REMARKS UPON A

CASE IN WHICH THERE WERE EVIDENCES
OF DILATED BRONCHI,

AND WHICH WAS COMPLICATED BY OCCASIONAL
EXPECTORATION OF THIN, FIBRINOUS, CUP-SHAPED,
FALSE MEMBRANES.

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JOHN CLARK, aged 35, bricklayer, has always lived in Colchester, and has never had any serious illness before the commencement of his present malady. He has never had croup nor chronic cough, and he states that he has always lived steadily and soberly. Six years ago he had a bad cold, which confined him to his bed for five or six weeks; he has distinct recollection of having suffered from dyspnoea and difficulty of speaking, but antiphlogistic remedies do not appear to have been used with any degree of severity. A month after his convalescence, during which his lung symptoms appear to have left him, he was suddenly seized with pain in the right mammary region, cough supervened, and during a paroxysm of it he expectorated a light-coloured fibrinous mass, cup shaped, and much resembling in thickness and shape, the top of the thumb of a good-sized kid glove. During his illness, which lasted three months, he expectorated thirty of these false membranes; occasionally three or four were ejected simultaneously, but generally but one at a time was produced. For five years after this attack his health was good, neither cough nor expectoration of any kind were complained of, and he followed his usual avocations. At Christmas, 1849, he caught cold, and has not been well since. In April of the following year he experienced great pain in the right mammary region, it was attended by cough, considerable fever and dyspnoea. After suffering for several days repeated paroxysms of coughing, he suddenly spat up five of the before mentioned cup-shaped false membranes and immediately after a considerable quantity of scarlet blood. His urgent symptoms were immediately relieved, but in a few days they returned, and he then came under my care.

He is a tall well-formed man, thorax well developed, and measuring thirty-four inches in circumference beneath the clavicles. The general expansion during deep inspiration is deficient, his breathing being chiefly abdominal. The mammary and infra-axillary regions of the right side expand less than those of the left, but in no part is there perfect loss of motion.

Beneath the clavicles the percussion-sound is healthy, but on the right side, over the mammary, infra-mammary, and infra-axillary regions, there is considerable dullness. The left side of the chest is resonant throughout. The natural bronchophony is heard considerably to the right of the sternum, and in all parts of the chest, except in the right mammary, infra-mammary, and the infra-axillary regions, the true and healthy vesicular murmur is audible. In these regions there is a Babel

of stethoscopic sounds. Tubular breathing, almost at certain spots taking on an amphoric character; loud, sibilant, and occasionally sonorous rhonchi, are heard low down posteriorly. In a spot little external to the right mamma, there was loud and tubular breathing during inspiration; a sharp "click" preceded the expiratory effort, which was accompanied by a distinct "burr." (After a fit of coughing which caused the expectoration of a very large false membrane, the sounds altered considerably in this spot. The tubular breathing was better developed, but the click and burr sounds were no longer heard. The voice, which was previously transmitted tolerably distinctly, now jarred the ear and produced perfect pectoriloquy.) The variations of the intensity with which the voice is transmitted through various parts of the infra-axillary region are very curious. In some spots the voice hardly strikes the ear at all, in others there are various degrees of pectoriloquy. There is no gurgling to be heard, and the expectoration is mucoid. He suffers slightly from dyspnoea and cough, but is never troubled by foetid breath or disagreeably tasting sputa.

The cup-shaped false membranes vary in size from that of half a pigeon's egg to that of half a filbert. Their thickness is tolerably uniform, being about one-sixth of an inch; of a pale straw-colour; they are singularly elastic and resistant to the finger, their cohesion being as great as that of a pericardial false membrane of some weeks standing. Pure fibrin appears to be their principle component. The microscope discovered a few of the colourless cells of the blood, a few blood discs and numerous refractile granules, which, when aggregated, form the exudation corpuscle of Glüge.

These cup-shaped membranes were shrunk by the influence of dilute alcohol. The sputa usually expectorated, consisted of a glairy albuminous fluid holding ciliated and pavement epithelium cells, in mechanical suspension. He was ordered low diet Pil. Hydrarg., gr. iij.; Extr. Coloc. Co., gr. v. Fiat pil. ij., o. n. sum.—R. Potass. Bicarb., gr. v.; Tinct. Hyoscy., min. xv.; Mist. Camph., oz. j., ter die.

