ORIGINAL COMMUNICATIONS.

ON SOME POINTS IN THE PATHOLOGY,
DIAGNOSIS, AND TREATMENT
OF PNEUMONIA.

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PART I.

ON THE PATHOLOGY, DIAGNOSIS, PROGNOSIS, AND THE
THEORY WHICH RECOMMENDS BLOOD-LETTING
IN THIS DISEASE.

I CAN scarcely hope in the present inquiry to bring much new matter to bear upon so vast a subject as that of Pneumonia in a paper the reading of which is, by the laws of this Society, limited to half an hour; but, looking upon this august assembly as one composed of practical men, I would rather seek, by a short sketch of this disease, to elicit the opinions of the Fellows present towards a more uniform and a less violent mode of treatment in this affection. At present, nothing is more varied: perhaps I should not be far wrong if I added, more unsatisfactory. After some fifteen years of professional life, a large portion of which has been sedulously employed in the study of disease in *continental* as well as *home* practice at the hospitals, and in private practice, I am forced to conclude that the word "pneumonia" is still made to include a variety of different diseases, requiring different treatment, and running a totally different course. I allude especially to simple inflammatory pulmonary congestion, capillary bronchitis, and some varieties of pleuritis. The treatment, besides, is far too energetic, too antiphlogistic, and, I fear, often founded on erroneous principles and theory.

The subject is so vast that I am compelled to divide its consideration into two parts. The first, that of this evening, will include some points in connexion with its pathology, diagnosis, prognosis, and, as a consequence, the unphilosophical nature of that theory which recommends blood-letting as a useful therapeutic measure. The second part, which I hope to be fortunate enough to bring forward before this Society on a future occasion, will relate to the *experience* of various plans of treatment, especially those of blood-letting, tartar emetic with and without blood-letting, the chloroform treatment, and the milder plans of practice, generally and statistically considered; to conclude with the plan of treatment which I would humbly and respectfully recommend. In this inquiry, I feel I shall need your indulgence, and would ask you to bear kindly with me if, in the course of this paper, I venture to oppose the views of many of the most eminent of our profession. I court philosophical inquiry: I seek only the elucidation of truth.

Pathology. It is not my intention to follow out the pathology, diagnosis, or treatment of pneumonia, in its second and third stages, except where incidentally necessary to illustrate a point under consideration; the more so as I believe that, in these two last stages, the diagnosis is comparatively easy, and all are agreed that general blood-letting is then comparatively noxious; to discountenance which is the chief object of this paper. It may, however, be right to premise by stating what I conceive to be, first, the pathological appearances of pneumonia in the first stage; and secondly, what are the principal morbid changes in the blood.

In regard to the first of these points, I may generally quote Dr. C. J. B. Williams's words as the exponents of my opinions, namely, that in the first stage there is not necessarily extravasation of blood in the air-cells, but that the vessels around these are engorged, dilated, and generally congested, in actual specific gravity heavier than healthy lung, but still floating in water. The mucous membrane of the smaller bronchi and air-cells may be redder than usual, but not remarkably so. When a portion of the lung is cut and com-

pressed, a frothy, rusty, or bloody serum exudes. The whole tissue is much reddened, and less elastic to the feel. This appearance is thus wholly due to congestion of the lungs. The occasional extravasation of blood in the bronchi is cadaveric, and analogous to what we observe in other hollow organs of the body, as in the intestines after peritonitis. (*Library of Medicine.*)

2. Looking to some of the pathological characters of the blood in this disease, when taken after death, it is not unusual to find it *fluid*, except in the immediate vicinity of the heart and large vessels; and, in some cases, again, *this fluid portion will not coagulate after death at all*, as in a case of Dr. J. Davy's. In other cases, the fibrinous coagula exist in large abundance; also with fluid blood, but which last coagulates very readily and firmly on exposure to the air. I believe the difference in these appearances is connected with the time during which the pneumonia has persisted, even although the disease may not have passed beyond the first stage; for then, and as the disease progresses, the state of the blood is intensely fibrinous; this tendency to hyperinosis being developed as the disease advances, and diminishing with the gradual cure. Thus Andral and Gavarret obtained, in fifty-eight cases of pneumonia (the healthy normal quantity of fibrin being 2.5 to 3.5 per 1000 parts of blood), the following result:—Maximum, 10.5; minimum, 3.4; mean, 6. From twelve other observations by various authors, the following result was also obtained:—

Rindskopf		No. 1		6.67
"		No. 2		12.72
"		No. 3		6.7
"		No. 4		5.9
"		No. 5		7.89
Simon		No. 1		9.15
"		No. 2		6.02
"		No. 3		5.63
Heller		1 case		4.3
"		—		6.1
Scherer		—		9.7
Becquerel and Rodier		—		7.4
Mean				8.18
Maximum				12.72
Minimum				4.3

This has been one of the reasons for which blood-letting, under an erroneous impression of its defibrinising tendency, has been so vigorously employed; whereas the hyperinosis may be due to special causes, in no way removed by venesection. I shall allude especially to some of these causes.

