and thereby increased the mischief. Condensed milk was often preferable to the cow's milk obtainable in towns. At all events, it was a matter of opinion, and condensed milk ought not to be condemned in the harsh terms often used. He had seen many children thrive very well on it until other articles of diet could be added.—Dr. DAWSON WILLIAMS agreed with previous speakers that the indiscriminate condemnation of condensed milk was a mistake; in large towns, during the prevalence of summer diarrhoea, the use of condensed milk was a valuable prophylactic, and it was well to have resort to it under other circumstances if the milk supply was under suspicion. Referring to infants' foods, he observed that in this country barley water, or some other starchy infusion, had long been recommended, as a diluent for cow's milk, in preparing the infant's bottle. The custom had arisen owing to a belief that the curds formed from such a mixture were finer and more easily digested. Dr. T. Rotch, of Boston, had recently published some experimental evidence tending to prove that this prevalent idea was mistaken. Dr. Dawson Williams had made a number of experiments on various infant's foods digested with artificial gastric juice, which had convinced him that the curd formed in the presence of starchy diluents under these circumstances was really finer, but it seemed probable that digestion proceeded less rapidly. He referred to the fact that after the first three or four months of lactation the quantity of milk secreted by a nursing woman in good health appeared to be increased, and expressed the opinion that this ought to be borne in mind in arranging the diet of hand-fed infants. The general custom seemed to be not to increase the quantity but the density of the fluid foods given after the age of 3 or 4 months.—Dr. JAMES MCNAUGHT pointed out that he had made a number of experiments with regard to the coagulation of milk alone and when diluted with water and different farinaceous substances, which were published in the *Medical Chronicle* of two years ago. It was necessary, in making experiments of that kind, to employ a very active and well prepared gastric juice, in which the rennet ferment was satisfactorily extracted, otherwise errors were likely to arise, and the experiments of different observers were liable to produce different results. He found that the really tenacious clot which gave trouble to the digestive organs was produced not by the acid of the stomach, as was often supposed, but by the rennet ferment. Farinaceous substances certainly lessened the tenacity and increased the digestibility of the coagula formed in the stomach, and were not themselves productive of the injurious results frequently attributed to them.—The PRESIDENT (Sir William Roberts) said that Dr. Yeo had spoken with such perfect good sense that he had nothing to say except that he agreed with him in all his recommendations. The subject of bread had been touched upon, and he wished to make a remark on this matter. Brown bread—bread containing the husk or bran of the grain—had been a good deal pushed into notice. This kind of bread was a pleasant variety for occasional use, and it also, like oatmeal, had a certain power of promoting the activity of the bowels. For these purposes brown bread had long been used; but of late more had been claimed for it. It was said that fine flour was inferior in nutritive value to coarse flour containing the bran, and it was alleged that we ought to retain the bran in some form or other in order to improve the quality of our bread; that bran contained more mineral matter, especially more phosphate, and that the loss of these by the use of fine flour was the cause of rickets, bad teeth, weak bones, and the imaginary general degeneracy of the race. There was, or recently had been, a society in existence with the high sounding title of the Bread Reform League, to purge this economic error. These attempts to make bread a more complete food were, he believed, all a mistake. Men did not live by bread alone, but supplemented its deficiencies by using other articles with it—milk, meat, fish, poultry, butter, eggs, cooked vegetables, salads, etc. Bread could not be made, even approximately, a complete food by adding the bran to it. It would still be all too poor in mineral matters, of which it would only contain 2 per cent. of the dry substance if every particle of the bran were retained in it, whereas meat, fish, milk, and vegetables, contained 5 to 6 or more per cent. of mineral matters (including the precious phosphates) in proportion to the totality of their dry substance. The real function of bread in our dietary was to furnish in as digestible a form as possible a supply of farinaceous matters and a vegetable proteid; and for this purpose bread made from fine flour was the best for ordinary use. The proper destiny of the bran was now, as it was 2,000 years ago, to feed the swine. There was no loss,

but real economy, in this, for not only did the bran come up again in the more digestible form of pork and bacon, but it had been shown that fine white bread was by far more completely absorbed than coarse bread, in regard not only to its starchy ingredients, but even in regard to its proteid and mineral constituents.—Dr. BURNLEY YEO said in reply he was glad to find that the observations he had made in introducing this subject met with such general acceptance, especially with regard to the dilution of food for febrile cases. He thought Dr. Sidney Martin's observations on the physiology of food digestion and absorption most interesting and important. He, like Dr. Mapother, had been struck with the excellent fluid, highly albuminous food, to be obtained, as he had suggested, from beating up egg with hot water and straining. He thought Dr. Radcliffe Crocker's objection to the use of starches in chronic intestinal catarrh might be greatly due to the habit of mixing milk with starch food in these cases. He had found arrowroot made with water very well digested in such cases. He emphasised Dr. Woods's caution as to the dangers of over feeding in the acute renal complications of scarlet fever. The President's remarks on the modern tendency to eat coarse bran-containing breads were in entire accord with his own observations.

A CONTRIBUTION TO THE SUBJECT OF BRONCHIAL PNEUMONIA (NON-PHTHISICAL)

IN THE ADULT LUNG :

WITH TWO ILLUSTRATIVE CASES AND A TABLE.

Presented to the Section of Medicine at the Annual Meeting of the British Medical Association held in Leeds, August, 1889.

By THOMAS E. SATTERTHWAITE, M.D.,

Professor of Pathology and General Medicine in the New York Post-Graduate Medical School and Hospital.