May 11th.—Is better; stethoscopic signs the same. No expectoration. Pergat.

14th.—Has suffered from pain in the axilla for some hours. He has considerable dyspnoea, slight cough and much anxiety of countenance. No breath-sound is to be heard in the greater part of the infra-axillary region of the right side, and in those spots where tubular breathing was heard, neither it nor any other sounds any longer exist. Believing that one of his larger bronchial tubes was plugged up by one of the false membranes, I was about to order him an emetic, when a paroxysm of coughing set in, and five fibrinous cup-shaped membranes were coughed up. The tubular breathing and breath-sounds returned and were audible in the infra-axillary region, immediately after the expectoration of the membranes. In the evening he coughed up a little scarlet blood, and complained of much pain in the chest. Cont. Mist.—R. Pulv. Opii, gr. $\frac{1}{2}$; Plumb., Acet., gr. ij., sextis horis formâ pil.

15th.—Is better.

16th.—Hæmoptysis stopped. Bowels constipated. Cont. Mist. Omit. Pil.

R. Hyd. Chlor., gr. j.; Pulv. Rhei, gr. x. Ft. Pil. iij. statim sumendæ.

17th.—Very much better. Bowels relieved. From this time he progressed towards health, gradually lost his cough, got flesh, and soon had appetite for full diet.

June 11.—His chest was examined before his discharge from the hospital. The dulness on percussion continues, but is more circumscribed; the tubular breathing is lessened in extent; all rhonchi and wheezing sounds have disappeared; pectoriloquy is audible here and there in the infra-axillary region of the right side, when a stethoscope with a small end is used.

August 1st.—Is in perfect health; the physical signs are still the same; he has neither cough nor expectoration.

It would appear, then, that our patient, a man of sober habits and apparently without any predisposition to chest affection, caught cold about five years ago and suffered for some weeks from bronchitis and some inflammatory attack about the larynx. A month after his convalescence he was seized with pain in the right mammary region, and during a fit of coughing expectorated a cup-shaped false membrane; his cough continued to annoy him for three months, and he occasionally expectorated three, four, or single false membranes, all cup-shaped and of different sizes. After the cessation of these symptoms he enjoyed nearly five years good health. At Christmas he caught cold, and after it had remained on him for four months he had another attack of pain, dyspnoea, fever, and cough, and expectorated numerous false membranes and scarlet florid blood. At the present time he is in good health, has neither cough, dyspnoea, nor expectoration of any kind. The physical signs determined that the lung in the mammary, infra-mammary, and infra-axillary regions of the right side was suffering from an altered state of its air-passages. Tubular breathing, almost at certain spots, taking on an amphoric character, was heard, and the voice jarred on the ear in those spots so as to give the notion of defined pectoriloquy. The normal bronchial sounds were heard far to the right of the sternum; and large sonorous rhonchi were audible low down in the infra-axillary region.

From these signs it is to be inferred that the main bronchial tubes of the lower and middle lobes of the right lung were more or less dilated; and that the more remote tubes were also dilated and occasionally sacculated. We have good evidence to the effect that the dilated bronchi were, and are now, sacculated. The cup-shaped fibrinous false membranes were evidently casts of the sacculi, and were analogous to, and probably formed in the same manner as, those tubular bronchial false membranes which occasionally are seen by everybody. The false membranes occasionally were unable to pass through the lesser dilated bronchi, and impeded the passage of air into the air vesicles and bronchial tubes remote from the obstruction. It is to be noticed also that there was considerable disturbance

of the bronchial mucous membrane during the illness, characterized by the expectoration of false membranes; for much mucous was secreted; and occasionally after the violent expulsion of the cup-shaped casts of the sacculi some blood was brought up. It would appear that the mucous membrane of the bronchial tubes secreted its normal product, whilst the sacculated dilatations eliminated a layer of pure fibrin. Before remarking upon the pathology of dilatations of the bronchi, it must be noticed that the stethoscopic signs of increased calibre of the bronchi, and of occasional sacculated projections from their interior, are still in existence, although the man's health is good, and he does not suffer from the cough and stinking expectoration of those affected with dilated bronchi.