The correctness of these views can be further confirmed and elucidated by (a) experiments on the lower animals, (b) by accidents in the human subject, and (c) by chemical investigation.

(a) 1. You may induce pneumonia by thinning the blood, or diminishing its vitality, or by internal remedies which have a similar effect.

2. The subsequent deposition and increase of fibrin is due to hyperoxygenation of this fluid, both by increased activity in the lungs and in the cutaneous function.

(b.) Pneumonia is frequently induced in typhoid diseases, and is a common cause of death after wounds or surgical operations.

(c.) It is probably connected with a diminution in solvents of the blood, specially the quantity of chlorides, and secondarily the alkaline phosphates.

(a) 1. The following experiments of Magendie prove that it may be artificially produced by defibrinising the blood. (See *British and Foreign Review*, vol. viii.) Eight ounces of blood were taken from the jugular vein of a dog, and the fibrin thereof extracted. This defibrinised blood was then reinjected. The animal became distressed, refused food, lay down, and made several attempts to vomit. The experiment was repeated. The same symptoms appeared—the animal became weaker, and finally died. On a *post mortem* examination being made the next day, the body

was found very rigid, and offensive in smell. The lungs were hepatized; reddish serum was effused in the cavity of the thorax; and there was no true coagulation of the blood.

The experiment was repeated in a more gradual manner, and during several days. The results were identical; but in addition, there was inflammation and ulceration of the cornea, and the skin was covered with petechiæ and scabies. Now, Magendie believes that the pneumonia in all these cases is due to want of fibrin in the blood. In like manner, in cases of poisoning by the injection of ænanthic ether, the blood becomes uncoagulable; as also where putrid matter is injected in the blood, and pneumonia is induced. In the latter case, Magendie found that if a few drops of water impregnated with putrid meat, were injected into the blood of a dog, the animal first presented symptoms of undue activity, and was affected with fever; then, after a time it laid down, refused food, and vomited an immense quantity of black matter, characteristic of yellow fever. Pneumonia is the common *post mortem* appearance observed in all these cases. The same explanation may be given to the production of pneumonia by the asphyxiating gases from common sewers; in which cases, owing to a chemical change of a similar kind induced in the blood, the pneumonia passes seldom beyond the first stage into the second, but into a half gangrenous, half purulent destruction of the lung.

(a) 2. The hyperinosis which follows is due to hyperoxygenation of the blood. Dr. B. W. Richardson, a fellow of our Society, has proved that if an animal be placed in an atmosphere of oxygen, intense hyperinosis follows; and, according to his views, this is one of the reasons which lead to the death of the animal by obstructions of the heart through fibrinous coagula. In pneumonia, too, circumstances contribute to the same result, viz., the increased number of respirations, and the increased activity of the capillary cutaneous function. The number of respirations in a minute in this disease is frequently very great. Instead of 16 in a minute, the usual extremes are from 30 to 50, sometimes as high as 70 or 80, when suffocation seems threatened; but 30 or 40 respirations per minute may exist without the patient being conscious of particular dyspnoea. (Walshe.) The rapidity of the respiratory function thus more than compensates for any obstruction in the lungs, a greater quantity of oxygen is absorbed in a given time, and hyperinosis results. But the increased activity of the cutaneous function assists to bring on the same result. The skin, as is now invariably admitted, contributes to the maintenance of animal heat, and, as such, acts like a respiratory organ to consume the oxygen absorbed. The experiments of Becquerel and Breschet on rabbits, have proved this. When the skin was covered by an impermeable varnish, the temperature of the body fell, although, *a priori*, the evaporation of the sweat being also thereby impeded, an opposite effect might have been expected. It is also remarkable, that in pneumonia the extreme heat of skin is a characteristic of the disease. Indeed, this increased heat of the surface is by some physicians considered as pathognomonic of the disease, reaching to 105° and even higher; the normal temperature being 98°. It is a curious fact, and one which I may observe *en passant*, that this increased heat of skin is usually observed chiefly in diseases where the blood especially may be considered as poisoned, as in scarlatina, typhus, remittent, and intermittent fevers, yellow fever, puerperal fever, etc.; and thus it may, perhaps, be considered as evidence of a similar state of blood in pneumonia. Most probably this increased oxygenation of the blood is an abortive attempt made by the *vis medicatrix nature* to burn or consume the noxious agent; and this view would explain the invariable presence of feverish excitement in the animals operated upon by Magendie. Some of these animals, if left to themselves, recovered spontaneously as by this process; others, in whom a larger amount of poison was introduced, seemed overcome by it; and pneumonia amongst other poisonous effects was induced. It is also interesting to note, in regard to the second stage of pneumonia, in which hepatization or fibrinous consolidation occurs, that there is, according to Bouillaud, invariably

formed at the same time solid and resistant coagula in the right cavities of the heart and pulmonary arteries, due doubtless to the same common cause.

