The forms of pneumonia / O. Sturges.

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from the Author -

(16)

THE FORMS OF PNEUMONIA.

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XV. THE FORMS OF PNEUMONIA.

In framing the statistical table of the Medical Registrar's Report for 1865, I was struck by the very slender bonds which held together under a common name affections of the most various kinds. This is especially the case with regard to pneumonia—a term which is made to comprehend diseases which in their nature and history are altogether opposite. The question occurred whether it might not be possible, by bringing together a large number of such cases, to effect some natural arrangement of the whole into groups, each of which should comprise instances of disease more or less obviously allied to one another. To test this point in the only practical way, it was necessary to review all the cases described as pneumonia in our hospital records. The result is epitomised in the accompanying tables, which show very remarkably how pathological conditions, sufficiently similar to warrant the application of a common name to the series, are associated with every variety of clinical phenomena. Before alluding further to these tables, it may be well to refer shortly to the views of writers on the subject of pneumonia and its modifications.

A modern author has sought to bring diseases within the limits of terse, dogmatic description, heading his subjects with a short definition after the manner of the exact sciences. The definition of pneumonia is as follows: "A disease expressed by severe febrile symptoms, which come on suddenly, attaining in a few hours a great intensity, and which undergo a no less sudden abatement or improvement between the fifth and tenth day." We read subsequently of its well-marked stages, of their pathological significance, and of the sounds belonging to each. The affection is described throughout with an unusual precision; it enters upon various phases in

a prescribed order, and each step in its course has its appropriate physical sign, the significance of which can be nicely appreciated. By one writer, indeed, the history of the disease has been sketched out in a programme of parallel columns, the sounds of each period being described alongside of the supposed morbid conditions which account for them. It would thus appear, at first sight, that in a given case the physician would not only recognise the stage the disease had reached, but be able further to give some account of its antecedent phenomena, and to forecast with confidence something of its subsequent history.

But we are next told that, although this is pneumonia, that pneumonia is not always of this form; that inflammation is wont to attack the lung insidiously and in the course of other diseases; that, so occurring, its features may be modified in various ways—so far modified, indeed, sometimes as to escape recognition altogether. It is then called (and no one can quarrel with the name) "latent pneumonia." One author, as angry with a disease which so escapes detection, speaks of it when it assumes this shape as "a low, sneaking inflammation."* Similarly, the writer whose definition I first quoted alludes later on to obscure and latent forms of attack which have no resemblance to his first description; and Sir Thomas Watson appends to his graphic description of the disease the caution: "All that I have hitherto been saying relates to acute pneumonia, as occurring in a previously healthy person; but pneumonia having that character and so occurring is a much less common disorder than most persons appear to suppose, or than I formerly thought it to be." "Inflammation of the pulmonary substance," adds this last author, "is apt to supervene insidiously upon various disorders which are of every-day occurrence—upon bronchitis, upon phthisis, upon disease of the heart, and upon fevers, especially the exanthematous fevers." In the more dogmatic enunciations of Dr. Walshe we find the varieties of pneumonia arranged in a table.† From this it appears that, in its "secondary or intercurrent origin," pneumonia depends on a list of acute diseases—in which rheumatic fever occupies the

* Dr. Sieveking in Jones and Sieveking's *Pathology*, p. 428.

† Walshe *On Diseases of the Heart and Chest*, 2d edition, p. 439.

first place, and acute diseases of the brain the last—and on a list of chronic diseases, in which pulmonary tuberculisation and cancer occupy together the first place and Bright's disease the last. Subsequently he writes :* "Instead of running the ordinary course, with marked subjective symptoms, pneumonia may be completely latent. Pneumonia occurs in this form solely under circumstances of general physical debility." He concludes by reminding us that, in treatment, "we must remember that the inflammatory character of the local malady is modified more or less seriously by the general state of the system." "It is exceedingly probable," he adds, "that various differences exist in the intimate constitution of many of the intercurrent pneumonias, though at present no absolute proof of the fact can be given." Other authorities might be quoted who thus vaguely allude to declensions from the typical form of pneumonia. Thus Dr. Stokes speaks of "typhoid pneumonia," which he explains to "include a variety of cases seen more frequently in hospital than in private practice; in which, whether from the low state of the constitution, the complication with other local diseases, or the pulmonary affection being secondary to a general morbid condition, we find a pneumonia often more or less latent, and accompanied by extreme prostration."†

All this seems to be a very inadequate account of the matter. First we are told at length of a disease which is characterised by certain well-defined symptoms. It is next intimated that these symptoms undergo modifications under various circumstances; and that, in fact, the modified disease is far more common than the simple one. It soon appears, moreover, that by "modifications" no less is meant than that the disease assumes an entirely new shape. The statement amounts to this. In certain cases, in a certain definite way, the lung becomes consolidated (inflamed, as some believe), and this change is accompanied by such and such symptoms; but much more often the lung is wont to become consolidated (inflamed or not) under very different circumstances from these; and although all these forms of lung-consolidation are called pneumonia, the description we give applies only to the least common.

* P. 443. † Stokes *On the Chest*, p. 338.

It is true, indeed, that some authors have described in order certain varieties of pneumonia, but these varieties refer only to as many distinct pathological conditions: they are not mentioned with any view to illustrate the various clinical aspects of the disease. For instance, Dr. Todd* recognises, besides the simple disease, four other forms—viz. pneumonia complicated with acute gout or rheumatism, strumous, typhoid, and traumatic pneumonia. He rejects the term lobular pneumonia, as referring only to carnification of the lung from absence of air. Dr. Fuller, on the other hand, enumerates five varieties. In the first he places cases which are especially apt to occur in rheumatic persons, and which are characterised by inflammation of the interlobular cellular tissue—a condition, it must be observed, which has never been actually demonstrated. Next comes lobular pneumonia—a form which Dr. Fuller admits does not occur so often as has been supposed. The third variety includes all those cases where the disease is of secondary origin. Fourthly, latent pneumonia is mentioned. "Its peculiarity," we read, "is simply that which the name implies, and which renders the mischief very likely to be overlooked." Lastly comes chronic pneumonia—a variety which is said to be extremely rare.† Here, therefore, in a list where a form of pneumonia not known to the pathologist occupies the first place, and lobular pneumonia—admitted to be rare—the second, the consecutive form of the disease stands third; and though it is admitted to be a very frequent cause of death, no attempt is made to sketch its clinical features, or to arrange in order the diseases most obnoxious to it.

In this obscurity, one is inclined to inquire whether the various conditions of lung here alluded to have really enough in common to entitle them to be called by a common name; to question whether that pathology is correct which speaks of "a low, insidious inflammation stealing upon the lungs" in

* Todd *On Acute Diseases*, p. 368.

† *On Diseases of the Chest*, p. 241. This mode of division may give rise to confusion from the clinical differences being mixed up with the pathological. Thus a case of pneumonia may belong clinically to the fourth variety as being latent, and anatomically to the second variety as being lobular; while pneumonia of the first variety is necessarily by its definition pneumonia of the third variety.

the course of fever or of structural diseases of old standing, and which ascribes to secondary pneumonia a large proportion of deaths after surgical operations and exhausting diseases. So long as inflammation has its present associations, such an inquiry cannot be without practical importance. I shall attempt to follow it first, and very briefly, by a consideration of the causes which may be supposed to influence, or, more or less directly, to produce, the various changes which the lungs undergo in so-called pneumonia, and the relation of these to the phenomena of true inflammation; secondly (and this is the main object of this paper), I shall endeavour, by a reference to the clinical records of our Hospital, to classify a number of reported cases of pneumonia, in all of which death was ascribed wholly or mainly to that disease.

Without alluding in detail to the several steps in the inflammatory process, it may be stated generally that the condition which immediately precedes it, viz. hyperæmia and congestion, may be closely imitated by mechanical means.* Venous obstruction, however produced, will give rise to an exudation which will be serous, or albuminous, or spontaneously coagulable, according as the pressure is less or greater. It is impossible, in short, to distinguish the hyperæmia due to this mere passive congestion from that which is due to commencing inflammation. It is only in the subsequent stages that the intimate characters of the exudation absolutely distinguish the two states. In reference to this point, Dr. Robinson has recorded some experiments of his own upon the renal circulation, designed to show that the character of the exudation which takes place under pressure may be made to approach very nearly to that which occurs as a product of inflammation. By obstructing in various ways the flow of blood in the renal vein, this observer obtained not only liquid albumen and blood, but fibrine. He concluded that "simple compression of the blood in its smaller vessels will, in a direct ratio to the degree of intensity of that compression, cause the exudation of an albuminous fluid, of coagulating lymph, or the extravasation of blood: its immediate effects, therefore, precisely resemble those of inflammation"—a result he would have been led to "infer from the primary

* Simon's *Lectures*, pp. 98, 99.

effects of inflammation being identical with those of and in compression of the blood and the mere consequences of that physical cause."*

But, it is said, the true inflammatory effusion thus rudely imitated has in fact characters of its own which distinguish it from any that can be mechanically produced. "In the first place, the inflammatory effusion tends to contain ingredients in larger proportion than that in which they exist in the blood. In the second place, the inflammatory effusion teems with organic life."† It is unnecessary to investigate just now these chemical and microscopic distinctions. It may be conceded that inflammation is a process which cannot be exactly imitated by any manipulation of ours; roughly, however, a resemblance between the mechanical result and the inflammatory seems to be admitted. I say, a rough resemblance; but really it is a very close one: under both conditions liquor sanguinis is poured out; in both the blastema undergoes changes and develops organic forms (only more forms in the one case than in the other).‡ The difference, it might perhaps be contended, is more of degree than of kind. It is enough for our purpose that to the naked eye the resemblance is perfect; that the changes which parenchymatous organs undergo, whether in size or increased solidity or in appearance on fracture or on section, may be due to the one kind of infiltration as much as to the other.

If these observations be applied to the lungs, it will be at once allowed that the phenomena which attend either mechanical obstruction or true inflammation elsewhere must occur with considerable modification in organs so constructed. For besides that the lungs form a second and in some respects

* Dr. Robinson's paper is in the 26th volume of the *Medico-Chirurgical Transactions*. It records the result of twenty experiments where artificial impediment obstructed the flow of blood through the renal vein. In the most striking of these, where the obstruction was incomplete, lymph was found in the bladder. "I am not aware," says the author, "that any other instance is recorded of coagulating lymph as a consequence of simple compression of the blood by venous obstruction." There follow fourteen further experiments, in which it was sought by various means to direct an increased determination of blood to one or both kidneys.

† Simon in Holmes's *Surgery*, vol. i. p. 27.

‡ Walshe *On the Lungs*, p. 346.

an independent circulation, by means of which the whole mass of blood is continually undergoing chemical change, consider that in them this fluid, abundant beyond comparison of other organs, is at all points and continually being brought into almost immediate contact with the external air, separated from it in fact by a membrane of such extreme tenuity, that physiologists are still disputing about its structure; consider that this vast network of capillary vessels is in connection, not as elsewhere, with an areolar tissue to surround and support it, but with the air-vesicles; or more truly, that the capillaries project into these cavities, their walls "exposed and bare," and with nothing save the thin membrane of the capillary itself between the air and the blood. In an apparatus so delicate, and charged with such functions, one would suppose that a very small disturbance, mechanical or otherwise, would suffice to produce changes, whether by exudation, or hæmorrhage, or what not, such as would occur only rarely and as an extreme result in the more compact and less blood-laden organs. If then we learn of the kidney, for instance, that by partial compression of the renal vein products are obtained which resemble nearly those which belong to inflammation, we should infer, I think, that such result would be obtained much more certainly and with much less inducement from any similar embarrassment of the pulmonary circulation. Still further, when it came to be considered that obstruction of a purely mechanical kind, rare elsewhere, is often obtained here by defect in the valves of the heart, we should be prepared to find that, in such cases more especially, exudation into the air-vesicles was by no means unfrequent. In a word, apart from the doctrine which would account for all morbid changes in organs by reference to inflammatory action, it would seem that consolidation of the lung might often be amply accounted for on physical grounds alone.

It is true, no doubt, that this purely mechanical explanation is not alone sufficient to account for all cases of hepatisation; to some even it may not be at all applicable. It is reasonable to suppose that there is a middle ground between consolidation mechanically produced, and that which is wholly the result of inflammation, where the same effect follows

from the joint operation of both physical and vital causes. Thus in some cases we may appeal to the operation of that law, so to call it, which requires that the circulating fluid, and the channels through which it flows, should bear a certain relation to each other. It may be conceived, that when this relation is disturbed by defect on either side, the blood, hampered and retarded in its progress through the capillaries, may on that account the more readily part with some of its constituents. It may be conceived otherwise; and in some cases I shall try to show presently that it can be proved that poisoned blood, or blood overcharged with some of its natural ingredients, may be so far impeded in its course that its activity is not sufficient to keep it fluid—it coagulates in the vessels themselves. Such a result, however it may differ intimately from pneumonia, leads at least to consolidation of the lung, and must give rise to physical signs during life identical with those ascribed to pulmonary inflammation. Lastly (it is unnecessary here to consider the solidification which results from external pressure), we may refer to those cases of protracted dying, where a sort of life is maintained long after the heart has ceased to be sufficient for its work. Here it is well known that in the remote and depending portions of the lung a condition is wont to arise which has often been described as the result of inflammation.*

In these several ways, some admitting of a physical explanation, others nearly allied to or identical with the inflammatory process, it is easy to conceive that the parenchyma of the lung may become infiltrated or consolidated. Such a result would appear to be no sure sign of inflammation. Do we then obtain more certain indications from minute pathology? I turn again for a moment to authorities. Dr. Todd indeed states his own opinion very plainly, that the

* It may appear, perhaps, that in this endeavour to restrict the term 'inflammation,' I am in fact only alluding to some of the circumstances under which it is allowed that certain products occur in the lungs which pathologists are willing to recognise as inflammatory products. I cannot think so. Inflammation, it must be remembered, has a programme of its own, in which mere physical agency plays quite a subordinate part. "A part does not inflame because it receives more blood; it receives more blood because it is inflamed. The afflux is due to an influence primarily exerted by the part" (Simon's *Pathology*).

post-mortem appearances due to passive congestion or to early inflammation are precisely similar, and relies on the lung being hepatised as "furnishing the only certain indication of pneumonia having existed during life."* Other authors would be more precise. Thus Dr. Walshe, in describing the difference between "passive congestion purely a result of failing vital power" and actual inflammation, writes: "As a general rule the distinction can be effected through the following characters: congested tissue is externally less deeply livid, internally shows blood-staining, partially removable by washing; it collapses more, crepitates more, furnishes more markedly frothy and less markedly red liquid on section; is of lighter specific gravity, and firmer in consistence than inflamed substance."† As yet, be it observed, no minute distinctions are mentioned; and to say that one condition is more markedly frothy or more markedly crepitant than the other is a difference of degree which must be worthless as a guide when it is allowed that the amount of frothiness and of crepitus is liable to variation in the several stages of both processes. But besides this passive congestion, we read of active congestion and mechanical congestion;‡ and the last form has three classes. Two of these "resemble anatomically the active variety, while the third more nearly approaches the passive." Of this third class it is said, moreover, that amongst its results are "low pneumonic exudation, hæmorrhage," &c.

