nonce the drug was discontinued. Three weeks had again elapsed since paraldehyde was last given, and, being unable to use sulphonal owing to the extreme after-depression and misery, I determined to again give paraldehyde a trial, but this time in repeated small doses. Beginning at 6 P.M., 30 minims were given, and this was followed at 7 P.M., 8 P.M., and 9 P.M. by 20 minim doses. After the second dose the patient was restful, lying quiet for half an hour though not sleeping. After the third dose the excitement and restlessness began to appear, and when the fourth had been given these were so great that the patient's friends were unwilling to have the drug pushed further. This state of matters lasted for an hour, after which the patient lay quiet for nearly four hours, during which time he had occasional and short intervals of sleep. This result I considered unsatisfactory and did not again use paraldehyde in this case. It is to be noted that at no time during the use of the drug, even when given in such considerable quantity, was there any diarrhoea—an after effect mentioned in the Year Book of Treatment for 1892 (p. 88) as sometimes occurring. From its non-depressing effects upon the heart's action, and since it seems to have diuretic properties, paraldehyde may become a useful hypnotic in cardiac cases where there is dropsy. Though I was induced to give up its use in this particular case I have not lost faith in the drug. The insomnia here was most intractable, and of all the hypnotics used, with the exception perhaps of opium, paraldehyde on the whole gave the most satisfactory results, and can be used where it would be decidedly risky to push the older drug. To sum up, then, it seems to me that, so far as my limited experience of the drug goes, paraldehyde may be looked to as a fairly reliable and safe hypnotic, that its administration is followed by a well-marked stage of excitement, that it does not depress the heart's action, does not interfere with the appetite or digestion, possesses probably diuretic properties and induces a sleep which is described as "refreshing."

Beith, N.B.

A DEMONSTRATION OF ACUTE PNEUMONIA, ITS FORMS AND ISSUES.¹

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ACUTE pneumonia in its pathological aspect is commonly regarded as the summation of a more or less definite and constant series of changes resulting from generalised inflammation in the lung. Thus viewed it is held to be made up of four stages, those namely of engorgement, of red hepatisation, of grey hepatisation, and of resolution. Here is a standard to which the disease is continually being referred, a mould into which it is constantly being cast, but the wider our study of pathology the more liberal becomes our conception of the scope of morbid processes. The stereotyped image gets dashed to pieces as disease manifests itself in its protean aspects. To this broad principle pneumonia forms no exception, its histological features are no less diverse and inconstant than are its issues.

Clinicians well know the various courses which this affection may pursue. Sometimes hepatisation declares itself only to vanish quickly and resolution is promptly effected; in other instances it proceeds wave-like over the organ, closely followed, it may be, by the symptoms of its breakdown. Or it may exhibit a faltering, a persistent, or even a recurrent character. And although the duration of the disease is by no means the measure of its fatality, the latter element will not infrequently yield its readiest solution to a microscopical study of the affected organ.

Under a microscope is placed a specimen of hepatised lung obtained from the body of a young and previously healthy adult. It is the so-called "red" hepatisation, the air-cells being filled with a meshwork of well-formed fibrin, which adheres with considerable tenacity to the alveolar walls. Scattered throughout this fibrin are variously sized epithelial elements, a few leucocytes and a fair proportion of red blood-cells. The alveolar walls themselves reveal but little alteration from the normal, and were attention directed to these alone it would probably be affirmed by most that they were in a state of health. A fibrinous network is further seen to exist in the lymphatics of the

adventitious structures of the organ, but beyond this there is little of note to be found. The next specimen reveals a very different state of matters. Herein the fibrinous network is still so far apparent, but it is no longer in contact with the alveolar wall. It appears to be disintegrating into a finely granular mass, which is separated frequently by a somewhat crescentic space from the wall. In this granular mass is a considerable sprinkling of fatty corpuscles with disintegrating nuclei, and somewhat similar though less degenerate corpuscles are likewise seen in fair numbers in the crescentic space. Closely inspecting the alveolar walls, it is found that they are lined with round and spindle-shaped bodies which are evidently the source of many of the corpuscles lying outside. Here, in fact, is seen the process of resolution in the hepatised lung. Very different, again, is the third specimen. It is a section of hepatised lung from an elderly and otherwise unhealthy man. The alveoli are densely packed with cells resembling leucocytes, amongst which not a vestige of fibrin is to be seen. In both lungs the hepatisation was everywhere uniformly thus. The walls of the air cells are, however, intact and their bloodvessels patent and containing blood. In this case also the alveolar walls are seen to be lined with round and spindle-like cells. Between these varieties many gradations are to be seen. Fibrin and corpuscles vary exceedingly. The earlier exudation may be chiefly fibrinous, and if resolution does not take place after the manner described the air cells will become crowded with cellular elements, replacing the fibrin. Sometimes, again, the corpuscles, shed early with the fibrin, are in considerable numbers, when they may exhibit the characters of true pus cells. Or it may be that the cellular element is the dominant one from the commencement, or even the sole element, forming the "yellow hepatisation" of Da Costa.