(b.) Pneumonia is frequently induced in typhoid diseases, and is a common cause of death after wounds or surgical operations. Its occurrence in typhus is so common as to need only to be mentioned. The recumbent position of the patient—whence the name sometimes given to this variety (pneumonia hypostatica)—favours the lodgment of the blood (in this disease unusually liquid and poisoned) in the small pulmonary capillaries, assisted also by the weakness of the cardiac action. But, from the time of Dupuytren to the present, it has been frequently observed after burns. Sir Charles Bell emphatically remarks that inflammation of the lungs is by far the most frequent cause of death after severe wounds, and especially in compound fractures. Mr. Erichsen, in some valuable observations on this subject in the *Medical Gazette*, observes that out of forty-one deaths occurring in the surgical wards of University College Hospital (in which an account of the state of the lungs had been kept), these viscera were found to be in the first or second stage of pneumonia in twenty-three cases. (Chevers on the cause of death after operations, *Guy's Hospital Reports*, viii, 83.) In these cases, the pneumonia is adynamic, and due to phlebitis; and the secondary absorption of unhealthy pus in the blood, by which its crasis is diseased.

(c.) This production of pneumonia is also probably connected with a diminution in the quantity of the solvents of the blood, especially the chlorides, and secondarily the alkaline phosphates. The albumen, casein, and fibrin, are doubtless in part held in solution by the alkaline phosphates and chlorides. In regard to the latter, in particular, if venous blood be made to flow in a vessel containing a solution of common salt, it will not coagulate; the fibrin is held in solution. But of the chlorides, the chloride of sodium especially is the salt which furnishes the soda base for the alkaline phosphates which maintain the alkalinity of the blood. It follows, therefore, that if the quantity of these salts is diminished in any way, it will lead to an impairment of the quality of the blood, and if carried beyond a certain point, to the deposition of the fibrin and albumen. It has been shown by Redtenbacher, from analysis in eighty cases, that during the stage of hepatization of pneumonia, the chlorides almost entirely disappear from the urine, but return on the resolution of the inflammation. Now, Liebig has shewn that analysis of the urine enables us to compare the incombustible constituents with those in the blood, and observation shews that in regard to the proportion of soluble salts with alkaline base, there is hardly any difference between them. If we incinerate the blood of a healthy person and also his urine, and lixiviate the ashes with water, the soluble salts of the blood ash are in nature the same as those of the urine; and it is extremely probable that in regard to their relative proportions also, a constant relation prevails (Liebig's *Letters*, p. 296). Whence he argues that we may, by reasoning backwards from the analysis of the urine, come to know the quality and composition of blood. Applying this argument to the chlorides, we may conclude that this salt is deficient in the blood.

Now the first effect of a diminution in the quantity of chloride of sodium in the food taken (and we may, therefore, infer in the blood, although from the usual presence of this salt in the blood in quantities almost normal, this diminution will not be very great), is to induce in man the phenomena of chlorosis, languor, debility, and pallidity, and even oedema and swelling of the legs (Robin and Verdeuil, ii, p. 186). Poggiale has, moreover, shewn that the number of blood-globules (on which the richness of the blood depends) is increased after the employment of common salt. The late Dr. A. T. Thomson informed me that purpura was common in those Russian soldiers who were unable to procure salt for their food. We infer, therefore, that the first effect of a diminution in chloride of sodium would be to *thin* the blood, and put the patient in a condition favourable to the production of pneumonia. But if this diminution continues, which the state of the urine

would seem to show, then it is not impossible a deposition of the fibrin would follow, not only by the diminution of the chloride only, as one of the solvents of the blood, but by the diminution of the alkaline tribasic phosphate. This diminished alkalinity of the blood, although from the character of the fluid difficult to measure accurately, would yet suffice to induce great changes in its constitution. That the serum exuded in some of the cavities of the body, as in some cases of puerperal fever, is acid, or that the pus effused in certain diseased states, as in anthrax and furunculus, is also acid, although both are in the normal state alkaline, is evidence of such a change being possible, although in the case of the blood, from its greater quantity and its uses, so positive a result is impossible. But that a minor degree of alkalinity may exist, appears very probable, from the acid state of the secretion generally in many diseases, as in rheumatism or gout, and their cure by alkalies. Probably hereafter it may be shown that pneumonia, as a result of the absorption of pus, is more or less connected with the degree of alkalinity. Mr. Henry Lee has shown that inflamed pus, the result of extreme inflammation, when injected into the blood, will produce fibrinous concretions, while that from chronic abscesses will not do so. I should, perhaps, here remark also, that the same absence of chlorides is observed in the urine of typhus, thus affording another link in the etiology of the two diseases.