The practical value of these fine distinctions is somewhat impaired by what follows. "In exceptional cases," we read, "where the blood is hypinotic, congested tissue may break as readily under pressure as if it were inflamed; and sometimes from the mode of decumbency after death, sometimes from inexplicable causes, either lung may be much more involved than its fellow." In this difficulty reliance is to be placed in "the presence of exudation-cells and blastema *in any quantity*," as "deposing unmistakably in favour of inflammation;" but it is presently added, "I believe cell-growth on a limited scale to be perfectly compatible with mere passive

* *Acute Diseases*, p. 371. † Walshe *On the Lungs*, p. 347.

‡ "Congestion of the lung is of mechanical, passive, or active mechanism." *Walshe*, p. 345.

congestion." So that here again it is a question of more or less. On the whole, I think, that this attempt to distinguish active from passive congestion, and both from early inflammation, altogether fails, and that in spite of these numerous divisions and subdivisions of Dr. Walshe, any ordinary observer would still find considerable difficulty in referring a congested lung, from the mere inspection of it, to its proper place in this complicated arrangement.

The case is somewhat different when the stage of consolidation is reached. Here, it is said, we have evidence of an exudation which has peculiar characters of its own, distinguishing it from all other exudations; "that it contains certain ingredients in larger proportion than that in which they exist in the blood, and that it teems with organic forms."* Here, at least, the distinction is broad and unmistakable between mere mechanical effects and that which is essentially a vital process. It is not necessary to deny it. But may not consolidation too occur under other conditions than these? Dr. Walshe seems to agree that in exceptional cases it may. Dr. Robinson's experiments seem to show that probably it very often does. And, indeed, by earlier observers, and apart from minute anatomy, pulmonary consolidation is spoken of as resulting from purely physical causes. Many years ago Dr. Williams wrote: "It seems to me that the same mechanical congestion which sometimes leads to an effusion of blood in the tissue, constituting pulmonary apoplexy in other cases, if long continued enough, terminates in an effusion of lymph and an obliteration and consolidation of the pulmonary tissue."† Stokes, also alluding to a passage in Andral‡ to a similar effect, says, "We must agree with him in the opinion that the solidity of pneumonia arises, not from any deposition of lymph, but merely from an excessive congestion of blood."§

Now let it be once allowed that hepatisation is not necessarily the result of an inflammatory process, and we need not stop to consider how far the microscope would be able to discriminate the inflammatory from the non-inflammatory forms

* Simon, loc. cit.

† Williams *On the Lungs*, p. 145.

‡ Spillan's *Andral*, p. 378.

§ Stokes *On the Chest*, p. 312.

of it. For, granting that minute anatomy should always be perfectly decisive upon the question, the actual fact is, that in the recorded cases to which appeal must be made on all points connected with the statistics of pneumonia we are furnished with no such information. So long, indeed, as the decision in any particular case remains of vital importance, the lung-tissue itself is not available for our inspection, and such physical signs as can be obtained during life must be identical, in character at least, in both cases.

The signification of lung-consolidation, then, must be sought by means of its attending phenomena, from its position and extent, from its duration and mode of access. Nor will these points alone be sufficient to enable us to form a judgment—"pathological appearances must be interpreted by the light of clinical knowledge."

The merest glance at tables of cases will serve to illustrate this remark. Here, for example, are two subjects whose lungs are described as exhibiting precisely similar appearances. The one died by gradual sinking with symptoms resembling continued fever, and but little embarrassment of respiration; the other was seized suddenly with urgent dyspnœa and acute pain, and died in a few days, suffocated.* Yet the disease, so far as pathological description is concerned, is the same in both—only active in the one, and latent in the other. Instances are numerous where the contrast is as striking. How far the pathologist of the present, ignorant of the histories of these cases, might succeed in classifying them in a manner which would correspond with their several clinical features, I know not. The actual fact is, that in our records no distinction is attempted. Yet every variety of disease would appear to be nearly associated with this common name—pneumonia. It attacks the subjects of chronic diseases of all kinds; it is a frequent attendant upon typhus fever; it occurs sometimes quite suddenly and unexpectedly as a "complication" in acute rheumatism; while not unusually patients who have been sinking bit by bit with slow and painful lingering, die at last of pneumonia "in its latent form." Only rarely does it fall with fatal force upon the healthy and robust; and then its course is so rapid, and its phenomena so marked and

* Compare, *e. g.*, Case 9 of Table IV. with Case 1 of Table V

uniform, that one wonders that so fierce and definite a disease can admit of so many modifications and varieties.

I take, then, a number of cases of lung-consolidation, omitting only those that are connected with tuberculosis or with secondary deposits—cases extending over a period of more than twenty years, and exhibiting, for the most part, that stage which has been called red hepatisation. Is any clinical classification of these possible; and if so, how far does the clinical classification serve to bring together cases which are pathologically similar? In the course of this labour of tabulating, one soon found that all the fatal instances of so-called pneumonia occurring in a series of years fell naturally, in view of their clinical histories, into four classes. The *first* and largest class would comprise patients who died of tedious and exhausting diseases of whatever kind, such as the constant drain of an abscess, or from the gradual extension of large areas of ulceration, as from bed-sores; or, generally, where lingering was unusually prolonged, and emaciation extreme. Lung-consolidation, indeed, is a familiar appearance in connection with this form of decay. It is remarkable that the condition is described in terms identical with those applied to the true pneumonic consolidation, proving, in fact, that death in these cases, or in most of them, was supposed due to a low insidious form of inflammation. A *second* class would consist of the subjects of a specific fever, or of some definite affection of a secreting organ and conspicuously of uræmic poisoning and the poison of typhus. In some of these cases, the lung-affection occurs with marked local symptoms, resembling in this respect idiopathic pneumonia, with which, indeed, it may be pathologically identical. In a *third* class hepatisation would seem due almost entirely to mechanical causes, and quite independent of any inflammatory action whatever; to such causes, for example, as would arise from defective power of the heart; from obstacles being opposed to the circulation, owing to some valvular imperfection; from the altered constitution of the blood itself; or from any combination of these states. I shall hope to show in the sequel that the phenomena which attend some of these cases of consolidation are quite inconsistent with any inflammatory theory. *Fourthly*, hepatisation occurs, there is reason to suppose, as the result

of idiopathic inflammation of the lung. It is then invariably connected with pleurisy, and often with pericarditis. It runs a rapid and tolerably uniform course, and would seem to be but rarely fatal.* I believe that each of these classes has features of its own which are perfectly distinctive, and that the more typical cases under each have little enough in common. At the same time, no doubt, there are many examples of a complex kind to which it is difficult to assign an exact place. And this is what we should expect, and is in accordance, I think, with our whole experience of disease, which has always resisted being wholly comprehended in precise definitions and classifications. It is the habit, indeed, of systematic writers to describe only typical differences, leaving out of view that middle connecting ground which lies between them; but when in a purely practical spirit we descend to actual cases, their lines of demarcation, abrupt as they appear from a distance, fade into the gentlest slopes, and we find ourselves often in an undescribed middle country which has as much in common with one type as with another.

Proceeding now to discuss shortly the several classes in the order in which I have already named them, I need say but little of the first—the hypostatic pneumonia of Piorry, the *péripneumonie des agonisans* of Laennec. Take such an instance of it as the following: a middle-aged woman, always greatly distressed by vomiting during her pregnancies, has that symptom occur in the fourth month of such a time, and with so much severity and persistence, that hardly any nourishment whatever is retained. After two months of this incessant sickness, she slowly sinks exhausted and starved to death. Whatever conjecture is formed as to the material cause of this result, certainly pneumonia is not thought of, since there was neither cough, nor pain, nor dyspnœa, nor other symptoms that could be referred to the lungs. Yet it appears that in this woman, who thus died inch by inch, enduring the want of food for more than two months, "the lower parts of both lungs were red, solid, and airless, from the early stage of pneumonia." Precisely similar is the appearance of

* There are a few kindred cases—cases, I believe, of great rarity—where this true pneumonia appears to be set up by the contact of inflamed bronchules.

these organs in many of the cases cited by surgeons of patients cut off by pneumonia after surgical operations. It is difficult to persuade oneself that the majority of the instances of the kind quoted by Mr. Erichsen* are not of this class. The same may be said of the so-called intercurrent pneumonia of typhus,† and of many cases of lingering death which used to be explained by saying that "an inflammation insidiously stole upon the lungs." Clinically, indeed, this form of consolidation is characterised by an absence of symptoms. Owing to this, and from its occurrence preceding death by so short a time, it is rare for it to be recognised by its physical signs. If the term pneumonia were applicable to it at all, it certainly deserves to be called latent pneumonia. Pathologically the features of this consolidation are such as we should expect from its supposed mechanical origin. The posterior and inferior portions of the lungs are most affected, and both participate in the change pretty equally, the solid tissue shades off gradually into that which is merely blood-laden and airless. Thus out of 45 cases of supposed pneumonia found in the bodies of those who have died from exhausting diseases, I find that in 25 the hepatisation occupies both lungs symmetrically at their lower and depending parts—in that situation, in a word, where it is usual to meet with signs of engorgement; in 9 only of the whole number was there any certain indication of recent pleurisy.‡ In 1 of these 9 (the only case of the kind out of the 45) pericarditis existed.

It is, I believe, from including cases of this class in the statistics of true pneumonia that we have been led to some erroneous notions as to the nature of that disease. We have got an idea of the preference of inflammation for the lower lobes. It would appear that no such preference exists for one portion of the lung more than for another. We have considered pleurisy as a common, but by no means necessary complication. There is reason to believe that acute pneumonia never exists without some accompanying pleurisy.

* *Med.-Chir. Trans.* vol. xxvi.

† See Murchison *On Fever*, p. 184.

‡ In 7 of these the pleurisy is single, and corresponds with the hepatisation, which is likewise confined to one side. In both these respects these 7 cases are exceptional.

There are 3 cases out of the 45 already alluded to as illustrations of this first class, where, with the symptoms of continued fever, consolidation is present, not, as with the others, at the lower lobes, but at the apex of one lung or of both. The same condition will be found more amply illustrated in the other Tables. It has, indeed, been especially alluded to by Trousseau as having special features; its symptoms being of a markedly typhoïd character, and the local affection often latent. From all that can be gathered of it, it would appear that most instances, at least of this form of disease, are in fact cases of specific fever, or granular degeneration of the kidneys, with this local manifestation. We know, indeed, that few typhus patients escape some symptoms of pulmonary congestion; and it is conceivable that these appearing earlier than usual in some cases, or at a spot where we are not accustomed to observe them (as at the lung's apex), may lead to a diagnosis of pneumonia; whereas, later in the disease, or in a different situation, the same signs would either be overlooked altogether or taken as a mere local indication of the blood-disease.* Somewhat similarly, the occurrence of violent delirium amongst the first symptoms of fever has not unfrequently led to the diagnosis of meningitis. Excluding these latent cases as of secondary origin, we have no evidence that simple pneumonia, attacking the upper portion of the lung, is distinguished by any other phenomena than those which attend the same disease elsewhere.

I have placed by themselves (under Class V.) 20 cases of lung-consolidation where death was ascribed wholly or mainly to low or latent pneumonia.† Only 5 of these seem due to hypostatic congestion: the remainder cannot be accounted for in any mechanical way. Thus there are 10 cases where the upper lobe is the seat of the consolidation; and it happens, strangely enough, that in all these cases it is the right lung which is so affected. Of the remainder, the whole of one

* In the first volume of the *St. George's Hospital Reports* I have alluded to a case with marked symptoms of lung-consolidation at one apex, which in its subsequent history bore the strongest resemblance to typhus fever. See vol. i. p. 344.

† See Class V.

lung is affected in 5; but in all save one of these the disease is most advanced at the apex. Recent pleurisy existed in 5 cases only out of the 20, though the value of this sign is greatly diminished from the pleura being often obliterated by old adhesions. Recent pericarditis was found in only one case in association with consolidation of the upper portion of the right lung, and dependent apparently on granular degeneration of the kidneys.

Consolidation of the lung is said to occur after this latent manner in connection with delirium tremens, and two such cases appear in the Table I am speaking of. Dr. Stokes alludes to it as one of the forms of "typhoid pneumonia."* "The disease," he says, "commonly attacks the left lung, particularly in its lower portion, and is constantly overlooked." It appears, on the contrary, that in the 5 cases answering to Dr. Stokes's description, to be found in all my Tables, both lower lobes were affected in 2, while the remaining 3 are marked cases of hepatisation of the upper lobe of the right side—2 of them having also pericarditis.

The question occurs, whether simple pneumonia, in this latent form, does ever occur "as the sole disease." Dr. Stokes believes that it does. The evidence which twenty years of hospital records supply does not contain a single undoubted case of it so occurring.

I say no undoubted case; but the authority of Dr. Stokes is so great, and the point itself is of so much practical importance, that, at the risk of being wearisome, I will allude very shortly to a case which bears upon it.† A woman, aged 36, was admitted into St. George's Hospital, after a week's illness, of which the first symptom was shivering and stitch in the right side. About the same time she got cough and dyspnœa. When first seen, she had a frequent pulse, hot and dry skin; the pulse was 120, respirations 52, the tongue dry and brown. A few moist sounds were heard at the apex of the left lung; the breathing generally was harsh and imperfect; there was neither ægophony nor tubular breathing. The case was at first regarded as one of pneumonia, and treatment by antimony was adopted. On the second day, however, though the respiration was less hur-

* Stokes *On the Chest*, p. 339.

† Case 15, Class V.

ried, and there was little cough and no expectoration, delirium had set in, and an ill-marked mulberry rash was visible. The chest-sounds were less marked than at first. The patient was now supposed to be suffering from fever, and in that view the treatment recommended by Dr. Dundas was followed—that by large doses of quina at frequent intervals. In this way more than eighty grains of quina were swallowed in twenty-four hours, by which time the skin was quite cool and the pulse had fallen to 72. At the very instant that these particulars were being noted down, the colour faded from the woman's face and she immediately expired, having been under observation for about four days. In this case it was found that nearly the whole of the right lung was in a state of gray hepatisation. The pleura was natural, both ventricles of the heart were uncontracted, and the blood was fluid.

Now I am not arguing that this patient must have had fever because she was treated for it; still, it must be allowed the history of the case corresponded in many points with that of fever (typhus was prevalent in the hospital at the time), even to the appearance of a mottled eruption; at the same time, there was nothing in the post-mortem examination to negative that view. On the other hand, if the case be regarded as one of simple pneumonia, latent during the greater part of its course, it is, so far as our hospital records go, a unique case; for in no other does simple pneumonia occur without the accompaniment of pleurisy. In other words, in every case where lung-consolidation, both from its pathology and history, may not reasonably be looked upon as secondary, there is pleurisy going along with it.