REMARKS.—The case recorded above presents several points of interest, amongst which those of dilatation of the bronchial tubes and fibrinous exudation from mucous surfaces are pre-eminent. Dilatation of the bronchial tubes, or bronchiectasis, appears to have escaped the notice of all writers before the great Laennec, whose history and pathology of the disease has been the theme upon which the variations of Andral, Stokes, Corrigan, and Hasse, of Zurich, have been founded. It appears to be a disease of tolerably frequent occurrence, and is well studied in children who have died from whooping cough; sufficiently rare for many years after puberty, it is occasionally found in the bodies of those dying from chronic lung diseases. The diseases upon which bronchiectasis is consequent are—chronic catarrh, bronchitis, tubercular disease, and pleuro-pneumonia. It appears to assume three forms; the first which I shall describe is a sequence of the last-named disease; the two other varieties follow after the three first diseases.

The first form of dilatation is that which is usually a sequence of pleuro-pneumonia; in it numerous bronchial tubes, sometimes every one in a lobe, are dilated along their whole extent; and the dilatation is greater towards the extremities of the tubes, where their structures are less dense and unyielding; the calibre of the tubes is greatly increased, occasionally to eight times its usual extent. The mucous membrane of the affected tubes is hypertrophied, thickened, and turgescient; it is occasionally elevated into highly vascular folds, and is usually covered with a thick muco-purulent secretion. The lower lobes of the lungs are those affected by this form; and when incisions are made into them the parallelism of the cylindrically-dilated bronchi is singularly evident. The proper parenchyma no longer exists; the air-cells in the neighbourhood of the bronchial tubes, which are nearly in close apposition, are obliterated, and a pale, flabby, soft tissue remains, being impervious to air and incapable of inflation. The dilatations end in blind sacs; the small twigs immediately supplying the intercellular spaces and the remote air-cells are obliterated. The cause of this obliteration is partly mechanical and partly attributable to vital changes. An old standing pleurisy may be cured, the fluid may be absorbed; but the lung may be found to

be unable to fill up the space occupied by the fluid, its power of expansion being lost on account of the alterations in the air-cells, consequent upon the pressure of the effusion and the loss of the exercise of their functions, One of two things, or both, must now occur; either the walls of the chest must be dragged in, or the calibre of the bronchial tubes must increase and gradually tend to the filling up of the vacant space, or both of these changes may happen. Inflammatory action in the mucous membrane would accelerate the bronchial dilatation by its paralyzing influence upon the muscular fibres of the tubes. In long standing pneumonia the destruction of air-cells, consequent upon their infiltration by exudation of fibrin, is the first step towards the production of cylindrically-dilated bronchi; the subsequent contraction and atrophy of the infiltrated tissue produces a space which is required to be filled up by the neighbouring healthy air-cells, and if there be none, by the bronchi; in the one case emphysema, in the other bronchitis is the result. This kind of bronchiectasis is accompanied by excessive secretion from the mucous membrane of the tubes, generally speaking of a viscid tenacious mucus, and Laennec ascribes the production of the disease to the efforts employed for its expulsion. There can be no doubt but that when the remote causes of the dilatation are at work, such an excitant would greatly enhance the progress of the disease.

Hasse gives the following summary of the two other kinds of bronchiectasis; the first of these "exhibits a single point of one or another bronchial tube, mostly of the third or fourth order of magnitude, dilated into a cavity of more or less regularly spherical shape. In the majority of cases the walls are at the same time attenuated, the cartilages and the fibrous texture being for the most part stretched asunder, if not made to disappear altogether, and the mucous membrane changed into a thin, smooth, almost transparent pellicle. In the second form we find a series of these cystic dilatations, occupying the course of one and the same bronchial tube, which in the intervals retains its natural width. The attributes of this form, and especially the manner in which it originates, prove it to be but a variety of the first form; for in neither instance does the dilatation constitute the extreme end of a bronchial tube—that is to say, a *cul-de-sac*, but either allows the cylinder to issue from it again, in its normal calibre, for further distribution, or else opens with small orifices into the lesser bronchi beyond." These two kinds of bronchiectasis are the result of long continued bronchial inflammation; they are not attended by that destruction of the air-cells which is found in the form described as following pleuro-pneumonia, for the degeneration of the vesicular structure is only found in immediate contact with the cystiform dilatation. There must be a peculiar idiosyncrasy in those who suffer from dilatation of the bronchi, for it does not follow upon hundreds of cases of chronic bronchitis, or upon the great majority of those diseases said to tend to its production. In the ordinary course of inflammation of the mucous membrane of the bronchial tubes, the capillaries in the