These considerations lead me to consider that pneumonia, as it occurs in the present day, is essentially an adynamic disease. It is clear, I think, that unusual thinness of the blood predisposes to this disease. A statistical table of the number of robust as compared with delicate adults who are seized with pneumonia is still a desideratum. Among young children from two to five years old, Rilliet and Barthez have shown that the most delicate are the most obnoxious to the disease. Again, the causes of pneumonia are chiefly those which would influence weak persons. Thus, from Grisolle's inquiries, we learn that the following is the per-centage of the exciting causes of this disease. Thus,—

	Per cent.
Contusions of thorax	1.4
Chill	24.5
Fatigue	9.6
Mental emotions	3.2
Excess or debauch	2.4
No cause traceable	41.3
	1000

Many other considerations, some of which I shall allude to hereafter, confirm this view. Besides those afforded by the comparative results of treatment, and the manner in which the disease proves fatal, to be alluded to in the sequel, we might mention the almost invariable adynamic type and fatality of epidemic pneumonia both in man and the lower animals, its varieties in localities, and among the black races, etc., and various other points of interest, which I cannot dwell on at present.

Diagnosis. I should be sorry to impugn in any way the accuracy of the diagnosis made by medical men, in every respect equally qualified to make it as I am,—perhaps much more so. But I am satisfied that the returns of deaths from pneumonia in the Registrar-General's reports is not a correct index of medical diagnosis. Amongst fatal diseases, capillary bronchitis is not even named, and pneumonia is more commonly a cause of death than bronchitis. The number of deaths registered in London from the former disease (pneumonia), years 1840-4 inclusive, is about half that registered from phthisis, and eight times that from bronchitis; and for all England, in the years 1847-8, nearly the same as that from phthisis, and nearly twice those from bronchitis. These years, generally, are indiscriminately selected. Both these returns cannot be correct, and they are certainly at variance with the general impression, and opposed to hospital practice.

Table exhibiting the numbers of deaths from Diseases of the Respiratory Organs.

	London.				All England.	
	1840.	1841.	1842.	1843.	1847.	1848.
Laryngitis	26	27	40	44	762	867
Quinsy	74	172	67	98	556	569
Bronchitis	500	578	683	812	16499	14472
Pleurisy	88	93	77	92	1176	1029
Pneumonia	3927	3725	3982	4282	23447	21868
Hydrothorax	277	210	219	257	—	—
Asthma	1325	1371	1123	1085	6456	3920
Phthisis	7366	7485	7283	7223	25088	24435
Other diseases of Lungs	738	783	781	787	2775	2645
All dis. of Lungs	14190	14413	14325	14690	60800	76754

This return does not include influenza: in many cases of which, individuals die from capillary bronchitis, as shewn by Dr. Peacock.

The deaths from influenza in these same years, and for the same places mentioned, was,—

In 1840	71
In 1841	94
In 1842	88
In 1843	207
In 1847	4881
In 1848	3810

Comparing this result with hospitals, take, for instance, Vienna and Glasgow, we find there died in the—

	Bronchitis.	Pneumonia.	Phthisis.
Vienna General Hospital	428	260	1567
Glasgow Royal Infirmary, 1844-7	38	27	110

which results bear no analogy to the Registrar-General's report. I am aware that it has been objected that the hospitals contain but few children, whereas the Registrar-General's reports include children, and this objection has doubtless its value; still, I am satisfied that the deaths from pneumonia therein recorded are over stated. If this were not so, why should the results of *all England* be so much at variance with those of London? It is, moreover, well known how commonly a child dying of some chest disease is said to die from inflammation of the chest, when the term does not specify if the disease was pneumonia, bronchitis, pleuritis, or even phthisis, and yet is probably registered as pneumonia. This supposition may account for some of the cases, and the apparent discrepancy with hospital returns.

Pneumonia, in its first stages, is usually described as fever with more or less pain at the side, and dyspnea, cough with rusty expectoration. The physical sign most depended upon is fine crepitation heard at beginning of inspiration and end of expiration; then during the whole respiratory act, and, finally, at the end of inspiration and beginning of expiration. Such is Dr. Williams' definition, which I think is the correct one. Dr. Hughes limits this sound to the end of inspiration; unfortunately all these symptoms are not invariably present in pneumonia; and conversely they may be all present, and yet the disease may not be pneumonia.

Permit me especially to dwell on one of the physical signs, I mean *fine crepitation*, or *crepitant rhonchus*, a sound which Dr. Williams aptly compares to that which is produced by rubbing a lock of hair near the ear, between the finger and thumb. This sound is that heard in the first stage of pneumonia. But it is by no means confined to this disease. In acute pulmonary congestion, a disease of not uncommon occurrence at present among children; in capillary bronchitis; in some kinds of phthisis and pleuritis, pulmonary apoplexy, and oedema of the lungs, it is likewise heard; and, excepting by the *period* of its recurrence, and when taken in connexion with other concomitant symptoms, it cannot be said to be diagnostic of pneumonia.