From this digression I proceed to the second class—that in which hepatisation occurs as a consequence of failure in the function of some secreting organ, or owing to any cause which gives rise to a poison in the blood. The illustrations under this heading differ from the latent ones of which I have just spoken, inasmuch as, for the most part, the implication of the lungs is sufficiently apparent during life, and recognised as consecutive on some definite disease elsewhere. The cause of hepatisation in these cases of blood-poisoning, or in some of them, is expressed by saying that the

relation between the fluid and its channels is disturbed, that the circulation is thence impeded, and that so exudation takes place. Take, for instance, the case where an interruption to the action of the kidney gives rise to acute anasarca. Here, following the kidney-derangement with a remarkable promptness, we get an infiltration into the cellular tissue of the whole body. Infiltration of what? That it is not a mere pouring out of water one can easily convince oneself, by the tenseness and firmness of the swollen limbs. It has been called, indeed, fibrinous dropsy, and, still earlier, was known as inflammatory dropsy. By whatever name we call it, it must at least be allowed, that in the lungs a similar infiltration would give rise to hepatisation.*

Consolidation arising in this manner would be general throughout the lungs, but most marked at their lower and posterior parts—resembling, in fact, that form which we have described as due to hypostatic congestion—though the rapidity with which the change was brought about would prevent any similarity in clinical respects. It appears, however, that this general and symmetrical consolidation is exceptional, and that in most instances the appearance of this kind of hepatisation resembles nearly that of true pneumonia: it is confined to one lung, its boundaries are sharply defined, pleurisy exists along with it, its chief seat is often in the upper part of the lung. Pathologically at least (however it may differ in its duration and general symptoms), it is unnecessary in such cases to dissever this consecutive disease from simple idiopathic pneumonia. As to its origin, we are left to suppose that the irritation of the vitiated blood excites in the lung, now in this part and now in that, a process which in its main features is strictly analogous to the inflammatory process. We read, indeed, that it may be "the

* See Jones and Sieveking's *Pathology*, p. 95: "Vogel does not seem to discriminate between fibrinous dropsy resulting from hyperæmia, and that resulting from unequivocal inflammation; and probably it is not necessary. The one condition is the inceptive of the other, and passes into it by imperceptible grades, or may exist in various degrees along with it." What is the exact force of the word "probably" in this quotation, or what is the meaning of the expression "unequivocal inflammation," when it is allowed in the same breath that such inflammation is not to be distinguished from hyperæmia?

tendency of the *materies morbi*, under certain atmospheric or epidemic influences, to excite irritation in particular portions of the respiratory apparatus."* Whatever such language may mean, it is to more practical purpose to observe that such cases of secondary pneumonia are oftenest met with in connection with diseased kidneys; its occurrence from failure or imperfection of other secreting organs (as of the liver) is far less obvious.

In tabulating 30 supposed examples of this form of disease (Class II.), 8 occurred in which it seemed probable that mechanical obstruction had its share in the result. Excluding such as of a mixed kind, we have, out of 22 cases, 8 in which the kidneys were markedly granular; in most of the remainder these organs were enlarged,—in some, as it would appear from the surrounding circumstances, owing to "amyloid" change, in others as a consequence of scarlatina or acute nephritis. In at least 12 of the 22 cases recent pleurisy is present. Of the rest it is usually stated, either that the pleural cavity is obliterated or that old pleural adhesions existed. 5 cases are of pneumonia of the upper lobe; in 4, of the right alone; in the 5th, of both apices. In a 6th case the left lung is hepatised as to its upper two-thirds; in 6 cases the whole of one lung (or very nearly the whole) is hepatised, viz. the right lung in 4, and the left in 2. Pericarditis co-exists in 6 cases. In one of these (22), an instance of advanced granular degeneration of the kidneys, the consolidation would appear to be due to hypostatic congestion; in the rest the right side is hepatised wholly in 3, and as to its upper lobe in 2.†

I have said that cases occur where, along with blood-poisoning, a mechanical obstruction seems to favour hepatisation. I come now to notice, according to my scheme, as belonging to a third class, some instances where this result appears to have depended almost entirely upon such obstruction.‡ This form of disease is best illustrated by cases

* Fuller *On Diseases of the Chest*, p. 231.

† Vide 7, 11, 16, 17, 18.

‡ Class III. It will be observed that some two or three of the cases of contracted mitral orifice do not exhibit *marked* hepatisation. They are included as helping to illustrate the general effect upon the lungs of this particular kind of obstruction.

of mitral valvular obstruction. I have recorded 15 such. Excluding 4 of these, where pericarditis was also present, and where the hepatisation seems to have been somewhat modified by that circumstance, we have 11 cases left as typical examples of the manner in which hepatisation takes place as the consequence of obstruction. As might be supposed, the two lungs participate equally, or nearly equally, in the change, which is most marked at their lower and depending parts. Often there is some amount of extravasation mixed up with it, and sometimes this "pulmonary apoplexy," as it is called, exists most in one lung, and hepatisation most in the other. Yet this hepatisation is in itself of the completest kind, and in some instances, as will be seen, is specially described as due to the infiltration of lymph. The stage of gray hepatisation seems rarely to be reached, and pleurisy is a rare accompaniment. The patients for the most part trace their first failure of health to an acute rheumatic seizure, and will describe former attacks of so-called pneumonia. Other cases in this class seem to have their origin in a feeble action on the part of the heart, owing to its fatty degeneration and the impediment of an adherent pericardium. (See cases 6, 9, 11.)

There is another way in which consolidation of the lung is apt to occur suddenly in cases of valvular obstruction. It is less purely mechanical than the foregoing, yet less dependent upon unknown causes than the hepatisation, which we have to refer to the agency of a *materies morbi*. Its causation appears to depend upon the fact that circulation is only possible so long as the constituents of the blood, the obstacles presented to it, and the muscular power of the heart, hold certain relations to each other. When these limits are past,—when, for instance, with a highly-fibrinous condition of the blood, we have a weak heart and a defective valvular apparatus,—we might suppose that any accident which should diminish the force of the blood-stream on the one hand, or tend still further to increase the quantity of fibrin which it held in solution on the other, would be sufficient to give rise to a blood-stasis either general or partial, and so to a form of consolidation which should resemble, roughly indeed to the eye, but in the completest manner to the ear, the hepatisation of pneumonia.

Now in acute rheumatism all these conditions are satisfied, and accordingly it is not very unusual in it to find consolidation coming on suddenly and without warning. Such an occurrence is commonly ascribed to inflammation; and amongst the diseases which are mentioned in books as destroying the subjects of rheumatic fever we find "intercurrent pneumonia."

This form of disease is indeed sufficiently uncommon for me to be allowed to allude very shortly to some illustrations of it.

In the year 1864 a girl 19 years of age was admitted into St. George's Hospital,* and under my observation, for acute rheumatism of no great severity, and without heart-disturbance. On the second day she had a fit of alarming dyspnœa, with catching, shallow respiration, pain in the left side and in the affected joints. Repeated auscultation failed to discover any alteration of the heart's action, except in its increased rate. As little could any sounds be detected suggestive of pneumonia, though it must be mentioned that the patient's extreme condition rendered it impossible to examine the chest very completely. After remaining in great distress from breathlessness for two days, yet free from any mental disturbance, this girl died. On post-mortem examination the pulmonary artery, as far as its third and fourth divisions, was found to be filled with decolorised coagulum, and there was a shred of lymph in the right middle cerebral artery. The lower lobe of the left lung is described as "much solidified from pneumonia, and sinking in water." The heart was uniformly covered with recent lymph. This, though a sufficiently well-marked instance of fibrin being deposited in the course of acute rheumatism, and giving rise to partial consolidation of the lung, is by no means the most characteristic case of the kind. In the previous year a boy of 9 was admitted with acute rheumatism.† After eight days' residence he was attacked with "double pneumonia." The exact manner and symptoms of this attack I have forgotten. He recovered from it in a week. He next gets a return of pain in the limbs and alarming dyspnœa; the pulse rises to 120, and there is much visible pulsation of the carotids; free respiration, however, is still heard in the chest. This attack too

* Case 21 of Class III.

† Case 11 of Class VI.

passes off, and he is up and about again, well, or nearly well. When so far recovered, and with perfectly unembarrassed breathing, he has one night a sudden attack of angina and dyspnœa, which carries him off in a few hours. Here the whole of the right lung, except quite the apex, was red, solid, and airless, "evidently," it is written, "in the first stage of pneumonia." The left lung too is affected in exactly the same manner, though less uniformly. The pericardium is adherent by recent lymph, the mitral valve very much thickened by fibroid matter.

Now the death of this boy and its attending circumstances must be accounted for, I think, in one of three ways. Either the consolidation had been going on for some time, had been "latent," as we say (and so latent that the child could play about as usual); or this was an instance of inflammation of the lungs attacking its victim with exceptional rapidity and exceptional symptoms; or else it was a sudden arrest of the pulmonary circulation—a sudden precipitation, so to speak, of the blood, the result of ever so slight a disturbing cause in a weakly boy with blood in a highly fibrinous condition, and with only a damaged organ to propel it. The first supposition cannot for a moment be entertained; and if the second is urged, some similar case must be adduced of acute pneumonia as suddenly fatal where the concurrent circumstances do not admit of the explanation I have submitted. In the first volume of the *St. George's Hospital Reports*, in a paper by Dr. John Ogle, there is a woodcut of a preparation in our Museum which illustrates, as I conceive, the pathology of this form of consolidation. It exhibits a section of a portion of hepatised lung, with minute branches of the pulmonary artery filled with firm fibrinous coagulum.*

It is obvious that in consolidation brought about in this sudden manner, and with no prior stage of engorgement or

* The specimen is from Series vii. No. 10, and is thus described in the Museum Catalogue: "Specimen showing red hepatisation of the lung, with extensive deposit of dark-red fibrin in the pulmonary artery. Double pleuro-pneumonia had existed, and there was tolerably firm adhesion between the layers of the pericardium; the cavities of the heart were dilated, especially the left auricle, which was lined by recent yellow fibrin. The margins of the mitral valve-flaps were occupied by recent fibrin also, and slight atheroma of the root of the aorta existed." See vol. i. *Hospital Reports*, p. 168.

congestion, there should be an absence of those auscultatory signs which have been so much dwelt upon in connection with pneumonia as the first indication by which the ear is apprised of the commencing mischief.* And accordingly we find that those writers who allude especially to "rheumatic pneumonia" as one of the varieties of that affection allude to the absence of fine crepitation. Dr. Fuller, indeed, suggests an explanation, which, though ingenious, can only be accepted upon actual pathological demonstration. "In some few instances," he says, "especially when pneumonia arises in connection with acute rheumatism, crepitation never occurs —a fact which I have verified on several occasions, and believe to be attributable to the occurrence of exudation into the interlobular cellular tissue, and consequent immediate occlusion of the air-cells. The mere non-occurrence of crepitation, therefore, is not a certain proof of the non-existence of pneumonia."†

Lastly,‡ I come to speak of 16 cases of simple, uncomplicated pneumonia, which are all the instances that I can discover in searching the records of 20 years; and in this number are included 4 where pericarditis coexists. It is true there are some few cases of a doubtful character to be found in the other Tables that ought perhaps to be included here, and so bring the number up to 20, or thereabouts. On the other hand, there are cases amongst these 16 where, although the pathological definition is satisfied, the clinical history forbids us to suppose that the patient had not long been the subject of disease. What first strikes us in glancing at this list is the constant presence of pleurisy§ along with the consolidation. Next, it is observable that in a majority of the cases either the whole lung is struck, or that it is the upper lobe which is solely or mainly affected. Where this is not so—where the lower portions of the lung are alone solid—we have just those cases which are most equivocal in their history, the most suggestive of some primary disease which has eluded us. In fact, the statement that simple inflamma-

* See also cases 15 and 18 of Class II.

† Fuller, p. 221.

‡ Class IV.

§ Pleurisy was absent in one case only (15), which was admitted moribund, and without history, and probably not belonging to this class.

tion attacks the lower lobes by preference seems to be the reverse of the truth. Thus in 5 cases the chief seat of the hepatisation is one apex (in 3 the left, and in 2 the right apex); and in 6 the whole of one lung is hepatised (the *right* lung in all but one case (5), where the whole of the left lung is inflamed along with the base of the right). Hepatisation is uniform throughout 5 of these 6 cases; in the sixth it is double, occupying the whole of the right lung and the upper part of the left. Omitting 3 cases (7, 11, and 15, two admitted moribund, and all without certain history), the average duration of illness in the remaining 13 is about 8½ days.

The association of pericarditis with hepatisation of the right lung, to which I have already made passing allusion, is again noticeable in 3 cases out of the 4 where pericarditis was present. In the fourth case the hepatisation is double, though the upper lobe of the *left* lung exhibits the most advanced stage of it. This connection is so unaccountable that one hesitates to accept it on light grounds. I will just give the result of an analysis of all the cases tabulated in which pericarditis and hepatisation coexist.

In 23 cases of recent pericarditis it is the right lung which suffers in 16, either solely (which is the rule), or with very slight participation on the part of the left lung. Of the remaining 7 cases, 4 are not distinctive, viz. one exhibits extravasation of the right lung without pneumonia. In 2 the lower parts *of both lungs* are hepatised (the right most in one of them), and in the fourth case the upper lobe of the right lung is hepatised along with all the left. In none of these 4 cases is the pericarditis stated to be recent; in two, at least, it is evidently of old standing. There remain 3 apparent exceptions. Two record hepatisation of the lower lobe of the left lung, together with a thin layer of recent lymph over the heart; the third has honeycombed lymph in the pericardium, along with hepatisation of the back of the left lung. From the situation of the consolidation, and still more from the *absence of pleurisy* in all these 3 cases, they would appear to depend on hypostatic congestion rather than on true pneumonia.

It may be stated generally, therefore, that wherever in the whole series of cases recent pericarditis is associated with

marked pneumonia, it is always the *right* lung which suffers either mainly or solely. In other words, *these Tables do not contain a single case of extensive hepatisation of the left lung only along with recent pericarditis, while they contain at least* 10 *in which that is the condition with respect to the right lung alone.*

The striking feature in this acute pneumonia, as our definition has already stated, is the suddenness with which violent constitutional disturbance, in which the nervous system largely participates, gives place to extreme prostration and very rapid sinking. That this is the natural course of the disease it would be unsafe to assume—at least in the earlier cases, since these relate to a time when all affections supposed to be inflammatory were very actively combated. Thus as lately as 1845 we read of a man of 32 suddenly seized with acute symptoms, which led to a diagnosis of inflammation of the right lung.* Upon this supposition he is bled twice within a few hours, 16 oz. of blood being taken each time. He is then ordered ½ a grain of tartarised antimony every 3 hours; and the report of the third morning states that he "has been unable to sleep, owing to vomiting caused by the antimony; he is also much purged." So the dose is reduced to a ¼ of a grain. As to nourishment, there was as little as possible of it, and that little of the most unstimulating kind. He is next blistered; and though the blister, it is said, "rose well," the man sank and died, violent delirium preceding death by a little, and giving place to insensibility. When this patient's body came to be examined, it was found that these energetic means had failed to subdue the inflammation, such as it was. The lowest lobe of the right lung was in various stages of red and gray hepatisation, and there was some recent pleurisy. The upper lobes of the right lung and the whole of the left lung are described as "remarkably healthy;" nor could any disease be discovered elsewhere in the body.

It will be seen that most of these cases died at the beginning of the second week, nearly half of the time being occupied in that gradual "typhoïd sinking" which is so common an expression in these reports. Sometimes, however, in the most rapid cases,† and notably in those where the upper lobe

* Case 1 of Class IV.

