.)

The pulse is small, feeble, rapid and frequently dicrotic. It varies in rapidity in proportion to the amount of fever as may be well seen from the chart of Case V. The temperature chart begins to show some of the hectic type owing to the increased daily remissions which may give a range of three to four degrees and even of 5.8 degrees as in Case V when one occasion night sweats, frequent diarrhoea and indifference to food soon show their effects in great prostration. Sleep is disturbed by cough, and delirium, rarely violent — more generally muttering is a very common night symptom in almost all of these cases. I noticed that a peculiar grunting or groaning noise was made, not necessarily during sound sleep but often also when the patient was simply dozing. On asking the night nurse in charge of the ward, the first evening that I remarked this noise, if this was an ordinary thing with such cases, I was astonished at the reply "Oh, yes! all these phthisis cases make that noise: I could diagnose a phthisis case in the dark by that grunt." On questioning a number of nurses, including a few who had had considerable experience in nursing pulmonary cases, I found that this "Pulmonary Grunt" as I would venture to call it, was not confined to cases of Phthisis, although it was generally heard best in these. It seems to be frequent in bad ~~case~~ cases of Pneumonia as well, but it is extremely rare in simple bronchitis. It is expiratory in its nature but what is its mode of origin, I cannot yet state. That it was not an ordinary "snore" I am certain. It partakes rather of the nature of a groan, it is inconstant in the disease and in the individual and seems to have some connection with the decubitus in severe cases where the pulmonary area is much diminished, as in double pneumonias and in advanced cases of tubercular phthisis and of bronchiopneumonia. In such cases the respirations are of a fixed type with a short sharp expiratory phase, and this may have something to do with the production of the sound.

Concurrent with the spread of the disease throughout the lung, the ~~respir~~

respiration in the healthy portions of the pulmonary tissue becomes exaggerated and puerile and soon, signs of commencing emphysema are manifested. The rales do not long remain dry but rapidly take on a manifestly moist character and soon we are carried into the fourth stage – that of softening. The clinical history already recorded, includes the first and second stages of the pathological changes – The fourth stage is the commencement, the fifth the completion of the pathological stage of Phthisis.

IV

The period of softening,

or ramollissement does not long delay its appearance in such a malignant form of bronchopneumonia. As new foci of lobular inflammation and obstruction arise in healthy portions of the lung, the rales in the original area of consolidation become more distant; fine bubbling and soon large bubbling. The expectoration now changes its appearance from that of a pithy purulent bronchitic sputum, to an evacuation composed of large rounded globular masses of muco-pus. These are of a greenish-yellow colour, are very tenacious and float like a thin glairy mucus which is as a rule only very slightly intermixed with air. In some cases fibrinous masses are expectorated but these are very imperfect in shape compared with those seen in true fibrinous bronchitis. Blood if present, is seen as streaks, there is rarely a rusty tint and in none of my cases was there a ~~fact~~ real haemoptysis. Berthier remarks of this period — "Il tousse davantage et par quintes. Les crachats sont muqueux ou muco-purulent, parfois striés de sang". 1

Sleep is much disturbed by cough. If the patient try to lie down upon the healthy side a fit of coughing is started which ends in the expectoration of a large quantity of muco-pus, and very often succeeded by vomiting. He prefers to sleep upon the affected side in a half recumbent posture. The amount expectorated daily varies from 25 to 80 grammes in Case IV, from 150 to 180 gram. in Case V and from 250 to 370 gram in Case VII, though in the late stages of the last case, 1450 gram were expectorated in one day.

1 Berthier. Thèse de doctorat de Paris.
"De la pneumonie caséeuse lobaire aiguë."

Pleuritic stitch is most frequently complained of. Strange to say in all these four cases i.e. III. V. VI and VII this pain was first experienced in the left subaxillary region. In case IV it appeared in the right mammary region. Pleural friction became quite evident to the ear at these areas, and soon took on a coarse grating character. In none of these cases was it accompanied with effusion. The process of softening goes on apace, and the consolidated tracts of lung and these rapidly develope the classic signs of pulmonary cavity.

V

The Formation of Cavities

now brings us to the fifth and last stage of the disease.

The percussion note over a limited area of the former dull patch undergoes an alteration. The dulness disappears to give place to a resonant dull tympanitic or even cracked-pot sound. The following is a report of the condition taken from my notes of Case V after the formation of a cavity in the left upper lobe. "Behind, the percussion note is distinctly dull at the extreme apex and at the lower third of the left lung. Just above the upper and inner angle of the scapula however, the note is hyper-resonant and upon strong percussion a cracked-pot tone is elicited. Here also there is egophony and fairly well marked whispering pectoriloquy. The metallic ring obtained by tapping with one coin, another laid upon the corresponding portion of the front of the chest is well transmitted here. The l.m. is cavernous and after coughing, there is usually a metallic ring with the rales which are fairly dry in character. During respiration frequent sibilant rales are heard. Above and below this cavity, the rales are quite subcrepitant especially at the extreme apex and base. Upon the right side of the chest, the l.m. is loud and harsh, though frequent subcrepitant, moist and sonorous rales accompany it. The vocal resonance and fremitus are not so well marked as upon the left side."

The sputum increases in quantity and consists of large nummular greenish yellow masses of purulent which frequently possess a very fetid odour. Upon treating it with caustic potash ~~it~~ fibrous tissue may often be noticed — significant of the destruction of real pulmonary tissue.

The general symptoms become still more marked of those of a Lethic type. Emaciation makes rapid strides. Respiration is almost continuous and diarrhoea further lessens what strength the patient may yet possess. The pulse is rapid, 120 or 130 or more, it is soft and small and frequent irregularities in rhythm are noted. The sound around the cardiac base through weak is relatively accentuated especially upon the right side where reduplication is also a common feature. Dilatation is difficult to make out ante-mortem on account of the overlapping of the emphysematous edges of the lungs. Cyanosis becomes more marked and the extremities suffer from cold and there is often slight oedema of the feet, legs and hands. The liver and spleen may be depressed as a result of emphysema but when the cardiac weakness is well marked, these organs are also found to be fairly enlarged. The renal congestion is shown by the presence of a small quantity of albumen in the urine and by occasional hyaline and even granular casts. The presence of urates and a high colour give further a febrile appearance to the urine.

Delirium becomes more marked at night and soon it extends throughout the day whenever a patient begins to doze. The temperature chart shows greater excursions. It may rise rapidly before death to 104° or to 105°7. as in case III and death takes place quietly from cardiac failure. In other cases, the thermic curve may run up to 102° or 103° or so at night and fall to normal or subnormal in the early morning. This goes on from day to day, the acme steadily becoming lower until for a few days before the end it may never reach the normal as in case V. Cheyne-Stokes respiration is also frequent and death seems in coma as in case IV where I have notes:— "July 9th. Today, the patient is very weak and ill. She picks at the bed clothes and wanders: the extremities are cold and the general surface of the body as well as the lips and mucous membranes are almost bloodless. There is marked orthopnoea, the breathing is short and catching. To day he died in coma."

The fourth course which a case of Bronchiopneumonia arising in a pulmonic fashion may take is a Chronic one, and this will be discussed in the following chapter.

Chapter VI

Chronic Bronchiopneumonia.

Chronic Bronchiopneumonia usually partakes of a lingering nature from the commencement. It is rare that the variety just described undergoes a partial arrest in its somewhat rapid course and still rarer is the fact of the destructive changes remaining in abeyance for a time. Of Chronic Bronchiopneumonia, Henri Roger says:— "It is established in pathology that chronic pneumonia apart from that found in tuberculous subjects is excessively rare in adults, (so rare that its existence is disputed) and that it is a little less so in old people; in childhood on the contrary, the chronic form of pulmonary inflammation certainly exists and is even comparatively frequent. It is the Bronchiopneumonia which so ends, and therefore as it is more frequent in childhood, chronic changes are then more frequent especially in that form of the disease which follows measles and whooping cough." 1 To these two diseases, I would add the Grippe. The prognosis of one is grave in any cases, judging from the duration of the cases, but is rather to be measured when the pathological appearances come to be considered. As I have already shown, interstitial changes appear early in bronchiopneumonia so that we was surprised to find that after death the lungs in Cases IV and V presented quite evident changes of cicatricial contraction, although the duration in the former case was only nine weeks and in the latter eight months. The production of fibrous tissue throughout the lungs cannot therefore be taken as a criterion of the chronicity of the case, but it may be said that

1 Henri Roger article "Bronchopneumonia" in Dict. Encyclop des Sc. Med.

subacute cases end usually in one, two or three months, rarely as long as eight months, and the chronic cases continue for one or two years and in a few recorded cases for a much longer period.

When the disease assumes a chronic course from the commencement, the general symptoms are such as to attract but slight attention. The patient apparently suffers from a "slight chronic bronchitis", a condition which is very frequent in the climate and which arouses no thoughts of apprehension in the sufferer. Cough is rarely troublesome at the commencement. Expectoration is scanty and mucous and scarcely ever does stitch in the side manifest itself. But as time rolls on, these symptoms become aggravated. The patient feels that he is not up to his usual form; that he becomes more readily tired on exertion and that he is somewhat breathless. Cough too is more readily excited and the spit has taken on a somewhat yellow tinge. He seeks medical advice when the following conditions are found upon physical examination.

The temperature tends to rise in the evenings to 99°, 100° or 101° and with this rise frequent night sweats may be present. The body weight has become reduced although in some cases this is not well marked. The spit is somewhat purulent and comes away after successive "bouts" of coughing. On examining the chest we may yet find no definite physical signs to account for the respiratory troubles (Sequin and Sisselle each 2 cases, Clement 1 case). Generally however we finds slight dullness at one portion of one side of the chest – most frequently at the base but occasionally especially in the aged at the apex. The condition of Case VII upon admission is fairly typical of the state of the system at this period. Here is the note upon admission: — "There is very little expansion at either apex in front during inspiration but upon laying the hands upon the chest, a well marked râle crépitus is felt. The percussion note all over the front of the chest, except just above the clavicle where it is hyperresonant is of a dull hypanitic nature which assumes an almost cracked pot character upon strong percussion over the apices. The left infra-clavicular region is dull to percussion and this intensifies as we descend. The spleen is normal to percussion. Above the clavicle, the note is hyperresonant upon

the right side until one approaches the apical area of dull tympanism. The expiratory portion of the R. lung is prolonged over the whole front of the chest. At the right side, the R. lung is tubular, almost amphoric at times, harsh and much louder and more distinct than upon the left side.

At the left side, the R lung is small and tubular and is accompanied by fine dry subcrepitant rales especially crackling and dry during prolonged expiration, when bronchial rales of a hissing nature are also frequently heard. Respiratory sonorous rales are more frequent and much louder upon the right side than down, where also large bubbling rales are often heard. The Vocal Resonance and Fremitus show practically no difference upon either side.

At the back, next one notices that the chest is retracted upon the left side towards the base. Upon measuring with a tape, held horizontally at a level of one inch above the nipple line, the circumference of the left side is found to be 17 inches during very gentle respiration. When asked to take a deep inspiration, there is only ½ inch of expansion. A similar measurement upon the right side gives 18½ inches with 3/4 inch of expansion. There is however no appreciable displacement of the scapula or of the ribs or spine. The percussion note is slightly dull at both apices, especially at the left. This dulness upon the left side lessens in the region of the upper 2/3rds of the scapula but is never absent. From this down, it rapidly increases in intensity until at the extreme base the note is absolutely dull and wooden. The Vocal Resonance and fremitus are markedly diminished at the left base. Here also the R lung in ordinary respiration is unheard except upon forced breathing. Faint tubular hissing rales are heard, while higher up as well as further out towards the subaxillary region dry crackling rales are more common in inspiration with sibilant hissing or sonorous rales during expiration. Towards the left subaxillary region there is distinct pleural friction. At the apices, the R. lung is tubular but is almost masked by abundant hissing and sibilant rales. The respirations are 39, short thorax regular and easy. The cardiac area is normal to percussion at its upper and inner limits but the axillary border is rendered indefinite by the dull note merging into the pulmonary area of dulness. The apex impulse is in the fifth space

in the nipple line. The first sound at the apex is somewhat booming and the second sharp and accentuated. The pulse 90, fairly full through compressible. The pulse-respiration ratio is as 1 to 2.5. Temperature upon admission was 101.4 but today it has fallen to 98.6 in the morning and to 98.4 in the evening. The bowels are regular and the appetite is good. The skin is dry though he occasionally perspires during the night. The expectoration is mucopurulent, tends to a greenish hue and is not today abundant. The interpretation of these signs denoted seventeen months after the initial attack of the grip is as follows:— condensation of the left base with considerable cicatricial contraction: condensation extending to a much less degree into the left subaxilla, the left apex and over to the right apex behind, diminishing in intensity in these areas in the order of enumeration: general tympanitic dilatation upon the left side to a marked degree; less marked at the right apex; pleural thickening at the left subaxilla without fluid effusion; general compensatory emphysema throughout the remainder of the pulmonary area; slight obstruction of the pulmonary circulation with compensatory hypertrophy and slightly of the left.

