An inquiry into the pathological anatomy of acute pneumonia / by J. Da Costa.

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AN INQUIRY

INTO THE

PATHOLOGICAL ANATOMY

OF

ACUTE PNEUMONIA.

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WITH A PLATE.

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ACUTE PNEUMONIA.

THERE is, perhaps, no subject within the wide range of medical pathology which has received more attention than Pneumonia. The frequency of its occurrence, the interest centred in its therapeutics, and the facilities which modern research has supplied to its diagnosis, have produced a larger number of exact clinical observations than most other diseases can lay claim to. Yet, notwithstanding the care with which its study has been pursued by successive medical generations, there are some points connected with it, vitally concerning its correct appreciation, which must still be regarded as unsettled.

That pneumonia, as a disease, was known to the ancients, cannot admit of doubt; but how far they understood its anatomical characters, and separated its symptoms from those of other acute thoracic affections, is not so easily decided. More especially does the distinction between it and pleurisy seem to have been to them a matter of difficulty, as may be gathered from the writings of Hippocrates, whose descriptions of the latter disease might with equal, if not with more, propriety, be made applicable to pneumonia. Aretæus, too, usually so clear a delineator of disease, is not more fortunate in this respect, and is exceedingly obscure when speaking of "peripneumonia," in the description of which he evidently includes morbid appearances and symptoms belonging to pleurisy. This confusion with regard to the two diseases lasted until the present century, since it can scarcely be said that the distinction which Sydenham introduced, of a *true* and a *false* peripneumonia, cleared up the anatomical characters of either of the affections in question. His own writings, indeed, bear ample testimony to the fact that, whilst he referred the term "peripneumonia notha," or *false* peripneumonia, to a disease at present known to us as bronchitis, or perhaps as bronchopneumonia, under the term "peripneumonia vera," he still confounded both pleurisy and pneumonia. Morgagni, Valsalva, Stoll, and even Hoffmann, followed him in this error, as becomes sufficiently apparent from a passage in the works of the latter, in

which we are informed that the difference between the two affections consists in pneumonia occupying the deeper portion of the lung, pleurisy generally the more superficial, and the pleura.[1]

Thus, notwithstanding the many observations made, and the time that had been spent on the study of pneumonia, the present century found the exact pathology in a very unsettled condition, and it required some of the master minds of this age to cut through the knot, tightened by medical controversy, and point out the true path on which observers were to proceed. Amongst the first who clearly distinguished pneumonia from pleurisy, we find Bichat leading the way by his anatomical researches. Yet to Laennec—great in pathology as in diagnosis—belongs the credit of having most clearly and satisfactorily investigated the pathological lesions, and, by careful anatomical examinations, as well as by a comparison of the results thus obtained with the stethoscopic signs, of having detached pneumonia, as a separate disease, from the other affections with which it had up to then been classed. Laennec divided pneumonia into three principal stages: the stage of engorgement—lung of livid hue, heavier, but still crepitant; the stage of red hepatization—marked by loss of crepitous feel, with increased consistence and weight; and, lastly, purulent infiltration, or the formation of pus in the inflamed lung. Besides these most prevalent anatomical conditions, we find in his work descriptions of rarer forms of the disease: as of chronic pneumonia; carnification of the lung; and even allusions to lobular pneumonia, the true nature of which, and its connection with bronchitis and collapse of the air-cells, form part of those important subjects which modern pathological research is now so brilliantly elucidating.[2] To inquire more minutely than this into the further historical development of the exact anatomy of pneumonia, would be here out of place, and is, indeed, in so far unnecessary, as the terms and distinctions of Laennec (if we substitute gray hepatization for purulent infiltration) are the ones still most generally adopted; and as, further, the opinions of the more distinguished pathologists of the day, and the additions made by them to our knowledge of the affection, will have to be considered in discussing the subject of this paper—the changes which take place in the lung in acute lobar pneumonia.

First Stage, or Engorgement.—We have, then, following Laennec, to admit, as the first stage of pneumonia, the "engouement" or engorgement. A lung in this condition is of a livid hue, less cohesive, less elastic, and less crepitous; it has lost its spongy character, and from it transudes, when pressed, a frothy serum. If a portion be subjected to a minute examination, the following

[1] "Vera autem pleuritis sedem suam habet in ipsa pleura, quae intus thoracis cavitatem investit, et plerumque in consensum trahit annexos subinde pulmones, et peripneumonia ipsam pulmonum substantiam occupat."—*Hoffmann, Op. Omn.*, tom. i. cap. iii. p. 294; Genevæ, 1748.

[2] See especially the valuable contributions of Dr. Gairdner, in the *British and Foreign Medico-Chirurgical Review*, 1853 and 1854, on Collapse of the Lung, Emphysema, etc.

appearance will be found: The fibrous walls of the air-vesicles (Fig. 1, *a*) are perfectly distinct, but seem to be more easily separated, and inclose, in some parts, a slightly granular matter. The epithelial cells are detached from the sides of the air-vesicles, and from those of the minute bronchi; their nuclei are unaltered, but the cell-contents are more granular (Fig. 1, *b*) than in normal epithelium. The minute capillaries are gorged with blood; yet the separate corpuscles in them are not distinguishable. Few of the latter, also, contrary to what might be expected, can be seen lying in the air-cells.

The congested bloodvessels soon permit a fluid to transude into the air-cells and their walls. Thin, frothy, and coagulable by heat, it presents, at the onset of the inflammation, but slight differences from the serum effused in ordinary congestions and œdema. As the morbid process advances, and the spongy texture of the lung becomes less distinct, the air-vesicles contain more granules; the serous fluid becomes of greater density, and shows distinct blood-corpuscles and new elements in the shape of small nucleated cells and exudation-corpuscles. (Fig. 1, *c*.) The effusion, indeed, commences, within the lung, to present the peculiar characteristics which we are in the habit of regarding, when expectorated, as the pathognomonic, rusty-coloured sputum of pneumonia. This completes the picture of a lung in the first stage, or the so-termed stage of engorgement. Before proceeding to describe the subsequent development of the exudation, it becomes incumbent upon us to examine whether the ordinary appearances, or the minute structure of lungs in this condition, present any signs which may fairly be considered to be undeniable evidences of inflammation.