† See cases 7, 9, and 12.

is concerned, or where the disease has fastened on a whole lung at once, the patient dies struggling for breath in a manner as torturing as that sometimes witnessed in those who are killed by acute laryngitis. This is only an illustration of the fact, that it is rather the rapidity than the extent of disease which gives rise to acute functional disturbance.

Delirium is so marked a feature in this affection, as to have received special notice in these fragmentary reports, in 7 out of the 12 cases of which there is a complete history. In 3 of these (all men) it was of violent character, resembling delirium tremens.

There are some other points in connection with this subject which I should gladly have noticed, and especially the rarity and exceptional characters of that pneumonia which follows injuries of the chest. I would have alluded also to that form of the disease which is supposed to arise from extension of inflammation in cases of acute bronchitis. I believe with Grisolle* that such extension is extremely rare, though one or two illustrations of pneumonia are given in the Tables which might be so interpreted.

But the length to which these remarks have extended warns me to conclude. In them I have endeavoured very imperfectly and in a limited space to give some account of the various ways in which, apart from tuberculisation, it seems probable that consolidation of the lung may occur. I may just allude in conclusion to some practical considerations to which the views I have maintained naturally give rise.

A very laudable attempt has been made of late years to test the comparative efficacy of various modes of treatment in certain definite diseases by means of statistics. Pneumonia has been especially chosen by Dr. Hughes Bennett for this experiment. The cases on which I have commented would seem to show that the choice is a most unfortunate one. Simple pneumonia is rarely a fatal disease under any mode of treatment; as a secondary disease its associations are so many and various, that it seems almost hopeless to attempt a classi-

* Grisolle, while rejecting this form of pneumonia, regards the bronchitis "as constituting rather a sort of predisposition, denoting a state of the organism which will be more easily influenced by the ordinary causes of pulmonary inflammation." Grisolle *On Pneumonia*, p. 430.

fication sufficiently precise to bring cases into fair comparison. In a word, if the term pneumonia be used to express all the cases of consolidation which I have been noticing, or the majority of them, no disease can so little be treated in this way or that because of its name. If, on the other hand, the word is to be restricted to pure uncomplicated cases of inflammation of the lungs, it is clear that our knowledge at present does not enable us always during life to discriminate such cases. The argument of treatment derivable from tables must concern itself with simpler diseases than this. Pneumonia, if that common name is to be retained, must be regarded as applying merely to a certain combination of physical signs. It must be forgotten that in strictness the term has a meaning beyond what these signs necessarily imply, and that it involves a theory of causation for all the cases commonly ascribed to it, which, at most, is true only of a few.

I have but one word to add regarding the Tables. From the great space these would occupy printed *in extenso*, I have been compelled in the present volume to omit the first class altogether. The diseases comprehended in it are very various, but have the common feature, that death is lingering. In many cases the patient is exhausted by some long-continued flux—by diarrhœa, or the constant drain of an abscess. Malignant disease is not uncommon; in one instance the subject died simply starved, owing to scirrhous cancer of the œsophagus. It is rare in these cases for any attention to be called to the chest during life. The Table contains 46 cases of hepatisation of this form. In most of these the consolidation is spoken of in language identical with that used in describing pneumonia elsewhere. The lungs are described as "extensively inflamed," "infiltrated with lymph," &c. Of the seat of this form of hepatisation, and of other points in connection with it, enough has been said. The condition is, in fact, that now recognised as "hypothetic congestion;" and from a general agreement in the present day as to its nature, it is the less necessary here to accumulate instances of it.

The cases under the other classes I feel bound to retain, because to omit any of them under a view of my own as to its import would be to give an imperfect account of the evi-

dence before us in this matter. I have, however, made each case as short as possible. About 8 have been excluded, from being imperfectly reported, or incomplete in some respect. As has been stated, some cases were difficult to classify, and may perhaps be considered as wrongly placed. For example, of Class II. cases 8, 9, and perhaps 10, may belong more properly to Class IV. On the other hand, cases 11, 12, and 13 of this last class ought probably, from their histories, to be placed under Class II.

The cases (which are very unequal in the amount of their information) are described in the language of the original narrators, with abbreviations, but without omission of any matter of importance.

In further apology for the length of these Tables, I may urge that as a faithful record of all the marked cases of hepatised lung (with the exceptions already adverted to) met with in our Hospital in the course of twenty years, they are appropriate to this volume, and may have a value hereafter quite apart from the present occasion, and when the purpose to which I now put them is forgotten.

OCTAVIUS STURGES.

Class I.

Table of Cases of Hepatisation occurring in the course of lingering Diseases, and ascribed to low or latent Pneumonia.

(Omitted for want of space.)

Class II.

Table of Cases of Hepatisation the result of Blood-poisoning.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
XLIX. 53. M. 63. 1.	Rapid sinking after simple fracture of tibia and fibula. Of early history he stated that for years he had been "a martyr" to gout. "No symptoms presented themselves to call attention to chest" (Surgical Report).	Left lung congested posteriorly; lower lobe of right lung much consolidated "from effusion of lymph and serum in its parenchyma," of a light dirty brown. Kidneys small, rough, and granular.	Some recent lymph on surface of hepatised lung. Pericardium not mentioned.
LII. 236. M. 57. 2.	Admitted at first under surgeons for cachectic rupia of six weeks' standing. Two days after, he was observed to be in a very depressed state, with cough and oppression of breathing; was stimulated and blistered. The following day he was in a typhoid state, with dry tongue and difficult articulation, but no confusion of mind; lying constantly on his left side. He was cupped to ℥x., and afterwards dry-cupped. He sank the same day. Three days (Medical Report).	The lungs on both sides full of reddish frothy fluid; but all parts floated, except the middle lobe of the right lung, which was almost entirely consolidated and of a light buff colour—the gray hepatisation. Bronchial tubes very vascular and full of thick mucus. Some old inactive cretaceous deposit at both apices. Kidneys weighed 8 oz., were highly granular, and with diminished cortical part and numerous cysts. The heart weighed 15 oz.	A little reddish fluid in both pleuræ, and some old adhesions in one; no lymph. Pericardium not mentioned.
L. 55. M. 44. 3.	Admitted with pain in the chest, cough, and dyspnœa. Had had dropsy of legs and face at beginning of illness four months before. Cough had come on during last fortnight with such	Upper lobe of right lung consolidated by recent inflammation; of grayish mottled appearance; the middle lobe red and condensed; the lowest lobe congested only.	Lymph and turbid fluid in left pleuræ, so that left lung

	great debility that he had kept his bed for that time. Treated by bleeding and antimony. Two days.	Heart hypertrophied ; valves healthy. Kidneys rather large, pale, mottled.	was compressed thereby, its lower lobe being impervious to air.
XLVII. 219. F. 67. 4.	Wretched cachectic person. Admitted for general pains about limbs ; severe pain in epigastrium ; frequent vomiting. The last symptom more especially for nine months ; pyrosis, &c. ; no cough. Died by gradual sinking ; vomiting being chief symptom. Six days in hospital.	Upper lobe left gorged with serum, but crepitant ; lower lobe throughout quite solid ; in parts with red hepatisation, and in parts with gray hepatisation, readily breaking down. Lower lobe right lung inflamed ; larger portion in red hepatisation. Upper lobes crepitant, loaded with serum. A small tubercle at one apex of chest. Kidneys much diminished ; cortex nearly all absorbed.	Old adhesions left side ; layer of recent lymph on lower lobe. Firm adhesions right side ; recent lymph at lower part.
LIV. 123. M. 28. 5.	First admission in March 1853 with severe bronchitis. He had been subject to cough and dyspnœa for five years. His face was dusky, and once the sputa were streaked with blood. He left the hospital in April, still with cough and rather dusky face. Re-admitted in May 1854, in a more depressed state. Had not been free from cough in the interval ; last taken suddenly worse two days previous, with pain in left side. Sputa were abundant, frothy, and muco-purulent. No mention of side-pain after admission. Pulse was weak and rather frequent. Stethoscopic sounds those of bronchitis, except some amount of dulness. Five days.	Body in good state. Lower part of right lung was emphysematous. Almost all remaining portion in a state of hepatisation, solid, and in places yielding a grayish fluid. Upper and middle parts presented a granular surface on section. At one part of middle portion of upper lobe a small amount of fluid within an irregular cavity. Left lung emphysematous ; at lower part congested, crepitant. The bronchial tubes vascular and containing much mucus.	On right chest recent adhesions, some fibrin, and yellow fluid. Heart and pericardium natural. Blood thin and watery.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
IX. 238. M. 31. 6.	An intemperate sailor. Four or five months before admission, he walked thirty miles in the rain. He then sat down in a public-house and let his clothes dry on him while he drank. The next morning œdema of legs appeared, and dropsy continued up to time of admission. Urine was then highly albuminous. After a few days, some blood-tinged sputum was observed. Progress of the case marked by orthopnœa and increased anasarca, death occurring, after long lingering, three months and a half after admission.	General dropsy. There is a little fluid in the left pleura; adhesions of right. The upper lobe of the right lung is hepatised and condensed from infiltration of lymph. Upper and part of lower lobe of left lung is in a similar state. The kidneys are small, smooth, and mottled; their cortex not diminished.	Fluid and old adhesions of pleuræ.
LIII. 73. M. 48. 7.	A fat, flabby, ill-conditioned man, evidently in the habit of free drinking. Admitted for pain in hands and shoulders of four days' duration. The hands were slightly swollen. He had great difficulty in moving; was bled twice (to ℥xviij. in all). The sweat had the usual acid odour (he had had cough for three months). At each apex sonorous râles were heard with the expirations; but there was no dulness. On the fourth day, tremor and tendency to delirium. Both these increased on the fifth day, and the sounds of pericarditis were heard. The wandering was now very similar to ordinary delirium tremens. The patient now gradually sank, and died on the twelfth day.	Left lung somewhat congested posteriorly; but crepitant throughout. The lining membrane of the bronchial tubes was inflamed and loaded with frothy muco-purulent fluid. The *upper lobe* of the right lung was completely solidified from red hepatisation. The tissue of the organ was soft and easily broke down. The heart was much enlarged, weighing 23 oz.; all its valves were healthy. The kidneys were large, weighing 15 oz.; healthy in structure.	Recent adhesions between pleuræ, corresponding to inner side of upper lobe of right lung and surface of pericardium. Three ounces of serous fluid in the pericardium; a shaggy layer of recent lymph in its visceral surface.

LV. 84. F. 28. 8.	Nothing said of early history. Ten or eleven days ago took cold. Had rigor, cough, and pain in *left* side; was leeched and blistered. On admission was very prostrate, fainting when raised; loud crepitation was heard at the lower part of the left side. Second day rubbing is heard at the base of the heart; the sputa were yellow, copious, muco-purulent (calomel and opium); loud gurgling was heard all over the chest. She died on the third day.	Very much emaciated. Left lung much compressed; numerous very old adhesions on the right side. All the right lung hepatised and easily breaking down.	Turbid yellowish fluid in pericardium. The serous membrane covering right auricle thick and opaque. Outer surface of left lung covered with thick layer of recent lymph. Pleural cavity containing large quantity of straw-coloured fluid.
LX. 136. M. 64. 9.	A hard drinker. Ill for a month with boils or superficial abscesses about nates. A week ago, when getting out of bed, slipped and broke his ribs. Three days before admission, dyspnœa. On admission orthopnœa and severe dyspnœa (dulness and large crepitus over upper of right lung). Ordered squills, bark, and ammonia. On second day, calomel and opium and wine. On third day was restless and wandering; breathing more difficult, and much tenacious matter sticking about the mouth. Died next morning. Three days and a half.	Body rather fat. Eighth and ninth ribs of right side fractured, with no attempt at union. Upper lobe of right lung throughout in a state of gray hepatisation (advanced); the lower lobe spumous. Heart healthy; extremely full of decolorised clots on both sides. Spleen diffluent; kidneys slightly granular; the pelvis containing much fat.	Pleuræ both adherent; not injured by fracture.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LV. 97. F. 59. 10.	A debilitated-looking woman. A month ago she was attending a neighbour in child-birth, and there caught cold. Two weeks before admission the cold became worse; cough, with slight spitting, succeeded, and she had shivering and pain in the loins. On admission she had pain in left side, and cough (her general condition is not further described); pulse was 106. She was blistered, and given calomel and opium. After six days she was free from pain, and the drugs were stopped. Next she was observed to be feeble and anxious, with rapid pulse and dry tongue; sordes on lips, &c. (twelfth day). Fourteen days in the house.	Emaciated. The lower lobe of left lung and the back part of its upper lobe in a state of gray hepatisation, solid, sinking in water; the parenchyma infiltrated with lymph and pus; the front of upper lobe healthy. Lower lobe of right lung like lower lobe of left; other parts of both lungs œdematous. Heart healthy. Other viscera healthy—not specially mentioned.	Old adhesions both sides, but much more extensive on the right. Pericardium not mentioned.
LV. 205. M. 26. 11.	A labourer, who had been six days ill 'with cold and cough,' and had spit a very little blood. On admission was flushed, and with full bounding pulse; not rational enough to give his full history; same night he became very noisy; the urine (suppressed for a time) was found to be albuminous; head very hot, great thirst; some crepitation and dulness at lower lobe of right lung. By the third day the physical signs and febrile symptoms had increased; fourth day twitchings of hands and low muttering; tongue now (fourth day) became black and fissured, with sordes on lips and congested face; fifth day some wine was given (antimony and the treatment hitherto being discontinued). The ex-	Body in good state. No œdema. Whole of right lung completely hepatised (gray); several small abscesses in various parts. The left lung was natural. The bronchial tubes are not mentioned. Kidneys were large and solid, with very yellow cortex, greatly mottled on the surface.	Right pleura obliterated; a little fluid (yellow) in left. Pericardium coated with thick layer of recent lymph.

	pectoration was now brownish, it had before been rust-coloured. Pulse became less frequent and tongue cleaner. Remained two days in this favourable state and omitted all medicines. On the eleventh day began to sink, and had constant vomiting. Eleven days.		
LV. 207. M. 42. 12.	An intemperate man. Seized five days before admission with pain in left side and vomiting. On admission, urine rather albuminous; tongue dry and dirty; full quick pulse. Vomited whenever he moved. Sputum frothy, muco-purulent (cough and some expectoration for years). Given stomachics, &c. Retching returned. He sank slowly. Six days.	Whole of left lung (except upper lobe, which was natural) firm and full, in state of gray hepatisation. Lining of bronchial tubes of affected part very highly vascular. The same tubes in upper lobe comparatively pale. Right lung quite natural. Kidneys cystid, granular, with adherent capsules. Liver pale. Other organs natural.	Old and recent adhesions left side, and much sero-purulent fluid. Heart and pericardium natural.
LXII. 43. M. 28. 13.	A labourer, admitted with febrile symptoms and weak pulse; pain under right nipple, and slight difficulty of breathing; spitting rusty mucus. Had been ill five days. The defined symptoms above only two days. No dulness detected till second day. From the physical signs, consolidation appears to have begun at the upper part of the lung. Given antimony wine; and later, calomel and opium every four hours. On the fifth day became very violent, and continued noisy that day and the following; vomiting after everything, and obstinately refused food, &c. No great dyspnœa now, and the spitting had almost ceased. Sixth day, became cold and collapsed, and died. Six days.	Right lung completely solid throughout from red hepatisation; in a few places (chiefly in the upper lobe) broken down. Left lung healthy. Both kidneys very granular on their surfaces; of large size; the capsules thickened. Heart not mentioned. Liver large. Other organs healthy.	Right lung surrounded by old adhesions. Left surrounded by adhesions (old?).