It is the purpose of my paper to discuss a topic that has been involved in much obscurity. So far as I know, bronchial pneumonia¹ in the adult lung has never been the subject of systematic description. It is not that medical men have ignored the existence of this adult variety, but rather that the few cases which have been published are scattered or fragmentary. My position will be made more apparent, when I quote from an article recently published by a distinguished fellow-countryman.² When treating of the general topic of bronchial pneumonia, he says: "There is strong reason to believe that the frequency with which adults are attacked [with bronchial pneumonia, T. E. S.] has been greatly under-estimated, in consequence of the failure on the part of the profession at large to clearly recognise this affection;" and, he continues, "while it is probable that the more severe and widely disseminated pneumonias of catarrhal type are commonly recognised nowadays, it appears undoubted that in very many instances of apparently mild sickness, of acute or subacute character, which are regarded as simple febrile colds or as the result of malaria, the true condition is one of circumscribed catarrhal pneumonia [bronchial or lobular pneumonia, T. E. S.], which, while threatening no immediate danger to life, will, if neglected, leave lesions of grave significance."

Since, however, the author quoted discusses the topic of bronchial pneumonia as taken from the combined standpoint of infantile and adult life, I have thought it would be interesting for the profession to consider these conditions apart, since I believe they are entitled to separate classification, both for anatomical and clinical reasons; and my experience for more than twenty years of continuous hospital service has furnished me with sufficient material bearing on the subject to make it appear desirable that I should lay it before the profession; especially as I hope that it will stimulate others to elaborate the matter more fully.

It may be correctly affirmed, paradoxical as it may at first appear, that bronchial pneumonia of the adult is first seen about the fifth year of life. At this time the lung has usually reached its full period of development, though not of growth; and hence all cases of bronchial pneumonia, not embolic in character, falling between the age of five and the limits of life naturally range themselves under this heading.

It may also appear somewhat singular that this variety of disease

¹ Bronchial pneumonia, as described in this paper, is not to be confounded with pulmonary phthisis of any form whatever.

² Dr. William Pepper, in an article on Catarrhal Pneumonia, *System of Med.* vol. iii, p. 353.

has heretofore received so little attention, for it is true that we find scant mention of it in our best manuals of pathology or pathological anatomy;³ in fact, I have only found one in which it has received a separate classification. This author,⁴ an American, has given a description of "disseminated" pneumonia as produced by pressure on the trachea or bronchi, and states that in such cases he found:—1. Intra-alveolar inflammation; 2. Collapse of air vesicles in groups; 3. Interstitial inflammation.

It is interesting, however, to note that isolated examples of the disease have crept at various times into our literature. Thus Aetius,⁵ of Amida, the great physician who flourished about the end of the fifth century, was perhaps the first to mention it. Under the caption, "De ea quae pleuritis esse putatur, quum tamen non est," he says: "Crude mucous and tenacious humours are generated in the body which sometimes discharge themselves into the cavity of the chest, and also into the lung;" and, he adds, "in such cases the respiration is not rendered difficult by pain, as is otherwise so commonly the case, but rather by obstruction of the air-passages through secretions;" and, further, "If they (the patients) are attacked by fever, it is very slight, the patients having little thirst. The pulse is slow, very small, and quite sluggish, altogether the opposite to what we usually find." This is, indeed, an imperfect description, but plainly shows that Aetius had seen such cases, although he does not really distinguish the disease sharply from bronchitis, and does not class it as a pneumonia; for, in another paragraph,⁶ entitled *De Peripneumonics*,⁷ which contains a description of acute lobar pneumonia, he does not allude to it.

Something over a century later, Sydenham, the "English Hippocrates," described it clinically under the head of "bastard peripneumonia," detailing certain features of a pneumonic inflammation that can only apply to a pneumonia of adult life, propagated along the avenues of the bronchi. "This form of lung disease," he says, "attacks by preference the stout and fat, and those who have been addicted to spirituous liquors;" and, in another place, he writes that "the matter which has excited the cough grows gross, blocks up the passages to the lungs, and preys upon the whole mass of the blood in the shape of fever." Now, bronchial pneumonia, as I understand it, is the only form of pneumonia that enters the lung parenchyma through the extension of a bronchial catarrh: and it is quite common in alcoholic patients, as in others of deficient vital force.

In the succeeding century, we find the views that had originated with the great English physician were sustained and extended by Boerhaave and his pupil, Van Swieten, and in the latter's *Commentaries*, section 867, vol. viii, the name "bastard" or "spurious peripneumonia" is retained.

Boerhaave is credited with saying that this form of pneumonia happens most frequently in "old people and such as are phlegmatic, of a cold habit and afflicted with catarrhs and colds;" that "by its deluding lenity at first destroys those who scarce thought themselves ill, for, beginning with a slight weariness, weakness, and prostration of almost all the faculties of the mind, and with a shortness of breath and oppression of the breast, it excites such gentle commotions that there is hardly any sign of heat or fever that warn one of the danger," etc.

The affection, according to the same authority, is due to running, injury, getting drunk, feasting, etc., which, it is true, may be secondary factors in the causation of the disease.

Morgagni,⁸ who wrote in 1760, does not appear to have ever seen a case, though he may have known of the infantile form, for he has written of a child, 14 days old, that died on the eighth day of a pneumonia, *post-mortem* examination revealing certain "black spots" in the posterior part of the lungs, that were possibly due to bronchial pneumonia.

There can be no doubt, however, that Andral knew of it, for in his *Clinique Médicale*, p. 357, after recording a case of bronchitis in a man aged 60, he continues as follows: "A few days

only before entering the hospital the cough became much more frequent and more intense, the difficulty of breathing increased, and fever was ushered in. When we saw this patient for the first time we thought him attacked merely with simple exacerbation of the bronchitis; in fact the expectoration indicated nothing else. It consisted of a clear glairy mucus, marked by opaque streaks. Auscultation detected in all parts of the chest a very loud mucous *râle*. The sonorosity of the thoracic parietes was everywhere equal. Pulse was very frequent, skin hot. On the following day the respiration seemed to be more free, pulse less frequent. But the following days the dyspnoea reappeared and went on increasing. The chest, when percussed, ceased to sound beneath the right clavicle and in the level of the inferior angle of the left scapula. Catarrhal expectoration still continued, mucous *râles* louder, orthopnoea, asphyxia, and death." "*Post mortem*: Mixture of red and grey hepatisation of a small portion of the lower lobe of the left lung; general redness of the bronchi, which were filled with mucus."