Characteristics of the First Stage.—Inflamed lung, as we have seen, exhibits to the eye a difference in colour and friability from healthy lung, as well as an augmented serosity. That the *colour*, although darker and more violet than that of the normal tissue, affords no certain distinguishing mark between the cadaveric and other engorgements, is pretty generally conceded; but this is not the case with regard to the increased *friability* of the lung, on which, indeed, some pathologists have laid, and still continue to lay, great stress. Thus, Lallemand regards the degree of friability as the only test by which an engorgement of the tissue, occurring shortly before death, can be recognized from that of an inflammation which has lasted for a day or two. In the first edition of his *Clinique Médicale*, Andral advocates the same view: "*Pour peu que l'un constate une plus grande friabilité de ce tissu, il doit être regardé comme enflammé.*" Further observations have, however, led him to modify his views; for, in the second edition of the same work, he states that more recent researches have convinced him that the increased friability of the pulmonary tissue may also be owing to the simple fact of the accumulation of blood, either during life or after death. Observations instituted with regard to this point, and made more especially on well-marked œdematous lungs, have led me to the latter conclusion; although it seemed to me, in many instances, that the lungs in which the congestion might fairly be considered to be *post mortem*, were wanting in that amount of friability which is found in lungs that have

been engorged for some time before death, or which ordinarily belongs to inflamed tissue.

Exact observations are wanting in regard to the character of the *serous* exudation, in the stage under consideration. Conditions of lung bearing a close resemblance to this first stage, are the engorgement of the tissue and the effusion of serum characterizing the disease known as "œdema," as well as the simple congestions occurring both before and after death, which may, perhaps, strictly speaking, not be designated as œdema, but which also give rise to slight exudations into the pulmonary structure. A question, therefore, of great interest arises here. Are there any signs which may serve to distinguish the effusion of the first stage of pneumonia from the ordinary serous effusion occurring in œdema? and, also, from the exudation observed in simple congestion of the lung? A solution of this inquiry is the more important, from the fact already stated, that the colour and friability furnish at the best but doubtful tests. Now, to arrive at a definite conclusion, with regard to this point, I have for some time past taken every opportunity of examining congested lungs, both with the unassisted eye and with the aid of the microscope, and of contrasting them with the appearances recorded of lungs in the first stage of inflammation. My observations have led to the following conclusions. To the unassisted eye, no difference between the effused fluids is perceptible at the onset of the inflammation; but as the pneumonia advances, the secretion becomes thicker and redder, until it ultimately approaches to the well-known rusty-coloured exudation. Neither are there, at first, microscopically, any very material distinctions. The fluids of both contain a few granules, whilst the structure of the lung is exactly similar, excepting that the walls of the air-vesicles are generally more granular in inflamed than in simply congested portions of lung.

In a more advanced stage, however, of inflammatory engorgement, the appearances are decidedly changed. We find a much larger number of exudation-corpuscles, and the epithelial cells seem to contain, on the whole, more granules, partly fat molecules, partly, perhaps, an imbibition of the inflammatory products. Again, there are elements in lungs which have been in a state of hyperæmia for some length of time previous to death, both in the tissue and in the exudations, which are not met with to any extent in the acutely inflamed organs, viz: peculiar large flakes and cells (see Fig. 2, *b*, *c*), inclosing blood-corpuscles, in numbers varying from one to twenty. These "blood-corpuscle-holding cells"—to use the term employed for similar bodies seen frequently in the spleen—degenerate readily into masses of pigment, which are seen to lie partly between, but most generally on the walls of the air-vesicles (*d*). The colouring matter of the blood which transudes from the broken-up corpuscles, imparts a uniform yellowish stain to these flakes or cells, as also to the surrounding epithelium. Many of the cells themselves are observed to be filled with a reddish or yellowish granular pigment (Fig. 2, *c*), not soluble in acetic acid, but destroyed by a strong solution of potassa; or there may be, when the

pigmental degeneration has been fairly established, no cell-walls at all visible, but merely accumulations of a reddish-brown pigment, or one of various colours of transition from that to an intense black.

In very acute congestions, and in cadaveric engorgements, these cells and the fresh pigment are mostly absent. In the much congested lung of a man who had been recently drowned, I met neither with these cells nor any new-formed pigment; whilst both I have found to be almost invariably present in the chronic engorgements of the pulmonary tissue accompanying cardiac affections. As occasional elements in these chronic congestions, I have met with granule-cells and a fatty epithelium (see Fig. 2, *a*); but never do I recollect to have observed them in what might with justice be presumed to be *post-mortem* engorgements. The granular cells themselves are probably the normal epithelium in a stage of fatty degeneration.

From these observations, therefore, it may be concluded that the number and variety of exudation-corpuscles, and perhaps the absence of blood-corpuscles undergoing peculiar pigmental changes, alone afford any evidences of acutely-inflamed pulmonary tissue.

The exudation of œdema, as well as that in inflammatory engorgement of the lungs, is known to be coagulable by heat and acids; the other *chemical* changes which accompany these conditions have, as far as I am aware, not been touched upon. My inquiries on this subject have been, up to the present, nearly altogether confined to one element—the amount of fat present; and have resulted in showing that in congested, as well as in inflammatory engorged lungs still permeable to air, this principle is invariably increased. The normal amount of fat in a healthy lung is stated by M. Nalatis Guillot (*Compt. Rendus*, 1847), not to exceed 6 per cent. My examinations, conducted with reference to this point, have yielded even less than that—not over 4 per cent. in proportion to the dried, or 0.65 to the wet tissue; whilst in primarily congested lungs, when dried, it ranged from 7 to 9 per cent. Subjoined is a table of some cases examined.