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LVI. 80. M. 13. 14.	Had scarlatina a month before, which left slight œdema. Four days ago he had a fit, and a second at time of admission. On admission was blooded (amount not stated); blood highly buffed and cupped; became highly delirious; urine was very albuminous. He lay wholly on the right side. Two days or less.	Right lung very congested. The lower lobe in a state of extreme red and gray hepatisation, very few vessels or tubes being visible. Kidneys pale; stellate on surfaces; very opaque; a yellow cortex. Other viscera natural.	Reddish fluid in both pleuræ; in right sac some recent soft fibrin.
LXII. 25. F. 21. 15.	Admitted fourth day of acute rheumatism, having had two previous attacks. Bled to ℥xij.; cal. gr. iij. and opium gr. j. every night; alkalies. Third day very pale and white, with pain in right side and cough. Fourth day, blistering and antimony wine. By the eighth day cough was much better; she was then up, and had been so two or three days. Twelfth day, ulcerated throat observed; this getting worse up to twenty-second day, when pain in right side and dyspnœa occurred; the sputa becoming blood-stained; the urine full of albumen; stridulous, breathing as from affected larynx. Died two days after the acute symptoms.	Right lung hepatised throughout; light buff, mottled with streaks of reddish-brown; infiltrated with cells like pus, except that acetic acid displayed numerous fine granules instead of the characteristic compound nucleus. All the air-cells of a thin section were completely filled by the exudation; the walls not thickened. Ulcers on tonsil, and a "diphtheritic" membrane on pharynx; soft palate and larynx. Left lung healthy. Right kidney with dilated pelvis and shrunken cortex; no obstruction being discovered. Other organs healthy.	Much serous fluid in pericardium, and some recent lymph. Recent lymph about mitral and aortic valves; the former much thickened and inflexible. Right pleura obliterated; much recent lymph at its lower part. A few old adhesions of left pleura.
LXII. 60.	Ill one year and nine months; illness beginning	Slight œdema of the body.	Tightly ad-

M. 11. 16.	with "rheumatic gout," followed by palpitation and dyspnœa. While in the hospital had frequent distressing fits of dyspnœa. Took antimony and calomel. Twenty-two days.	The whole of the right lung red, solid, and granular on section, except some circumscribed patches in the lower lobe; these were very conspicuous from their white colour, and arose apparently from emphysema of some lobules. Some small amount of hepatisation, lower lobe of left lung.	herent and thickened pericardium. Many layers of deposition, of which the inner was the more recent. Heart pale, its cavities dilated; recent fibrin on its valves. Heart and pericardium weighed 20 ounces.
LXII. 107. M. 18. 17.*	Acute rheumatism with heart-affection at the age of 11. Present attack began with wandering pains, two weeks ago. Ten days ago had pain in the præcordial region, which remained up to time of admission. Admitted gasping for breath, and with orthopnœa; pulse 108; much tenderness over the heart and left chest generally. Treated by alkalies and calomel and opium. Some amendment took place, and the pulse sank to 94. Fifteenth day, some œdema of the legs is noticed. From this time he varied from day to day as to dyspnœa, &c., the swelling increasing. He died exhausted. Forty-six days.	Legs œdematous. The aortic valves thickened with old fibrous deposit; in one of them was a small hole; an abrupt ridge of deposit of several dates on the inner surface of the mitral valve. The right valves were healthy, and the left lung. The right lung slightly solidified, as if in the earliest stage of pneumonia. Heart weighed 1 lb. 15¼ oz. The kidneys were increased in size.	Pericardium greatly thickened, and adherent to the whole surface of the heart, whose walls were hypertrophied, and cavities dilated. Pleuræ not mentioned.

* The three preceding cases have more in common with Class III.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LXIII. 132. F. 16. 18.	Chorea; violent convulsions, dying soon after admission as if falling asleep. History of rheumatism. Some hours in hospital.	Middle and upper lobe of right lung in a state of incomplete hepatisation ('the first stage'). The spleen dotted with small white specks smooth to the touch.	Old lymph on pericardium. Recent lymph in valves of left side of heart.
LXIII. 137. M. 50. 19.	Good health till two months ago, then œdema and cough, the urine becoming scanty and bloody. Admitted wheezing, and with râles audible, and with bloody urine. Diuretics given. Œdema subsides in four days. On the seventh day sputum becomes blood-tinged; pulse rose; pain in right chest. He became very livid, and died. Eleven days.	Whole of left lung consolidated, and apparently swollen with gray hepatisation, except the apex. The bronchial tubes healthy. Kidneys smooth, large, 16½ oz. in weight; ecchymoses throughout their cortex, and much congestion. Large heart (23 oz.); its left ventricle much thickened; soft atheroma in the aortic valves.	Much turbid fluid in left pleura, and thick recent lymph.
LXIV. 43. M. 2. 20.	Admitted moribund. Scarlatina a month before, followed, it was thought, by recovery. Two weeks ago chronic twitching. Day before admission dyspnœa and croupous breathing. A few hours.	Lower three-fourths of right lung solid, so as to sink; it had a dark brownish colour, mottled with light brown spots like circumscribed gray hepatisation. Lower lobe of left lung contained a few similar spots. A little recent lymph on inner surface of mitral valve; small speck of the same on the aortic valve. Kidneys very pale, and mottled with irregular vascularity; the cortex increased; weight 4½ oz.	Recent lymph in right pleura, and a little serous fluid. Left pleura natural.
LXIV. 175.	Ill a month. Debility, œdema, pain in the chest. Two weeks' cough; thick uncoloured mucus	All upper lobe of left lung and greater part of lower lobe firm, red, and granular; the tissue	Pleuræ not mentioned

M. 28. 21.	and some specks of blood; respiration something hurried. Given squills, digitalis, and acetate of potash. Death quite sudden and unexpected. One day.	friable and heavy; most parts sank. Right lung full of serous fluid. Kidneys enlarged (weight 20 oz.); smooth, vascular, a few stellate veins on surface. The cortex much increased; it had a spotted appearance, and was very full of blood, though paler than natural, owing to the presence of some opaque whitish matter. Heart natural, except for one or two beads of recent lymph on the mitral valve.	(natural?).
LXIV. 259. F. 45. 22.	Admitted in state like fever. Only partially conscious. No history could be obtained. Lived in hospital some hours only.	Lower lobe of left lung and greater part of upper in condition of diffuse hepatisation, exuding purulent fluid in many places, the consolidation not generally complete. Lower lobe of right lung in state of red hepatisation. Pericardium completely filled with recent lymph, layers thereof in adhesion to each other. Left ventricle hypertrophied, valves natural. Kidneys finely granular, shrunken, and with diminished cortex. The organs chiefly consisting of fibrous tissue.	Some flakes of recent lymph on surface of left lung.

Class III.

Table of Cases of Hepatisation from Obstruction.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LII. 95. M. 47. 1.	Out of sorts for three weeks. Supposed healthy before. Some years ago had rheumatic fever (marks of leech-bites over heart). Symptoms: cough, dyspnœa, and anasarca. Heart's action quick; difficult to appreciate. Urine very albuminous; excessive dyspnœa. Rather rapid death. Two days.	*Exceeding contraction of mitral orifice* and dilatation of left auricle. Weight of heart 8¼ oz. Lower lobe of left lung hepatised; not so the right; both congested. Slightly granular kidneys.	Old adhesions in right pleura; in left, both old and recent adhesions. A little fluid only in pericardium.
LI. 89. F. 16. 2.	The subject of frequent rheumatic attacks. Admitted for palpitation; afterwards some dropsy appeared. There was a bluish flush on cheek; a cardiac bruit, and oppressed breathing (no further lung-symptoms mentioned). Rather gradual sinking. Fifteen days.	Auricles enormously distended by semi-clotted blood; vegetations on mitral valve; *mitral orifice contracted.* Other orifices and valves natural. Lungs were friable and sank in water; dark red fluid could be expressed from them. The left was congested merely.	A little clear yellow fluid, and some very firm adhesions of pericardium. Pleuræ very vascular; in right sac 5 oz. of reddish fluid.
LVI. 72. F. 24.	Looking 12 or 13 only; had never menstruated. "Biliousness" for some time. On admission, blue, pulseless; heart beating 170; diarrhœa	Back part of right lung œdematous and containing a brown circumscribed patch the size of a walnut. In left lung a firm circumscribed	Fluid in both pleuræ, in right compres-

3.	and vomiting; much dyspnœa and jugular distension. Vomited some blood; got rapidly worse. Eleven days.	reddish mass at the apex, and two others in the lower lobe, the result of extravasation. *Mitral orifice contracted to size of end of little finger;* tricuspid also contracted; some hypertrophy of walls.	sing the lung. Pericardium universally adherent.
LVII. 22. M. 26. 4.	Subject to palpitation and dyspnœa since an attack of acute rheumatism seven years before. Ill with those symptoms six or seven days on his admission; supposed from wet. Nine days.	Lungs in places emphysematous; at lower parts consolidated; firmer and darker patches here and there, as from old inflammation or old-standing extravasation of blood; but in no place was any recently extravasated blood. Liver large and nutmeggy. Heart very large (27 oz.). *Mitral orifice narrowed* by calcareous matter and thickening of flaps; two fingers could not pass. Left auricle very large.	Pleuræ not mentioned.
LVIII. 20. M. 37. 5.	Dyspnœa only; no pneumonia mentioned or treated; intemperate; congested face; frothy sputa. Ill five weeks in a similar manner before he came under treatment. Nearly one month.	Red hepatisation of upper lobe of left lung; emphysema of the lower lobe. Right lung congested. Bronchial tubes dilated. Hypertrophied heart (20 oz.).	
LIX. 79. F. 17. 6.	Account of an illness (ill-defined) with delirium and increased action of heart, three years before (rheumatism ?); has had palpitation ever since. On admission, rheumatic pains about the shoulders; a loud systolic murmur; pulse regular, not very unnatural; no œdema; constant orthopnœa, but no obvious dyspnœa. Given bark and wine. Latterly, œdema and emaciation; mental wandering. Lingered long. Died as fainting. Forty days.	The lower part of each lung in a state of red hepatisation; the inflamed part melting down gradually into the healthy. The pericardium firmly adherent. Heart much hypertrophied; its muscular fibres in advanced stage of fatty degeneration. Some deposit of lymph on aortic valves. Other organs healthy.	Adherent pericardium.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LVII. 225. F. 40. 7.	For twelve weeks dyspnœa and pain between shoulders. For a month has had œdema of the feet. Urine albuminous; heart 150 in the minute; pulseless; much anasarca, and a livid face. Three days.	Lungs generally pale and bloodless; but two patches of pulmonary apoplexy in the substance of the right lung near together, each about a cubic inch. Kidneys rather granular. Some hypertrophy of left ventricle. *Mitral orifice exceedingly contracted*, admitting only point of forefinger.	Adhesions of right pleura.
LVIII. 148. M. 22. 8.	Palpitation and diarrhœa (the former ascribed to overwalking three weeks before); in bed for two weeks. No history of rheumatism. Breathing very distressed; urine albuminous; anxious to be left alone. Died suddenly, after apparent improvement. Six days. (Very imperfect account.)	Good condition. Heart very large (21 oz.); ventricles very large; wall of left much hypertrophied; wall of right rigid and leathery; both filled with gelatinous clot. Lower lobes of both lungs in state of red hepatisation. Large fibrinous vegetations on aortic valves, which were small, rigid, and insufficient for closure. *Mitral orifice hardly admitting a finger;* its flaps rigid; the ventricular surface covered with fibrinous vegetations. Spleen and kidneys containing large blocks of fibrin.	Old pleural adhesions on right side.
LVI. 67. Lad—age not given. 9.	Admitted dying. History states that he had been ill eighteen months; much worse for the last six months. An attack of acute rheumatism (date not stated) had left him very anasarcous. Died shortly after admission.	Considerable œdema of lower extremities. Red hepatisation of lower part of upper lobe and of the whole of lower lobe of the right lung. Lower lobes on both sides contained several large, firm, solid, circumscribed patches, where, apparently, blood had been extravasated.	A good deal of bloody fluid in left pleura, and little in right. Recent pleu-

		Heart of enormous size and universally adherent to pericardium. Liver and spleen congested.	risy right side.
LVII. 276. F. 27. 10.	Dyspnœa for several years. Admitted with that and general œdema; the jugulars being greatly distended. Ten days.	Apoplexy in left lung. The right lung gray and quite solid. The right auricle and its appendix very much dilated; orifice of tricuspid very large. Heart weighed 16 oz.	Fluid in right pleura.
LX. 26. M. 62. 11.	History of acute rheumatism twice, and now for seven weeks. Cough and dyspnœa, with dropsy. Urine is albuminous; sputum rusty, "pneumonic;" delirium at night; pulse 54, very irregular. Antimonial wine, and later, stimulants. Death by asthenia without dyspnœa. Five days.	Lower lobe of right lung hepatised. Left lung healthy, but for a patch of apoplexy the size of a nut. Heart large; its cavities much dilated; the walls hypertrophied; decolorised clot in right cavities. The left valves rigid and atheromatous; extreme atheroma of root of aorta. Remaining organs natural.	Serum in right pleura, and a very general lining of lymph. Serum, but no lymph, in the left pleura.
LX. 144. M. 22. 12.	Cab-driver; had had rheumatic fever four years ago, and ever since palpitation. A cough came on, "from a cold," three weeks before admission, with spitting and increased palpitation. Urine very scanty and dark; œdema shortly supervened. On the day of admission sputum was blood-stained. He was very sallow and œdematous, and had orthopnœa; irregular heart and pulse (82). Given squills and saline diuretics; twice dry-cupped. The sixth day dropsy increased, he became delirious and violent, and pulse became frequent. Ten days.	Lower extremities very œdematous. Heart very large, chiefly from distension of its cavities, the walls being weak and thin. The *orifice of mitral diminished* and perfectly rigid from large mass of calcareous matter in both flaps. Aortic valves healthy. Left lung much congested. Whole of lower lobe of right lung in a state of red hepatisation. At its lowest part, amongst the inflamed tissue, a small spot of pulmonary apoplexy. Liver soft and pale. Spleen contained block of fibrin.	Much fluid in right pleura; left pleura adherent. Bloody fluid in pericardium.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LX. 225. M. 17. 13.	Ill with rheumatic pains for four days previously. For years subject to cough and slight hæmoptysis. On admission, rather acute rheumatism, with tumultuous heart-sounds. No pain in chest; a very little viscid light-coloured spitting. Bled to ʒxij.; given calomel and opium; also ten leeches over the heart (physical symptoms being those of pneumonia). Continued to get weaker and more short-breathed. Always on his back. Sank rather rapidly. About six days.	cavities. The mitral rigid; *its orifice barely admitting point of forefinger.* Rather large heart; decolorised clot in all its Whole of lower and part of upper lobe of left lung in a state of red hepatisation. Back of right lung in similar state, and much loaded with serum. Other organs healthy.	Much clear serum in pericardium. Old adhesions right pleura; left pleura healthy.
LXI. 171. M. 50. 14.	Case nearly illegible. Dyspnœa and cough, sonorous râles being audible in the right chest. Had been in previously with supposed effusion into right pleural cavity. Anasarca came on, and the legs were punctured when sloughing commenced. He lingered four months and a half.	A deep and extensive ulcer on the right leg. A few miliary tubercles at apex of left lung, and some pulmonary apoplexy at its lower part. Right lung entirely consolidated by means of a gray granular deposit, which infiltrated its entire substance. Heart much hypertrophied. Both auriculo-ventricular valves closely embraced by strings of yellow fibrinous coagula, *the openings both somewhat contracted.* Kidneys enlarged, and a little rough.	Fluid in lower part of right pleura. No recent pleurisy. Pericardium adherent by stratified lymph.
LXI. 305. F. 40. 15.	Admitted with dropsy. Sputa generally bloody. Account mostly illegible. One month.	Some parts of the lower lobe of the left lung were hepatised; some cretaceous masses at the apices. Heart of natural size; *extreme contraction of the mitral orifice*, which barely admitted the end of the little finger.	