"This case," he continues, "is an instance of what would formerly have been called suffocating catarrh [capillary bronchitis T. E. S.]. The cause of all the bad symptoms and of death lay in the inflammation of the pulmonary parenchyma, but the symptoms characteristic of this inflammation were so little marked that without percussion nothing would have been recognised during life but simple bronchitis. The expectoration remained catarrhal; the *râle* occasioned by the accumulation of mucus in the bronchi prevented us from detecting by auscultation the state of the pulmonary parenchyma. Hence," Andral deduces, "in old persons affected with chronic bronchitis the appearance of an unusual dyspnoea and of fever very often indicates a greater or less inflammation of the pulmonary parenchyma."

Andral also instances other patients that had bronchial pneumonia in conjunction with phthisis, hepatitis, enteritis, and small-pox. In several of these he did not detect the disease during life, because the symptoms of pulmonary trouble were so slight.¹⁰

Laennec also knew of bronchial pneumonia under the name of suffocative catarrh, but found it rare in adults.¹¹

Now it is interesting to observe (from a historical point of view, at least) that in a late edition of Rilliet and Barthez¹² it was shown that capillary bronchitis and broncho-pneumonia are the same thing (p. 497), or rather that *pneumonie lobulaire généralisée* is the successor of capillary bronchitis. Previously (about 1825) Lanoix¹³, and shortly after several of the French school¹⁴ noted the similarity between the pneumonia of children and of the aged; but otherwise the contributions of the present century have been meagre enough, and I have but to add a very small number of names. Thus Frank¹⁵ seems to have known it as the *peripneumonie fausse* of old people. Wilks,¹⁶ of Guy's Hospital, reported one case, and the late lamented Fagge described it theoretically, though it is not clear in my mind that he had seen a genuine case.

I regret exceedingly that I have not at my command a greater number of facts upon which to base conclusions, but the twenty cases that furnish the basis of this paper represent a continuous and personal study of pneumonia, and I feel, therefore, that they are worthy of being put on record. Each of the cases will be found in the table at the end of this article, and though several are incomplete in matters of importance, yet, taken together, the data are instructive from several points of view, revealing the fact that we are dealing with a type of pneumonia that merits separate consideration. This becomes at once apparent when we observe the age at which it occurs. Contrary to the statement of almost all well-known writers, bronchial pneumonia (excluding now all cases under 5 years of age), is most common in the middle and later periods of life, chiefly between the fourth and sixth decades.

According to my figures, males largely preponderate. On this point I differ from Jürgensen, who finds that sex has no influence on catarrhal pneumonia. Males preponderated in nineteen of my cases in the proportion of twelve to seven. This ratio may not have any significance, however, but it is interesting at the same

³ Cornil and Ranvier, *Path. Hist.*, Phila., 1880, p. 414. Coats, *Man. of Path.*, Phila., 1883. Zeigler, *Path. Anat.*, New York, 1887, s. 610.

⁴ Delafield, *Studies in Path. Anat.*, vol. i, p. 71, New York, 1888.

⁵ Aetius, *Tetrabiblion* II, Sermo IV, cap. 66, Lugdun. 1549. This book is to be seen in the library of the New York Hospital. (Translation by Jürgensen.)

⁶ *Op. cit.*, cap. 68.

⁷ The term "peri," when used as a prefix, means acute or severe, as explained by Celsus, when he says: "A compagine corporis ad viscera transeundum est; et imprimis ad pulmonem veniendum. Ex quo vehemens et acutus morbus oritur, quem Περικνημονίαν Græci vocant. Lib. quart., cap. vii. [This book is in my own possession. T. E. S.]

⁸ *Works of Sydenham*, vol. i, p. 251. London, 1848.

⁹ Morgagni, *The Seats and Causes of Diseases*, 205, 15, London, 1769.

¹⁰ *Clinique Méd.*, London, 1836. (English translation).

¹¹ *Diseases of the Chest*, N. Y., 1830, p. 86.

¹² *Maladies des Enfants*, Paris, 1853.

¹³ It is but proper to state that Lanoix's article (*Essai sur la Pneumonie des Enfants comparée à celle des Vieillards*, Paris, 1825) was based upon a study of only three cases.

¹⁴ Weiss in Gerhardt's *Handbuch der Kinderkrankheiten*, p. 731.

¹⁵ *Méd. Prat.*, vol. i, p. 188, Paris, 1842.

¹⁶ *Guy's Hosp. Reports*, 1865.

time to observe that this preponderance of the male sex is found in every type of pneumonia.

In considering the etiology of this disease we do well to study its antecedent conditions; but, before doing so, let us note that the season of the year has an important bearing on its origin; thus, between October and June 60 per cent. of my cases occurred. It is, therefore, a disease of the autumn, winter, and spring months most particularly. On this point I am glad to agree with most writers. Singularly enough, occupation, whether outdoor or indoor, was apparently an important matter. Thus in 67 per cent. it occurred in persons who were leading an outdoor life. It may be noted that in acute lobar pneumonia there is no such disproportion, and that in embolic pneumonia the reverse ratio holds good. In this respect, again, there is evidence that the disease we are now considering has a distinct place in the categories of pneumonia.