areolar tissue beneath the basement membrane, which supports the epithelium, become distended, the current of blood is retarded, and the calibre of the vessels is irregularly increased; their office is suspended, they no longer give to the epithelium cells the wherewithal to produce the natural mucus. The air passages are speedily stripped of their epithelium and further changes ensue, exudation of the blood from the walls of the capillaries of the colourless pleura occurs, (occasionally blood corpuscles are set free,) owing to the aberration of the nutrition of the sub-basement tissues, the growth of the epithelium is rapid and irregular, and in those places where these cells are not reformed, a cell growth of a lower nature is produced—pus; or, owing to some peculiarity, no cell forms are produced, and the fluid whence they ought to have been formed is simply poured out, it is by nature spontaneously coagulable and consists of fibrin and more or less albumen. It is tolerably evident that an exudation of such a fibrinous mass would tend to plug up a smaller bronchus, and that it would be assisted in doing so by the mucus and pus also secreted. A certain amount of bronchial tube would be then exposed to the distending effects of muscular action upon retained air, and bronchiectasis may ensue; or the plug might simply cause the collapse of the neighbouring air-cells and consequent partial dilatation of a near bronchial tube.

These mechanical cases are, however, only the stimuli to a series of perverted vital actions; simple distension by accumulation of secretion will not of itself cause dilatation of gland-ducts; for instance, it merely acts as a stimulus to an irregularity of growth; the dilatation may be accompanied by hypertrophy or by atrophy of the structures implicated. In the case before us there was a series of sacciform dilatations, and the calibre of the bronchial tubes between the dilatations was also increased, so that there was a combination of the varieties of the bronchiectasis—an union of that variety characterised by simple cylindrical, and of that characterised by a series of cystic dilatations. It is tolerably evident that in this case the dilatations were occasionally cylindrical, and that at other spots the dilatation did not affect the whole bronchial calibre, but that a lateral dilatation occurred constituting a greater or less lateral cul de sac. The physical signs and the negative evidence proved that the fibrinous, cup-shaped, false membranes, came from the lateral sacciform bronchial dilatations, and the long attacks of bronchitis from which the patient suffered accounted for the formation of the alterations in the calibre of the bronchial tubes. It would be out of place to do otherwise than simply mention, that bronchitis is met with, in all its forms, in the lungs of those dying from phthisis pulmonalis, and that it occasionally effects a cure.

The subject of bronchial false membranes—polypi as they were formerly called—is one of great interest; for an excellent sketch of the bibliography of the disease I must refer to a paper by Dr. Ranking in this journal, (June 13th, 1849, p. 316,) but I am not aware that a case in which the false membranes were cup-shaped has been recorded, unless the case of Phricides, mentioned

by Hippocrates, in which *γαλακτώδια* were expectorated, was one. We are all familiar with the false membrane of croup, and the exudations of certain forms of laryngitis, such as occasionally occur in measles and scarlet fever. False membranes are also found, under certain circumstances, in the fauces, on the schneiderian membrane, in the œsophagus, stomach, certain portions of the intestinal canal, biliary passages, tubuli uriniferi, ureters, urinary bladder and urethra. Exudative processes in the uterus, fallopian tubes, and vagina, are constantly presented to our notice.

Were we to say that all mucous membranes are capable, under certain circumstances, of producing false membranes, we should be pretty well correct. They are most commonly seen in the throat and air passages. Rokitsky gives an excellent description of the disease as found in the other mucous membranes. But as Dr. Ranking remarks at the conclusion of his capital paper:—"But why fibrinous rather than mucous secretion should take place, remains a mystery." In one organ, the kidney, simple inflammation invariably produces casts of the uriniferous tubes—transparent, fibrinous, microscopic effusions, which exactly correspond with the inside of the affected uriniferous tubes—in no other organ is this the case. There is more required than that aberration of nutrition which we call inflammation, to replace the mucous secretion by a fibrinous exudation.