I believe it gives rise to a good deal of confusion, by the great stress so frequently laid upon it.

Acute pulmonary congestion. This disease is not usually described otherwise than as forming part of the first stage of pneumonia; but I nevertheless believe it to be sometimes distinct. It appears very rapidly, in one or two hours, usually after exposure to cold. There is a feeling of oppression in the chest, if not pain, pulse very frequent, dyspnoea very urgent, aspect of face very anxious, and great restlessness. The physical signs are those of pneumonia in the first stage. These symptoms may disappear as suddenly as they appeared, under the influence of a drastic purge, or after a warm or a foot bath, or they may remit almost as a paroxysm of remittent fever, and generally are worse at night. It not unfrequently accompanies the bronchitis of children, in whom, while there is no actual evidence of tubercle, the sputa are nevertheless distended with blood, and there is great debility. I shall best illustrate the diseases by the three following cases.

CASE I. A gentleman, aged 18 years, having been engaged in fishing all day, and wading in the water, having retired to bed, was seized with a sense of suffocation at the chest, great dyspnoea; indeed, he was gasping for breath. The pulse was very rapid. A practitioner was called in, and the patient was bled *ad deliquium*. The relief was instantaneous; but the convalescence was tardy, and, finally, in the course of it purpura supervened.

CASE II. A medical student, after much walking and fatigue, was exposed to cold. All the above symptoms, with fine crepitation, were developed the next day, only the dyspnoea was less urgent. A pediluvium at night, quiet, and a drastic purge removed the disease in a couple of days.

CASE III. This was a hospital case. A patient, in whom no phthisis was detected, after exposure to cold, came in with partial hæmoptysis and dyspnoea. There was no marked dulness observed, but in several parts of the chest, here and there, there was fine crepitation, intermixed with larger crepitation. There was also general anorexia present. The case was put down as one of pneumonia, and treated with tartar emetic. The recovery was long and tedious.

The diagnosis between this variety of disease and pneumonia may be stated as follows.

By the general symptoms. Its rapid occurrence, absence of any marked lividity, occasional remittent character, generally the dyspnoea is greater. There may be no cough present. The expectoration, at first wanting, is not frothy, usually viscid and mucous, and not unfrequently tinged with blood.

By the physical signs. No abnormal dulness anywhere. The fine crepitation not constant, and even varying occasionally in position and mixed with larger crepitation. In some chronic cases its persistence for a longer time than would be likely had the case been one of pneumonia without consolidation supervening, will help the diagnosis.

Capillary bronchitis is a disease fearfully fatal, and frequently mistaken for pneumonia in the first stage. I cannot do better here than reproduce the words of Dr. Peacock, who, in his work on Influenza, has so ably given the signs of diagnosis between this disease and pneumonia. These are—

"1. *General symptoms.* The less sthenic character of the febrile derangement which accompanies all stages of the disease; the greater degree of lividity of the face and extremities; the more rapid prostration of strength, and the earlier appearance of symptoms of asphyxia.

"2. *By the physical signs,* the absence of dulness on percussion, and the occurrence of abnormal clearness at the late periods, the fine crepitation generally, first audible in the inferior parts of one or both dorsal regions, and then rapidly spreading over all parts of the chest, and the tendency of this sign to pass into the subcrepitant or mucous rhonchus, rather than give place to condensation, such as bronchial respiration, and increased resonance of voice and cough." (Dr. Hughes distinguishes this kind of crepitation from that of pneumonia, because it is not con-

fined to the end of inspiration. This statement, however, does not accord with Dr. Williams', before quoted).

"3. By the peculiar character of the dyspnoea, cough, and expectoration. The respiratory movements are rapid, short, and hurried, rather than laborious, irregular; and there is a sense of constriction in the chest without pain. The cough comes on generally in paroxysms, and is fatiguing from its frequency, rather than painful and very severe. The sputum consists of whitish-coloured and viscid pellets, free from air, which have a tendency to become aggregated with tenacious, solid, and irregularly-shaped masses, and wants the russet colour, glary adhesive quality and small air bubbles which characterize the expectoration in pneumonia."

Pleuritis. There is a peculiar variety of pleuritic disease, of which I have seen a few examples, in which, with most of the symptoms of pneumonia, this fine crepitation is heard to a remarkable degree. I remember such a case occurring in the practice of my late lamented friend and teacher, Dr. John Taylor, at University College Hospital. It was the case of a female, Jane Fife by name, aged 45 years, who came in with cough, palpitation, great prostration, double pleurodynia, and urgent dyspnoea. There had been general pyrexia; there was general emaciation; the physical signs were those of general dulness posteriorly and inferiorly, with bronchial respiration; there were no signs of effusion. The case was, therefore, put down and treated as pneumonia in the first and second stage. She was consequently treated by blood-letting, cupping, etc., but the fine crepitation persisted for weeks, and she left the hospital with it, although she had then neither cough nor expectoration. Dr. Taylor could not certainly conclude the cause of this, whether it was one of persistent pneumonia, oedema, or pleuritis, although it was believed the crepitation was finer than oedema, and less so, if anything, than pneumonia.