In my twenty cases I found, in all but two, antecedent conditions that aid somewhat in our comprehension of the disease. The most important of these were as follows: in one, pericarditis with effusion; in two, fibrinous pericarditis; in two, blows on the chest; in two, blows on the head with meningitis; in two, bronchitis; in one, thoracic aneurysm; in one, resuscitation from drowning; in one, typhoid fever; in two, diphtheria; in one, measles; in one, phthisis; in five, alcoholism.

Now, starting from the well-grounded assumption that infantile bronchial pneumonia depends for its cause upon a bronchitis, we may conclude that a similar condition in the adult lung will be capable of causing a similar pathological condition. But in infantile bronchial pneumonia we reason that inflammation of the parenchyma follows immediately upon occlusion of the ultimate bronchioles by a swollen mucous membrane, or irritating matter detained at those points. In other words, there is obstruction of a certain number of the bronchial tubes. And yet this state of things may be found in pericarditis with effusion, aneurysm, after resuscitation from drowning, and in phthisis sometimes, mechanical pressure from retained substances in all these cases being sufficient to excite bronchial pneumonia. Indeed, of this condition we have experimental proof. Another class of cases may be due to feeble respiratory efforts, for we know that collapse of the lung may be produced in like manner; or rather, we know that atelectasis in the newly-born, which is a parallel condition with collapse of the lung, will be permanent unless the infant be compelled to breathe lustily. Now collapse of the lung, we have reason to believe, is the forerunner often, if not always, of broncho-pneumonia. Such feeble respiration exists in states of advanced alcoholism and chronic nervous affections, and in typhoid fever. Or, it is fair to suppose that it may originate simply as an extension of some systemic disease, such as measles or diphtheria, the antecedent affection being in these cases a bronchitis.

Finally, bronchial pneumonia in the adult may develop by direct extension of an inflammatory process. Thus, I have seen two cases develop from fractured ribs, where the injury, though severe, was attended by no septic symptoms. At this point I will mention that Fagge's cases, so far as his description of them goes, were perhaps not true cases of lobular bronchial pneumonia; that is, according to my present classification. Thus he speaks of a young woman who developed bronchial pneumonia eight days after a miscarriage, and died in ten days. Such a case should be, it is reasonable to suppose, one of embolic pneumonia. Of course it is not always easy, even with all the evidence at hand, to classify each individual case. Fagge tells of another instance of bronchial pneumonia in an old syphilitic, who, he supposes, contracted the disease by inspiring the foul discharges of an ozena. If this were a real case of pneumonia and not lung syphilis the pneumonia might have been termed inspiratory or pythogenic and yet be a bronchial pneumonia. At this point it is proper to explain that the bronchial pneumonias of the aged may very reasonably be imputed, as has been done, to the inspiration of bronchial secretions, if the patients have suffered from bronchial catarrh, and under the same heading may be classed some recorded examples of what has been termed "Buffalo pneumonia," a disease produced by the inhalation of dust among the shovellers and scoopers of the great grain elevators of the city of Buffalo.¹⁷ It has also been held that heart disease is a cause of pneumonia, but precisely in what way has not, I think, been clearly shown. That it may occur, as I have indicated, through pressure in pericarditis is not so difficult to understand, or that it can be a contributing

cause or factor in weak or fatty heart is easily understood; but it must be remembered that, as a rule, organic diseases of the heart produce either an embolic or an interstitial pneumonia.

In taking up the morbid anatomy of bronchial pneumonia in the adult lung it is indispensable to consider briefly a number of allied topics, among which are tracheitis, bronchitis (general and capillary), pulmonary collapse, oedema, bronchiectasis and emphysema, interstitial pneumonia, pleurisy, abscess or gangrene, and phthisis, one or more of which may have its part in the production of the adult form of bronchial pneumonia as in the infantile, which latter relation I have shown elsewhere.¹⁸

In some cases the origin of the disease can be traced to some inflammatory condition of the upper air passages, just as in children. There is, to be sure, less activity in the symptoms, and the glandular implication is less because the glands are now not so large and active in their functions; but, in exceptional cases, the origin and development of the disease may have essentially the same character. But there is always all the difference that arises from the alteration in the structure of the lungs at these different periods of life, for, at the time when the lung is fully developed, namely, in the fifth year, we have a right to class its diseases among those of adult life; and besides, bronchial pneumonia now finds results that previous diseases have established in the lung. Thus, it is to be presumed that the sequel to old attacks of pneumonia, but especially of pleurisy and bronchitis, have established here and there bronchiectatic foci, and it is from these especially that the disease may most easily spread by the processes I have already hinted at, namely, by the blocking of the tubes, and later, by the inspiration of bronchial matters.

At first we may have collapse of the lung, a condition corresponding at its commencement with the foetal state, when as yet the lung has not been aerated. The *rationale* of this pathological process has often been described. A little pellet of mucus or muco-pus, or secretion of some sort, or a foreign particle, is adherent near the bifurcation of a bronchial passage. At each expiration it passes further into the large tube; at each inspiration it passes back until obstruction takes place. Thus, by a succession of respiratory movements, a lobule or group of lobules is pumped dry of air. But even if the plug does not move with inspiration, the oxygen and other gases are absorbed in obedience to the law of the diffusion of gases; consequently there is a falling in of the walls of the lobule or lobules, and now there is pressure from the circumambient vessels with a necessary transudation of their liquid or solid contents, and more or less alteration of the pre-existing cellular elements of the vesicles.

This constitutes the first stage of this form of pneumonia. I may add here that the first stage of lobular pneumonia and collapse of the lung are indistinguishable, pathologically.