An experiment by J. A. Albers, does not really, although it seems to do so, militate against the opinion. He excited artificial inflammation in the air-passages of animals, and produced fibrinous false membranes. It must be noticed, however, that brute pathology is hardly to be identified with, or to be argued upon like, that of man; and the manner in which he produced his inflammatory effects, tended to cause great effusion of blood into the parenchyma and into the smaller bronchi. Its coagulation would account for what he describes as spurious membranes. It is also notorious that acute bronchitis, or in fact any simple inflammatory attack of a mucous membrane, excepting in the kidney, is very rarely indeed accompanied by the formation of false membranes. But during certain epidemics of measles, and not in those characterised by great malignancy, tubular expectoration from the air passages is common. There is a note to a passage in "Hasse's Pathology":—"The most strongly marked case I ever met with of tubular expectoration from the trachea and bronchi, occurred during the prevalence of a very mild epidemic of measles." In mild cases of scarlet fever, however, membranous exudations on the fauces and larynx do not occur; they are only found in cases characterised by their peculiar malignancy. The tough plugs found in mucous canals after their exposure to corrosive agents, are not fibrinous exudations, although if these masses of mucus, coagulated serum, and blood, be removed, and the patient recover from the immediate effects of the injury, the damaged mucous membrane is very liable to produce fibrinous layers instead of mucus, (epithelium, &c.,) for some time. Many forms of croup and of exudations in the œsophagus, stomach, and intestines, never occur as primary affections, but

according to Rokitsky as *sequelæ* of exanthematic processes, in variola, in typhus, in the absorption of pus into the circulation, and particularly in puerperal inflammation of the uterine veins. He has also observed, with great care, the varieties of exudation in the uterus in puerperal fever, and describes with his usual minuteness the exudative processes in the mucous membranes consequent on the puerperal poison.

It would appear, then, that certain diseases which originate by the absorption of a poison from without, or by the formation of noxious matter within a cavity, and their consequent absorption, predispose to fibrinous exudation from mucous membranes, and that certain poisons produce false membranes in the bronchial and tracheal mucous membranes, whilst others affect the genito-urinary and digestive mucous tracts.

Where we cannot trace the presence of a poison, and where there are structural changes in the mucous membrane affected with the fibrinous exudation, it must be admitted that structural changes tend to the production of false membranes; but the absence of these in most cases, where the calibre or the nature of the mucous tube has undergone a change, forces us to believe, that either idiosyncrasy or a peculiar state of the mucous membrane with which we are unacquainted, has much to do with this production.

Cases occur in plenty where idiosyncrasy appears to be the cause of the replacement of a mucous by a fibrinous secretion.

The circumstances under which mucous membranes pour out a fibrinous product, which coagulates and forms a false membrane, exudation or polypus, are when the system is influenced by—

1. Idiosyncrasy.
2. Idiosyncrasy; structural changes.
3. Morbific agents in the circulation, (pus, typhus, measles, scarlet fever, poisons.)
4. Simple inflammation.

It is, of course, implied, that there is an exciting cause to enable the two first influences to act, as hyperæmia, of greater or less intensity. The toxic agents carry with them the power of inducing certain kinds of inflammation.

I may here remark, that in my experience cases in which bronchial false membranes, (I allude especially to those which plug up the various ramifications of a third or fourth rate bronchus,) have been expectorated, run a very peculiar course, and rarely terminate fatally. Death generally is caused by the effects of occlusion of the affected bronchi, and need not happen for many years, the patient being carried off by emphysema and dilated right side of the heart. Intensity of inflammation does not seem to have any thing to do with the production of false membranes, nor can we take refuge under the explanation of its occurring as an effect of some peculiar irritation, for by doing so we only transfer our difficulty.

We know nothing concerning the state of the blood in this disease, and cannot imagine that even a highly fibrinous condition of it could produce aught but a tendency to the exudation. From what I have noticed,

I am inclined to believe that before a fibrinous exudation can occur, the epithelium of that part of the mucous membrane covered by it, is destroyed, and that the basement membrane, and the vessels of the areolar tissue beneath it, are all that remain of the mucous membrane. The areolar tissue is invariably infiltrated with serum, and the whole tissue is softer than usual. This is notably the case in the exanthemata. Occasionally the fibrinous exudation extends from one spot, whence it arises, and covers the surrounding mucous surface.