When an acute or a subacute attack passes into a chronic condition the ultimate conditions are quite similar to those just described. According to Vulpian it is in children in which such a transition is most often seen. The acute attack undergoes an apparent resolution and the patient begins to feel much better. The evening rise of temperature however still persists in most cases. The pulmonary condition undergoes very little if any change and soon an exacerbation of the general symptoms arises. The cough and expectoration increase, the temperature rises higher and physical examination reveals a condition of affairs similar to that which has just been described. In these cases however the rales are usually of a more bubbling character. (2)

1 Vulpian. Des pneumonies secondaires. Revue d'agrég. 1860 Paris.
2. See two cases reported by Regimbeau (loc. cit) from the clinical records of Garrod and of Vulpian.

As the case progresses in a chronic fashion, the local signs become more marked. The area of dulness increases in extent and in density of tone. The respiration becomes more cavernous and the rales louder and more bubbling. When the bronchial dilatation is great, the voice sounds take on the character of cavernous, of bronchophony etc., such as we find in case VII. To quote from a note made three months after admission to the hospital:— "Today, the rales behind are minute and moist bubbling upon the left side especially just beneath the angle of the scapula. Elsewhere, however, they retain their crepitant character described in previous notes. Between the angle of the scapula and the vertebral column the vocal resonance partakes somewhat of the nature of aegophony, and here the whisper is fairly well and distinctly heard. Elsewhere the vocal resonance is neither so punctate nor so loud as upon the right side". This last mentioned change in the voice sounds, notwithstanding the increasing dulness is seen in many cases and would seem to be due to pleural adhesions and thickening such as were found in this case = Case VII upon post mortem examination. Signs of bilateral contraction and pulmonary obstruction become more marked. Hypertrophy of the right ventricle such as seemed here was note by Bastian 10 times in 30 cases. When the retraction of the lung becomes more marked the heart may be drawn towards the affected side and according to Traube, the diaphragm may be drawn upon higher on the left side. The chest two becomes more drawn in, the intercostal spaces are narrowed the ribs approach one another in turning upon their longitudinal axis and in very advanced cases the shoulder falls and the scapula tends downwards and inwards, giving rise to a remarkable distortion of the thoracic cage.

Vesicular emphysema increases not only upon the sound side but also in the intervals between the affected portions of the affected lung.

In Case VII I note that "along the left side of the spine, there is an area two inches wide of clear percussion continuous with the clear percussion upon the right side. The note is hyper-resonant and the R. Rh. heard". The bronchial dilatation also reveals its presence by the nature of the sputum and by its mode of evacuation. The quantity increases considerably,

DISEASE.

Notes of Case.

Name Case VII

Age

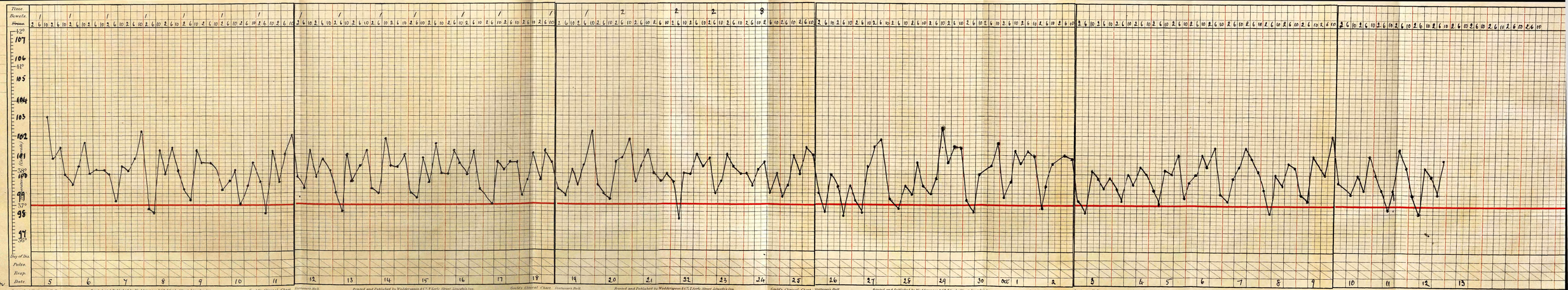
Diet

Case Book N°

Temperature taken every four hours.

Date of admission September

Result



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DISEASE.

Typhoid

Notes of Case.

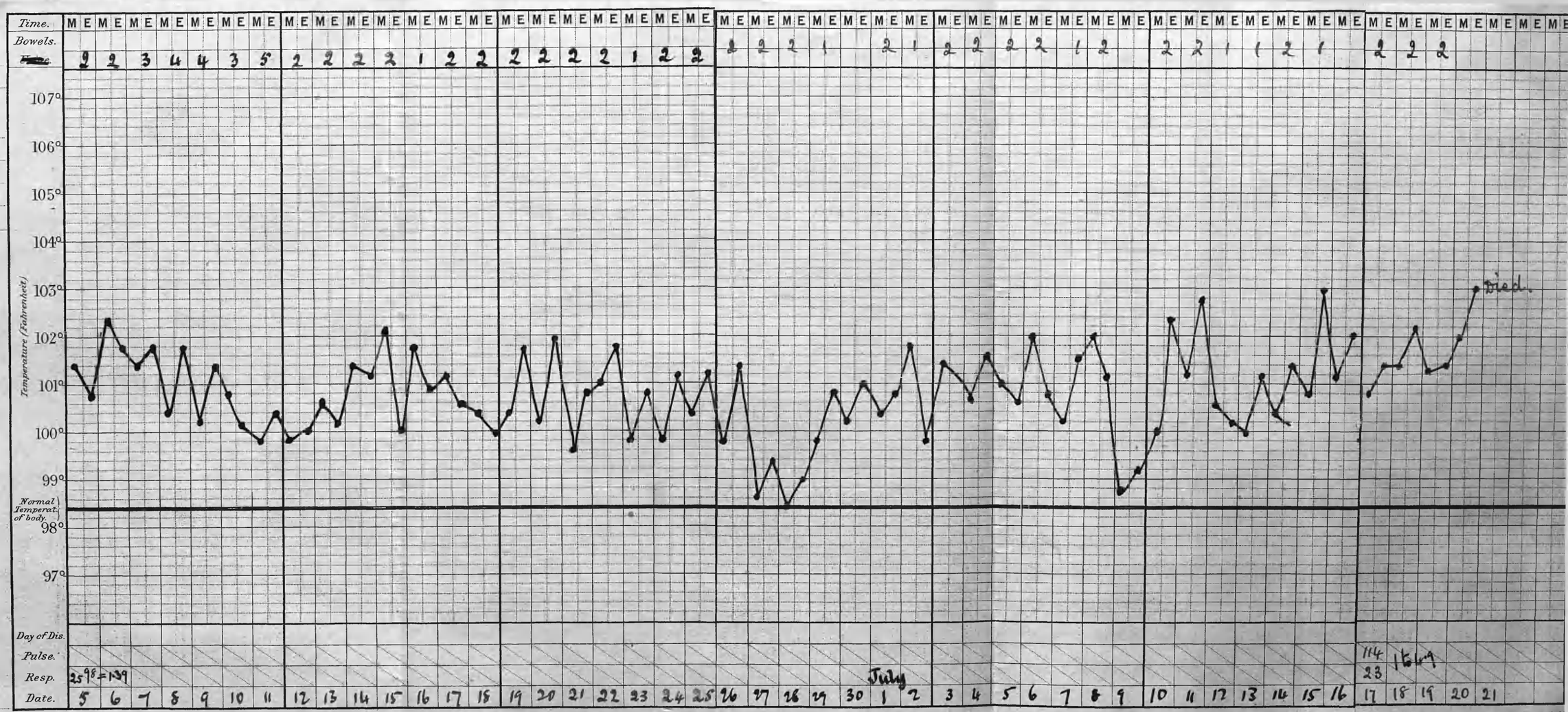
Name

Age

Case IV

Diet

Case Book N°



Date of admission.

June

Entered at Stationers Hall. Printed and Published by Wodderspoon & Co. 7 Serle Street, Lincoln's Inn. Gould's Clinical Chart Stationers Hall. Printed and Published by Wodderspoon & Co. 7 Serle Street, Lincoln's Inn. Gould's Clinical Chart Stationers Hall. Printed and Pub

cases however as in Case VII the cardiac action remains remarkably steady and able for the requirements put upon it to almost the very end. The duration of the disease may be years. Summer brings a temporary relief but every winter the condition becomes more aggravated and finally unless when complications set in, the pulmonary lesions take on a more rapid course and ultimately prove fatal.

Death may occur from marasmus and bed sores, from progressive asthenia, from sudden heart failure, from haemoptysis, from diarrhoea or from other of the various complications which will be dealt with presently.

A few words may be said with reference to the temperature, pulse and respiration in the subacute and chronic forms.

In Case IV upon admission and when consolidation was becoming evident, the pulse was 98·7 and the respirations were 25, giving a ratio of 1 to 3·9. Again in Case V in the early stages of softening a pulse rate of 118 and a respiration rate of 20 gave a ratio of 1 to 5·9. Such ratios were most frequent in all these subacute cases. The pneumonic ratio which was noticed in the acute type is absent here. The normal balance between pulse and respiration is not only returned to but as we see in many cases there is often an exaggeration of the pulse rate. Now and again throughout the course of the disease as a result of a fresh inflammatory condition the respirations increase in frequency while the acute pattern of the bronchitis, bronchopneumonia or even pneumonia, as the case may be, lasts. In an average of 20 readings taken at 2, 6, and 10 a.m. and the same hours p.m. from the 14th to the 18th. inclusive in case V, the following figures were obtained:—

	a.m.			p.m.		
at	2	6	10	2	6	10
Pulse	112·4	96·3	123·6	146·4	116·5	126
Respiration	23	20·4	24·5	25	21·4	25·6
Pulse-respn. ratio	1 to 4·8	1 to 4·6	1 to 5·15	1 to 5·8	1 to 5	1 to 4·9.
Temperature	100·8	99·2	100·7	100·5	99·4	102·2.

DISEASE.

Notes of Case.

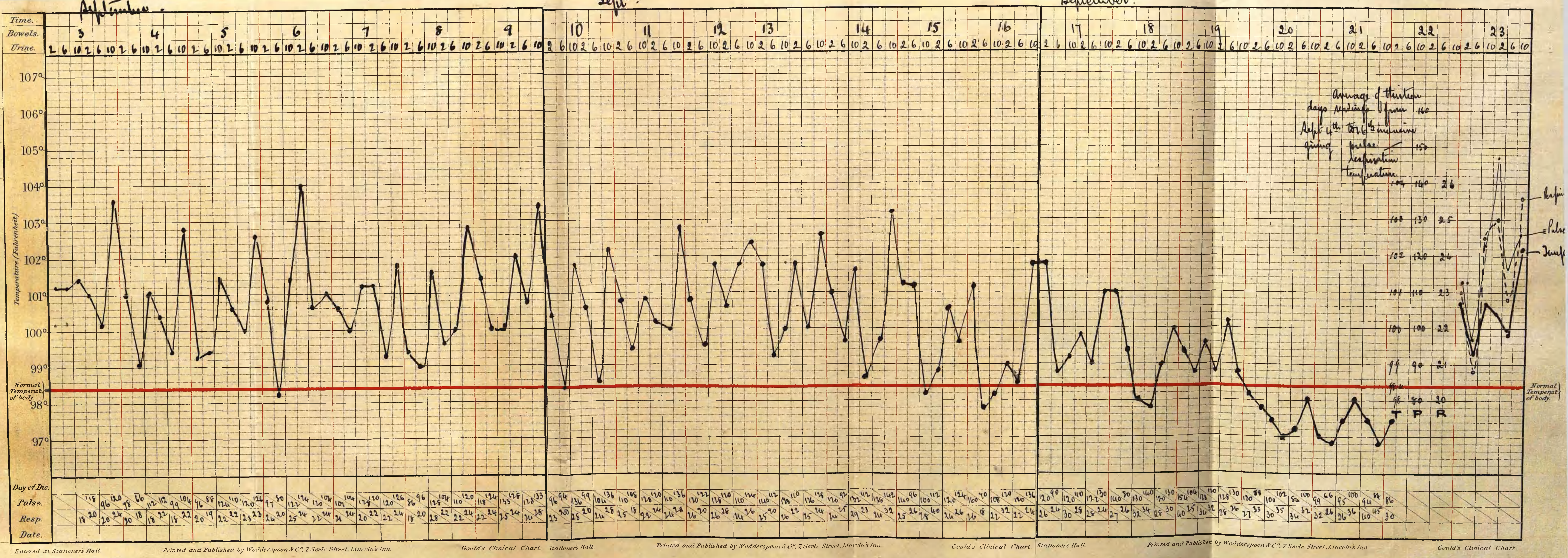
Name { Chas Hinchey.

Age

Diet

Case Book No.