Age of patient.	Lung weighed; wet.	Lung weighed; dried.	Mass analyzed.	Amount of fat extracted.[1]	Per cent. of fat in wet lung.	Per cent. of fat in dried lung.	Disease patient died of.
	grs.	grs.	grs.	grs.			
40	175.5	32	32	2	1.13	6.25	Chronic pleurisy.
38	111.65	11.75	11.50	1	0.896	8.695	Tubercular meningitis.
48	56	10.3	10	.7	1.25	7	By inhaling hot air.
42	155	22	22	1.76	1.135	8	Drowned.
23	162.5	27.25	27.25	2.25	1.384	8.25	Disease of mitral valve.
31	65.5	10.5	10	.85	1.29	8.5	Phthisis.
45	658	.....	658	4.40	0.668	.....	Chronic pleurisy.
2	380	.....	380	5.6	1.47	.....	Meningitis.

[1] The fat was extracted by repeated treatment with ether. It need scarcely be stated that parts, entirely free from tubercular or other morbid changes, were selected for analysis. Several of the cases presented well-marked signs of congestion of the lung for several days before death.

Now, as the engorgement was not cadaveric in any of these cases, this table proves a marked increase of fat in hyperæmia of the lung occurring some time before death. My observations with reference to the increase of this substance, in the first stage of pneumonia, are as yet too limited to permit of my publishing here a comparative table; but the cases examined lead me to the belief, that the accumulation of fat occurs in this stage even to a much larger extent than in simply congested lungs. In one well-marked specimen, in which the portion of the lung analyzed had commenced to lose its spongy character, but still floated in water, although some parts in its vicinity were already markedly consolidated, the amount of fat present in 360 grains of wet tissue was 7.85 grains, bearing the proportion of 2.18 per cent. to the wet, and 15 per cent. to the dried structure; a much larger percentage than was obtained from any congested lung examined.

A recognizable condition of the pulmonary tissue is stated, by a most distinguished observer, to *precede* the stage of engorgement of Laennec. Dr. Stokes describes, as the true first stage, an "irritation" of the lung, in which the pulmonary tissue is drier, and of a bright vermilion colour. However convenient, in many respects, it might be to admit such a morbid condition, pathological demonstrations have not borne out this author's view. Nearly all eminent pathological anatomists of the day deny this stage as an antecedent of inflammation of the lung; nor does, indeed, Dr. Stokes himself seem to have based his assertion so much on anatomical as on clinical grounds.[1] He states, it is true, that he has met with this condition in pathological specimens, and relates (*op. cit.*) a case of an extensive burn in a child, where nearly the whole of the lung was in this state; but he fails to point out that this appearance is of necessity connected with inflammation. I have had recently an opportunity of examining the lung in a case similar to the one he describes. The organ, in this instance, was dry, and presented an unusually bright colour; yet nothing could be discerned in which it differed from ordinary congested lungs, nor did it, when minutely examined, afford any evidence of inflammatory action.

Another morbid change which has been much dwelt upon by writers, as connected with this first stage, is *splenization;* but so varied are the descriptions given of this condition, and it has been associated with so many diseases, that there is no little difficulty in referring any characteristic appearance to it, save that very general one of a resemblance to the spleen.

[1] "No one can doubt," he writes, "that the crepitating is but the diminutive of the mucous râle; it is a phenomenon produced by the passage of air through a viscid fluid secreted by the irritated cells or terminal tubes, and its peculiar characters result from the bubbles being necessarily so minute. The existence of this sign, then, points out that secretion has taken place into the cells and minute tubes; so that Laennec's first is, in reality, the secretive stage of the inflammation, and every analogy favours the opinion that a stage of irritation has existed previous to the secretion which caused the crepitus."—*Stokes on the Diseases of the Chest*, Am. ed., p. 266.

Andral represents it as a more intense engorgement of the lung, and states it to be an intermediate stadium between the first and second stage of pneumonia. Rokitansky refers it to a "stasis" or stagnation of blood in the lung, which may lead ultimately to its hardening. Meriadec Laennec, in a note to his cousin's treatise, describes splenified lung as sinking in water, highly congested, yet devoid of granulation, and thinks it arises from a hemorrhagic congestion. Some authors state to have met with it in several pulmonary diseases, some only in pneumonia, whilst others, again (Bouillaud, Lallemand, etc.), make it a synonym of the stage of inflammatory engorgement. Considering, then, the wide scope given to the name, and the different pathological considerations splenization is made to include, it is not easy to determine what changes in the tissue produce the only universally admitted feature—the more intense congestion. Several lungs from typhoid fever patients, which were violet-coloured, yet devoid of granulations, and which, therefore, according to Rokitansky's description, I considered as splenified, exhibited no marks by which they differed at all from ordinary congested lung, excepting, perhaps, the increased number of blood-corpuscles and of granules found in the fluid contained within the air-cells.

Splenization, when apparent in inflamed pulmonary tissue, shows, superadded to the already described microscopical appearance of this, a larger number of blood-corpuscles and exudation-cells, and vessels more intensely engorged.

Second Stage, or Red Hepatization.—As the inflammation advances towards the second stage, the lung becomes heavier, its spongy character disappears altogether, the air-vesicles are more and more distended with exudation-corpuscles, and the whole structure acquires an apparently solid condition. This second stage, or *red hepatization*, being fairly established, the lung-tissue presents the following well-known characteristics: It is heavy; dense; resembles in appearance the liver; is very friable;[1] sinks in water; if incised, a red, thick fluid transudes, and the cut surface appears distinctly granular. Between the small granulations—which are of varying size in different parts of the lung—masses of a darker colour may be observed, due generally to extravasated blood or to black lung-pigment. These and the white lines of the minuter bronchi and vessels divide the hepatized lung into numerous small sections, giving to it sometimes a distinctly marbled appearance. The granulations themselves are generally round, and difficult to isolate, except by the most careful manipulation.

Now, what are the *minute* changes which take place in the exuded mass, or

[1] The great friability of the lung, in this stage, has led Andral to propose for it the term of red softening (*ramollissement rouge*). Laennec condemns this, on the ground that, in reality, an induration occurs, whilst the humidity of the tissue becomes augmented, and adds: "Je crois que pour rendre complètement l'idée, qu'a voulu exprimer M. Andral, il faudrait dire, au lieu de ramollissement, augmentation de l'humidité." A very doubtful improvement, since increase of humidity occurs without inflammation in many affections of the lung.

in the parenchyma of the lung, which produce the appearances described? We have above traced the effused matter from a frothy serum until it attains a greater tenacity, and found it to consist mainly of granules and exudation-corpuscles, with but few blood-globules. These, indeed, only become fairly obvious at the commencement of this second stage, and are owing to rupture of the over-distended bloodvessels. In many parts of the lung, small lobules may be observed, redder and more densely congested, with their capillaries ruptured, and the air-vesicles full of blood-corpuscles, whilst in other portions no trace of the kind can be discerned. It must, thus, be admitted that the characteristic red sputum does not proceed from the whole of the inflamed lung, but only from those lobules in which the distended vessels have given way, and permitted the corpuscles to mix freely with the exudation.