The consolidation did not vary much. Now a French writer in the *Archives Générales de Médecine* for October 1843, has there shewn that this fine crepitation may be, and often is, produced in the pleura, as well as in the lungs, and is only a variety of the friction sound. In regard to the period of the pleural affection, he has observed it in the beginning of pleurisy to be gradually succeeded by the ordinary friction sound. 2. At a later period, when false membranes only remained, the serum having been already absorbed, he has also observed it in the neighbourhood of a friction sound. This interpretation was subsequently given to Fife's case.

Dr. Walshe mentions a case of phthisis in his *Manual* in which there was slight serous effusion in the meshes of adventitious cellular tissue produced on the pleura, as proved by a *post mortem* examination; and in which, during life, he had heard fine crepitation. The case mentioned of Jane Fife was admitted Nov. 30th; and yet as late as Jan. 13th, fine crepitation was heard. It is highly probable, therefore, that some of these cases of pleuritis with so little effusion are sometimes mistaken for pneumonia; and how frequent partial pleurisy is, in which little or no notice of the disease has been taken by the patient, is evidenced by the *post mortem* appearances on the pleura. In former times, these cases would have been treated as pleurodynia; and in these stethoscopic times, perhaps, more heroically as pneumonia. The means of diagnosis here would be in the history of the case chiefly, and in the character of the dyspnoea, absence of lividity of the face, and the nature and extent of the crepitation. The French author above alluded to, states, the crepitations in these varieties of pleuritis are less numerous, less frequent or instantaneous, than those of pneumonia; they also occur less in puffs, and are not quite so fine. They either resemble the dry crepitation observed by Laennec in emphysema, or returning crepitation after consolidation in pneumonia; although he acknowledges that the diagnosis is very difficult. He supposes that, when the pleura is moist, no sound is produced by the rubbing of one upon the other; but when inflammation occurs, then certain isolated points are formed on the

pleura, which become rough, and then, by rubbing one against another, produce this fine crepitation. When these points are more numerous, then the ordinary friction sound only is heard.

There are two other diseases, both of which give rise to fine crepitation. But I need not dwell much upon either of these. In the first, oedema of the lungs, the disease is usually accompanied with general anasarca, and the crepitation scarcely so fine as that of pneumonia. In the second, hæmorrhage from the lungs, in which the uncoagulated blood may have penetrated to some of the finer bronchi, fine crepitation may be induced.

Dr. Hughes, of Guy's Hospital, distinguishes this kind of crepitation still further. In these cases, the crepitation is moist, and accompanies the expiration; whereas that of pneumonia is dry, and heard only at the end of inspiration, and is finer. Looking, however, to the nature of these diseases, and their concomitant symptoms, a mistake in the diagnosis can only be the result of inadvertency; yet I have seen the mistake made by an eminent stethoscopist, by whom a case of hæmorrhage in the lungs was mistaken for one of pneumonia, and treated accordingly by tartar emetic, etc.

Prognosis. If I am right in asserting that cases of simple pulmonary congestion, capillary bronchitis, pleurisy, etc., have been treated as cases of pneumonia, it is not to be wondered at that the experience of different practitioners should be very different. On one side, the eminent Dr. Williams asserts that pneumonia is at all times to be considered as a serious disease; while, on the other, Chomel asserts that it is in most cases a benignant disease; and, when the individual attacked is of favourable age, he will almost invariably get well under any kind of treatment. Dr. Walshe, admitting the disease to be most serious within unfavourable ages, states that there are periods of life at which it is not easy, with common prudence, to lose a sufferer from idiopathic or sthenic pneumonia. I am myself inclined, after personal inquiry for some years, both in London and in the larger continental hospitals, especially Paris and Vienna, to believe that genuine uncomplicated pneumonia is a benignant disease. I am ready to admit that pneumonia is most fatal in England and Scotland; but this chiefly arises because the proportion of complicated to uncomplicated cases is about fifty-three per cent., and because the disease is generally too actively treated. I shall, however, again allude to the mortality in the sequel.

Treatment. I cannot here enter at length into the various methods of cure adopted in pneumonia—in other words, the experience of practice. I shall speak of three only—1, that by active blood-letting, local and general; 2, that by tartar emetic; 3, that by chloroform, emollients, and milder measures generally; and, lastly, I shall allude to that which I follow out myself, and which I would venture humbly and respectfully to recommend.