LXIII. 17. F. 50. 16.	Urgent dyspnœa and cough for years; present attack three weeks. Admitted blue and gasping; coarse crepitation at base of right lung, &c. Given diuretics. Forty-four days.	Lower lobe of right lung (the greater part) hepatised; the bronchial tubes congested. Walls of heart thickened and yellow, fatty; *mitral orifice narrowed*, admitted but one finger. Heart weighed 16 oz. Old lymph on the visceral pericardium. Kidneys slightly granular; some depressions and cicatrices on their surface.	Recent lymph in pleura corresponding to hepatisation.
LXIII. 164. F. 28. 17.	Dyspnœa and cough for six months (after a cold); never had rheumatism; latterly some dropsy has appeared. Admitted very short of breath, with irregular pulse and loud systolic bruit. Four days after admission the sputa became bloody, but not for long; pulse was always full and sharp, and generally above 100. Cupped and dry-cupped; antimony given and digitalis. Forty-six days.	Belly distended and fluctuating. Whole of lower lobe of right lung solid, of buff colour, mingled with small patches of extravasation. The left lung, in the same situation, contained a smaller extent of the same charge. The bronchial mucous membrane was thickened. Kidneys were increased in size; buff-coloured and mottled; the cortex increased in thickness; their weight 18½ oz. *Mitral orifice considerably contracted* and thickened by means of calcareous deposit—only the little finger could be passed; ventricles uncontracted. Some recent adhesions about intestines.	Fluid in left pleura. Heart covered with false membrane of some standing.
LXIV. 335. M. 10. 18.	No case. About a day and a half.	*Mitral valve thickened and narrowed;* beads of recent lymph along its auricular aspect, also on the aortic valves. Both lungs largely occupied by red hepatisation irregularly diffused, natural tissue being interposed; left apex natural, right solid. Heart hypertrophied (9 oz.).	Much old lymph in pericardium.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LXIV. 101. M. 36. 19.	Caught cold, as he supposed, six weeks before from exposure to wet; health previously good but for frequent attacks of rheumatism; rheumatic fever three years ago. Admitted with œdema; ascites and blood-stained sputum; mitral bruit; action of heart regular, and not excessive; highly albuminous urine. Diuretics and elaterium given. Sputum abundant and blood-stained. Day before death erysipelas on left side of nose and some delirium. Fourteen days.	Slight jaundice; legs œdematous. Greater part of upper lobe of right lung consolidated with hepatisation; the tissue soft and friable, and in some places gray, yielding much sero-purulent fluid on pressure. Small portions of the lower lobe of the left lung friable; bronchial tubes red, not containing fluid. Aortic valves very atheromatous, so that they must have been permanently open; around the mitral was an enormous mass of stony deposit, which *narrowed the orifice so as only to admit the little finger*, and probably to prevent its complete closure. Liver nutmeggy. Kidneys natural; in places a little yellow.	Old adhesions and half a pint of fluid in each pleura. Pericardium uniformly adherent by old adhesions. Left ventricle contracted and hypertrophied. Heart weight 25 oz. Spleen large and firm.
LXIV. 189. M. 13. 20.	Anæmic and wasted; orthopnœa and slight œdema. Three months ill from dyspnœa and chest-pain. Admitted with pulse 120, and much heart-impulse. Ninth day pericardial friction audible, œdema increased, and pains occurred in the limbs (rheumatic). Rather gradual sinking. Thirty-six days.	Left lung infiltrated with serous fluid. A considerable part of lower lobe of right lung in a state of red hepatisation, sinking in water. Heart hypertrophied; mitral valve thick and stiff from fibroid growth in its substance. Liver congested.	Left lung surrounded by old adhesions. Pericardium universally adherent by a succession of alternate layers of black coagulum and recent lymph alternately (about two of each).

LXIV. 167. F. 19. 21.	Admitted with acute rheumatism, not very severe. The apothecary was called to her on the second day. He found her breathing with much difficulty, in a shallow, catching manner, and complaining of pain in the left side, with increase of the rheumatic symptoms. No friction-sound could be heard. Six leeches applied to left side; calomel and opium given every four hours. Next day she was tender, cautious of slight movement, and very breathless; pulse 104. Nothing was made out by auscultation of the chest anteriorly. Heart-sounds were quite clear. Painful breathing, restlessness, and general distress continue till death, the mind remaining clear. Four days (or two from the seizure of dyspnœa).	Very fat. A small shred of decolorised lymph at beginning of the right middle cerebral artery, not closing the channel; some of the small arteries were irregularly distended with fluid blood. The lower lobe of the left lung was much solidified from pneumonia, and sank in water. The right lung was natural. Along the inner edge of the mitral was a layer of soft recent lymph, of which a few beads were seen on the aortic valves. The pulmonary artery, from its origin to its third and fourth divisions, was filled with coagulum, as was the right ventricle; in many parts of the artery the coagulum was opaque, granular, and friable, evidently of some standing; in some places it was elastic and semi-transparent, and was mixed with ordinary black clot; the coats of the artery were quite natural, the clot but slightly adherent. The right ventricle contained a large quantity of yellow elastic fibrin, continuous with the clot found in the pulmonary artery. Some patches of fatty degeneration detected in some of the columnæ carniæ.	Both pleuræ contained a little fluid. Some recent lymph on right lung. The heart uniformly covered with a thin layer of recent lymph.
LXIV. 343. F. 39. 22.	Admitted prostrate, with frequent pulse, dry tongue, &c. Lived some hours only.	Both lungs irregularly mottled, with little red patches approaching hepatisation; here and there patches of complete hepatisation. Both lungs emphysematous. Spleen contained some fibrinous blocks—some hard and recent, others old and soft. *Old thickening and narrowing of mitral*, which was covered with a quantity of shaggy recent lymph.	

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LXIV. 93. F. 11. 23.	Anæmic; had suffered acute rheumatism two years before, and had dyspnœa ever since. On admission violent action of heart, mitral murmur, much pulsation of cervical vessels, pulse 136; child not greatly distressed. Given digitalis and paregoric. Did not keep bed till near her death; ate well; latterly had occasional fits of dyspnœa, pulse continuing excessively frequent, with no new symptom. Sank and died. Twenty-nine days.	Slight general œdema. The lower lobe and anterior depending tip of the upper lobe of the left lung in a state of red hepatisation. In the right lung precisely the same portions were in the same state, its lower lobe less uniformly affected than that part of the other side. Mitral valve covered with small, prominent, ragged vegetations, very soft, covering both edges of the valve. Partly decolorised clot in both ventricles. In the right kidney two small fibrinous blocks.	Old adhesions and a little fluid in both pleuræ; beneath the left a few spots of ecchymosis.
LXIV. 21. M. 28. 24.	Death so soon after admission that no history was obtained.	No œdema. Anterior edge of right lung a good deal solidified, as if from pneumonia, but not sinking. Front of left lung and almost the whole of its upper lobe consolidated, of brown colour, devoid of air, sinking, serous fluid in bronchial tubes. Heart, 1 lb. 5 oz.; one of aortic valves perforated by two ragged holes; left ventricle contracted. Kidneys enlarged and hardened. Liver and spleen enlarged and congested.	Fluid in pleuræ; in the left old adhesions.
LXIV. 115. M. 21.	Vigorous healthy man. Awakened one night by pain in chest and shivering, having been at cricket for a long time on the previous day	The right lung compressed by the distended pericardium against the back of the chest; the anterior portion of its upper lobe was of a dark	A few adhesions in each pleura.

25.	(in March); perfect health previously. On admission dyspnœa, tubular breathing, &c., in left chest, and dull percussion; rusty sputum. Calomel and antimony every fourth hour. Gradual amendment. After six days, lung-sounds nearly natural; three days later erysipelas attacks chest, back, and shoulder; weak, irregular, faltering pulse. Bark and ammonia. Lay on right side. Sank. Twenty-five days.	colour, quite solid, and sinking in water; the other parts of a pinkish colour, almost solid. Left lung compressed, but otherwise healthy. Heart healthy.	Pericardium much thickened, and containing two quarts of pus.
LIX. 79. F. 17. 26.	Account of an illness (ill-defined), with delirium and increased action of heart, three years before (rheumatism?). Has had palpitation ever since. On admission, rheumatic pains about the shoulders; a loud systolic murmur; pulse regular, not very unnatural; no œdema; constant orthopnœa, but no obvious dyspnœa. Given bark and wine. Latterly, œdema and emaciation; mental wandering. Lingered long. Died as fainting. Forty days.	The lower part of each lung in a state of red hepatisation, the inflamed part melting down gradually into the healthy. The pericardium firmly adherent. Heart much hypertrophied, *its muscular fibres* in advanced stage of fatty degeneration. Some deposits of lymph on aortic valves. Other organs healthy.	Adherent pericardium.

Class IV.

Table of Cases of simple Pneumonia.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
XLV. 200. M. 32. 1.	Well formed and in good condition. On day of admission seized with severe rigors, acute pain in right chest, and fever. When admitted skin is hot and dry; face flushed; pulse 108, not easily compressible. Bled to ℥xvj. Second day, pulse 120, small and weak; very hot and thirsty; a little brownish-red viscid mucous sputum. Cupped to ℥xvj., and given ½ gr. of ant. pot. tart. every three hours. Third day, pulse 108, weak; unable to obtain any rest, owing to vomiting caused by antimony; is also much purged. Dose is reduced to ¼ gr. Fifth day, in very weak state, spitting mucus mixed with blood; blistered. The blister rose well, but gave no relief. Herpes labialis now apparent. Jumped out of bed delirious. Soon after became insensible, and so gradually died. Six days.	The upper lobe of right lung and the whole of left lung "remarkably healthy." The lowest lobe of the right lung in various stages of red and gray hepatisation, adherent to the costal pleura by slender bands of lymph. All other organs healthy.	Recent pleurisy in connection with hepatisation.
LIV. 110. F. 18. 2.	Cough for a fortnight, with dyspnœa, pain in chest (earlier history not given). Admitted in state of considerable depression; the face flushed, skin dusky, pulse weak, tongue dry and furred; movements tremulous; feeling of great weakness and lowness; evidently unable to bear active treatment. She somewhat rallied subsequently (same day); but still there was such a feeling of faintness, that it was	Body somewhat obese and well made. Besides the marks from bleeding and the vesication from the blister, some leech-bites were visible on the chest. There was a little fluid in the left pleura. The left lung was somewhat congested. The right lung was perfectly solid and non-crepitant throughout; it gave out at its lowest part a turbid fluid as from gray hepatisation.	Right pleura obliterated by recent adhesions.

	thought necessary to give her wine. Second day, much hesitation felt as to propriety of bleeding. The pulse was weak and rapid, but the breathing very greatly oppressed. A small bleeding was made with great care. The pulse rather increased in power, and diminished in frequency the while. The blood was of course intensely buffed. Calomel and opium now given every three hours, and a large blister applied to the side. She died the following morning. Less than three days. [Verbatim report, with some curtailment, chiefly from omission of stethoscopic signs.]	The heart and all other viscera were natural.	
XLVII. 81. M. 32. 3.	A free drinker. Supposed to have been ailing some time with cough and symptoms of fever; but about his work till three days before; then rigors and pain, especially in right chest. On admission, skin hot; tongue coated, rather dry; pulse 120, small and weak; aspect that of a man suffering from fever, headache and general pains. Eighteen leeches to chest; ¼ gr. ant. pot. tart. every four hours; calomel and opium thrice daily. Vomited the medicine. Third day after admission, tongue dry and coated; some wine given. (Blistered on second day.) Fourth day, delirium decided. Fifth day, unconscious; passing motions, &c. involuntarily. Some brandy given. Continued to sink, and died on sixth day. Little expectoration, and that muco-purulent.	Upper lobe left lung gray hepatisation throughout, with specks as of commencing abscesses. Lower lobe compressed and dark red; portion only crepitant. Right lung loaded with red frothy serum; lower lobe in state of red hepatisation. Kidneys rather coarse.	Left pleura generally adherent by recent lymph at back part; much purulent serum, with flakes of yellow lymph. Right pleura contained small quantity of lymph, mixed serum and lymph adhering to lower lobes. Lymph and some fluid in pericardium.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of heptatisation.	Co-existence of pleurisy, or of pericarditis.
XLVII. 95. M. 30. 4.	Three days before admission, rigors, headache, vertigo, vomiting, general pains, cough with white frothy expectoration; no pain in chest; no exposure. On admission, flushed, dusky yellow, pulse 120, feeble; tongue white and rather dry; no chest-pain, even on deep inspiration. Second day, much heat of skin, and stitch in left side; tongue dry; pulse 120, sharp; rusty pneumonic sputa. Third day, bled to ℥xij.; and same evening to ℥viij.; tart. emetic. gr. ¼ every four hours; blistered. Quite gradual typhoid sinking, and death on the ninth day.	Upper lobe of left lung perfectly solid and in state of gray hepatisation; lower part equally solid with red hepatisation. Right lung generally crepitant, but at back gorged with frothy serum. Both kidneys rather large and slightly granular.	Left side, extensive recent lymph, the more recent at lower part. Right pleura not mentioned (healthy?). Slightly turbid serum (a little) in pericardium.
LV. 149. M. 25. 5.	A robust-looking man. Caught cold three days before admission; aching in back, &c.; cough and pain in the right side. On admission, face was flushed, skin hot, pulse hard; there was pain on deep respiration at lower part of right side. Leeches, calomel, and opium. Two days later, the face was livid, the other signs remaining, pulse being more frequent. Leeches were repeated and medicines given every three instead of every four hours. Third day, there was more pain at the upper part of the chest; the expression was pinched and anxious. He was now blistered, and mercurial ointment was applied to the vesicated surface. He became delirious and much lower. Fourth day, wine was ordered, and he seemed to rally. Fifth and	Body in good condition, well made. The whole of the left lung was hepatised; the front part was in a state of gray hepatisation. The lower part of the right lung was hepatised. The bronchial mucous membrane was vascular, with mucus in the tubes. Kidneys congested; healthy. Other parts healthy.	Left pleura full of turbid fluid and shreddy particles. The membrane highly vascular. The diaphragmatic portion of the right pleura coated with recent fibrin.