In the fully-established second stage, round or slightly oval inflammatory cells, from 0.02 to 0.016 of a millimetre in size, may be detected, filling up nearly the entire cavity of the air-vesicle. (Fig. 3.) These cells are extremely varying in contents and shape. Some inclose one or two distinct nuclei, others are entirely filled up by granules. Some are round, others very irregular; although not near so much so as the imperfectly-formed bodies which have been denominated tubercle-corpuscles. Acetic acid leaves, in many, the granules untouched, but renders the nuclei more distinct. Maceration of the tissue in water for several days does not alter the cells materially. Many contain small fat-globules, which may be dissolved out by ether; the granules are not influenced by this reagent. In the majority of instances, the cells lie closely together on the basement-membrane of the vesicles, and are here in such numbers that, by a careless observer, they might readily be taken for a layer of granules. These, in reality, are the only exudation visible in some air-cells (Fig. 4); whilst others, again, contain numerous compound inflammatory cells (Fig. 3, *c*), stained more or less red by the colouring matter of the blood, and generally destroyable by caustic potassa. Besides these elements, many irregular flakes may be discerned (Fig. 3, *b*), probably formed by concrete albumen; also oil-granules and free fat.

Other new-formed substances found in the exudation in pneumonia, and to which attention was more particularly drawn by Remak,[1] are the *fibrinous casts*. These are described as ramifying in the smallest bronchial tubes and in the air-vesicles; their mode of arrangement being suggestive of areolar tissue, whilst their behaviour under acetic acid serves as the distinguishing mark. They are stated, further, to appear in the sputum, between the third and seventh day of the disease, and are considered by Remak as a sure sign of progressing exudation. He did not find them absent once in fifty cases. My own observations on this subject have not been very satisfactory, as I have frequently sought in vain, both in the sputum and the

[1] Diagnostische und Pathogen. Untersuch. Berlin, 1845.

air-cells, for these ramifying coagula. May not the frequency of their existence, as well as their import, have been overrated by their able discoverer?

All the elements enumerated are mainly found in the air-vesicles, and bear a relation, presently to be examined, to the formation of the granulations.

In the dense fibrous *walls* of the vesicles, and in their basement-membrane, but little alteration is perceptible. The former are slightly more granular, contain a few rather elongated cells, with nuclei rendered distinct by acetic acid, and occasional fat-drops; some of the elastic fibres may present a peculiar unravelled appearance. The smaller bronchi are also generally found involved, and within their walls deposits of granules and newly-formed elements are apparent. The vessels, in this as in the first stage, are gorged with blood; but they seem more compressed by the tissues, and their walls are infiltrated with albuminous granules. The epithelium, when present, is in a complete state of fatty metamorphosis.

Site of the Deposit.—These observations would go to confirm the views of those who contend for the almost exclusively vesicular site of the pneumonic deposits, and account for the granulations by the complete blocking up of the air-cells with an inflammatory lymph. Ever since the anatomy of pneumonia commenced to be fairly understood, the questions with regard to the exact situation of the exudation, and the nature of the granulations, formed subjects which gave rise to the most interminable dispute. Laennec[1] considers these granulations as characterizing the stage of hepatization, and describes them as the air-cells converted into solid grains by the thickening of their parietes and the obliteration of their cavities by a concrete fluid. Andral[2] shares to a certain extent this view of Laennec, at least as regards the secretion of a fluid. Williams,[3] on the other hand, inclines to the opinion that the granulations, and hence also the consolidation of the lung, are caused by a mere swelling of the membranous tunics; whilst Stokes, influenced by the rapidity with which the physical signs change from vesicular to bronchial respiration, conceives excessive congestion of blood to be the cause of the granulations, and quotes Andral (correctly?) as in favour of this view.

The opinion cited, with regard to the granulations, throw, then, very little light on the nature or exact site of the pneumonic process. Laennec foreshadowed, to a certain extent, as we have seen, by his description, the view of the air-cells being primarily affected; but nearly all subsequent writers, besides those quoted, preferred admitting as the cause of the hepatization mere swelling of the tissues, and derided the idea of an exudation into the vesicles. When, however, the minute anatomy of the lung began to be more closely investigated, it became evident that mere swelling of the parietes of the cells could not account for the consolidation in pneumonic lungs, or for their undoubted increase in size; and the inquiry, an exudation being

[1] Laennec, by Forbes, p. 184.

[2] Clinique Méd., iii. 465.

[3] Cyclop. of Pract. Med., vol. iii.

admitted, was directed with renewed care to its exact seat. Some, then, contended for the vesicular, some merely for the interstitial tissue, whilst others believed both to be equally affected. The majority of the pathologists of the present day incline to the view that the air-cells receive the pneumonic deposits, although there are not a few who still embrace the opposite sides of the question. Rokitansky takes very decided ground in favour of the exudation of lymph into the air-vesicles in ordinary acute pneumonia, and contrasts the whole process to a parenchymatous croup. He admits, however, besides this "croupy" pneumonia, as a distinct variety, a form in which the interstitial tissue alone is the seat of an effusion which gradually compresses and obliterates the air-cells.