In the second stage there is more or less complete consolidation of each lobule implicated, so that it becomes comparatively firm and dense, though usually less so than in lobar pneumonia. The colour is now deep red. But the deposit does not differ so much in kind from that seen in the other or "croupous" form. It is rather in degree that we see a difference, and herein I think many of us have been led away under the influence of Continental authorities. In recognising the "fibrinous" character of acute lobar pneumonia we have been disposed to think that therein was a distinguishing mark. Let us at once dispossess ourselves of this erroneous idea. Bronchial pneumonia may be, and often is, fibrinous. If we want pathological criteria we can find them in this, namely, in the one, the bronchial, there is a comparative excess of liquid and corpuscular elements over the fibrinous; in the other, the lobar, a comparative excess of the fibrinous over the other solid and the liquid elements. The solid contents are altered blood, epithelial corpuscles, pus, fibrin, and perhaps granular matter; air and fluid are usually entirely absent. This subject has been treated of *in extenso* at another place.¹⁹

In the second and third stages of pneumonia the process is very easily identified on the *post-mortem* table. We have here to distinguish chiefly from lung syphilis and lung infarction; or, to be more exact, to exclude gummy tumour and embolism. In the former instance we should have the syphilitic history of the patient, and the deposit should be nodular and encapsulated. In the latter we find the characteristic phenomena of infarction, easily detected by the experienced pathologist, even if the history of the case be defective. I may add, parenthetically, that the diffuse interstitial pneumonia of syphilis (white pneumonia) is

¹⁸ *Virginia Medical Monthly*, June, 1889.

¹⁹ *Virginia Medical Monthly*, June, 1889.

¹⁷ *Buffalo Med. and Surg. Journal*, December, 1879.

A Table Showing the Phenomena of Lobular Bronchial Pneumonia in the Adult Lung.

No.	Age.	Sex.	Month.	Locality of Disease.	Antecedent Affection.	Coincident Affections.	Supposed or Apparent Cause.	Duration.	Auscultatory Signs.	Sputum.	Pulse Respiration.	Pain in Side.	Urinary Symptoms.	Nervous Phenomena.	Abscess or Gangrene.	Cause of Death.	Range of Temperature.	Place of Record.	Initials.	Remarks.
1	—	M.	Oct.	of each lung	Phthisis	Phthisis	Inspira- tion of morbidity	—	—	—	—	—	—	—	—	—	—	Path. Reg., 1, p. 21	E. W.	A case of inspiratory pneumonia.
2	55	F.	Feb.	Upper lobe	Extensive pericardial effusion	See pre- ceding column	Pericardial effusion	3 days	Regular signs present	—	Pulse, 128-152	Present	Albumen 20 per cent.	—	—	Heart failure	—	Path. Reg., 1, p. 99	C. K.	—
3	48	M.	Sept.	Lower lobe	Fibrous peri- carditis, chr. diffused	"	Pericarditis	—	—	—	—	—	—	—	—	—	—	Path. Reg., 1, p. 152	I. F.	—
4	27	M.	Dec.	"	Nephritis Biphysema, injury to chest,	"	Blows upon the chest	15 days	Regular signs present	Rusty	—	Present	—	Delirium	Gangrene	Septi- cemia	Not above 102° F.	Path. Reg., 3, p. 58	P. D.	A typhoid pneumonia.
5	27	M.	"	Bilateral	Fracture of ribs	"	Injury to chest	4 days	"	—	—	—	—	Delirium	—	Pulmo- nary hemor- rhage Heart failure	—	Path. Reg., 3, p. 152	C. K.	—
6	32	M.	Mar.	Base	Alcoholism	—	—	—	—	—	—	—	Granular casts	—	—	Internal hemor- rhage Heart failure	Highest temp., 104	Path. Reg., 3, p. 517	J. B.	—
7	69	F.	Oct.	Lower lobe	Thoracic aneurysm	See pre- ceding column	Rupture of aneurysm	3 days	Regular signs present	Bloody	Pulse, 100-120	Present	None	—	—	Internal hemor- rhage Heart failure	"	Path. Reg., 4-1-48	E. D.	—
8	35	M.	June	Bilateral	Cirrhosis of liver	—	—	4 days	Dyspnea, bronchial breathing	—	Pulse, 135	—	Chlorides present	—	—	Heart failure	Highest temp., 105	Path. Reg., 4, p. 84	S. H.	—
9	42	M.	"	Dissemi- nated	Chronic diffused nephritis, alcoholism	Resuscita- tion from drowning	Inspira- tion of foreign matter	5 days	Regular signs present	Rusty	Pulse, 120; resp., 52	—	Albumen 15 per cent., casts	Delirium; stupor	Tendency to gangrene	Heart failure	Highest temp., 108.7	Path. Reg., 4, p. 160	H. C.	A case of inspiratory pneumonia.
10	36	M.	"	"	Malaria, typhoid fever	See pre- ceding column	Typhoid fever	6 days	Dulness and bronchial breathing	Bloody	Pulse, 120	—	—	Delirium	—	Heart failure	Highest temp., 108.75	Path. Reg., 4, p. 186	E. R.	—
11	53	M.	Aug.	"	Meningitis, locomotor ataxia, endocarditis	"	Bronch- itis	2 da., s	Mucous râles, dulness	—	Pulse, 130; resp., 40	—	—	Delirium	—	—	Highest temp., 105.5	Path. Reg., 4, p. 204	H. H.	—
12	78	F.	"	"	Pericarditis bronchitis	"	"	10 days	Crepitant and sub- crepitant bronch- ovesic	None	Resp., 42	Present	Albumen	Delirium	—	—	Highest temp., 103.4	Path. Reg., 4, p. 253	M. C.	" Pneumo- nia in the aged."
13	6	M.	Feb.	Lower lobe	Diphtheria	"	Diph- theria	—	Ordinary physical signs	—	Pulse, 95	—	"	—	—	Heart failure	Highest temp., 100	Path. Reg., 1, p. 160	—	Patient of Dr. J. Lewis Smith.
14	—	F.	May	Bilateral	Measles, bronchitis	"	Measles	—	Not detected	Pur- ulent	—	—	—	Gangrene right mid. lobe	—	Septi- cemia	—	M&J. Case, B. 1, p. 115	B.	A child about 7 years old.
15	—	F.	Mar.	Dissemi- nated	Diphtheria, fatty heart	"	Bronchial diph- theria, bronchitis	A few days	Those of bronchitis	—	—	—	—	—	—	—	Highest temp., 105	Path. A. & P., p. 84	H.	A lady about 50.
16	67	F.	Sept.	Lower lobe	Alcoholism chr. Bright's, cirrhosis, fatty heart	"	Respira- tory weakness	—	Regular signs	—	—	—	Albumen	—	—	—	—	N. Y. Post- grad., 1, p. 16	D.	—
17	—	—	Jan.	Dissemi- nated	Injury to head	Mening- itis	—	4 days	—	—	—	—	—	Convul- sions	—	Heart failure	Highest temp., 103	Path. Reg., 2, p. 62	J. H.	A young child.
18	29	M.	July	Lower lobes	Alcoholism	—	Respira- tory weakness	—	Cough, dyspnea	Bloody	—	Present	Albumen 7 per cent.	—	—	—	—	Pres. Hosp., 4, p. 272	G. V.	—
19	50	M.	Nov.	—	Alcoholism	—	Respira- tory weakness	7 days	Cough, dyspnea	—	—	—	—	—	—	—	—	Postgrad C.B.I., p. 2	M. D.	—
20	—	F.	Feb.	—	—	—	—	—	Cough	—	Pulse, 120; resp., 24	—	—	—	—	—	—	H. & P. Re- ports, p. 58	—	—