Case V



Date of admission

Result

In the notes upon Case V it is noticed that "the daily range of temperature has not been according to what is generally described as the normal course, but in place of a simple rise beginning in the forenoon and continuing until 6 p.m. or so and then falling until six or seven the following morning, there has been a rise from six A.M. until 10 A.M. and then a fall until 6 p.m., and a second rise to 10 p.m. when the maximum for the day is usually obtained." Such was the case in case IV though not to such a marked degree where it was noted that "the temperature has shown a similar course to that described in the other cases. As a rule, the morning and evening readings are very close and during the first fortnight in July they generally remained beneath 100°F. Upon the 13th. however, they rose above 100°F. and from this time until late in August the readings fluctuated between 100°F and 101°F. being occasionally above or below these two points. From August the 28th. until today September 6th the temperature has been almost uniformly over 101°F. The chart shows an irregular line during these two months, but the greater areas of depression are not diurnal. Each manifest rise and fall embraced two three or more days, the fall being as a rule by a crisis and more sudden than the rise. There is no regular ~~intermittency~~ remittency in these rises such as one would have in any of the various types of intermittent fever." These rises from the ordinary line of the temperature curve, mean as a rule a fresh attack of bronchopneumonia. A new focus of the disease has become established or it may be that is a more virulent bronchitis. In other cases, a limited lobar pneumonia or a pleurisy is the active agent in the production of the rise. All charts show this tendency to an irregular intermittency from day to day. The irregularity however precludes a comparison with the cases of Intermittent Pneumonia such as have been described by Fröhlich.[1]

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[1] Fröhlich. Wien. Med. Press. 1878. N° 15.
also in Surg. Abdom. 25th. Oct. 1878. p 688.

Chapter VII.

Primary Subacute Bronchopneumonia.

In introducing the subject of the subacute form of this disease it was remarked that this rarely is generally grafted upon a preexisting bronchial catarrh, and hence might be classified as a secondary bronchopneumonia were it not for the fact that on rare occasions we find a case developing and progressing to a fatal termination in a subacute manner throughout its whole course.

Case VI is a good example of which the report is briefly as follows:—

B——. Coachman. aged 42 was admitted to the Hertford British Hospital Paris, upon the 28th of March 1891 suffering from great weakness, pains in the head and limbs and from fever.

A fortnight before entering the hospital, he contracted Influenza of which he presented most of the usual symptoms.

Condition on admission:— The temperature is 100°7. He seems to be very weak, he sweats heavily and the pulse is very feeble. The extremities are cold and along with the lips, cheeks and ears, present a livid tint. This cyanosis of the face is perhaps the most striking feature in the case. He has no cough and physical examination reveals nothing abnormal in the lungs or heart. He complains now of no pain when in bed but simply of a feeling of extreme prostration. There is neither sugar nor albumen in the urine. The bowels have been regular without any looseness.

Upon April 5th ten days after admission and twenty four days after the onset of the grippe, the following note was made:— The general condition of the patient remains pretty much the same as when he came in. The heart is rather stronger but still the lividity of the face and extremities is very evident, and he sweats heavily. The highest point upon the temperature chart was reached upon the evening of the 30th when the thermometer registered 102.6°7. Hence it has fallen, though irregularly to 99°7. upon the morning of the 3rd and 4th, but upon the evening of the 4th it rose to 101.6. Tonight it has reached a similar figure after having fallen in the morning to 100.2°7. On examining the chest today, percussion revealed a distinct

wound freely in the line over the left apex in front. The note seems flat and the expiratory portion prolonged. There is still no cough however.

April 8th. There is now dulness over the left apex in front. The N.M. is tubular and is accompanied by a few subcrepitant râles best heard during expiration. An occasional râle of a little coarser is heard at the right apex although no dulness to percussion is noted here.

April 17th. The dulness is increasing in density upon the left side, both front and back, and is rapidly descending. The note over the right apex is now dull also and here too the same rapidity of extension is noted. The râles have assumed a liquid metallic ring. For the last few days he has complained of pleuritic stitch in the left subaxillary region. The temperature has been rising and falling between 100.6°F as the lowest point since the last note was made to 104°F upon the evening of the 16th as the highest point.

April 22nd. The dulness is down towards both bases now, the râles are quite moist bubbling and metallic towards both apices in front, especially at the left apex where the lung seems to be breathing down very fast.
The heart is becoming smaller and the lividity more evident. The emphysema and the expectoration is muco-pus. There has never been any haemorrhage nor even a tendency to the rusty sputa of true pneumonia.

April 23rd. The patient died quietly this morning from heart failure.

The treatment was Stimulation, attempts to reduce fever, and local irritation in the form of counter-irritants so soon as the first chest signs were discovered.

The post-mortem appearances:- Cadaveric rigidity still present. The lividity of the parts already described still exists. The body is considerably emaciated.
Thorax. There is a considerable amount of fat around the heart. The pericardium contains a small amount of serous fluid. In the left ventricle, there is a large white thrombus. The right ventricle is full of venous clot as also are the pulmonary arteries and the superior and inferior venae cavae. The heart muscle is pale but the organ is otherwise normal. All the valves are competent.

Lungs. There are slight adhesions upon the right side of old date. The lungs are deep livid red but are universally crepitant especially towards

both apices with yellowish grey nodules ranging from the size of a pea or smaller. In the upper lobe especially the left these nodules tend to run together and so form firm areas of condensation which present a yellowish grey cheesy aspect. Cavities are forming at both apices. On squeezing the lung a pretty purulent fluid exudes from all these yellow spots as well as from the grey tubercles and lymph. The lungs float in water.

Abdomen. The liver is large and deeply congested giving a very distinct nutmeg appearance. There is a considerable amount of serous fluid in the peritoneal cavity. The spleen is large soft and deeply congested. The kidneys show marked venous engorgement.

Brain. There is over ~~one~~ one ounce of fluid in the skull cavity. The general venous engorgement is evident here also both in the membranes and in the cerebral tissue as well. Otherwise there is nothing remarkable to record.

Microscopical Examination of sections of the affected lungs, revealed the presence of a caseating bronchopneumonia. Here and there liquefaction of the caseous foci was taking place and cavities were developing. The tubercle was found either here or in the bronchial glands.

With regard to the primary physical signs at the apices in this case, it may be interesting to quote a passage from Berthier's thesis[1] which describes a somewhat similar course in what he describes as a case of "Pneumonie Caséeuse Lobaire aigüe". Of course to a faithful disciple of Laennec, such a Bronchopneumonia as he describes at length, is to be considered a tubercular affair and the natural corollary in his mind is the synonym which he employs - i.e., "Bronchopneumonie tuberculeuse aigüe Pseudo-Lobaire" although he bases his conclusions solely upon clinical facts with the support of bacteriological or microscopical evidence.

Without digressing further upon this wide subject we shall simply quote the following from a very admirable thesis. "M. Jaccoud dans une "observation remarkable suivre dès le premier jour et sur laquelle "nous insistons, ayant présenté une affection de poitrine, dirigea ses "recherches de ce côté, mais par la percussion, ni l'auscultation ne

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1 loco citato.

" lieu fournissent de renseignement. Le lendemain surement il
" constatait à la base gauche quelques râles sous-crépitants fins et du
" son tympanique; la troisième jour apparait le souffle avec le point
" Est-ce la règle au début ? "

From the clinical history and post mortem report it will be seen that once the disease becomes fully established, the further course of this variety differs in no way from the secondary forms of the pulmonic type except that the duration is generally less. In this case the total duration was forty one days reckoning from the onset of the grip & eighteen days after the first pulmonary symptoms were detected.

Chapter VIII.

Complications and sequellae of the disease.

Pulmonary - Abscess of the lungs occasionally occurs during the acute attack in severe cases. According to Kundrat "these bronchopneumonic foci were extraordinarily numerous. In the superior parts of the lungs they attained the size of a nut, they were besides purulent in their central parts. At the first look at the affected lungs, one might have imagined that he had to do with a metastatic abscess. These abscesses recall the pneumonia in the influenza of the horse".[1] To quote another authority M. Drasch " the pneumonias and pleurisies of influenza have absolutely special characteristics the foci of bronchopneumonia ending often in suppuration or in gangrene". 2.

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1. loco. citato.
2. M. Drasch. Allg. Medizine March 13. 1890. p 289.
also Royal & Imperial Society of Vienna 7th. March 1890.

Although M. Kahler maintains that "these acute pulmonary abscesses are almost always primitive appearing in the early stages of the disease and in consequence they are the result of the direct action of the influenza poisons and not of a secondary infection"[1] still we often find them in the subacute as well as in the chronic forms of the disease. (Roger[2], Balzer[3]).

Gangrene is reported by Hendrat in thirteen cases of pneumonia following upon influenza, and as already quoted a similar termination has been noticed by Drasch.

Pleurisy may develope in any of the three forms of the disease. In the acute attack it may often takes on a purulent character as described by Hendrat, Laveran[4] (4 cases) Drasch, Kahler, Eade[5], Menetrier[6], etc. In the subacute forms there may be present a slight pleural effusion of a serofibrinous nature but most frequently the pleurisy is of a dry nature.[7] Stitch is generally not complained of until the stage of softening has commenced. This was the case in these four cases III, IV, V and VI and in all with the exception of Case IV where it commenced in the right mammary region, pleural friction and pain first manifested themselves in the left subaxillary region. In none of these was there ever any definite signs of pleural effusion.

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1 Kahler - Med. Mo. Feb 27 1890. p 196.
2 Roger Article "Bronchopneumonie Chronique" in the Dict. Encyclop. des Sciences Médicales.
3. Balzer. Article Bronchopneumonie subaiguë et Chronique" Dict. de Med. et Chirurgie.
4. Laveran. Med. Mo. 30 Jan. 1890 p. 111
5 - Sir Peter Eade. British Med. J. Aug 8th 1891 p 308.
6 Menetrier. loc. cit.
7. Consult Léon Bassé "De la pneumonie lobulaire chez l'adulte" Thèse de Paris.
& Reginbeau - loc. cit.

the date of origin and the character of the pleurisy which is rarely of a purely acute nature, point apparently to its infective origin. General infection of the system which is rare in children is the rule in adults when the case is of some duration and the primary cause of the infective process is through the lymphatic channels. The pulmonary lymphatics converge towards the pleural cavity — which is practically a huge lymphatic sac, and hence when the alveolar exudations began to break down, the products of disintegration with their accompanying microorganisms are carried along the lymph channels to the pleura giving rise to irritative processes all along their course. Marked thickening of the interstitial pulmonary tissue as well as of the pleurae results, leading to those firm semicartilaginous envelopes which have been described in the post-mortem records. Adhesions of visceral, parietal and interlobar layers readily occurs and give rise to various changes in the physical signs such as were found in Cases IV and VII where chest movements were restricted, the percussion note was markedly dulled, but the vocal vibrations were diminished and the R. Br. was almost abolished. It is only in chronic cases however such as Case VII in which the fibroid contraction of the pleurae as well as of the pulmonary tissue leads to deformity of the thoracic cage.

Pneumothorax — the result of the bursting of an abscess through the pleura was reported in one case by M. Laveran.[1]

Bronchitis is a frequent complication in the subacute and especially in the chronic cases. This is specially the case when bronchial dilatation has become established and when, therefore, the secretions get leave to remain stagnant and to decompose. Perhaps the most frequent cause of the occasional rises of temperature which have been described, is an attack of bronchitis. In Case VII this was noticed again and again not only in the affected upper but very often in the other lung as well.

Pneumonia of a true lobar type is not an infrequent cause of death when it arises in the healthy lung in subacute or chronic cases. As we have already seen in Case II it may occur in the acute forms and may prove a much more serious affair than it was with that

1. M. Laveran. Mil. Med. 30 Jan. 1890.

patient.

Emphysema has been frequently mentioned already and when it develops to any great extent it reveals itself by its usual definite symptoms and signs and is an important factor in the disablement of the cardiac and general circulation.

Haemoptysis unless in very bad cases is not a dominant feature in the acute forms. There is usually as in Cases I & II only a mere streak of blood if there be any. The absence of a true rusty tinge is one of the factors in the differential diagnosis between Bronchopneumonia and lobar pneumonia. In malignant cases however, almost pure blood may form the bulk of the sputum. Bouchereau reports such a case in which he states "il cracha du sang pur". Later on in the same case he further remarks "En outre les crachats avaient l'aspect de jus de pruneaux." The presence of prune juice expectoration has been frequently noticed in treating of the history of Grippal Broncho-pneumonia. In subacute cases, haemoptysis is rarer. In none of my cases was it present although in Case II there was a tinge of blood for many weeks and well on towards recovery. In all the other cases it was to employ a Hibernian phrase, "conspicuous by its absence". In chronic cases reported by Ch. Bastian it was present in 15 out of 30 cases and may be due in some cases to rupture of aneurysms of branches of the pulmonary artery such as Barth describes.[1]

Asthma as I have already remarked was noticed by me in a young woman of 24 and where it continued for nearly a month after convalescence had become established. The attack was an acute one in her case.

Cardiac Complications mainly manifest themselves as the result of obstructed pulmonary circulation.