My own observations on the minute anatomy of pneumonic lungs, as well as the clinical facts, connected with the renewed permeability of the lung-tissue after an attack of acute inflammation, have, as already stated, convinced me that the exudation is nearly entirely confined to the air-vesicles, and that only a very limited portion remains in their walls. That the interstitial tissue alone should, under any circumstances, be the sole seat of the deposit, without this reaching the air-cells, is, considering the great minuteness of the walls of the vesicles (for thus the term interstitial must be interpreted), very improbable. No author, moreover, has exactly defined what he means by the interstitial tissue, whether the tissue uniting the lobules, or the elastic tissue forming the walls of the air-cells and lobular passages. It seems, indeed, much as if this view, sanctioned to a certain extent by the high authority of the illustrious pathologist last cited, had been copied from book to book without any exact anatomical or pathological meaning having been attached to it. On the whole, then, we may be permitted to regard as established that the lymph in pneumonia is poured mainly into the air-cells, and only to a slight extent into their walls.

A form of consolidation of the pulmonary tissue, which has been variously described and explained, is that of the dense, dark, yet flabby lung, which, from the resemblance to meat, has been termed *carnification*.

This state of the organ results from the compression by a pleuritic effusion of a pneumonic, or even of a previously healthy lung; whilst, according to some (Rilliet and Barthez), it is most frequently owing to a chronic inflammation. Minute examinations, in cases capable of being restored to nearly their natural condition by inflation, exhibit the fibrous tissue infiltrated with masses of granules, insoluble in ether; with pigment, and sometimes with oil-globules. The epithelium, also, is more granular: some cells inclose a yellowish pigment-substance.

As the second stage of pneumonia progresses, the colouring matter of the blood is absorbed, and the lung becomes of a slightly more yellowish colour. Its surface is mottled; yellow masses are distinctly visible between the discoloured red spots, and, commencing as isolated points, they soon become general, until we have the peculiar soft, grayish-yellow lung produced, which

has been termed *gray hepatization*, or *purulent infiltration*. The pulmonary parenchyma itself has then become more friable; the granular appearance is in a great measure lost; and a thick pus-like fluid may be pressed out from the apparently suppurating lung. Or if, on the other hand, the pneumonia end by *resolution*, we have the organ becoming of a paler hue, the granulations, lighter in colour, mix with a freshly exuded serous fluid, which gradually dissolves them, leaving the lung itself more infiltrated with serum. The deposit is then thrown off with the expectoration, which itself shows many exudation-corpuscles, granules, and free oil. Or, again, we may have, after an acute pneumonic attack, the lung substance becoming indurated, by portions of the exuded lymph being transformed into lung-tissue.

Before considering the *minute* changes which accompany the formation of the third stage, it will be necessary briefly to recall how exudations degenerate, and what effect inflammation has on tissues. The exudation, the result of a stoppage to the free circulation, may be (see *Paget's Lectures* or *Wedl's Pathological Histology*) of various kinds: either capable of undergoing organization, or not; either firm and fibrinized, or serous—the more special character being determined in individual tissues by the state of the blood, and the seat and degree of the inflammation. The serous effusion is thin and watery, and not a frequent result of inflammation; more generally do we meet, under these circumstances, with the fibrinous. This latter, or the true inflammatory lymph, presents itself, according to Rokitansky, either as the simple or plastic, or as the croupous lymph. The former is generally more fluid, and may readily be entirely absorbed by an effusion of fresh serum, or else it changes into fibrous tissue; the latter, the "corpuscular lymph" of Paget, is firmer, of a yellowish colour, usually excessive, extends over a larger surface, and is prone to break up with great rapidity into a pus-like fluid, exerting a corroding property on the tissues with which it is in contact. These two principal forms of lymph are frequently found mixed in recent exudations.

If any of the varieties of lymph go on towards a further development, they demand, as a necessary condition, the cessation of the inflammatory process. If, on the contrary, they degenerate, the fibrinous part withers up or liquefies, undergoing, ordinarily, a fatty, or in some cases, a pigmental change. The corpuscular elements, to use Paget's term, may also dry up, or undergo a pigmental or calcareous degeneration, but most usually, they change into pus, or are subject to a true fatty metamorphosis, similar to the one observed in the cells of the liver.

On the tissues themselves, inflammation shows its results in several ways. It leads mostly to their softening—from a true disintegration, or from their penetration by the inflammatory products—or to their fatty degeneration.

This last named result, which may affect all normal, as well as abnormal structures, is one of the most interesting, as well as one of the most frequent of all pathological processes. Its most essential feature is the substitution of

minute granules, or oil drops, for the elements of the organ it attacks. In cells it leads to the formation of granule-cells and granular masses.[1]

Fatty degeneration may be the effect of a slow chronic affection, in which the nutrition of the part has been gradually impaired, but it may also occur as the result of acute inflammation, and of a sudden disturbance of the nutritive function. Fibrinous exudations, whether simple or croupous, are especially prone to undergo this change, as are also pus and formed tissues. In the latter, when acutely inflamed, fatty degeneration goes, as proved beyond doubt by the researches of Virchow and Reinhardt, hand in hand with softening. Thus Virchow[2] observes that, in acute inflammation of muscle (excepting when exceedingly violent, in which case softening of the fibres occurred without a change of the exuded lymph), a fatty molecular mass takes the place of the exuded protein substance and of the fibres, leading, within a short space of time, to their complete disintegration. The same changes in the constituents of an organ were produced by the experiments of one of his pupils, who found artificially excited inflammation of the cornea to be followed by alteration in its texture, and especially by the appearance within its elements of fatty molecules.

Third Stage, or Gray Hepatization.—To return from this digression, the bearing of which the reader will presently see, to our legitimate subject. The changes visible in the stage of "purulent" infiltration, or *gray hepatization*, are generally believed to be owing to suppuration having occurred in the parenchyma of the lung and in the exudation. But are pus-corpuscles the elements most prevalent in cases of gray hepatization? By no means. The number of these bodies present is, on the contrary, generally remarkably small, whilst other elements are observed to abound, which cannot be referred to a suppurative degeneration, viz: distinctly marked corpuscles, much larger than those of pus, and filled with granules and small oil drops (Fig. 5). But few of these cells contain nuclei, which, when present, are generally oval and apparent without the use of acetic acid, while the cells inclosing them are distinct, and not very granular. These nucleated cells are perhaps due to a more recent fresh exudation (which undoubtedly sometimes occurs in this stage) still in progress of development. In "yellow" condensation, a lesion presently to be described, they exist in larger proportion. In the ordinary "gray" hepatization, the non-nucleated granular cells are, however, far more predominant. They are uninfluenced by maceration in water, and not broken up by acetic acid. Ether

[1] These granule-cells (*corps granuleux* of Donné, compound inflammatory globules of Gluge) are not always necessarily caused by fatty degeneration of previously existing cells, whether inflammatory or normal. They may form from exudations devoid of corpuscles; although this is the exception, and not the rule.