quite unlike any form of lobular pneumonia in its pathological characters.

In lobular pneumonia the lesion is usually somewhat elevated above the surface of the surrounding lung tissue. In size each deposit may not be larger than a hazel nut; it is seldom as large as a pullet's egg, though, if the attack has been protracted, the several diseased foci may become amalgamated, so that half a lung or even more will be involved.

The duration of an attack is governed almost wholly by the antecedent disease. Lobar pneumonia, when uncomplicated, terminates invariably within two weeks, usually in much less time. Lobular pneumonia, whether in the adult or infantile form, is extremely variable in its duration. It may last a few days only or a month, possibly longer, because it is apt to be characterised by a series of invasions. After a certain length of time chronic pulmonary changes are inevitable, such chiefly as interstitial fibrous deposits, bronchiectasis and emphysema, which will inflict more or less permanent damage. For each pneumonic deposit is at first the centre of a zone of congestion and oedema, while a sort of compensatory emphysema of the vesicular variety is sure to be established in order to make room for the inspired air that has been defrauded of space. But this form of emphysema presumably disappears when the distension has not been too complete, and when the primary disease has yielded. And indeed, there is reason to suppose that the entire lung will return to its original condition in cases of not too protracted a character, for we know that in acute lobar pneumonia, even when pleurisy has been a marked feature, complete resolution may occur with entire absorption of the exudation, whether fluid or fibrinous. Unfortunately lobular pneumonia is a disease that attacks individuals whose strength has already been undermined by the primary affection. Hence it is not surprising that resolution should be delayed, even if partial destruction or, at least, alteration of the lung does not occur. And hence, too, it is not remarkable that the lung is liable to suppurative or gangrenous change about as frequently as in secondary lobar pneumonia (10 per cent.). We have also a right to expect a sort of lung cirrhosis²⁰ in severe cases.

With these vital changes incident to a long-protracted malady we should expect that miliary tuberculosis would be readily initiated. This does not, however, appear to be the result, although in the lobular pneumonia of the infant lung we have been told that suppuration changes are often coincident with the development of phthisis. This special topic requires, however, new and careful study, and I think that the pathology of the future will show that in this respect we have been greatly in error. There is no reason to suppose that the products of the inflammation may not become cheesy. When abscess or gangrene occurs the suppurating matter may be expelled by the bronchi, or by bursting through the pulmonary pleura we may have an empyema, or possibly a pyo-pneumothorax.

At any rate, very soon after the pulmonary disease touches the pleura we shall have pleurisy, which will progress coincidentally with the pulmonary disease, and regress also with it. It is therefore always in such cases a secondary affection. Now, this contemporaneous occurrence of bronchitis and pleurisy, affections that clinically are often seen in unison, invites the question whether or not we did not have a lobular pneumonia to establish a bond of connection between the two. A study of our clinical and pathological records will indicate that this surmise is correct. The physical signs of pneumonia have simply eluded us. How often does the diagnosis of "a patch of pleurisy the size of a five-franc piece" or "of a silver dollar" represent in reality a bronchial pneumonia?

The disease is often confined to a few lobules, or groups of lobules, and seldom involves half a lung, as I have already said, though as a rule both lungs are attacked (in about 70 per cent.). Where a single lung is involved it is the right or left indifferently, but when both are attacked, the inferior lobes, or the posterior portions of the lungs, suffer the most.

I have already alluded to the attendant morbid conditions that I have found associated with lobular pneumonia in the adult.

From the foregoing it will be seen that the physical signs may be uncertain; in fact, an ordinary examination may fail to reveal them, though a finely crepitant *râle* is sometimes heard in the first stage, and yet we may have to provoke a fit of coughing in order to produce it. In the second and third stages there may be dullness on percussion if the condensed area be large or if the focus

be near the surface of the lung. If it is small or centrally located there will be difficulty in detecting it. In those cases where there has been a preceding bronchitis, acute or chronic, or where there has been an exposure to the inspiration of solid matters, such as dust in unusual quantities or foul matter of any sort, we shall have our attention directed to the probable seat of the affection by more or less dyspnoea, with mild suffocative attacks and short cough, with, perhaps, temporary arrest of the secretion; generally there will be a sudden rise of temperature and pulse, but not necessarily a chill. Pain in the side was noted in one quarter of my cases.