In acute malignant cases, syncope may terminate the case in a sudden manner. In subacute and chronic cases too, death often arises from the same cause. In cases which run a subacute or

1 La Bronchopn. chronique of Henri Barth. Dict. Encyclop. d. Sc. Méd. also Balzer. Les hemorrhagies pulmon. dans la bronchopn. Bull Soc Anat de Paris, LIII 269 274 1878.

chronic cases the amount of wall thrown upon the right heart becomes gradually greater owing to the progressive obstruction to the onward flow in the diseased lung or lungs. Hypertrophy of the right ventricle suffices for a time to maintain the balance but soon dilatation indicates the commencement of cardiac failure. Cli. Bastian reports this in one third of the chronic cases. Then now develops a series of changes which pushes from dyspnoea and lividity of the lips, &c., to manifest cyanosis, oedema of the limbs, ascites, &c. On auscultating the heart, accentuation of the second pulmonic sound was noticed early in my cases and later on along with the signs of dilatation of the right heart, a slight systolic whiff was occasionally noticed. In Case V the 2nd sound was reduplicated on the right side for some weeks before death. In most of these cases two irregularity in rhythm was frequent towards the end although in Case VII the heart remained remarkably regular up till the end. As the anaemia and cardiac weakness increase a dicrotic pulse develops and a tracing from that of Case V is given.

Cheyne-Stokes respiration was present at intervals for a few days before death in Case VII.

Displacement of the whole cardiac organ as a result of traction by a contracting lung is seen in many chronic cases. The direction of displacement will evidently depend upon the lung involved. In one case in which the right lung had greatly shrunk, the heart's beats were felt to the right of the sternum. The diaphragm and the abdominal organs may be drawn up (Traube) and the chest deformed as already described.

Thrombosis of the veins as a complication of grippe has been reported by many observers such as Duchesneau[1], Bouveret[2], Ferrand[3], Moure[4], Vaillant, by various English observers[6] and by the author[7], apart from any actual

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1. Duchesneau. Sur la gangrene des membres consécutives à l'influenza. Gaz. Hebdom. N°24. p 371 | 1890.
2. Loc. Cit. 3. Ferrand. Méd. Moderne. Feb 27. 1890. p 192.
4. Med. Mod. 5th. June 1890 p. 762. 5. Soc. Méd. des Hôp. Paris. 24 June 1890.
6. Practit. Med. J. March 1st 1890. and April 26th 1890.
7. A case of symmetrical gangrene following upon influenza by the Author. MS. Brit. Med. J. July 18 | 91.

association with the disease in question. The probable pathology of these was discussed in the paper above quoted but that in many cases a septic phlebitis was the starting point is proved by the researches of several bacteriologists, conclusions which have been already described in treating of the pathology of the disease. In the acute form of the disease several cases have been reported by Duchesneau of acute Bronchio-pneumonia complicated by venous thrombosis and phlebitis of the inferior extremities especially. One fatal case in particular is of peculiarly painful interest in that the patient was a medical man. It is in the subacute and chronic cases of Bronchio-pneumonia that thrombosis becomes more frequent. The sites of its occurrence par excellence are the pulmonary arteries and veins and the veins of the lower extremities. Some described its presence in the cerebral sinuses. In case IV we have a good example of its occurrence in the subacute stage towards the termination of the illness. Pathologically it has been already discussed and has been shown to partake of the same nature as various cases described by Bouchut, Vaquez & c. The clinical notes of its advent in my own cases are as follows:— "July 14th 1891. (4 weeks and 2 days after the onset of this illness.) Yesterday, patient complained of shooting pains and a sensation of numbness all down the left lower limb, and on examining it today the following appearances are evident:— the limb is swollen and large compared with the right leg which is thin and shows well all the bony prominences. The left limb is rounded, edematous but pits only on firm pressure. Scattered over the leg and thigh there are deep purple purpuric spots of subcutaneous venous haemorrhage. This is most apparent upon the postern and inner surfaces of the thigh and calf, where the spots have largely run together and become confluent. In these parts also pain is complained of upon pressure. No thickened veins are felt although pain is quite distinct along the course of the popliteal vein. The limb is very cold and blanched except where the subcutaneous veins show themselves purple and blue. The left calf measures 11½ inches in circumference, the right 9½". The femoral artery is felt beating quite well. July 20th: the left leg is warmer today than the right as it has been wrapt in cotton wool and laid upon a pillow. There are some purpuric spots round the right knee now. She died tonight." ..... "At the last autem examination the measurements were left thigh 39 cm. right thigh 28·5 cm.
" calf 33·5 cm. " calf 26 cm.

Cerebral Complications. In the acute stages sleeplessness is almost an invariable symptom but all degrees of mental disturbance from slight occasional wandering to continuous muttering delirium or even acute mania have been recorded.

DISEASE.

Notes of Case.

Name Case III

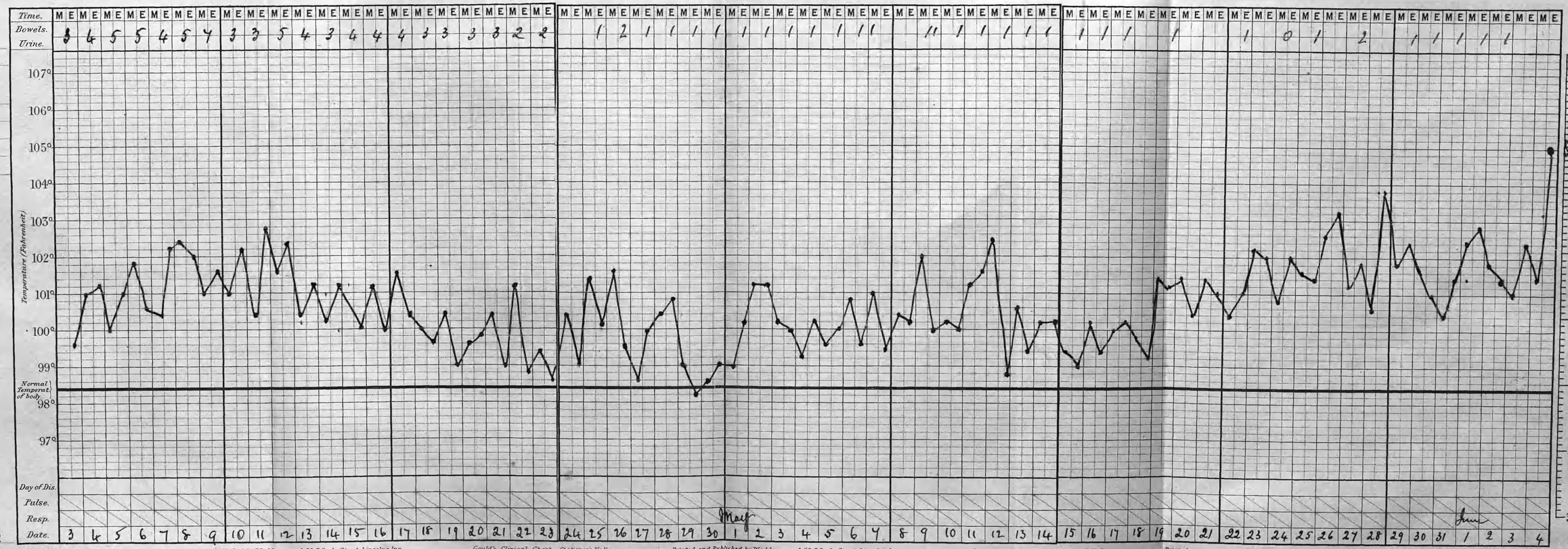
Age

Diet

Case Book N°

Date of admission. April 3rd

Result



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In the subacute and chronic forms of the disease, muttering delirium is very frequent when the circulation comes to be interfered with. This as is usually the case in cardiac cases is most at night but becomes progressively worse as the necessity of the blood increases. Cheyne-Stokes respiration was present at intervals for a few days before death in Case III.
Abscess of the brain has been noticed by Barth in some chronic cases. 1.

Enlargement of the Spleen and Liver from passive engorgement are often evident to physical examination. This was markedly so in Case V.
Renal engorgement is present too and manifests itself by a slight trace of albumen in a highly colored urine and the frequent presence of hyaline or finely granular tube casts. There is often too when the temperature is high a heavy deposit of lithic urates.
Ecthyma as in Case VII, exhibiting of the fingers in Cases V & VII bed sores (Bakes) gangrene in the mouth found especially in children (Legendre) and profuse perspirations are all purpose lesions of frequent occurrence.

Diagnosis

From Typhoid Fever. That the disease may at first be mistaken for a case of typhoid fever especially when the onset is at all acute will be seen well by glancing at case III. This patient was admitted suffering from symptoms of bronchitis, from severe diarrhoea, and from great weakness. On admission physical examination revealed some slight dullness to percussion at both bases with abundant moist crepitant and bronchial rales. There was pain and tenderness in the right iliac region with considerable gurgling upon palpation. The bowels were moved thrice upon the day of admission and the stools were very fluid and bright yellow in color. The symptoms were only of four days duration and as yet there were no rose spots upon any portion of the skin. Although the temperature was only 99.6 we did not feel justified in denying the presence of enteric fever at this early stage and in the face of such a group of symptoms. Now that we can look upon a fortnight series of morning and evening temperature readings and now that we have the whole history of the case before us, the diagnosis is sure, but let us look solely at the state of affairs up to and including the evening of the 23rd of April. Here we have a gradual temperature angular rise from the 3rd April to an acme upon the evening of the 11th and then a fall by crisis to 98.6 upon the evening of the 23rd. which was exactly the 21th day of his illness so far as the patient knew. According to Wunderlich the fall of the temperature to 100.7 upon the morning of the

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1. Barth. Cinthese des processus. La Semaine Méd. Chron. Bibliog. des Sc. Méd.

5th which was one degree lower than the reading at the same time the previous morning would, assuming as it did during the ascent, of itself prove emphatically that we had not a case of typhoid fever to deal with. Again, upon the evenings of the 6th and the morning of the 7th the readings gave lower figures than the two previous readings. Wunderlich further says, (quoting Hillier Tapp) "enteric fever might generally be excluded from the diagnosis if the temperature rose during the first two days to 104°: if (at least in a patient under middle age) it did not rise between the 4th and the 6th day to 103°7: if it stood the same level on two successive mornings or on two successive evenings; or if it were ever lower than that at the same hour on the previous day. ..... Jenner remarked to the Clinical Society in 1875 that in private practice there were found [illegible] numerous exceptions to one of Wunderlich's rules and thus certainly accords with my own limited experience in the matter. ..... In 1878, a girl was admitted who had been carefully watched from the 3rd day of her illness by Dr. Ingoldsby; the morning and evening temperatures were on that day 103·4 and 105·2 respectively; on the 4th day they were 103·4 and 104·2; on the 5th day 103·4 and 102·3; on the 6th day 103·5 and 104·2." 1.

The fall of temperature to the normal upon the 4th day of this case still strengthens the similarity to Enteric fever but here the comparison fails. The general appearance of the chart is so irregular, the rise is so gradual that notwithstanding the fact that there was abdominal pain and tenderness with gurgling in the right iliac fossa and diarrhoea with pea soup motions the idea of enteric fever was nevertheless set aside. The future course of the disease substantiated the correctness of the diagnosis. No rose spots were ever seen upon any portion of the body, the tongue never showed any characteristic appearances and the pulmonary condition steadily increased in severity and so demonstrated the true nature of the case. The redness, vomiting and diarrhoea, apparently typhoid signs yielded gradually to suitable treatment and now the stools lost their pea soup character.

In an Acute Phthisis, the diagnosis is often extremely difficult. As we have seen the initial site of the lesions may be apical in a great many cases and the rule that phthisis begins at the apex and lobular pneumonia at the base is one subject to many variations. The family history as well as a personal disposition towards pneumonia or tubercular affections point to Phthisis. The bacteriological examination of the sputa is an important aid, but the presence of elastic fibres is not a pathognomonic sign of tuberculosis as destruction of lung tissue alone may have two. The association of tubercular

1 Hillier Tapp. Principles and Practice of Medicine. 1st Edit. Vol. I. p 187.

conditions in other parts of the body such as the peritoneum, the joints, brain, intestines etc are powerful evidence for phthisis.

Acute Pneumonia is said to simulate acute Bronchi-pneumonia but the resemblance is never very close. The physical signs and many of the general symptoms are distinct. Herpes Labialis is very rare, herpes Labialis is frequent and the Chlorides in the urine are undiminished as a rule and the temperature chart differs materially in Bronchi-pneumonia. Resolution is slow, the temperature falls by a crysis and the physical signs are slow in disappearing. The remittent nature of the daily temperature readings is of itself sufficient to distinguish this disease from acute Lobar Pneumonia even when it assumes a quasi-lobar aspect. The only cases in which Lobar pneumonia might present a somewhat similar chart are those described by Polich[1] and by Bernard[2]. Intermittent forms of bronchitis have also been reported by Abeille[3] and by Bouyard[4] but these with any degree of care can be easily distinguished from subacute Chronic Bronchi-pneumonia. Between Chronic Pneumonia and chronic bronchopneumonia the distinction is however somewhat difficult. Chronic pneumonia according to Charcot, Bulyer and others may arise in two different ways:— first, either by a regular continuous and progressive course, succeeding a first attack of pneumonia, or second, by a process of successive phases of interrupted evolution which establishes itself as a result of a certain number of relapses of acute pneumonia in the same portion of the lung.

Auld[5] in the latest work upon the subject, holds that some cases are primarily of a chronic nature.

Chronic Bronchopneumonia on the other hand is always of a secondary nature and follows as I have already stated the specific fevers, measles, whooping cough, diphtheria, etc.