[2] See for this, and other points bearing on the subject, his admirable Essay on "Parenchymatöse Entzündung," in *Archiv. für Path. Anat.* Bd. iv. p. 266.

clears them of their granules, and renders them almost transparent vesicles (Fig. 5, *b*); in some it shrivels up the cell-walls. Other elements met with, are flakes of albumen or fibrin, some containing small oil-globules; in exceptional cases, masses of pus-cells (Fig. 5, *e*) predominate. The fibrous tissue in the walls of the air-vesicles is very indistinct, and more or less densely infiltrated with granules (Fig. 5, *c*), which are mostly soluble in ether. Epithelium is entirely absent; nor are compound granule-cells in advanced gray hepatization noticed in the same quantity, as in the stage of red hepatization, whilst free fat molecules and globules abound. The vessels are generally dark brown, and their walls (Fig. 5, *d*), coated with large masses of granules, destroyable by the caustic alkalies.

These minute structural appearances, then, point conclusively to the fact, that gray hepatization is, certainly, in most instances, not a suppuration; but rather a gradual breaking up of the exudation (and, secondarily, of the lung-tissue), allied perhaps in its nature to fatty degeneration, such as we have stated to occur in other parts of the body, as a result of acute inflammation, and commencing with the earlier stages of the pneumonic process. In support of the latter assertion, besides the appearances presented by the microscope, may be cited the fact, that hepatized lungs yield a much larger quantity of fat than the normal organs, or even than such as are in a state of engorgement. Thus Natalis Guillot (*loc. cit.*) writes, that, in hepatized lungs, he finds from 18 to 40 per cent. of fat, whilst in engorged lungs, whether inflammatory or not, the fat, as we have above seen, rarely exceeds 10, or at the highest, 15 per cent. As he does not inform us in what stage of hepatization the lungs examined were, we cannot make any further use of his observations for ascertaining the relative amount of fat in the two stages. Yet there is a strong probability that the organs were mostly in the third, or in a far advanced second stage, as pneumonic lungs in the earlier conditions are more rarely attainable for pathological researches. My own comparative analyses are as yet too few to justify a conclusion on purely chemical grounds; nor do I conceive that this—considering that we have proved the fatty metamorphosis to be a gradual one, and shown that a large amount of oil and granules are visible in the epithelial cells, and in the exudation in the earlier stages of red hepatization—should be as decisive as the microscopic appearances. These last exhibit the exudation-cells much more oily, and further—what most concerns the proposition advanced—the fibrous tissue more indistinct, softer, broken up, and infiltrated with fat-granules, the nearer we approach to the yellowish-gray appearance of the third stage; which we thus can regard, in many respects, as simply a continuation of the second.

The opinion expressed with regard to the non-suppurative nature of most cases of gray hepatization derives further support from a fact which every pathologist has admitted—the extreme rarity of pulmonary abscesses following pneumonia. In over 100 *post-mortem* examinations, extended over a space of 20 years, Laennec only met five or six times with a collection of pus in

an inflamed lung. Andral, when he published the first edition of his *Clinique Médicale*, had seen but one. Chomel,[1] in 25 years, has met with but three. Stokes, too, although he conceives the rarity of the formation of pulmonary abscesses to have been overrated, bears testimony to the infrequency of their occurrence.

The difference, finally, between the matter, in cases of purulent infiltration and ordinary pus, has not escaped the attention of some of the most distinguished pathologists. We find, indeed, in Laennec, that the pus, when first exhaled, is "concrete or plastic, like the false membranes, and passes rapidly through different degrees of softening before it acquires its proper mucilaginous character. When it begins to soften, if the part containing it is pressed or scraped, it escapes under the form of a greasy substance, which a superficial observer might mistake for fat." Rokitansky admits the fact that the third stage does not always bear the aspect of suppuration, and states that the lung has a peculiar yellow appearance before it becomes entirely gray. Lallemand[2] even affirms that when the pus replaces the blood the lung has the appearance of a fatty liver; whilst Addison,[3] evidently struck by the frequent opaqueness and solidity of the matter, expresses his belief that the tissues of the lung are, in the third stage, reconverted into albumen, or undergo, to use his own term, albuminization.

Gradual Changes in the Exudation and Tissue in Pneumonia.—Now, let us attempt, according to the views advanced, a *rationale*, to account for the successive steps of the degeneration of the exudation and of the tissue. In pneumonia, we have an arrest of circulation in the bloodvessels going to the inflamed part, and as a consequence defective nutrition, which is admitted to be one of the most powerful causes of atrophy and fatty degeneration. The morbid condition progressing, interferes at the same time with the supply of oxygen, and changes, as the microscopical examination proves, the character of the normal cells first, and then of the exudation, by converting (to follow the generally received chemical hypothesis of fatty changes) their nitrogenized basis into fat. As the hepatization advances, the same causes operate on the walls of the air-cells and true tissue of the lung, and permit them to become gradually more granular and to become atrophied; which process is accompanied by softening, and, as may be readily seen, frequently by a fatty substitution. If the second stage end in recovery, the effused and degenerating matter is expectorated, and the tissues, which have scarcely commenced to be involved, restored to their natural condition. If, however, the morbid process runs on, it produces a complete state of softening and disorganization of the textures, while sometimes, even in this last stage, a fresh, dense infiltration may occur into the

[1] Dictionnaire de Médecine, vol. xxv. p. 151.

[2] Recherches Anatomico-Pathol. sur l'Encephale, etc.

[3] On Pneumonia, and on its Diagnosis, in vols i. and ii. of *Guy's Hospital Reports*. 1843 and 1844.

air-cells of a portion of the lung. In cases of recovery from a far advanced second stage, or from the third stage—the possibility of which is proved by Stokes[1] and Andral[2]—loss of tissue and depressions would probably be observed in the lung. What the minute appearances accompanying these changes are, I am unable to state, as I have never had an opportunity of examining lungs in this condition, and as I am equally unaware of any examinations of the kind having been recorded.