In speaking, however, of blood-letting as a remedy, it is necessary I should allude to some of the theories upon which it is based. By premising what I have to say on the experience of that practice, by such an inquiry, I shall be in a better position to disprove the utility of its employment. The advocates of blood-letting in pneumonia recommend its use chiefly on the following grounds—

1. It diminishes the amount of fibrin, which is in excess in this disease.
2. It removes the fever and general pyrexia.
3. It removes the local pain, dyspnoea, and congestion.
4. Patients affected with pneumonia bear depletion well, and it seems therefore unphilosophical not to afford them by it the relief which it ensures.
5. It diminishes the mortality. This last point can only be answered by comparing the results of different methods of practice, and, as such, forms part of the second portion of this paper.

Let us examine these four several points.

1. It is now admitted that it is only when blood-letting is carried to an excess that the fibrin is diminished.

In pneumonia, according to Andral and Gavarret, and

other observers, the blood, on analysis, was found, after repeated venesections, to contain less solid constituents and blood-corpuscles, but more fibrin and more solid residue of the serum.

Analyses of Andral and Gavarret.

	Venesection.	Day of disease.	Amt. fibrin.
1st case	1	2	4.0
"	2	3	5.5
"	3	5	6.5
"	4	7	9.
2nd case	1	3	5.2
"	2	4	7.0
"	3	5	6.9
"	4	6	8.
3rd case	1	4	5.5
"	2	5	6.8
"	3	9	6.4

Analyses of Rindskopf.

	Venesection.	Day of disease.	Amt. fibrin.
1st case	1	not stated	6.7
"	2	"	7.7
2nd case	2	"	5.9
"	3	"	7.7
"	4	"	10.3
"	5	"	8.1
3rd case	2	"	7.9
"	3	"	0.1
"	4	"	9.5

In another case of pneumonia biliosa, analysed by Scherer—

1 venesection	fibrin amounted to 9.7 in 1000 parts
2	" " 9.4 "
3	" " 12.7 "
4	" " 8.8 "

The same fact has been observed by Andral and Gavarret in rheumatism.

Venesection.	Day of disease.	Quantity of fibrin.
1	8	6.1
2	9	7.2
3	10	7.8
4	13	10.2
5	17	9.
6	28	7.

And Simon has observed the same fact in peritonitis. Indeed, the above statistics are taken from his work; and it is only when bleedings are carried to a great extent that the fibrin is diminished. Moderate blood-letting is therefore useless. Magendie's experiments, moreover, before quoted, go to prove that defibrinisation of the blood is a common cause of pneumonia; and, if blood-letting had the effect of diminishing its quantity, it might not cure, but rather give rise to the disease.

But, 2. It is said that bleeding diminishes the severity of the general symptoms. Granted: but, to do this, the blood-letting must be begun early, and taken in full quantities; and the secondary consequences, namely, debility and incapacity to follow out any occupation for a long period afterwards, are the results also obtained.

3. It is said to diminish the severity of the local symptoms. But such a result is rather the exception than the rule. Grisolle, the great authority on pneumonia, admits this to have been the case in some 131 cases out of 232, to which I shall again allude, and in which blood-letting had no effect. In these, however, the disease has passed the second stage. Remarking on these cases, he says, "We may hence conclude that the blood-letting had no effect on the progress of the disease, or if it had any good effect (which he believes probable), that it was only in disposing the system to absorb with better advantage the antimonials." Indeed, he himself admits that if the pneumonia has attained a certain degree of intensity, it cannot be checked, or cut short by blood-letting, though practised ever so freely, and these statements are perfectly borne out by the experience of Chomel and Rasori. Among ourselves, Dr. Todd (*Medical Times*, vol. xxv., p. 484) believes that whether in sthenic or asthenic pneumonia, the tendency is to depress the general powers of life, or (p. 486) pneumonia has a fatal tendency in proportion as it tends to exhaust;

for which reason he discards active treatment by blood-letting.

Laennec advised moderate blood-letting, and then only in strong and robust patients. The great Louis of the Hôtel Dieu remarks, "It is an error to believe you can regulate pneumonia by blood-letting. In all cases, if the bleeding is practised in the period when the disease is on the increase, the local symptoms are thereby aggravated, although the general symptoms may improve." He believes, however, it will in the end shorten the duration of the disease. From his cases, he concluded that the disease lasted only eighteen days, when bleeding was resorted to before the fourth day of the disease, when after that period it lasted twenty-one days. This is the result obtained at La Charité and Pitié. The experience of these hospitals proves that by blood-letting early, you only anticipate the period of cure, in some of the cases which yield to it.

4. Patients affected with pneumonia bear depletion well. It seems, therefore, unphilosophical not to afford them that relief which this operation ensures. But the truth of this proposition is founded in part on the truth of the three former; and if these be disputed, there cannot be said to be relief from depletion. And, again, patients do not bear depletion well in the present day. The type of existing diseases is, and indeed has been for some years back, adynamic, and in none is this more true than for pneumonia, which is fatal exactly in proportion as it is adynamic. Patients do not bear depletion as formerly; thirty years ago powerful medicines and heroic blood-letting was the order of the day, and eminently successful. The practitioner who would now follow the practice of his forefathers in this respect would meet with universal reprobation. Hence one of the reasons that that archquackery, homoeopathy, has taken so well among many. Typhus and low fevers have been upon the increase, and generally those diseases arising from cachectic blood, a better instance than which I could scarcely give than the extraordinary frequency of furuncular and carbuncular disease.