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1. "La Pneumonie Intermittente" Polich. Wein. med. Press. N° 15. 1878
2. "Observations de pneumonie continue rémittente - Union Méd. Paris 14. p165. 1855. also. Bull. de Soc. Hôp. de Paris 1862
3. Bronchite et Pneumonie Intermittente guéries par le sulfate de quinine. Abeille. Paris Méd. XXXI 1874. page 423.
4. Bouyard. Bronchite aiguë intermittente. Journal de Méd. chirurg. et pharm. de Bruxelles XXV page 212. 1857.
5. Auld. The Pathological Histology of Bronchial affections, Pneumonia and Fibroid Pneumonia by A.S. Auld M.D. 1891.

Furthermore, Chronic Bronchopneumonia gives rise to dilatation of the bronchi especially those of lesser calibre while this is rare in Chronic pneumonia. In Chronic Bronchopneumonia too, the deformity of the thoracic cage is much more evident than in the chronic lobar pneumonia. (~~Balf~~ Balzer after Charcot).

Other conditions of the lung have also been described by various authors, which upon post mortem examination have shown similar subacute or chronic forms of bronchopneumonia associated with non-tubercular destruction of lung tissue.

Syphilis and cancer may simulate chronic bronchopneumonia but in the former case we have generally the history along with other lesions to aid us, in the latter we have a marked cachexia, the pain is more intense than in non malignant conditions and the physical signs are somewhat different.

Cases where tumours have compressed the bronchi and have led to destructive changes in the lung have presented marked resemblances to the less subacute or to the chronic types of the disease. Percy Kidd records a case of "destruction of lung following upon obstruction of the bronchi".[1] J. P. Irvine[2] too writes in the Lancet a paper "On the occurrence of collapse, emphysema and destructive pneumonia in association with tumours compressing the bronchi". The clinical aspects of such cases must have presented considerable difficulties during life with respect to their differentiation from chronic tubercular disease or from simple Chronic Bronchopn. Waters seems to have had to deal with cases of subacute Bronchopn. ending in cavity and cure when he wrote his "Clinical lecture on certain diseases of the lung resembling acute phthisis".[3]

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1. Pathological Society of London. P. Kidd. April 21st. 1885.

2. Lancet. Vol. I. p. 415. ~~and~~ et seq. 1878.
Lancet Vol. I. page 123. 1879.
British Medical Journal Vol. I. p. 114. 1879.

3. British Medical Journal. page 1074. 1886.
also Strauss and Bruguy. Rétrécissement de l'artère pulmonaire (symptômes ressemblant tuberculose phthisis) Gaz. hebdom. 28th Dec. 1877
also Blachez. Du rétrécissement avec insuffisance tricuspide ayant donné naissance à des infarctus pulmonaires multiples simulant une phthisie caséeuse. Gaz. hebdom. xxxvii 586. 1877.

Prognosis

In the acute form our prognosis depends upon many points — upon the age of the patient, the previous condition of health, the presence of any chronic disease such as Phthisis, Chronic Bronchitis Emphysema, Heart and Kidney disease and also upon the severity of the initial attack of the grip. Bouchereau makes a remark which must be evident to every medical practitioner whatsoever who has had any experience of influenza when he writes "l'ésprouve tardive des prins est un des facteurs essentiels de gravité"[1] he proves his point by referring to cases which he had under his care in Val de Grâce hospital. These were persons from Bicêtre et Cherche - Midi who were removed immediately the symptoms of the grip had shown themselves. Some of them remained in the hospital wards longer than forty eight hours. On the other hand, the ten fatal cases which he reported had gone about six days before getting medical advice. How often has the same thing been seen by all of us, where business men have continued at work trying to fight out the disease but have at last succumbed to the inevitable. The natural result is that the general strength has been greatly reduced, that the cardiac muscle has been weakened, that the lungs have been subjected to exposure while in a state in which they are least able to resist such a trial and that in short the resistance to septic infection has been much diminished. What wonder is there then that so many strong and healthy adults have succumbed to the pulmonary complications of influenza while confirmed invalids and the weak in general have so often escaped with impunity. When the disease takes on rapidly such conditions of congestive oedema of the lungs with failure of the respiratory and cardiac functions as have been described, a fatal termination is almost inevitable. In subacute cases our opinion is influenced by the extent of the pulmonary tissue involved by the previous condition of the lungs, by the temperature, the sputum and the general health of the patient. It is not enough to recall of the character of the sputum as demonstrated by microscopic appearances; microscopic examinations must be frequently made especially with reference to the presence of micro-organisms. The quantity of expectoration in the 24 hours ought to be carefully measured. I am convinced by experience that if this were done more as a general rule, we would gain valuable evidence as to the progress of the pulmonary lesions. In such cases as these subacute forms of

1. Bouchereau. loc. cit.

bronchiectasis, where not only do the intracalicular expectorations be[illegible] and become ammoniacal in the expectoration but also true pulmonary tissue as well, owing to the breaking down of coalesced areas of lobular condensation which have undergone caseous degeneration, the quantity of the daily evacuation gives the only reliable basis of opinion with regard to the spreading or the cessation of the destructive process. As we have seen already in treating of the bacteriology of the subject, the staphylococcus pyogenes aureus was the principal microbe found in these cases. As a condition of recovery approaches these become scarcer and the prognosis becomes quite sanguine but in some cases along with an increase or a renewal of the bacterial condition, tubercle bacilli make their appearance and at once confirm the invasion of a very frequent and very distressing sequela of many of these cases. Chronic cases may last for many years. Occasional acute exacerbations occur but so long as these are localised to a small portion of one lung and especially to the portion previously attacked, so long as they undergo somewhat of a resolution and so long as the general health remains good and the temperature continues normal between these attacks, one may hope for a fair duration of life. This is more especially true when the social condition of the patient is such as to allow of the best possible food and surroundings and of his remaining in a warm dry climate. When bronchiectasis occurs to such an extent as to ~~prevent~~ permit of the evacuations lying in the cavities for any length of time, we have a new source of danger in the great liability to decomposition especially in the form of butyric fermentation (Bamberger). The fetid odour of the expectoration may set up sickness vomiting and much dyspeptic trouble while septic absorption is almost inevitable. One of the most common signs of the latter, is the frequency of stomatitis during the latter periods of a chronic bronchitis pneumonia.

Chapter IX.

Treatment.

The old adage "prevention is better than cure" applies as appropriately to the bronchopneumonia of influenza. Prophylaxis in this case however, involves the question of the treatment of the initial disease — the first, the only subject with only of two past portions to deal with now, but also one foreign to the matter in hand. There are a few main points to be attended to in all cases of influenza and these I shall briefly touch upon. Absolute rest in bed should be strictly enforced in all cases until the temperature has remained at the normal for some days. In the majority of cases there is an intermediate period succeeding the fall of the temperature and antecedent to the return to the normal - characterised by a condition of subnormal readings. This fact I have noticed very many times. A recent communication to the British Medical Journal with regard to the experience in the French hospital and dispensary practice reported by Louis Vinikas, B.Sc. Paris &c. confirms these observations of my own made six months previously. This is a dangerous time for the convalescent as it marks a very depressed state of the system and especially of the nerve centres which govern the heart and of the vaso-motor system as well. The slightest exposure to chill may lead to discussions ~~result~~ respiratory troubles and to marked increase of these if they already exist. Rest then, in bed preferably, at least in an uniformly warm room must be maintained until all signs of danger have passed. Stimulation with light nourishing diet and with the addition of alcohol in some easily absorbed form if necessary is called for. Systematic disinfection of the respiratory and alimentary tracts even if there be no marked ~~respi~~ respiratory trouble goes a long way in preventing these too frequent complications, and thus applies much more forcibly when these have arisen, for then as we have seen the streptococcus is hard at work. As to the way of disinfectants perhaps no one has been "boomed" so much as oil of Eucalyptus! Professor Fairbrother in an address to the students of the Leeds Medical School prophesied that in the near future we might not be astonished to see the "antimatic-supply" principle carried into the sacred domains of the healing art. These bantering words of his have received an amusing and I am sure to the prophet, unexpectedly early verification.

1. British Med. J. March 14th. 1891. Vol. I. p. 601. "Subnormal temperature in the convalescence from influenza." Louis Vinikas B.S. Paris. L.R.C.P. M.R.C.S. Edinburgh.

By dropping a penny into the micrometers "slot" one was enabled to have sprayed upon one's handkerchief a few drops of oil of Eucalyptus — an elixir of life which would not only prevent but even cure all cases of Influenza. Many practitioners, however, have made continued and careful use of this drug, but the general opinion seems to be that any good obtained by it has been more than counterbalanced by the nausea, the headache and other disagreeable sensations which it has produced. Thymol, terebine, menthol, camphor, carbolic acid, have been all employed and in fact the whole list of remedies official and non-official has been ransacked in the search for some 'real cure'. In my experience, pure creasote has proved of great value. It was prescribed in the form of two parts creasote to one part of Chloroform, ten drops of which mixture were placed upon a glass-oral respirator. The chloroform allayed the slight irritation which the inhalation of even the purest obtainable creasote so often sets up. Where the staphylococcus was found to be very abundant and where the cough was very troublesome the inhalations were ordered to be as nearly continuous as was compatible with the comfort of the patient. The patients soon felt the benefit of the treatment and then it was rare that we received any complaints as to the medicine not agreeing with them. During convalescence nerve tonics are called for and generally there is great anaemia to be remedied as well.

When bronchio-pneumonia shows signs of developing, the treatment must be immediate and energetic. Rest in bed, warmth, stimulation, light nourishing diet, a purge at the onset if required and the administration of sedatives and drugs which will increase the pulmonary and bronchial secretions, are all called for. Local applications are of great use. The skin over the affected area may be freely though very lightly touched with the finest point of a Paquelin's cautery. This acts quite as well as a blister, is not nearly so painful as one might imagine, it is applied into Cave and gives no trouble in the way of dressings. Blisters or sinapisms do in place of the cautery when it cannot be obtained. Large jacket poultices containing mustard in the first one or two and changed every three hours often give great relief when the condition at all tends to be suffocative from. Stimulation in almost all cases is called for especially in the aged, and I have been often surprised at the amount of alcohol which is required to give any definite results as recognised by the pulse. We must never forget the fact of a septic toxin in these cases. Inhalations charged with terebine and Chloroform were administered by me when the cough was of a very irritable nature. In other cases the glass-oral respirator was used freely, a mixture of creasote and Chloroform as already described being the antiseptic and sedative employed. Capsules, each containing one minim of pure wood creasote were administered internally. The dose was pushed until disturbing effects of the drug were noticed and then the quantity was reduced for a few days and steadily increased and so on.

Case II, I consider, owed her life to the remedy. On Nov. 3rd two capsules were ordered to be taken thrice a day. The temperature was rising and the quantity of sputum was increasing daily at this time, and the chest symptoms were in no way improved. On the 9th November, 250 grammes were expectorated but from this date, the quantity steadily diminished with the exception of a rise to 225 grams on Nov. 12th. The temperature was now falling but on account of the increase of sputum on the 12th the patient was ordered 6 capsules 3rd daily. On the 17th Nov. an increase to 16 capsules and on the 28th Nov to 20 capsules daily (equivalent to 2 ounces) was ordered. By the 18th Nov. the temp. fell to 99°7 in the morning and 100°8°7 in the evening and from this time until dismissal nine then a month later it continued normal. From the same date the sputum fell from 120 grams to 30 on dismissal besides entirely changing its character from a blood tinted purulent mucus to a mucous with scarcely any purulent taint. At the same time Creasote and Chlorform were inhaled by this patient at least four times in the 24 hours and in fact when awake the respiration was scarcely off her mouth. She occasionally complained of slight sickness and once of slight griping pains but as will be seen by the chart no such thing as diarrhoea was ever produced. In this case besides administering creasote as well as in four other cases, I tried a new form of treatment which was greatly lauded by some of the French and German authorities. This was the hypodermic injection of a solution of Camphor in olive oil in the proportion of 1 in 10. One gramme of the solution was injected into the subcutaneous tissue daily under careful antiseptic rules. No local disturbance has ever set up but slight paleness of the head soon began to be complained of after the fourth or fifth injection. A day was missed then and the remedy continued. In this case as well as in two other cases of a like nature the temperature seemed to fall after the first four days of the treatment and certainly the sputum lessened somewhat also, but the improvement if small there were ever any was of a very transient character and finally, I gave up the treatment in all the cases, seeing that it was proving of no use. In these dangerous cases of the Acute or Subacute type, where the heart seems to be becoming rapidly weaker and where failure of the respiratory centre is imminent, the use of Strychnine by the mouth or hypodermically along with the employment of inhalations of Oxygen has produced marvellous results in some cases such as these reported by Lauder Brunton and Marmaduke Pricket and others.[1] The strychnine stimulates the respiratory nuclei

1. Brit. Med. J. Jan 23rd 92. Vol I. p172. On the use of Oxygen & Strychnia in Pneumonia. Brunton & Pickett also B.M.J. Vol I p327 — A.N. Gilchrist M.D. also B.M. Journal. Vol I. p436. J. Cowper Griffith M.D. also in treatment of Influenza Pneumonia J. Rochard. La pneumonie Méd. Feb 13. 1892.

in the medulla while the oxygen inhalations supply the necessary amount of ~~that~~ gas required by the organism even under the most adverse pulmonary and cardiac conditions. By means of this treatment we may tide a patient over a severe and otherwise fatal crisis by immediately strengthening the muscle and so giving time for the organism as a whole to respond to the double stimulation. In subacute cases, when the conditions tend to become localized some portion of a lung counterirritants may be employed but in my hands they proved of very little service. In those repeated slight attacks of pleurisy, pneumonia or pericarditis of a acute nature which are so apt to occur in the latter chronic cases, counterirritants seemed however in many cases to materially assist in cutting short the attack.