As a rule, when the epithelium is removed from a mucous membrane, the vessels of the areolar tissue beneath the basement throw out the colourless blood plasma as usual, and pus cells are formed for a while in it; subsequently the normal mucous cells are developed. We must account for the production of false membranes instead of mucus or pus, by supposing there is an inability on the part of the blood plasma to develop cell life; it coagulates rather, and forms a fibrinous mass. The circumstances producing the above inability, and tending to the primary destruction of the epithelium, are those, then, which I have mentioned as being of four kinds.

In the case recorded at the commencement of this paper, the formation of false membranes covering altered tracts of mucous membrane was constant; and as now, none have been expectorated for many weeks, it may be supposed that epithelial cell growth is formed from the blood plasma, and that the sacculated dilatations are now covered by a healthy mucous membrane. Nature seems to have done everything in this case; the few doses of acetate of lead were required to control a slight hæmorrhage from the bronchial tubes, excited as much by the presence of passing false membranes as by the exertions employed to get rid of them.

CASES OF OVARIAN DROPSY.

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Read at the Quarterly Meeting of the Bath and Bristol Branch, Dec. 19, 1950.

THE attention of the medical profession being at this time much directed to the subject of ovarian disease, and many of the leading men amongst us appearing still to differ in opinion as to the extent of surgical interference admissible in such cases, I consider it the duty of every surgeon to record those cases which have occurred in his practice, where operations have been attempted for the radical cure of ovarian dropsy, and to report faithfully the result of each case, whether favourable or unfavourable.

Influenced by this consideration I have thought it right to lay before this meeting a brief account of four cases of ovarian dropsy, treated by me since October, 1849, and to detail a mode of operating which differs, in some important respects, from any hitherto described,

and which, unless I am mistaken, will be found to be attended with less risk than any hitherto made public.

Previous, however, to describing this particular operation, I will briefly notice my first case, in which a cure was effected by the formation of an oviduct, (as this process has been called) by means of a caustic issue. The case stands thus in my note book :—

Elizabeth Antrobus, aged 38, spinster, consulted me in October, 1849, with an ovarian tumour, which had reached an inconvenient size, and which she wished to have tapped. I had tapped her twice before—the first time in October, 1840, when three and a half gallons of opalescent dirty-looking fluid were evacuated. The cyst refilled slowly, not requiring to be emptied again till May, 1845, at which time a smaller quantity, (not quite two gallons) of similar looking fluid was drawn off. The cyst again filled slowly. An interval of four years and seven months had occurred between the first and second tapplings, and now an interval of four years and five months passed before she again sought relief in that way.

As her health had been remarkably good during the whole of both intervals already named, much better indeed than it had been previous to the first tapping, I thought it a fair case to try to effect a radical cure, and, having been favourably impressed by the reasoning adduced by Dr. Tilt, in recommending the treatment of these cases by caustic issue, I proposed that proceeding to my patient, and having obtained her consent, proceeded to carry it into practice on October 29th. Selecting a spot about two inches below the umbilicus, in the median line, a stick of potassa fusa was rubbed upon it, over a space about the size of a shilling. A portion of Vienna paste was then applied to the same surface, and confined by lint and plaster, and allowed to remain until the next day, by which time an eschar was formed, not much less than half a crown in size. The eschar proved very deep, and did not require to have the caustic renewed, but it was eight weeks before it separated sufficiently to discharge the water. At first no injection of any kind was used, but in three or four weeks from the evacuation of the water the discharge became purulent and foetid, and my patient's health declined so rapidly that I feared I should lose her. Under a generous diet, with quinine internally, and the repeated injection of the cyst with warm water, she rallied, after having lain a month or six weeks longer, in a very precarious state. At that time a weak solution of iodine, (one drachm of the compound tincture to six ounces of water,) was occasionally used, without producing any ill effect, and a portion of gutta percha tubing was fitted to the opening of the wound. This was fitted with a wooden plug, so that the discharge could be drawn off at stated times. Before this the wound showed a disposition to close permanently, and required to be opened by a probe, to evacuate the fluid that accumulated, the patient always suffering until this was done. From the time the gutta percha tube was introduced, and the iodine injection used, the cyst began to contract, and the patient to improve