The following are the number of deaths from typhus and pneumonia for the years 1839—1850, taking typhus as the measurement of debility in the population.

Year.	Typhus.	Pneumonia.
1839	1819	3687
1840	1262	3776
1841	1151	3608
1842	1174	3923
1843	2083	4224
1844	1696	4064
1845	1501	3896
1846	1796	3151
1847	3181	4290
1848	3569	3409
1849	2479	3503
1850	1923	3108

Thus blood-letting, in theory at least, does not seem advisable. The reason it sometimes does good is probably stated correctly by Dr. B. W. Richardson, namely, by diminishing for the time the flow of blood to the lungs, and thus arresting its hyperoxygenation.

To recapitulate, therefore, I think I have proved that—

1. Pneumonia is characterised at first by unusual fluidity of the blood, and may be produced by causes which tend to poison the blood, as proved by experiments on the lower animals, accidents in the human body, and chemical examination.

2. It is characterised by an increased amount of fibrin, from hyperoxygenation, both in the lungs and skin, and a deficiency of chlorides, in the blood.

From such facts I conclude the disease is essentially adynamic in kind.

3. In regard to diagnosis, there is reason to believe it is mistaken for simple pulmonary congestion, capillary bronchitis, and some kinds of pleuritis.

Lastly, the theory which advocates blood-letting in this disease is both physiologically and pathologically unphilosophical.

Montague Square, March 1855.

NOTE ON THE FORAMEN OVALE.

By J. L. W. THUDICHUM, M.D.

[Read before the Physiological Section of the Medical Society of London, March 12th, 1855.]

Two hearts which I have lately removed, one from a sheep, and the other from a human female, have presented certain peculiarities in reference to the communications between the right and left side of the heart. I therefore propose to give a brief description of the specimens, and then to make a few remarks on the closure of the foramen ovale.

1. *Heart of a sheep.* The organ is pale; and all parts show fatty degeneration. The *foramen ovale proper* is closed, but there existed an oblique canal of about half an inch in length, passing from the right to the left auricle, and bounded by folds of membrane projecting into each cavity.

2. *Heart of a woman, aged 38, who died of pneumonia.* It showed the same transverse canal as the heart of the sheep, ending in the left auricle under what might be called the remnants of the free valve of the foramen ovale. The latter is attached to the front border of the fossa ovalis by three bridges of membrane and also by several filaments. There is an opening of the size of a pin's head through the fossa ovalis itself.

REMARKS. These preparations have suggested to me two questions: 1. As to the mode of closure of the foramen ovale; 2. The time at which it is closed.

1. *Mode of Closure of the Foramen Ovale.* There are two distinct modes of occlusion of the foramen ovale in man and animals. The first is what has been termed

1. *Occlusion of the Foramen by Filaments.* They are said to exist in the calf and the foal. Flourens says, that in the calf they exist as early as the second month of embryonal life. In his little work, *Histoire de la Découverte de la Circulation*, at p. 67, he gives a description of the mode of occlusion by filaments, which has a direct bearing on the preparations on the table.

"The filaments are always developed along with a membrane which grows from the posterior border of the foramen. From the opposite or front border the filaments rise, to the number of twelve or fifteen at least. But they almost directly join each other, separating again to join anew, and then forming a network, the meshes of which increase in size in proportion to their distance from the margin. This net is, so to speak, suspended in the left auricle, and ends with three or four filaments, which are inserted in the left wall of the advancing septum of the auricles, on a spot about one-fifth of an inch distant from the front margin of the foramen, from which the filaments grew.

"As the animal is developed, the membrane and the network of filaments become thicker, in consequence of which the meshes become smaller and disappear. The inserted ends always remain on the same spot, and do not vary in number. After a certain time, there are only three or four arches left, formed by the free border of the membrane and the filaments, which now have become very much shortened and thickened. These arches disappear in their turn by the same process; and there is no longer any communication left between the two auricles. Before this communication is completely closed, there is a very oblique canal left, which extends from the right into the left auricle. Sometimes this canal persists in the adult."

Flourens states this for the cow and the sheep. He is borne out by the specimen on the table; and we have, moreover, a preparation which shows that the same may be the case in man.

2. *Occlusion by the Valvula Foraminis Ovalis alone without any filaments.* Here the foramen closes by the hypertrophy of the membrane and its insertion into the left wall of the septum auricularum. After this mode of closure there may be an oblique canal, which, according to Flourens, can persist in the adult. This persistence takes place in the dog, in the rabbit, and in man. Bischoff (*History of De-*