The main point in the chronic cases is to maintain strength and to prevent auto-infection. Light nourishing diet, stimulation, rest in bed or if able quite exercise in an equable temperature are all demanded. To lessen perspiration at night and to insure sleep, sulfonal has proved invaluable. Given in doses of 20 grains in hot milk or hot whisky and water as a potion, one to two hours before falling asleep, it checks sweating, gives a calm night's rest and is not followed by thirst or other disagreeable symptoms such as are only too often complained of when atropine has been administered. To prevent auto-infection from the decomposition of the bronchial secretions in the bronchiectatic cavities or in the pronicae as well as in the alimentary canal owing to the patient swallowing the sputa, creasote was given by me in large doses and with the most happy results. I have already described its ~~use~~ mode of administration in Case II. Its utility in all these other cases which presented a more or less chronic is proved by the fact that in more bad ~~cases~~ sputum even a disagreeable odour. Even in case VII which lasted for two years and which presented such a cavity as has been described in the post-mortem records this showed a splendid result.

As the various complications which have been enumerated, they call for their own appropriate treatment which need not be discussed now.

Surgical interference would prove of service in acute or subacute cases where a local abscess or focus of gangrene has been diagnosed. In such cases a portion of rib may be removed the abscess or gangrenous area cut down upon, the contents cleared out and the cavity drained and allowed to heal by granulation. Brilliant results have rewarded this somewhat recent feat of modern surgery.

Thos Bond.

Synopsis of Cases not already recorded — of the subacute and Chronic Forms.

	Case III. C—r.	Case IV C—s	Case V H—y.	Case VII Mc—n.
Debut	Symptoms of influenza: great weakness, diarrhoea, right iliac pain and gurgling: no rose spots bronchitic symptoms: signs especially at bases where percussion note tends to be dull. temperature 99.6 on admin anaemia marked.	Giddiness, weakness, painless diarrhoea. cough & pain in the side a week afterwards. Great anaemia with some cyanosis. slight cough and spit.	Influenza, then caught cold, cough & spit. pulmonary condition gradually increasing.	Influenza followed in a few days by cough & spit.
First stage of Pulmonary Inflammation. Bronchitis with pulmonary engorgement.	Four days after catching cold. there were noticed abundant bronchial râles of a moist type with slight dulness at both bases. Spit bronchitic	Two weeks after cough and pain arose and three weeks after onset of influenza, we noticed dulling of percussion note over left apex front and back & at left base. R.M. weakened. Subcrepitant & bronchitic râles.	Apparently bronchitis at first.	apparently bronchitis slight at first but increasing in severity.
Second Stage of pulmonary inflammation. Consolidation	Bases fairly dull with somewhat moist crepitant and bronchial râles. at apex of each lung dulness and subcrepitant râles	Three weeks after onset of grip at left apex, R.M. weakened tubular. subcrepitant râles during both phases of resp. with piping râles especially in expirat when they become sonorous upon prolonged breathing. percuss. note dull. R.M. cogwheel. At right apex	Seven months after influenza flattening of note all over front of chest. R.M. weak expir. prolonged. subcresp. râles during insp. with piping râl. in exp. pleuritic friction in left infraaxillary region. Behind whole left side gives same flattened note of heightened pitch. dry resp. râles becoming subcresp. toward apex. pleuritic friction at base	Seventeen months after influenza. diminished expansion at both apices râle percutus marked. percuss. dull tympanitic point with tendency to a pit at apices. dull in left subclavi sound in point prolonged: R.M. tubular and harsh. puerile upon right. weak left apex with subcresp. fairly dry r

		whistling & piping bronchit. rales. Behind dulness of whole left side. duln. at apx. & base. Voc. sounds unaltered. R. M. weak all over & absent at extreme base towards spine where only a thin pipe is heard upon forced breathing. deep insp. echoes upon whole left side bronch. rales. On right side puerile exaggerated R. M. occasional bronch. rales. temp. oscillates about 100 to 102. evening exacerbations. resp.-ratio 1 to 3·9. (25 to 98) Spit mucopurulent. cyanosis. appetite poor. bowels still loose.	towards infra axill. region. upon right side R. M. tubular under. subcrep. râl. in insp. piping at end of exp. R. M. puerile in both axillae. Decubitus sitting. sallow earthy pallor with lividity. hard purpuries. nails clubbed. resp. 19. p. 115. 116 64 temp. fallen from 106 to 101·4, oscillates. sputum tenacious semigelatinous rounded masses of greenish muco-pus with occasional fibrinous casts. flattening of apices.	with somewhat of a bronch. creak in insp. and piping in exp. = prolonged. Upon right, loud crackling sonorous & large bubbling bronch. r. Voc. R. & F. somewhat equal in both sides. Behind diminished expansion at le[ft] slight duln. at apx. distinct at scapul[ar] angle to base where absolute. Voc. F. [illegible] absent at very base. Voc. Res. lessened. R. M. weak and tubular with subcrep. rales in insp. a crackling bronch. r. in exp. No R. M. at very base but pipe. r. in [illegible] exp. pleural fr. at left subaxill. region [illegible] note to percuss. is dull. On right side R. M. is breezy and tubular with few subcrep. r. here and there. Resp. 32. p. 90. ratio 1 to 2·8. Heart apex impulse in 5th space & also in nipple line. 1st sound booming. 2nd rather accentuated. pulse good. some cyanosis of extremities. skin more jaundiced than of earthy pallor. bowels reg. very little sweating.
Third Stage of pulmonary inflam. Softening	Twenty four days after gasp. duln. at apx. increasing as well as at base. rales muster & bubbling. profuse sweating. tendency to delirium at nights. earthy pallor of face. sickness & often vomits after fit of coughing. emaciation. temp. daily remittent. cough hacking. often in severe kinks. pleuritic stitch in left subaxill.	Six weeks after gasp. 5 after onset of symp. rales moist at all parts especially at left side. pleurit. stitch. planing at right mam. reg. emaciation rapid. cough bad. spit nummulo-globular green masses / mucopurulent without much air. no blood. Resp. 23. p. 114. 1 to 4·9 resp. laboured. catching. decubitus sitting up. temp. remittent erratically. 103 often at night. resp. often 32.	Seven months and one week after onset of illness emphysema in front especially at right side. rales rather muster at left apex.	nineteen months after onset of sy[mp.] rales more frequent & more moist upon left side. spit muco-pu[rulent] pulse resp. ratio 31/52 1 to 2·87 temp. rises & falls as in other cases but rarely to 100 or 101·7.

Synopsis of Cases — Continued.

	Case III. C— r.	Case IV. C—s	Case V. A— y.	Case VII. O— n
Fourth Stage. Formation of Cavities and Fibroid Contraction.	Spit more abundant and more purulent. hectic state worse. râles at apices more bubbling. delirium. frequent. Cheyne-Stokes resp. Death in Coma. temperature fell rapidly before death.	Large bubbling râles at left apex. profuse sweating. dyspnoea. great weakness. Thrombosis of left iliac and fem. veins two days before death.	Behind at upper portion of inner border of Scap. there is a cavity which gives all the usual classic signs. Above and below this, the râles are bubbling especially at extreme left apex and base. Upon right side the râles are dryer. subcrep. Rhonchi. r. more common. [illegible] loud & harsh. Voc. Res. & Frem. not so well marked as upon left side. Inspiration of chest. Heart. [illegible] of 1st. sound at apex especially right. 2nd sound accentuated. area of dulness. [illegible] lungs, liver and spleen painlessly enlarged & somewhat depressed. [illegible] heavily. bowels tend to be loose. average p. resp. ratio is 21/104, 1 + 4.95. temp. varies from 98.2 in morning to 104 as evening maximum. Cyanosis increasing. oedema of feet & legs. some delirium. death from heart failure. duration of disease 8 months.	Apparently a cavity within angle of left scapula. there moist bubbling râles, aegophony & some whispering pectoriloquy. left apex dull. also subaxilla & whole base. râles moist at apex. subcrep. at rest of left side. contract. of lower 2/3rds of left chest. only 1/4 inch of expansion on insp. circumference 17 inches. On right side 1 3/4 & 3/8 inch of expansion. Right side of chest. dulness commencing at apex & slightly at base. moist râles at apex. emphysema at other parts very evident. Spit greenish round masses of muco-pus. lie only on left side or sits up. temp. intermittent. occasional sweating. [illegible]. bowels tend to be loose. Died Jan 18th with acute [illegible] on right side.
Complications.	At debut, diarrhoea and griping with tenderness at right iliac region.	Began with giddiness, weakness & diarrhoea of painless nature. later on 14 days after admiss. griping & diarrhoea. latterly [illegible].	After some months, dyspeptic symptoms. never any haemoptysis. pleuritic friction in left subaxill. region. emphysema.	Emphysema upon opposite side. pleural friction in left subaxilla, retraction of chest. but not enough to depress scapula.

	loss of appetite later on. occasional sickness & vomiting. delirium & Cheyne Stokes resp with coma before death. frequent pleuritic stitch in left subaxilla. grunting during sleep.	has been occasionally rusty, never any real haemorrhage. 2 days before death thrombosis of left iliac & fem. veins. slight delirium. death in coma from heart failure. grunting during sleep.	accentuation of 2nd sound & reduplic. of same at pulm. area. liver & spleen depressed & enlarged. oedema of feet & legs. with heart failure before death. grunting during sleep.	accentuation of 2nd heart sounds. Ecthyma upon back & also in axillary glands. colic & diarrhoea once. Grunting during sleep.
Duration.	9 weeks 3 days or 66 days.	9 weeks 3 days or 66 days.	almost 8 months.	almost 2 years.
Post-mortem appearances.	Heart pale & flabby. much venous clot. Lungs adherent by recent membranous adhesions easily broken down. Appearances show that the second and third stages of bronchopn. have been attained at the apices while both bases still retain the appearance of carnification. Caseation & cavities at both apices. Liver. nutmeg. Spleen. enlarged & engorged. Kidneys venous congestion. Brain " " with slight basal meningitis.	Chest. 3ii fluid in pericard. heart muscle pale & flabby: cavities full of clot. Lungs. adhes. slight at mammary reg. at upper lobes in each lung. caseous bronchopneumonia in 2nd and 3rd stages. several large cavities. carnification at bases. Pleura - visceral layer - much thickened. much emphysema. Liver - nutmeg. enlarged. 1 kilo. 710 gramm. Intestines venous engorgement. Spleen. enlarged 200 gramm. Kidneys venous congest. Simple thrombosis of veins. Brain venous engorgement.	Heart. slight mottling of endocardium. full of clot. Lungs both upper lobes firmly adherent. Caseous bronchopn. in both. with cavities at apices. and this fades away towards base through areas of carnisation to simple oedema. much emphysema. bronchial calculi frequent. Liver nutmeg. 1 kilo 980 gramm. Kidneys. congested. Spleen. large. soft purple.	Heart. enlargement of right side with some dilatation of right ventricle: heart soft. Lungs. Right lung slightly adherent at apex. lung presents a fairly general & acute bronchopn. passing towards the end of the first stage especially at apex. left lung. Caseous bronchopn. with large irregularly walled cavity at left base. Spleen. engorged. enlarged. Liver - nutmeg.

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Bibliography.

Historical. Besides the authors already quoted the following may be referred to specially. For an exhaustive bibliography of the Grippe the reader is referred to the Dictionnaire Encyclopédique des Sciences Médicales. 1888: to the Dictionnaire des Med. et de Chirurg. Pratiques. 1880. Paris. The Index Catalogue of the Library of the Surgeon General's Office U.S. Army. Washington. 1881. "Authors & Subjects".

Althaus on the Pathology of Influenza. 1892. London. Thompson. Synoptic table of Influenza & Epidemic Catarrh of 1239 to 1837 in Med. Review Vol XX 1st series.

also:— Valleriola. Loc. med. comm. append. J. Epidem. Catarrh. France 1557.

Buoni Segni. Epidemic Catarrh in Italy in 1323. Baillou — Ephemerid. et Epidem 1762. Paris. (Epid. of 1570. 71. 74. 78.)

Willis — Opera Omnia — (de febribus Vol. I.) Amsterdam 1682. Epidemic of 1658 in England. Ettmüller Mich. Febres et morbi catarrhales epidemici Leipsic 1683. Sydenham Opera. London 1676. Lancisi. Opera. Epidem. 1709 & 1717. Essays & Observations of the Medical Society of Edinburgh — Epid. of 1732-33.

Fordyce. The Catarrh. Edinburgh 1758. Fothergill. Sketch of the Epidemic disease which appeared in London in 1775. Whytt. The Epidemic of 1758 in Scotland.

Monro. Medicine in the Army Epidem of 1743. 1762. Macbride. Epidem. in Dublin 1729 to 1762. Watson. Philosoph. Transact. Epid. of 1762.