Again, it will help in the diagnosis to know that the disease is usually symmetrical, and that there will be scattered foci of disease chiefly in the posterior and inferior portions of the chest.

We may remember that the temperature, pulse, and respiration will all be abnormally high, and, though the pulse may be as low as 100, it is more apt to rise above 120. I have known it to reach 158.

Expectoration may be bloody, rusty, or purulent, or all three in any one case. In a very small number of instances there may be no expectoration. In something less than half (40 per cent.) I have found abnormal urinary signs, such as indicated functional or organic renal lesions. In a little less than half there were abnormal nervous phenomena, such as delirium, convulsions, or coma. The temperature was found to average 104.7°, a little less than in acute lobar pneumonia, a little more than that in secondary lobar pneumonia. It may even in this form run very high, and in one instance at death it reached 108.75°. If, however, we realise that the regular physical signs were found in about 55 per cent, there is some encouragement in believing that a diagnosis can usually be made. Pathological data alone give little clue to a knowledge of the duration of the disease. Nor do clinical data help us always, for reasons that we have already seen.

In studying the determining cause of death we have a complicated problem, quite unlike that of acute lobar pneumonia. For we have an underlying affection that is sapping the very springs of life, and which very possibly may be in itself the actual cause of death. Who can say in diphtheritic pneumonia whether it is the pneumonia that kills; or in an extensive pericarditis, with an effusion embarrassing the heart's action, whether death takes place at the heart or in the lungs. Sometimes it will be certain that the pneumonia is not concerned primarily in the cause of death, which may be plainly due to sepsis, possibly to uræmia. But with all that can be said on these points, I am of opinion that death is usually due to heart failure, though I find myself on this point in opposition to so brilliant a writer as Jürgensen.

First in order of causes comes sepsis. Least of all in order of importance stands uræmia.

From a review of the natural history of lobular pneumonia it will now appear that the treatment involves questions of by no means so simple a character as those in acute lobar pneumonia, lung syphilis, or pulmonary embolism.

If, for example, we have to do with a pneumonia in the weak and aged, possibly even in a phthisical patient, following upon a bronchitis acute or chronic, we must realise that there is a chance of checking the disease by subjugation of the bronchitis or by such a fortification of the system that the minute bronchioles and bronchi will reject their natural secretion, and with it the altered matters held by them in suspension. By successfully treating these indications, collapse of the lobules will be obviated, and a step successfully made in [resisting the inroads of the attacking disease. So in any case of obstructed bronchi, whether it be from the causes mentioned, from the bursting of an abscess, the escape of food into the air tubes, as in cancer of the œsophagus or larynx, our aim should be to stimulate respiratory action, so that the foreign matter may be expelled as speedily as possible.

Again, if the pneumonia can be referred to an antecedent or concurrent disease, it should be considered whether or not the primary affection can not be made tractable. If a pericarditis with effusion pressing on the bronchi, it would obviously be the proper treatment to endeavour by appropriate means to remove the fluid.

On the other hand, where the lung has sustained a mechanical injury, as in the case where a portion of rib had been driven into the lung, operative surgical treatment is plainly indicated. If, again, there is danger of extension of the disease from a diphtheritic bronchitis, it is plain that all human means should be resorted to to prevent its extension to the finer tubes. But prophylaxis having failed, or the disease having attained a seat in the

²⁰ This has been alluded to by Jürgensen.

lung, it is well established, I think, that it will go through its regular stages, and in their regular order. Now, of course, the plan of treatment is not essentially different from that of lobar pneumonia, except that there is less need of the active energetic treatment pursued in such cases, because the area of lung involved will be smaller, and because the disease is less sthenic in character. The patient should remain in a temperature that is absolutely uniform, and not far from 70°. A certain degree of moisture will enable him to respire with more ease and with less pain. If sepsis is present the air may be charged with some disinfectant, and for this purpose I have employed listerine very advantageously. Ipecacuanha has no sphere in the pneumonia of adults, if used as an emetic, but as a sialogogue it has desirable qualities, though I use it less than formerly. Chiefly do I depend upon the muriate and carbonate of ammonia suspended in a mucilaginous liquid. I am inclined to think ammonia has a larger field for use than alcoholics, and for two reasons. The first is that it can be employed with advantage for a longer time continuously; and, secondly, because it has a positively favourable action upon the renal secretion, which is a matter to be considered in every form of pneumonia. In addition, I always use the oil silk jacket lined with ordinary cotton wadding, and I keep it in place during the continuance of the disease. The patient is now bathed constantly in perspiration, and there is no likelihood of his taking cold. The bronchitis, if present, is likewise improved.

In some instances I use poultices, which can easily be slipped in under the oil silk. They are to be renewed every four to six hours. A moderate use of calomel in 1-20 grain doses repeated every two or three hours I find an excellent remedy to relieve congestion of the liver and deplete the engorged lung. In uræmic cases we cannot be too prompt in detecting its early symptoms so as to direct our efforts vigorously in that quarter. In some cardiac cases digitalis or some heart stimulant may be imperatively necessary. I am inclined to think that under these circumstances digitalis continues to be our safest and most efficient remedy if administered with caution, and only when necessary.

The following are illustrative cases:—

BRONCHIAL PNEUMONIA IN CONNECTION WITH ALCOHOLIC CIRRHOSIS AND ABDOMINAL DROPSY.