Before bringing this paper to a conclusion, a few questions of great interest, and which have a direct bearing on the subject, remain to be considered. Does the deposit in pneumonia always undergo this degeneration, or does true suppuration ever occur? Are the different terminations of the inflammatory process owing to the kind of lymph deposited? And again, may any variety of exuded lymph give to the eye the peculiar yellowish appearance of the third stage, without having been preceded by red hepatization?

Now, with regard to the *first* question, it is undoubted that the exudation into the lung may be transformed into pus, without any other degeneration of the inflammatory product occurring. For, although the majority of lungs, in the so-called third stage, do not, as stated, present any evidences indicative of suppurative action, we also meet with others in which there is no appearance of any other process. The air-cells are then observed to be filled with pus-corpuscles and molecular masses, whilst the tissues themselves, partly perhaps from admixture with the purulent fluid, are in a state of complete degeneration. Yet these cases are not the most frequent, and are probably such in which the disease occurs most acutely and in depraved constitutions, thus causing the lymph to be rapidly transformed into pus-corpuscles.

The *second* inquiry is not easily answered, as opportunities for examining the effusion, when it first occurs, are extremely rare. The lymph, in most cases of pneumonia, is, as far as can be ascertained, of a mixed character, both fibrinous and corpuscular. Reasoning from analogy renders it not improbable that the more the latter elements abound, the greater would be the tendency of the exudation to a complete and rapid fatty degeneration, or even, in individual cases, to a suppurative process; the more the former, the increased liability to permanent indurations: but any opinion on this subject must, in the present state of our knowledge, be extremely hypothetical.

The *third* question, finally, demands an affirmative answer, as I have met with specimens in which the lung, in "gray" hepatization, was firm, yellowish, and granular, and exhibited, under the microscope, no elements indicative of true suppuration, and but slight traces of any fatty degeneration, but, on the contrary, distinct nucleated cells of varying shapes, a solid blastema, and protein granules. (Fig. 6.) Lungs in this condition are heavy; evidently enlarged; the air-cells distended; and the yellow appearance remark-

[1] Op. cit., p. 269.

[2] Cours de Path. Interne, par Latour, tom. ii. p. 7.

ably uniform.[1] In no portion of the affected lobe is a transition stage from red hepatization visible, although a red crepitating tissue (the first stage?) may surround the yellow mass. Chemically examined, these lungs yield comparatively only a small proportion of fat; in one specimen I found not more than 7 per cent. The exudation causing this peculiar "albuminous" lung, or *yellow condensation*, if I may so term it, is probably a peculiar kind of inflammatory lymph, deposited rapidly, and allied to the croupous or corpuscular variety. It may break up into pus or into fatty molecules, and evidently gives rise to a distinct pathological species of consolidation, which has incorrectly been classed with gray hepatization, from which it may be distinguished by the greater density of the tissue, a more uniform extension and yellowish colour, and a different structural appearance, when microscopically examined. This yellow condensation may also sometimes occur near portions of lung-tissue already in an advanced state of inflammatory change. Whether it be owing to a peculiarly defective nutrition; to nervous depression; whether its metamorphosis bears any relation to the formation of tubercle, are questions which a more advanced pathology, and a more intimate acquaintance with inflammation and its products will have to solve; as, on the other hand, close clinical scrutiny may learn to connect these different varieties and terminations of acute pneumonia with different symptoms, different diagnostic, and, perhaps, with different therapeutical indications.

[1] For drawings of lungs representing the *yellow* condensation, as above set forth, see *Bock's Atlas*, Part 3d, Pl. I. Fig. 3, and Cruveilhier's *Anat. Pathol.*, Vol. ii. Part I. Livr. xxix. Pl. 5. The latter plate more especially depicts the lesions, as generally observed; it is, at least, the best delineation of the condition referred to, which I have met with in the current works upon Pathological Anatomy. Cruveilhier describes the pulmonary tissue, in the case from which the drawing was taken, as very heavy and dense, and calls the affection "concrete suppuration" terminating a subacute pneumonia. Dr. Hodgkin, in his Lectures on the *Morbid Anatomy of the Serous and Mucous Membranes*, London, 1840, classifies this "yellow" condensation as a *non-plastic* variety of pneumonia; a name adopted on account of the general properties of the lymph deposited. He is, as far as I am aware, the only writer who does not confound this form of hepatization with purulent infiltration. Yet even he does not indicate sufficiently closely the minute appearances of the lung-tissue in these different conditions, and this may explain the fact, that his divisions of pneumonia have been so generally overlooked.

PLATE III.

Fig. 1.

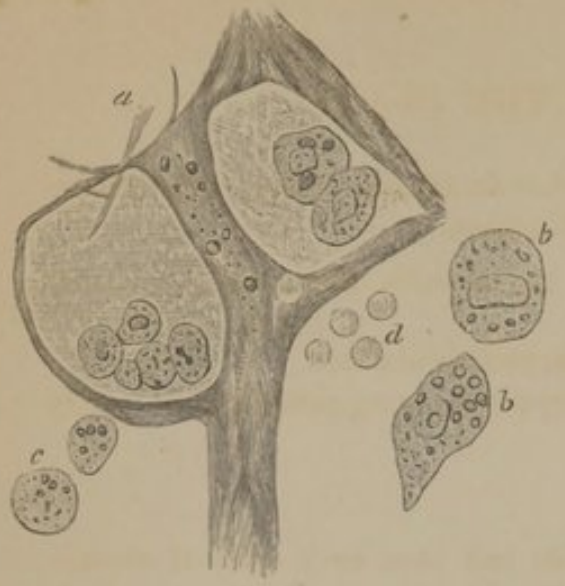


Fig. 3.

Fig. 2.