Gilchrist Eben. Observations on the Cat. Epid. of 1762. In Ess. & Observ. Phys. & Lit. Soc. Edin. Vol III 1771

Baker G. De Catarrho et dysenteria Londinensi epidemicis utrisque an 1762. libellus. London 1764.

Huxham. J. Essai sur les fièvres (de peripneumonia notha etc. p 268. Paris 1764.

Heberden. The Epidemical cold in June & July 1767. Med. Transact. London

Jussieu. Thèse med. Paris 1733. Grant. A short account of the epidemic cough & fever 1775 in a letter to Dr. Delacour of Bath. London 1776. Cullen. Synopsis nosolog. method. Edit. tertia Vol. II. Class I. Ord. V. Influenza gen. catarrh. catarrhus contag. Edinburgh 1780.

Hamilton R. Some remarks of the Influenza that appeared in Spring in 1782. In memoirs of the Medical Society of London. Warren. Description of the Influenza of America 1782. Memoirs of Med. Soc. London. Falconer. Account of the epidemic catarrhal fever called the Influenza. London 1782. Currie. A short account of the Influenza which prevailed in America in the year 1789. Annals of Influenza. Watson letter to John Huxham, Gilchrist etc. Epidem. of 1762. Annals of Influenza { Thos Thompson. Pringle. Heberden. Baker. Revell. Reynolds. Cumming. Glass. Lithe. Ash. Haygarth. etc Epid. of 1775.

Annals of Influenza. Gray. Leith. Houlston. Clephorn. Cammichael Smyth. Haygarth. de epidem. of 1782. John Warren Epidem. of 1789. 90. Memoirs of Med. Soc. London. Epid. of 1803. Vol VI. Dunglison. Synoptic table of Epidem. Catarrh of 1239 to 1837. in Med. Review. Vol XX. 1st series.
Annals of Influenza or of Epid. Catarrh. Fever in Great Britain from 1599 to 1837 & Theophilus Thompson. Nonat. Recherches sur la grippe et sur les pneumonies observées en Fev. 1837. in 8°. 1837. Landau. Mémoire sur la grippe de 1837 et sur la pneumonie considérée comme symptôme essentiel de cette epidem. In Arch. de Med. 1837. Sandras. note sur la nature et le traitement de la pneumonie bronchite epidemique qui a accompagné la grippe. In Bull. Gen. de Therap. 1837. also Considerations sur la pneumonie qui a regni à Paris conjointement avec la grippe et sur son traitement. Epidemic of asphyxiating grip at London 1835. Dujardin Bronchites fébriles epidem. Thèse de Paris 1847. Influenza at Dublin 1846-47. See account of acute Bronchitis in Churchill's diseases of Children page 368. 3d edit. James R.T. Lessons on clinical Medicine 1862. Durand Fardel Bronchopneumonia in adults during an Epidemic of grip in winter of 1852-53. Paris. Pidoux. Considerations sur la variété de la phthisie 1860 et introduction à une doctrine nouvelle de la phthisie pulmonaire – Union Med. 1865. Lovett. T. Remarks on the epidemic influenza of 1861 and of 1863 with notices of some malignant forms of this disease. American J. of Med. Sc. Philadelphia 1864. new ser. XLVII p 65–80. Henderson. On the influenza recently prevalent in Glasgow. Glasgow Med J. 1847. Trapenard. G. De la Grippe de l'hiver 1880. Soc. de Sc. Méd. de Gannat. Compt. Rend. Paris 1881. ~~XXV~~
Vogel. Die Influenza vom Jahre 1881. in Wurtemburg. Report de Hirsch. Stuttg. 1883. Saillant Tableau historique et ~~[illegible]~~ raisonné des epidémies catarrhales vulgairement grippe depuis 1510 jusqu'en 1780 Paris. 1780. Annals of Influenza 1775 to 1782. Bergmann – Bronchopn. Grip. et endocardite ulcereuse à pneumocoques etc. Bull. Soc. anat. de Paris. LXI p 253. 1886 also Progrès Med. Paris IV p 621. 1886.
Alison. La grippe. Arch. gen. de Med. April & May 1890. Vol I. p 451. 571.
Bunnet. Pyaemia and suppuration in Influenza. Lancet. 8th Feb. 1890. Vol. I. No 6.
Peter. Complicat. pulmon. de la grippe. France Med. Jan 1890. p 52. 1890. Grasset. Leçons sur la grippe de l'hiver 1889-90. Montpellier and Paris 1890. Villard. Leçons cliniques sur la grippe à propos de l'épidémie de 1889-90 faites à l'Hotel Dieu de Marseille. Paris 1890.

Pathological. Traumatic Pneumonia and effects of section of the Vagi.

Morgagni. Epistolae Anatomicae XIII Chap. XX. 1740. Legallois. Experience sur le principe de la vie. Paris 1812. also Oeuvres complètes avec notes de Pariset. Paris 1830.
Magendie. Précis élémentaire de physiologie Vol. II. 1825. Reid. Edinb. Med. Surg. J. 1839-39.
Traube. Die Ursachen und die Beschaffenheit derjenigen Veränderungen welche das Lungenparenchym nach Durchschneidung des Nerv Vagi erleidet. Berlin 1845.
Schiff. Die Ursache der Lungenveränderung nach Durchschneidung der pneumog. Nerven. In Arch. für physiol. Heilk. p 691. 1847. also "Ueber den Einfluss der Vagusdurchschneidung auf das Lungengewebe in Arch. f. physiol. Heilk. p 624. 1850. Billroth. De natura et causa pulmon. affectionis quae utroque vago dissecto exoritur. Diss. Berlin. 1852.
Chauveau. sur le rôle de pneumogast. dans la déglutition. Comptes Rendus 24 March 1862. Boddaert R. Recherches expérim. sur les lésions pulmonaires consécutives à la section des nerfs pneumogast. Gand 1862. in Journal de physiol. Vol VI p 323. 442. 1865.
Rugenberg. Ueber den angeblichen Einfluss der Nervi Vagi auf die Lungen. In Stud. des physiol. Laborat. zu Breslau p 47. 1863. Ogle. Remarks on changes in nutrition dependent on altered nerve influence. Med. Times & Gaz. Nov. 1866. Robin Ch. Sur les nerfs dits nutritifs ou trophiques. In journal de l'anatom. et de la physiol. Vol IV p 276 1867. Handfield Jones "Are there special trophic nerves" St George's Hosp. Rep. V. III p 89. 1868.
Section of Vagi Brown. Pn. Article "Trophiques Nerfs" Dict. Encyclop. des Sc. Med. 1871.
Traube Gesammelte Beiträge I p. 1. 1871. Genzmer. Gründe für die pathol. Veränderung der Lungen nach doppelseitiger Vagusdurchschneidung. In Ateplice etc 1873 Berlin.
Frey O. Die patholog. Lungenveränderungen nach Lähmung der Nervi Vagi Leipzig 1877.
Brown-Séquard. Recherches sur une influence spéciale du système nerveux sur les vaisseaux. Comptes Rend. Vol XCIV No 8. 1882. Gaskell. On the structure, distribution and function of the nerves which innervate the visceral & vascular systems. Journal of Physiol. VII. No 1. 1886. "Are there trophic nerves? Yes" in Med. Rec. 2/76. pp 382. 385-388. also Med. Times 2/79. p 451. 2/83 p. 183. also Med. Rec. 83. p 309. ~~Lancet~~ also Lancet 1/78. p 207. Pneumoparesis in Influenza. Asclepiad. 1890. p. 40.
Pneumoparesis distinct from pneumonia. Asclep. 1888. p 1. Lancet 2/87. p. 1221. Ed. Med. J. 1/1888. p. 28. M. Gley. Physiologie du pneumogast. Soc. de Biol. Compt. Rend May Aug. 1885. Guerrier and Amorio. Pneumonia after section of the pneumogastrics Anal. M. D. O. H. Huchard. Angine de poitrine. Remarques sur les ~~syncope~~ Synergie morbides du pneumogastrique. Union Med. 1879. Anstie. Pathol. & Therap. relations of asthma of angin. pect. & of gastralg. Brit. Med. J. 1872.

Haberslon. Gulstonian Lectures on pneumogast. nerves Lond. 1877. p. 46. ~~Teale~~.
Teale A ~~treatise~~ on neuralgic diseases dependent upon irritation of the spinal marrow & ganglia of the sympath. nerve. Philadelph. 1830. Eloy. Ch. Les synergies centrales du nerf pneumogast. Union Méd. No 9. Jan 20. 1883. Balzer. Pulmonary haemorrhage as a result of trouble of innervation, article "Poumons" (Haemorrhagie). Dict. de Méd. et de Chir. Prat. No XXIX. M. P. Cogne. Broncho pneumonia in children & old people from passage of food and drink into bronchi. Gaz. d. Sc. Méd. de Bordeaux. No 14. 1880. Henri Huchard. Angine de poitrine cardiaque et pulmo. paralysie consécut. du nerf pneumogast. Union Méd. 16th Sept. 1879. No 108.
Masaloff A. Contribution à l'étude expérimentale de la pneum. et de la bronchopn. Arch. de physiol. norm. et pathol. Paris. VI p. 526-532. 1888. Vaillard A. Origine nerveuse et parasitaire de la pneumonie franche et de l'angine herpétique. 8° Paris 1887.
Crane. Case in which prune juice & rusty coloured sputum were present without pneumonia. Med. Times Lond. 1852. Vol V. p 234.

Bronchopneumonie Aiguë & Chronique.
Besides consulting "Les Maladies des Enfants" by Rilliet & Barthez, the treatises of Damri, Bouchut - West. d'Espine et Picot, etc. Jaccoud. Roger in Dict. Encyclop. d. Sc. Méd. & Balzer in the Dict. de Méd. et Chirurg. etc etc. see also the following.
G. Sée Indications comparatives dans les bronchopneumonies et les pneumonies in "Les maladies des poumons" Vol II. Eustace Smith. on Diseases of Children.
Sopporitch D. Contribution a l'étude de la pneumonie catarrhale. (Angine de poitrine). 4° Paris 1886. A. Macdonald. Two cases of puerperal pneumonia Tr. Ed. Obst & Soc. 1876/77/78. IV. 349. 365. Kehra. pneumonia puerperalis. Amtl. Ber. d. K. K. Allg. Krankenh. zu Wien. 1867. 68. p. 122. Delafield. The pathology of bronchopn. Bost. Med & S. J. 1884. p 111. Oreste. Potain - La diagnostic différentiel de la broncho et de la pneum. congest. Rev. gén. de clin. et de thérap. Paris 1887. Vol I. 609. also Gaz. Méd. de Picard. Amiens. 1888 Vol VI. 567.
L. Mackey. Case of bronchopn. with high temp. lowered by warm bathing. Lancet. 1885. 2. p 617. Belcastro G. Storia d'una grave peripneumonia puerperale con anasarca acute idrotorace. Giorn. anat. di med. Milano. IX. 35. 43. 1828.
Baulle. puerperal pleuropn. Dublin Hosp. Gaz. N.S. VI 370. 1859.
Thompson W. H. Bronchopn. Med. Rec. N. Y. 1878. XIV. 327.
Vulpian. Bronchopn. chez un alcoolique: point de côté avec violent: frisson -

Clin. méd. de l'hosp. de la charité. 8°. Paris. 358-360. 1879.
Vulpian. Des pneum. secondaire. 4° Paris 1860. Serio. Cy. Epidem. di broncho-pneumonia tifo osservata in Rivalta Joniese. Ungagni Napoli XVII 903-916 1875. Brollman. E. F. Nine cases of bronchopn. Madras Monthly J. of Med. Sc. Vol III. 191. 200. 1871. Anderson A B. Broncho-pneumonitis. Atlanta Med. Surg. Journal XV. 329. 331. 1877. Joffroy. A. Des différentes formes de la bronchopn. 4° Paris. 1880. Balzer. F. Contributions à l'étude de la bronchopn. Versailles 4°. 1878. Mosny. E. Étude sur les lésions histologiques et les causes bactériennes de la bronchopn. Thèse d'agreg. Paris 1891. Sée. G. Maladies spécifiques non tuberculeuse du poumon. Paris. Hamilton. Bronchopn. practitioner 1881. Mosny. Bronchopn. érysipélateuse. Acad. de Médecine Feb. 11. 1890. Fauvelle. Formes cliniques de la pneum. 1887. Thèse. Paris. Gillet. Pneumon. chez les jeunes recrues Thèse 1887. Lossowitch. Pneumon. catarrh. Thèse de Paris 1888.