	sixth days, he was very violent and noisy; then low typhoid, delirium, and death. Eight days.		
LVIII. 64. M. 52. 6.	Good health till eight days before; then sore throat, cough, pain in left chest. Day or two after, pain shifted to right chest. It was severe at time of admission. Pulse 90, weak; respiration weak, hurried (auscultation). Blistered over chest in front; cupped on right side. Antimony and nitre every three hours. Better till the third day, when he became very delirious. On fourth day pain in right side increased and friction was heard. Cupping repeated. Calomel, opium, and antimony every three hours. On the fifth day a large blister to the right side; difficulty in spitting. Death on the fifth day.	Left lung and pleura healthy. Lower lobe of right lung extensively inflamed, the whole of its tissue, except a small part of the outside, being infiltrated with a whitish-yellow deposit, which in parts had broken down into well-formed pus. Other viscera natural.	Pericardium distended with turbid fluid and coat of lymph, the whole membrane lined with it. Heart healthy, but for a little atheroma. Soft lymph in pleura, corresponding with hepatised lung.
LVII. 277. M. 35. 7.	Little or no history. Appeared to have been under treatment for pleuritic pain in left chest, for which he had been leeched and bled. Some account of a blow two weeks before. Suffering intensely. Less than one day under observation.	Lower of upper lobe of left lung in a state of gray hepatisation; the apex healthy; the lower lobe much congested. The right lung crepitant throughout. Liver and kidneys healthy; also heart.	Left pleura quite obliterated. Surface of right lung coated with lymph half an inch in thickness. Some turbid serum.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LVI. 120. M. 30. 8.	A "navvy," who had been ill three days with catching pain in the right side and short hacking cough. Previously quite well. Face flushed; pulse 96, feeble; tongue coated; respiration very short and hurried; crepitation of various degrees all over back of right side, and universal dulness. Salines and antimony. Bleeding on second day. On fourth day, renewal of dyspnœa and much pain in left side. Antimony omitted. Great exhaustion from this time; and he died on the fifth day.	Body somewhat emaciated. The right lung completely hepatised and solid; nowhere crepitant. The bronchial tubes occupied by light-coloured coagula. The left lung was in a similar condition; but the lower part of it was only to a slight extent affected, much of it being spongy and natural. Capsules of kidneys somewhat adherent; kidneys otherwise natural.	Tolerably recent adhesions, to some extent, on both sides of chest. Some turbid fluid in left pleura. Heart natural. Numbers of shreds of fibrin in the pericardium.
LX. 74. M. (?) 9.	Formerly a coachman. Good health till he got wet six days before admission. This followed by rigors and dyspnœa. Admitted blue, gasping, and speechless for want of breath; the extremities cold; dyspnœa did not diminish. Died in the evening, quite sensible to the last. Death almost entirely by apnœa. Seven or eight hours.	Back of the right lung in a state of gray hepatisation as to its upper and middle lobes. The opposite lung congested, but crepitant. Structure of kidneys confused, but nothing more. Other organs healthy.	Uniform membranous layer of lymph, on both sides, over nearly all the pleural membrane.
LXII. 148. M. 62. 10.	A labourer. Attacked suddenly on his way to church, five days before admission. Giddiness and shivering; pain in left side; dyspnœa. In very weak state on admission. Pulse 100, very small; hands and feet cold. Ordered ipecacuan-wine in ammonia saline, and wine. Rallied a	Good condition. Lower lobe of left lung in a state of gray hepatisation; pus could be squeezed out of it. The bronchial tubes were very red. The right lung healthy. Heart quite healthy.	Left lung surrounded by large quantity of lymph. Old adhesions on right side.

	little; then became delirious, dyspnœa more urgent. At noon of third day was dying, respiration being extremely rapid. Died gradually. Three days and a half.	Capsule of right kidney slightly adherent. All other viscera healthy.	Considerable quantity of fluid in pericardium.
LXIV. 128. F. 50. 11.	Admitted moribund. Said to have been ill about five months. Died in a few hours.	All right lung, except a very small piece at its apex, converted into a mass of gray hepatisation, very granular in fracture, and pouring out pus abundantly on pressure; its weight, 3½ lbs. Weight of left lung natural, 15½ oz. Liver slightly fatty. A small cyst in right ovary. Heart, &c. healthy.	On right pleura, between lung and diaphragm, much gelatinous lymph.
LXIV. 117. M. 28. 12.	A single man of dissolute habits; in failing health for three years, and often laid up with lung-symptoms. In his usual health morning before admission; went to work at 5 A.M.; at 10 A.M. brought home in violent fit of rigor; put to bed; soon began to ramble, and then to be violent, requiring to be kept in bed by force. On admission, continued to ramble; with difficulty kept quiet; bowels very loose; tongue dry and brown; breathing rapid; no orthopnœa; neither cough nor sputum; dulness, tubular breathing on right side. Opiate enema; calomel and antimony; brandy. Soon obstinately refused medicines. The bowels continued rather loose; much raving. Fourth day, he began to sink; occasional returns of raving; respiration very hurried. Five days. (Six days ill.)	The left lung had some patches of ecchymosis on its surface; part of its upper lobe was congested, yet contained air, and floated. Weight of left lung 18 oz. The right lung as solid and unyielding as liver. It occupied all the pleural cavity, and was entirely hepatised, save a very small patch close to its lower edge, which still contained air; elsewhere the lung was quite airless. The entire organ sank. The upper lobe was of an uniform buff colour, infiltrated with pus. The lower lobe was browner. Everywhere the texture was remarkably granular. Weight of right lung 3 lb. 11 oz. Kidneys large, smooth, coarse. Transparent coagula in heart's cavities. Liver large; its surface very uneven; capsule thickened; the tissue gathered into sphericles, with large quantity of fibrinal matter between them.	On left pleura a small quantity of serous fluid. Both layers of right pleura coated with recent lymph.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LXIV. 36. M. 23. 13.	Ill for a month; pain and lumps about larynx, and dysphagia; and then pain under left nipple (not "stitch"). History very imperfect, from his want of voice, and pain which speaking caused. Stout; manner natural (auscultation, signs of left pneumonia); sputum rusty; partial orthopnœa; face venous. Second day, pulse 80. Third day, pulse 88; distressed breathing; has had severe pain in left side and epistaxis. Six leeches, calomel and opium. Died on fourth day, after two severe epileptic fits, ten minutes before and at death, respectively. Four days.	Upper lobe of left lung much congested; its lower lobe in a state of gray hepatisation, exuding pus-like fluid. Lower lobe of right lung congested. Cortex of kidney increased (natural appearance under microscope). Other organs healthy.	Left pleura contained a pint of purulent fluid. Recent lymph on surface of lung. A little clear fluid in the right pleura.
LXIV. 161. F. 47. 14.	Ill two weeks with cough and pain in left side. These came suddenly with rigors. The sputum at that time "like currant-jelly," and some unmixed blood. On admission, much distressed for breath; frequent pulse; much thirst; cool skin. Left side dull, and vocal fremitus absent; blowing breathing. Respiration became worse, and she died. Rather over one day.	Lower lobe of left lung completely hepatised, so as to sink as a whole; it had a grayish-brown colour, and a finely granular fracture. Posterior part of right lung in a state of early hepatisation. The small bronchial tubes were packed full of very thick muco-purulent matter. The muscular fibres very conspicuous. Heart natural; left ventricle imperfectly contracted.	Thin layer of recent lymph in the left pleura.
LXIV. F. 1 y. 10 m. 15.	Admitted moribund. No history.	Right lung much congested; especially its upper lobe, which was nearly solid, and contained little air; red; not sinking. Small dark coagula in right pulmonary artery. Yellow coagula in right ventricle.	Sub-pleural extravasation back of right lung.

LXIV. 231. M. 23. 16.	Seized with shivering and febrile symptoms two weeks before; kept bed four days; has much the aspect of fever; pulse 132; skin warm; a little thick, bile-stained sputum; averse to food; delirious. Calomel and opium every four hours. Second day, 6 oz. of port-wine; blister; delirious and violent during night; hot skin and tympanitic belly; respiration quickened, but no distress from dyspnœa. Pill stopped third day. Continued to sink till death. Four days and a half.	Emaciated. Tinge of yellow on skin and caynaction. Whole of right lung in state of gray hepatisation; as solid as liver, and exuding much pus; sinking as a whole. Weight 2 lbs. 10 oz. Left lung natural; weight 12 oz. Liver congested in patches. Liver-cells opaque, granular, of a faint brown. Other viscera natural.	Small quantity of purulent fluid in left pleura. On right, some clear fluid and some recent adhesions. Recent lymph in pericardium.

Class V.

Table of Cases of Hepatisation where death was ascribed wholly or mainly to low or latent Pneumonia.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
XLVIII. 262. M. 33. 1.	On admission presented all the appearance of an ordinary case of fever, extreme deafness having come on at time of illness two days before; the other symptoms being shivering, headache, sickness, dyspnœa. On admission he was very dirty, p. 120; had headache and much wandering (attention not called to the chest, which was not examined till six days later); had some cough, with scanty adhesive sputum; urine was retained in bladder; delirium occurred; face became livid, and cold sweats occurred. Death, by gradual sinking, on the twelfth day.	Dark serous fluid in left pleura. Posterior part of left lung was much congested and solidified; the whole of right lung was quite hepatised, firm and solid; small deposits of pus in this lung. The other organs healthy.	Some recent lymph at base of right lung.
LII. 173. M. 39. 2.	Reported of very dissipated habits, and to have had attack of "inflammation of stomach" (there was mark of blister on right side of chest), for which calomel and opium had been given. On admission was very restless and excited, so that he could hardly be kept in bed; state like delirium tremens. After two or three doses of laudanum, he slept for two hours; woke more collected; his face, however, was now dusky, and breathing oppressed. He sank very rapidly, having been about a day in hospital.	The upper lobe of right lung solidified completely, light gray fluid escaping on section; supposed to be in early stage of gray hepatisation; the lower parts of lung more solid than natural. On left side, lower part of upper lobe the same as lower lobe of right; the bronchial tubes very vascular. Kidneys congested. Other organs natural.	Pleural cavities natural.

LIII. 62. M. 27. 3.	A history of having caught cold, and having been ill in consequence for three or four months. The illness ascribed to standing in water. Four days before admission, acuter symptoms. Was admitted much emaciated, with rattling sounds in bronchial tubes. About two days in hospital.	The right lung solid; reddish gray *as to its upper lobe;* other parts of lung very congested. On left side, the lower lobe in the same so-called hepatised state; the upper part congested. Much thick mucus in the bronchial tubes.	Some firm pleural adhesions on both sides, but no lymph.
L. 114. F. 64. 4.	Nine days ill, the main feature being great debility and prostration, with but little cough. Sunk after seven days.	Upper lobe right lung consolidated and gray, approaching to suppuration. The left lung and lower of right lung emphysematous.	A few old adhesions only.
LIII. 66. M. 17 5.	Of remarkably bloodless expression. Ailing about sixteen months, "some slight illness" from which he had not recovered. No cause could be made out for his condition. Some hæmoptysis occurred (amount not stated) seven days after admission; great prostration followed. He died suddenly the following day. [No record of examination of urine during life. It was believed that it had been tested, and found healthy. After death, that found in bladder was distinctly albuminous.] Eight days.	Exceedingly exsanguine hue noticed at autopsy; the blood very fluid; all viscera exceedingly pale, but otherwise natural. Lungs very œdematous at apices. In middle and lower portions exceedingly consolidated, and of a dark reddish-brown colour, sinking in water. The heart natural.	Pleuræ and pericardium natural.
LIV. 115. M. 45. 6.	A Frenchman, from whom an imperfect history was obtained of eleven days' illness, viz. pains in limbs and bilious vomiting, the pulse being "pretty quiet;" no chest-pain. From yellow he gradually became jaundiced, got cough and muco-purulent expectoration. Died after eleven days. (No diagnosis of pneumonia.)	Whole of right lung, except the base, inflamed; the texture infiltrated with sero-purulent fluid. The left lung emphysematous in front, congested and œdematous behind. Kidneys large and congested; their capsules rather adherent.	Recent cavity in right pleura, with thick recent lymph.

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Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LV. 209. M. 62. 7.	Much emaciated; a year out of health. Seven weeks lumbar pain, œdema of legs, vertigo, and great debility. On admission, urine pale and albuminous; vomiting frequent. Fourth day, diarrhœa; afterwards gradually sank. Ten days.	Body in good condition. Œdema of legs. Lower part of right lung in a state of gray hepatisation. Lower part of left lung slightly hepatised.	Sero-purulent fluid in right pleural sac. Pericardium and heart natural. Kidneys too much decomposed for examination.
LVII. 20. M. 42. 8.	A butler of good general health till five weeks before admission, when he had a "cold" and dyspnœa. At this time he was knocked down by a severe blow on the left thigh; considerable swelling and partial loss of power of that limb followed. There were no symptoms to indicate chest-mischief. Fifth day he had severe rigor, followed by great collapse. Seventh day, a second rigor and collapse. Death on the eighth day. Purulent urine.	Left leg greatly enlarged. The whole of the left lung hepatised; one or two masses of fibrin in its upper part. The same condition, to slighter extent, in upper part of right lung. Femoral and popliteal vessels, and thin branches, occupied by dark-red fibrin. The cavities of the heart dilated.	Softish rough fibrin over the pericardium; old cystitis. Left pleural sac obliterated.
LVII. 43. F. 54. 9.	Admitted for old sloughing ulcers; began to complain of sternal pain; stomach was leeched, &c. Lingered on, and became almost comatose a day or two before death. Thirty-four days.	Gray hepatisation of upper and lower lobes of right side. Left lung emphysematous. Kidneys small, and with adherent capsules.	Recent pleurisy of right side. Left pleura healthy. A little turbid fluid in the pericardium.