CASE I.—S. C., carpenter, 35 years of age, was admitted into the Presbyterian Hospital in January of 1886. He had previously suffered from varioloid, intermittent and yellow fevers, and had contracted syphilis five years before admission, and was in addition a confirmed victim of the tobacco and alcoholic habits.

Symptoms pointing to thoracic aneurysm, the patient was put upon Tufnell's treatment. At the end of a month it was thought best to order a regular diet, as symptoms of weakness became apparent. Two weeks later a chill occurred without apparent cause. It lasted thirty minutes, the temperature rising suddenly to 103° F., and an hour later to 105° F. The fever was at first reduced by antipyrin in 30-grain doses. On the second day the temperature rose to 105° F., but was this time reduced to 102° F. by antipyrin (grains xv) in repeated doses with thallin (grains iv). The patient then began to expectorate blood, and on the same day bronchial breathing was detected in the left lower lobe. The patient died on the fourth day of his pneumonia, and apparently of heart failure. At the *post-mortem* examination it was found that the heart was somewhat hypertrophied and that its cavities were dilated. Weight of organ 15 ounces. The aorta and large vessels were quite free from apparent disease. Both lungs were firmly bound down by old pleuritic adhesions, and were congested and oedematous, the right weighing 30 ounces; the left, 27. Both lungs contained areas of lobular pneumonia consolidations. The liver was enlarged, cirrhotic, and pigmented. The peritoneal sac contained a large amount of serous fluid. Here there was a case of alcoholic cirrhosis with abdominal dropsy, where the cause of death was due to bronchial pneumonia. To be sure the patient had gone through many of the phases of constitutional syphilis, but none of its lesions were detected at the necropsy. In such an instance what produced the pneumonia? An answer will only be conjectural.

BRONCHO-PNEUMONIA IN CONNECTION WITH DIPHTHERIA.

CASE II.—The following case is interesting as showing how broncho-pneumonia may be the final lesion in diphtheria. In March, of 1887, I made a *post-mortem* examination in the presence of Drs. J. E. Janvrin, E. H. Peaselee, W. H. Porter, and C. E. Billington, on the body of a lady who had been suffering for about

a week with diphtheria, taken presumably from the grandchild that she had been nursing. She died suddenly with suffocative symptoms, and the following lesions were found. In the trachea from immediately below the thyroid cartilage to the smallest divisions of the bronchi, a membranous exudation, diphtheritic in character. It was thick and tolerably adherent to the mucous membrane, forming a veritable cast of the tubes. No membrane in the buccal or nasal cavities, or in the larynx. Here there was one of those very rare examples of truly diphtheritic bronchitis. The lungs both right and left were studded with zones of bronchial pneumonia in the stage of red hepatisation. The heart was enlarged, fatty, and dilated. These areas were so small and so scattered, that they could hardly have been detected by physical signs. The pneumonia in this case might not have been a very serious affair, had there not been fatty heart, and profound blood infection from the diphtheria.

THE HISTOLOGY OF BRIGHT'S DISEASE.

Read in the Section of Pathology and Physiology at the Annual Meeting of the British Medical Association, held in Leeds, August, 1889.

By GEORGE F. CROOKE, M.D.,

Physician for Out-Patients to the Queen's Hospital, Birmingham, and lately Pathologist to the General Hospital, Birmingham.

THE material examined consists in considerably over a hundred individual cases of nephritis of all forms, and represents the work of ten or twelve years; but the subject of the paper includes only Bright's disease proper; all the suppurative and other forms of kidney mischief, due to local causes, are purposely excluded.

The examples of acute nephritis number sixty, of which only three were instances of an idiopathic affection, the rest being associated with other diseases mostly of a specific febrile nature.

While rejecting the term parenchymatous in Virchow's strict meaning of it, I am disposed to retain it for those cases where the parenchymatous changes undoubtedly predominate. Interstitial changes are undoubtedly also present, but in so slight amount that they might in places easily be overlooked. I have met with a number of such cases where the parenchymatous changes constituted the main anatomical features. I quite subscribe to Cohnheim's classical dictum that glomerular changes alone may be responsible for the cardinal symptoms of Bright's disease, and in a few cases these changes may be so prominent and extensive that the nephritis merits the title of acute glomerular nephritis.

The term acute diffuse nephritis I apply to those cases of nephritis where the parenchyma, the stroma, and the vascular apparatus are almost equally affected, and the extent of the changes indicates the intensity of the inflammation. Cases of acute nephritis of a hæmorrhagic or septic character usually show these changes.

Then there are cases where the stroma is principally affected by a round-celled infiltration, and these I term acute interstitial nephritis. Cases have been described by Dr. Coats (our President), by Biermer, Wagner, Klein, myself, and others in scarlatina.

Coming, now, to chronic nephritis, I take the large white kidney first. As regards its histogenesis, it develops as a chronic nephritis usually in connection with diseases of a subfebrile nature, such as phthisis and chronic suppuration, and it is in these examples that amyloid changes are found associated with the parenchymatous.

Regarding the relation of the two, I coincide with the view recently expressed by Dr. Saundby in his book, that the two processes are concomitant or co-ordinate, and traceable to the same cause. In other words, so far as my observation leads me, the extent of the amyloid changes found in some of these cases is so slight in comparison to the extent and intensity of the parenchymatous, that the two cannot be regarded in the relation of cause and effect. This view has been more recently expressed by Orth in the first part of his second volume on *Pathological Anatomy*. Cases, however, are met with where the organ of the chronic parenchymatous nephritis is distinctly traceable to an acute attack, or to the effects of, perhaps, several intervening attacks. Other noteworthy histological features in cases of subacute or chronic parenchymatous nephritis are the glomerular and interstitial changes present, which have previously attracted the atten-