Val la Berge. Recherches sur la pneum. lobulaire observé à l'hosp. des enfants malades de Paris. Journ. hebdom. des progrès de Sc. et Institut. Med. 1831. Vol II & III p. 1014. 1015. Valleix. Clinique des Maladies des nouveau-nés. 1838. p. 140. Fauvel. Recherches sur la bronchite capillaire purulente et pseudo-membraneuse (catarrhe suffocante, croup bronchique) chez les enfants. Thèse. Paris 1840. No 112. also De la bronchite capillaire suffocante chez l'adulte. Arch. gén. de Méd. Series 3. Vol IX p. 261. 1841. also recherches sur la bronchite capillaire suffocante (catarrhe suffoc.). etc. Mem. de la Soc. méd. d'observat. Vol II. p. 433. 1844. Gairdner. On the pathology. state of the lungs in connection with bronchitis etc. Monthly Journal XI & XII 1850-51. Addison. Capillary Bronchitis & Pneumonia Pn. Lancet Vol II 1855. p. 357. Franke France Méd. 26th July 1856. Güntherer. de pneumon. infant. et de temperatura differentiis in ea observat. 1859. Vulpian. Des pneumonies secondaire. Thèse de concours d'agreg. Paris 1860. Bremner. Zur theorie und anatomie der Bronchien-veiterung. Virchow's Arch. für pathol. anat. Berlin 1860. Bartels. Studien etc. Virchows Archives of Path. Anat. p. 21. 1861. Jeunisen. Pleuritis und Pneumon. in Kindesalter Berlin 1862. Damaschino. Des différentes formes de la pn. des enfants. Thèse Paris. 99. 1867. Buset. Pneumonie lobulaire chez l'adulte. Thèse. 1872. Paris. Rindfleisch. Traité d'histology pathol. trad. franc. Paris. 1873. Friedländer. Untersuchungen über Lungenentzündung. Berlin 1873. Roeber. J. C. Parallèle différentielle entre la pneum. franche.

et la bronchop. chez les enfants. Thèse Paris 1875. Consult also
Charcot's thesis, Résumé in Revue mensuelle de Med. et Chirurg. 1878. also
Charcot's leçons clin. sur les malad. des viellards et les maladies chroniques.
1867. invédit 1874. also Rilliet et Barthez. Mem. sur la pneumonie
vesiculaire chez les enf. rev. med.-chir. XV. 129. 136. 1852. also their
Maladies des Enfants. 1838. also their Mem. sur quelques parties de
l'histoire de la bronchite et de la broncho-pn. chez les enfants. Arch. gen. de
Med. 4th series Vol. 28. Page 129. 272. 1851. Legendre. F.J. Recherches
anatom.-pathol. sur quelques maladies des enfants. p. 157 Paris 1846.
Legendre et Bailly. Nouvelles recherches sur quelques maladies du poumon
chez les enfants. Arch. gen. de Med. 4th series Vol IV. p. 55. 186. 235. 1844.
also their Bronchopn. chez les enfants. Arch. de Med. 1844.

For Chronic Pneumonia. consult

the works upon Pathology anatomy of Hasse, Rokitansky, Lebert,
Förster, Cruveilhier, Rindfleisch, Cluld, etc. also article –
Cirrhose du poumon by Henri Barth in Dict. Encyclop. des Sc.
Med. – F Balzer. Subaiguë & chronic Bronchopn. – Dict. de Med. et Chir.
Prat. See both for bibliography especially of French authors. also
Stokes. Diseases of Chest. Dublin. pp. p. 353. 1837. Corrigan. Cirrhosis of the
Lung. Dublin J. of Med. Sc. Vol XIII 1838. Morgagni. Lettres. 19. V. 7.
Chomel. Dict. in XXI vols. Aumbruzger et Corvisart. Nouvelle méthode et
le squirrhe du poumon. p. 297. 1818. Hope. Principles and illustrations of morbid
anatomy. p. 15. London 1834. Bouillaud. Dict. de Med. et Chir. Paris. Vol viii 1835.
~~Stokes~~ Andral. Anat. Path. Vol III. p. 516. & Clin. Med. Vol III. p. 462.
Corrigan. Cirrhosis of the lung. Dublin J. of Med. Sc. Vol XIII 1838. Cruveilhier, Atlas
d'anatom. patholog. I. Raymond. Thèse de doct. de Paris 1842. Addison. W. Observations
on Pneum. & its consequences. Guy's Reports. 2nd Series 2. p. 365. Oct. 1843.
Legendre et Bailly. Nouvelles recherches sur quelques maladies du poumon chez
les enfants. Arch. de Med. 4th Series Jan. Vol IV. 1844. Rat. Thèse Paris 1845.
Rayer. Gaz. med. de Paris 1846. Lacour Thèse de doct. Paris 1847.
Marquet. Soc. Anat. p. 238. 1847. Paget. Lectures on inflammation and
ulceration. Sect V 1850. Bricheteau Traité des maladies chroniques de
l'appareil respiratoire. 1851. Durand Fardel Malad. des viellards 1854.
Cotton. Med. Times & Gaz. March & April. 1855. Peacock. Ed. Med. J. 1855.

Wunderlich. Pathol. und Therap. Band III. 1856. Walshe – Case of
cirrhosis of lung & bronch. glands. Med. Times & Gaz. Vol I. p. 156. 1856.
Heschl. Vierteljahrsschrift für pract. Heilkunde XIII 1856. Mugnier in
Gaz. Hebdom. Paris. p. 675. 1856. Lebert – Physiol. Patholog. 1845.
Vol. I. p. 137–421. Traité d'anatom. path. Vol. I. p. 668. 1857.
Corrigan. Dublin Hosp. Gaz. 1857. Bennet. Clin. lect. Edinburgh 1857. p. 643.
Charcot. De la pneumonie chronique. Thèse de concours d'agrég. Paris. 1860.
Pneumonie chronique double avec dilatation des bronches et néphrite album.
Soc. Anatom. Path. de Bruxelles. 1861. Brian. Rech. sur une forme parti-
culière de pneumonie chronique. Gaz. Hebdom. 1862.
Cornil. Pneumon. parenchymateuse aiguë entée sur une pneumonie interstitielle
chronique: érysipèle erratique: phlébite: vaginite suppurée: autopsie.
Rec. de Trav. Soc. Med. d'Obs. de Paris. II 574. 610. 1859. – 1863.
Pauli. Gaz. Hebdom. 1862. Grisolle. Traité de la pneumonie 1841. 2d Ed. 1864.
Cornil. Anatom. pathol. des diverses espèces de pneum. aiguë & chronique.
Gaz. Hôp. de Paris XXXVIII 426. 1865. Chvostek. Beiträge zur pathol.
des Greisenalters Induratio pulmon. Oester. Zeitschrift für prakt. Heilkunde
1866. Chvostek. Studien über die primäre chronische pneumonie
Oester. Zeitschrift für prakt. Heilkunde. 1867. Jaccoud. Clinique med. Paris 1867.
Skoda. Ueber chronische pneumonie. Allg. Wien. Med. Zeit. 1868.
Cavasse. Pneum. interstit. du sommet du poumon. chez les vieillards. 1868.
Wilson Fox. Pneum. chronique Reynolds' System 1870. Bastian Ch. Cirrhosis
of Lung. in Reynolds' System. 1870. Henri Barth. Cirrhose du poumon.
Dict. Encyclop. des Sc. Med. 1871. J. Balzer. Sub-ac. Pneum. Bronchopn.
Dict. de Med. et Chir. Green J. H. Pneumonia. Trans. Path. Soc. XXIII 37. 42
1872. Thierfelder & Ackermann. Ein eigenthümlicher Fall von Hypertrophie
und epithelialer. Hepatisation der Lungen. Deutsche Arch. Arch. für Klinik.
Med. Leipzig. Band X. p. 207. und Tafel V. 1872. Green. Interstitial Pneum.
Trans. Path. Soc. London. 1872. Renda. Bulletin de la soc. anat. p. 207 1872.
Brouardel. Note sur la pn. interstitielle qui accompagne la pleurésie et les
indications qui en résultent au point de vue de la thoracentèse. Bull. de la Soc.
Med. des Hôp. 1873. Achard de Salandière. Pneum. fibreuse à résolution
lente. Thèse de Paris. 1873. Rendu et Tuchapet. Angine ulcéreuse chronique
considérée d'abord comme syphilitique plus probablement de nature scrofuleuse.

dilatation des bronches et pneumon. chronique, s'accompagnant pendant la vie de vomiques purulent et fétides. Bull. Soc. Anat. de Paris. I. 11. 17. 1875. Greenhow. Lancet March 10th p. 350 1875. Tust. Pneum. dans les affect. chroniques de coeur. 1875. Moxon. Patholog. Transactions 1872-76. Traube. Deutsch. Klin. 1859. Gesam. Beiträge zur pathol. Band II. p. 443. Berlin. 1877. Trousseau Clin. Med. de l'Hotel Dieu. 5th Ed. Vol III p. 714. 1877. Nothnagel. Diagnost. et étiolog. de la retract. pulmon. unilateral. Volkmann's Sammlung Klinisch Vorträge. Charcot. Des pneumonies chron. Leçons resumées par F. Balzer. Rev. Mensuelle. 1878. Guyenheim. Bull. de la Soc. Anat. 1878. also Relation in same. Régimbeau. J. Les pneum. chroni. Paris 1880. De Castel. Sclerose pulmonaire. Soc. med. des hôp. March 16. 1882. Debove, Pneum. Chron. Ulcereuse. Gaz. Hebd. Feb. p. 74. 1884. Lecey Kidd. MD. Subacute Indurative Pneum. Lancet April. 1890. p. 740.

Caseous Pneumonia

Delbarre. Pneumonie Caséuse. dissection des bronches et des vaisseaux. Bull. Soc. anat. Paris XI. 151. 153. 1866. Traube. Pn. Caseous. Ein Fall von acut. Verlaufener tuberculösen (käsigen) pneum. und rasch etc. Berlin Klin. Woch. IV. 1867. Crando J. De la pneumonie Caséeuse. 4° Paris. 1868. Campenon. Pneum. Caséeuse lobaire. Bull. Soc. Anat. Paris. XLIV 445. 1869. Fourrié. J. Considérations sur la pn. caséeuse consécutive à une pneumonie non résolue. 4° Strasbourg. 1869. Stevens. Caseous pneumonia - a case. Cincin. Lancet & Obs. 1871. XIV. 579. Lépine R. De la pneum. caséeuse - 8° Paris. 1872. Reyt. Caseous pneumonia in its relationship to tuberculosis. Tr. Ohio Med. Soc. Dayton. 1873. 99. 117. Chauffe. A. Contribution à l'histoire clinique de la pneum. cas. lobaire aiguë chez les adultes. Arch. gen. de Méd. Paris. II. 5. 171. 1874. Colomiatti. V. J. Contribuzione allo studio del carcinoma e della pneumonite caseosa. 8°. Torino. 1877. Charcot. note sur la pneum. cas. Bull. Soc. Anat. de Paris. 1877. III. 476 also Progrès Méd. 1877. p. 947. Dieulafoy. La tuberculose pulmonaire et la pneum. caséuse. Gaz. Hebdom. de Méd. et de Chirurgie. N° 25. p. 389. 1877. Traube Fall. von subacut verlaufender käsiger pneum. Ges. Beitr. z Path. u Physiol. Berlin. 1878. III. 383. Berthier. A. De la nature de la marche clinique de la pneum. cas. lobaire aiguë chez les adultes. 4° Paris. 1880.

Hamilton's papers in the Practitioner already quoted. Lancaster & Wilson – Caseous pneumonia of entire lung. Med Times & Gaz. Lond 1884 II. 1660. also Tr. Path. Soc. Lond 1884-85. xxxvi 115. Cardarelli A. Broncopolmonite con infiltramento esteso del lobo superiore e porzione del medio, possibile caseificazione, centrale enfisema polmone e sinistra aderenze pleuriche.

Riv. Clin. di Napoli 1884 I. Duhamel, Pneum. aiguës chez les tuberculeux. Thèse de Paris 1886. Kopp – Abfühl. Pneumonia. Deutsch. Arch. für Klinik. Med. XXI. Chopin Pneumonie caséeuse chez l'enfant. Thèse 1890.

Calculi in the lung.

Consult the thesis of Poulalion (already quoted) for full bibliography: also Martorelli – Storia d'una bronchite polmonia calcolosa. Gior. d. R. Accad. Med. Ch. di Torino. 1852. XV. 529.539. Smith E. Chronic Bronchitis with suspicion of phthisis – expectoration of small masses compared to bits of coral from their hardness: probably composed of cholesterine. Lancet. II. p 677. 1856. Blachez. Calculs et concrétions bronchiques. Dict. Encyclop. des Sc. Méd. Crisp. Plum-stone. haemoptysis 15 minutes before death: pulley cavity near to apex of lung filled with lung deposit. Tr. Path. Soc. Lond 1874-75. XXVI. 16. Calcification of the lung. Med Record 1879. p. 189. J. Déjérine. Rech. des bacilles dans la tuberculose calcifiée et caséo-calcifiée. La Rev. d. Med. Dec. 1884. Juhel-Rénoy Concrétions crétacées rendues pendant expectoration. Soc. méd. des Hôp. 11th April 1890.

Intermittent Pneumonia. – (see page 96.)

Bugard. Bronchite aiguë intermittent J. de Med Clin. et Pharm. Brux. XXV. 212. 215. 1857. Bernard C. Observation de pneum. continue rémittente. Union Méd. Paris 1855 IX. 145. also Bull Soc. méd. des Hôp. de Paris 1862. Abeille. Bronchite et Pneumonie Intermittente guéries par le sulfate de quinine. Méd. Paris. XXXI 423. 425. 1874.

Treatment:

Les injections sous cutanées d'huile camphrée. Med. Mod. Oct 8 1891. p 162 of supplement.