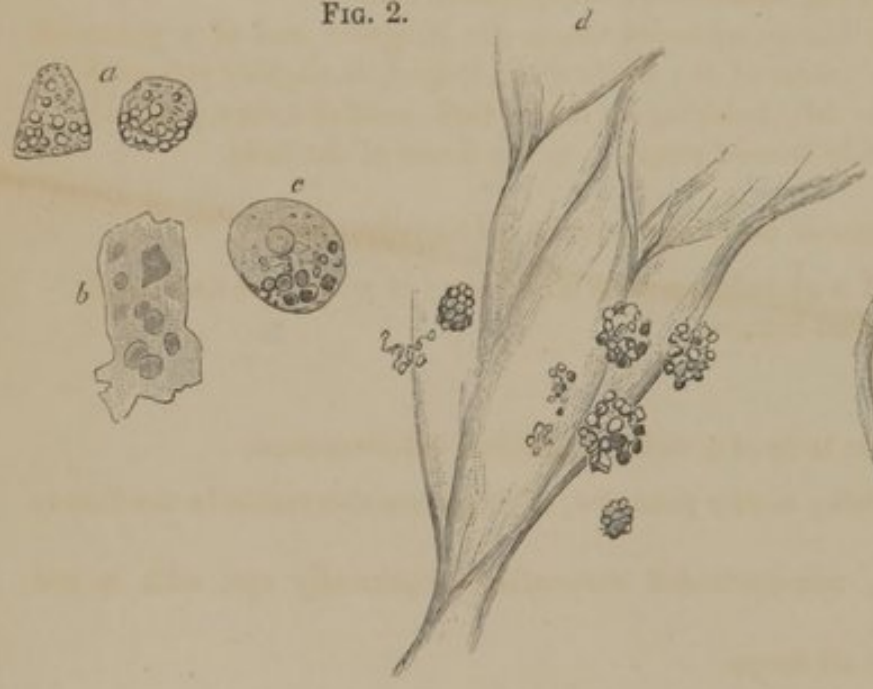
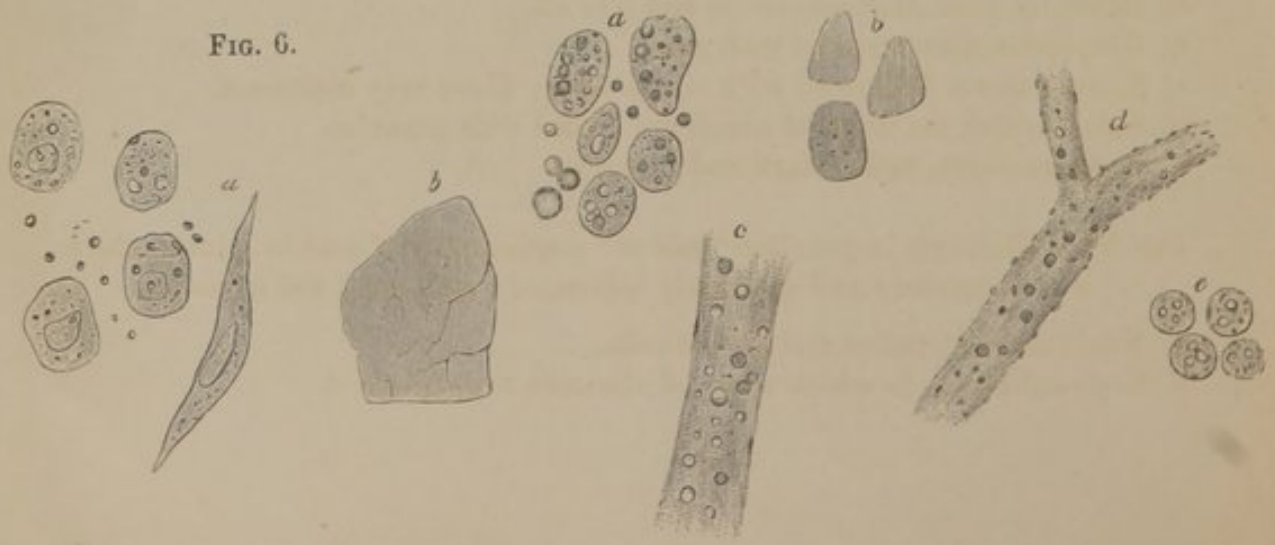


Fig. 4.

Fig. 5.

Fig. 6.



DESCRIPTION OF THE PLATE.

FIG. I.—Drawing taken from a lung in rather an advanced stage of inflammatory engorgement, magnified 430 diameters.

a. The fibrous tissue forming the walls of the air-cells, distinct and inclosing a few granules.
b. Very granular epithelial cells of bronchi and air-vesicles.
c. Small nucleated cells and exudation-corpuscles, lying partly within, partly outside of the air-vesicles.
d. Blood-corpuscles.

FIG. II.—Elements observed in lungs that had been in a state of chronic hyperæmia.

a. Epithelial cells being transformed into granular cells.
b. Flakes inclosing blood-corpuscles which are irregular and of a yellowish colour. The colour of the whole mass, indeed, is slightly yellowish.
c. Blood-corpuscular cell, breaking up into a dark, reddish-brown pigment.
d. Situation of freshly formed pigment, in the tissue of the lung.

FIG. III.—Appearance of the lung-tissue in *red* hepatization.

a. Air-vesicles filled with inflammatory cells, some of which are nucleated.
b. Concrete albuminoid masses.
c. Granule-cells.

FIG. IV.—Pneumonic lung of a child, magnified 430 diameters.

a. Air-vesicles inclosing mostly granules; oil drops are observable in the fibrous walls.
b. Irregular, small, non-nucleated corpuscles occasionally met with in red hepatization.
c. Granule-cells and oil drops.

FIG. V.—Elements found in the lung in *gray* hepatization.

a. Granular exudation-corpuscles and free oil.
b. Corpuscles when treated with ether.
c. Fibrous tissue infiltrated with oil-globules; fibres very indistinct.
d. Small vessel, the walls of which are coated with granules.
e. Pus-corpuscles, rather dark and granular.

FIG. VI.—Elements in peculiar "yellow" condensation, found in a lung which was completely and uniformly infiltrated; magnified 480 diameters.

a. Nucleated corpuscles and fibroid cells.
b. Basis-substance in which many of the cells are imbedded.