LVII. 176. F. 24. 10.	On admission, very much exhausted and very dirty; diarrhœa and vomiting for past three weeks. At present headache, brown tongue, weak pulse, a loud systolic murmur. Given astringents and prussic acid. The urine slightly albuminous; sensations referred to the abdomen. She lay groaning and calling out night and day. Fifteen days.	Body well formed. Posterior flap of mitral very much thickened. Heart otherwise healthy. The upper lobe of right lung much inflamed; its posterior part in a state of red hepatisation. The lower lobes and the opposite lung much congested, infiltrated with fatty serum. Spleen large, 16½ oz. Left kidney rough, small, mottled; diminished cortex. Other organs healthy.	Firm pleural adhesion right side.
LVIII. 37. F. 29. 11.	Admitted with extreme debility, after child-bearing one month before. Rigors had occurred daily since that event; a dry cough and profuse sweating. Given bark, brandy, and stimulants. Thirteen days.	The body not emaciated. Lower lobe of right lung in a state of gray hepatisation; its tissue in part broken down, so as to form small abscesses. The opposite lung contained several masses of extravasated blood, also breaking down in parts. Kidneys pale and smooth, with stellate veins. Uterus as usual a few weeks after parturition. Heart large, with healthy valves and firm white clots in its cavities. Spleen almost diffluent.	Fluid and recent lymph in right pleura. Left pleura healthy.
LIX. 131. M. 42. 12.	Had sore-throat three months before admission. No history of syphilis obtained. The man slow and muddled and hesitating in speech; left pupil rather the smaller throughout; distinct loss of power in both hands; pulse 112. Given ammonia, brandy; later, iron and calumba. Gradually lost memory completely, and got delusions of all sorts; violent at night. Two days before death became comatose; pupils very contracted; pulse gradually accelerating up to 160. Ten days.	Emaciated. Slight softening of corpus callosum and septum ventriculorum (whether post mortem, or otherwise, doubtful). Lower lobe of right lung in a state of red hepatisation; rest of lung spumous, as was the left. Heart and remaining organs healthy.	Pleural adhesions at left base and right apex.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LIX. 164. M. 39. 13.	A great drunkard; suffered delirium tremens more than once. Out of health now for two weeks, with lassitude, headache, and pain in the limbs. Gave up four days before admission, and took to bed. Admitted with muttering delirium; not violent, but fidgety and restless; skin cold; pulseless; no spots. Neck blistered; wine and ammonia given. Died in a few hours.	Body in good condition. Right side of heart filled with decolorised clot; extensive old pleural adhesions on both sides. Left lung healthy. The whole of the two upper lobes of the right in a state of gray hepatisation, sinking in water. Bronchial tubes full of mucus. A few cysts on the surface of the kidneys; their capsules adherent.	Old adhesions both sides. Pericardium natural.
LIX. 193. M. 48. 14.	Admitted dying. A hard drinker, who had had repeated attacks of delirium tremens.	Heart dilated and flabby, apparently fattily degenerated. Lungs much congested; lower lobes on both sides inflamed and very friable. Liver and kidneys healthy.	Pleuræ and pericardium natural.
LXII. 208. F. 36. 15.	Admitted 28th July. A week before, seized with shivering and acute catching pain in right side. Cough came on about the same time, and, with dyspnœa, had continued ever since. On admission, aspect distressed and anxious; face flushed; respiration 52; pulse 120, and very weak; skin hot and dry; tongue dry and brown; lay towards the left side. In right side of the chest breathing was very harsh and imperfect; no ægophony or tubular breathing. A few moist sounds were heard at the apex on the left side; breathing was free. Both sides were equally resonant. Ordered antimony-wine in ammonia saline and port-wine (℥vj.) daily.	The body was plump and in good condition. There were no spots on the skin. The brain was perfectly healthy in all respects. The whole of the right lung was in a state of gray hepatisation, except a small portion near the apex and a small patch at the lower sharp margin. The texture was gray and mottled, and in parts softened as if on the point of breaking down. Pus could be readily squeezed out. The bronchial tubes were congested, and contained frothy fluid. The left lung was healthy, except that it contained near the apex two minute cretaceous tubercles. The left ventricle was uncontracted; it contained	The right pleura was natural. About the left there were a few old adhesions.

	Last day, much the same; face still anxious; a slight cough, but no spitting. Ordered ammonia salines; port-wine ℥xvj., daily.—30th. Quite delirious; respiration not so rapid; pulse 120, with some power; tongue dry, brown, and chapped. In front of the chest on both sides air was admitted, but imperfectly on the right, and very harsh was the breathing that was heard. A few small indistinct spots (supposed to be the mulberry eruption of typhus) appeared on the belly.—31st. Delirium continued; gums and tongue covered with sordes; respiration between 50 and 60; pulse 120; skin very hot; face flushed; the eruption just as it appeared the day before. Ordered 3 ℈j. doses of quina in three successive hours; afterwards two grains of quina every four hours.—August 1st (twenty-four hours after the quina was ordered). The skin was quite cool; pulse 72; respiration 60 a minute. She put out her tongue when told, and helped herself to some water. The next instant, within half a minute of the pulse being taken, the colour faded from the cheeks, and she was dead. About four days.	fluid blood, with two or three little pieces of black coagulum. The right ventricle was also uncontracted; it contained a decolorised clot of great length and tenacity, extending into the auricle and cavæ. There was a trifling amount of atheroma on the aorta and the mitral valve. The spleen and supra-renal capsules were soft. Kidneys and liver healthy. Large and small intestines perfectly natural. Uterus contained a little purulent matter.	
LXIII. 125. M. 33. 16.	He and his wife said to have been lying ill a fortnight. His appearance that of a fever-patient, but no eruption; flushed; semi-comatose; dry parched tongue; hot dry skin. Died without change. Ten hours.	Signs in brain as of a very old clot. Upper lobe of left lung in state of red hepatisation, approaching gray. At the back of the left lung a patch of pulmonary apoplexy. Thin layer of recent lymph in the heart, and beneath it old lymph deposited. Peyer's patches congested. The kidneys granular.	Adhesions (old?) in right pleura.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LX. 5. M. 42. 17.	Admitted in a state resembling fever. Said to have been losing flesh, and out of health for three months; had shivering ten days ago, and, shortly after, a fit like epilepsy. On admission, pulse 80; general state like that of phthisical patient with fever; no cough or expectoration, or pain at chest. Lay picking at bed-clothes. Two days.	There were a few small unsoftened masses of crude tubercle at the apex of the left lung; remainder of that lung was quite healthy. The whole of the right lung was hepatised, except a layer an inch thick at its lowest part. At the upper lobe the hepatisation was gray; lower down, red. Decolorised clot occupied the right side of heart. Kidneys finely granular; capsules adherent; cortex somewhat diminished.	Recent lymph was smeared over the right lung.
LX. 108. M. 52. 18.	A free liver, who had long suffered from eructations and epigastric pain. The day before admission, he was thrice attacked with hæmatemesis, each time vomiting about half a pint of blood. On admission, had slight cough with mucous spitting. Bronchial sounds were heard about the chest, and the liver was felt enlarged somewhat. Gallic acid and dyspeptic medicines were given. He got better after eleven days, and took an airing. The evening after, had a rigor, with great depression and want of breath. He lay from that time on his right side, evidently dying. No treatment was attempted, save stimulants. Fifteen days.	Rather emaciated. A patch of consolidation middle of back part of left lung, and another near the apex. In these parts the lung was very spumous, contained a good deal of blood, and only just floated. Whole of right lung in a state of gray hepatisation, except a very small part at the base. Other organs healthy, save kidney, which had two cysts on its surface, in one of which was coagulated serum having a whitish succulent mass of fibrin.	Extensive old plural adhesions on both sides.
LXI. 92. M. 65.	Suffered privations for three months, and for as long had pains in the loins extending down the left leg. He was much emaciated. The pain	Heart healthy, but for slight atheroma; right ventricle and pulmonary artery full of decolorised clot; red blood-clot in the left ventricle.	Right pleura obliterated. A very large

19.	continued and was very acute. Two days before death, severe cough came on with viscid dark-brown sputa. The day before death there was much pain in the left chest, and dyspnœa. Calomel gr. iij., and opium gr. j., every three hours. Thirteen days.	Right lung healthy. Nearly all lower lobe of left in a state of gray hepatisation. Double inguinal hernia. Remaining organs healthy.	collection of purulent serum in the left pleura, and recent lymph smeared on the surface of left lung.
LXIV. 186. M. 35. 20.	Epilepsy; delirium; aspect, &c. like that of fever. Slowly died; exhausted. Five days.	Lower lobe of right lung consolidated as in first stage of pneumonia. Congestion of brain-substance. Other organs appeared quite natural.	

CLASS VI.

Some exceptional Cases, mostly resembling Class IV.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LVII. 39. M. 32. 1.	A clerk, who had been out of health for some months; latterly he had "caught cold," and for a week kept his bed; later (exact periods are not given in the Report), acute pain occurred in the left side, like "stitch." Was admitted two days after, with pinched features, in great distress from dyspnœa. On auscultation fine crepitus, &c. Given calomel and tart. antimony every two hours; blistered; some wine occasionally. Wandering; death. Under four days.	Body in good condition. The lower and posterior parts of both lungs in a state of hepatisation. Lining of bronchial tubes very vascular. Kidneys large, but smooth.	Old adhesions of both pleuræ. Recent soft fibrin in both. Pericardium and heart natural.
LVII. 93. M. 35. 2.	A potman, who had had cough and hæmoptysis for three or four months. Admitted unconscious, very prostrate, hot skin; condition like continued fever, but no spots; urine collected in bladder. Thirty-six hours.	Middle lobe of right lung in a state of gray hepatisation. The lower lobe was red, softened, and breaking down. One-third only of the whole lung floated. The liver was large, mottled, and greasy. Kidneys large, coarse, healthy. Heart was large.	Old adhesions in left pleura. Right pleura universally adherent. In front and at upper part these adhesions were recent; posteriorly lower down they were jelly-like, apparently older.

LVII. 95. F. 3. 3.	Admitted in state like scarlatinal dropsy. Unhealthy aspect, large head, &c. Three weeks ill, described as "a cold." Large quantity of bronchial mucus. No examination of urine.	Bronchial tubes inflamed, and full of frothy mucus. The lung in various parts solidified, *e.g.* at both apices. Fatty liver.	Pleural cavities natural.
LVII. 304. M. 28. 4.	Represented as quite well three days before admission. Drank neat gin largely on the 26th December. Breathing was affected on the 27th. Was admitted on the 28th, when his manner was quiet and natural; pleuritic friction was then audible, and mucous râles masked the sounds of the heart. Antimony, calomel, and opium. Night of admission had restlessness, which soon passed into furious delirium. Two days.	Lower lobe of right lung in a state of red hepatisation. The upper lobe in a state of gray hepatisation. Some emphysema at edges of both. There was also bronchitis. The vessels of the lungs were plugged with lymph. Liver congested. Kidneys healthy. Brain not examined.	Recent lymph in both pleuræ and in pericardium.
LXIII. 20. F. 40. 5.	(Imperfect history from her husband.) Sticking to her work (though falling down from "pain" and exhaustion) till four days before admission. On admission, dulness of right side, &c.; the patient dusky, and quite exhausted. Soon died. Under two days. (Lay low in bed; respirations very rapid and shallow; scarcely, if at all, conscious.)	Belly tympanitic. The whole of the lower lobe of right lung in a state of red hepatisation, verging on gray; the whole sinking. Left pleura and lung healthy. Liver slightly fatty. Kidneys slightly rough on surface. Other organs natural.	Fluid in right pleura.
LXIII. 291. F. 30. 6.	A large flabby woman, ill since her confinement two months ago, much hæmorrhage following delivery; sore-throat later; dropsy set in two weeks back; kept bed for last ten days; winter cough for many years, and severe dyspnœa. Admitted with extreme dyspnœa and orthopnœa; sputum yellow, and finally rusty; delirium. One day and a half.	Upper lobe of right lung in a state of gray hepatisation. In several places small circumscribed deposits of pus, which might have been regarded as minute confluent vomicæ, had any tubercle been discovered. The whole lobe solid and livid. The lower lobe and left lung natural.	Sero-purulent fluid in right pleura, and a thick layer of recent shaggy lymph.

Reference to Post-mortem and Case-books.	Nature of case, &c.	Post-mortem appearances: site and extent of hepatisation.	Co-existence of pleurisy, or of pericarditis.
LVIII. 145. M. 30. 7.	Cough for eight days before admission, followed (in two days) by rigors and pain in the left side; anxious; face flushed, skin hot, pulse frequent. Auscultation. No albumen in urine; rusty sputum. Antimony and calomel. Twelfth day revived somewhat, and was given wine, bark, &c. Rather sudden relapse and unexpected death. Twenty-two days.	Whole of back of left lung hepatised; the right lung somewhat congested and œdematous. Kidneys rather large, with adherent capsules.	Purulent fluid and honey-combed lymph in the pericardium. Substance of heart dilated and soft. Lining membrane blood-stained. Pleuræ not mentioned.
LXII. 104. F. 28. 8.	History of rigors and pains in the limbs thirteen days before admission; then cough and bloody sputum, the quantity of which "had decreased very much." Was told she had fever. Admitted with acute symptoms, hot skin, flushed face, husky voice. Ordered wine. Expectorated a very little dark-coloured sputum. Second day, on auscultation, friction was heard (pleural or pericardial?). Pulse 120. Ordered calomel and opium, and wine increased. Third day, pulse more frequent. Left side was blistered. She soon tore the blister off, and talked wildly. Dyspnœa was now urgent. Wine and brandy given. She gradually sank, and died the next morning. About four days.	Lower lobe of left lung perfectly solid, and devoid of air. Of a light fawn-colour and compact texture. The upper lobe quite natural; the transition from one to the other quite abrupt. Slight emphysema of the right lung. One kidney dwindled into mere fibrous tissue. The other healthy, pale.	A little straw-coloured fluid in the pericardium; and a little recent lymph on the surface of the heart. Pleuræ not mentioned.

LIII. 254. F. 22. 9.	Ill only a week, with cough and sore-throat, with pain in left side. On admission, face very dusky; breath very hurried; pulse 130, full, firm. Bled to 10 oz. Auscultation next day; coarse, moist sounds universally, but no dulness. Blood not buffed nor cupped. The expectoration scanty and purulent. On the 5th day there was some approach to fine crepitation or friction at lower part of left lung without marked dulness. She sank gradually, with typhoid symptoms. Nine days.	Right lung healthy and crepitant throughout, but the bronchial tubes of both lungs vascular, and full of muco-purulent fluid. Back part of lower lobe of left lung solid and compact, breaking down under pressure. Gall-stone blocking up cystic duct.	A single band of adhesion in left pleura.
LXIII. 178. F. 11. 10.	Dyspeptic symptoms; sudden accession of dyspnœa, and death in a few hours. Twelve days. (Had had acute rheumatism some years before, and cough for two months. Frequent pulse, hot skin, and throbbing carotids noticed before the sudden collapse of which he died.)	On lower part of left lung, and throughout the right lung, were patches of lobular pneumonia as big as peas. Old thickening and recent blood-stained lymph on the cardiac valves.	Old adherent pericardium.
LXIII. 265. M. 9. 11.	Delicate. Admitted with acute rheumatism. After eight days has all the physical signs of double pneumonia, mostly of right side. Makes a good recovery in about eight days more. Convalescence interrupted, seventeen days later, by return of pain in limbs and alarming attack of dyspnœa; pulse 120; visibly pulsating carotids; free respiration posteriorly. Soon up and about; well, or nearly so. Then, eleven days after the last, a sudden attack of angina, which carried him off in a few hours. Forty-three days.	All the right lung, except quite the apex, was red, solid, and airless, "evidently in the first stage of pneumonia;" portions only sunk in water. Left lung was affected in exactly the same way, but less uniformly. The consolidation is scattered through the lung, so as to give to a section a marbled or mottled appearance.	Pericardium universally adherent by recent lymph. Mitral valve very much thickened by fibroid matter. Aortic valve slightly so. Adhesions of some standing in both pleuræ.