From the consideration of these phenomena I am led to recognise a typical resolving pneumonia, represented in the three stages of engorgement, hepatisation and resolution; and likewise a pneumonia whose leading feature is a corpuscular hepatisation terminating either in resolution or in destruction of the lung. In the first case we have resolution accomplished without the intervention of this corpuscular consolidation. The fibrinous exudation no doubt loses colour and becomes more cellular in the period of resolution, but this is distinct from corpuscular hepatisation. Hence the term "grey" hepatisation, though convenient, is otherwise objectionable and apt to mislead. The exudation in this speedily resolving pneumonia is identical with that obtained by Paget in the blisters of healthy individuals. On the other hand, in corpuscular hepatisation, the exudation resembles that obtained by the same illustrious observer in the blisters of cachectic persons and called by him "corpuscular lymph." The term "corpuscular" therefore seems to me to be the most authoritative definition for this form of consolidation. As to the light gained from a study of these exudations respecting the gravity of the disease, it may be said generally that when the disease proves fatal in the absence of corpuscular hepatisation we have to look to the severity of the constitutional symptoms, the blood poisoning, or the diminution of the breathing space in the lungs leading of course to failure of the circulation. When, however, the corpuscular lymph is abundant there is a local condition which in the absence of severe or sthenic symptoms is sufficient to cause a protracted resolution (and it is undoubted that this form of pneumonia may resolve) or else cause a destruction of the lung with its invariably fatal termination.

Regarding the derivation of the cellular elements found in the air cells in pneumonia, when these are abundant many of them have no doubt proceeded from the bloodvessels; but careful examination reveals—and this is particularly applicable to the stage of resolution—that a goodly proportion are derived from the epithelial elements lining the walls. Cornil and Ranvier, Rindfleisch, and others thus regarded pneumonia as terminating by a catarrhal pneumonia—a view which I cannot but regard as a travesty of inflammation. On the other hand, I believe that these corpuscles are merely the outcome of a superabundant growth of young epithelium, just as a similar overgrowth of new epithelium is formed on the surfaces of healing wounds.

Is this disease, then, to be regarded as a genuine inflammation? Inasmuch as no changes occur in the connective-tissue cells of the alveolar walls, the process can only be regarded as superficial, the local expression of a general disease. It is this absence of germination in the fixed tissue cells which is the sole anatomical distinction

¹ Substance of Remarks on Microscopical Preparations exhibited to the Pathological and Clinical Society of Glasgow, May 8th, 1892.

between acute pneumonia and acute broncho-pneumonia. I have elsewhere compared the pulmonary lesion of acute pneumonia to the joint lesion of acute rheumatism. As the former differs from broncho-pneumonia, so does the latter from acute synovitis. It is alleged—as for instance by Sturges and Coupland—that hepatisations of lung exist resembling that of acute pneumonia, which are in fact secondary to other diseases or own but a mechanical origin. But such cases are to be suspiciously regarded, and I am inclined to think that they are mostly genuine pneumonias, modified no doubt by the peculiar circumstances under which they occur.

Now as to the issues of this affection. Passing over destructive softening of the lung, abscess formation and gangrene, there remain two conditions which are no doubt rare and consequently ill-understood, but which are none the less highly important. One is somewhat vaguely termed “delayed resolution.” By this it is implied that dulness, râles and deficient breathing persist over a greater or less area of lung after the complete subsidence of all acute symptoms. This condition may be present for weeks or months and yet the lung may gradually “clear up” and become restored to health. This is an occurrence of considerable rarity, and, according to Wilks, in ninety-nine cases out of a hundred in which dulness persists after pneumonia it is due to pleurisy. In many cases there is no doubt empyema; but post-mortem, cases are likewise found showing persistent pulmonary consolidation, the air cells containing granular fibrin, mucous flakes and fatty cells, and the alveolar walls being lined with several rows of spindle cells. In such cases there is absence of interstitial changes, at least at first. If, however, complete recovery does not take place there remain two other possibilities. The hepatised area may break down into small abscesses and a form of phthisis is set up, or else the lesion contracts in area and cicatricial changes ensue, leading to a fibrous node, which becomes ultimately buried in emphysematous tissue.

The other issue of pneumonia to which I refer is in induration. Not, strictly speaking, in induration of the lung, but in induration of the pneumonic lung. The first step in this process is the germination (atypical to the disease) of the connective-tissue corpuscles of the alveolar walls. Next, the fibrinous exudation (never the corpuscular) becomes organised. To this form of induration I have applied the term “fibroid pneumonia,” for reasons which it is impossible here to discuss. Easily recognised at first, this remarkable transformation of the pneumonic products exhibits changes both of growth and of decay. To trace microscopically the evolution of fibroid pneumonia from its inception to its various terminations is a task of considerable difficulty. Outwardly indeed and to the naked eye this lung is sufficiently distinguished from Corrigan’s cirrhosis in its bulky character and the comparative absence of bronchiectasis. Further, it is found as an independent affection not consecutive to an acute pneumonia. But many cases of what is termed chronic pneumonia or interstitial pneumonia or Clark’s phthisis are in reality advanced cases of this fibroid pneumonia. No doubt this statement may be criticised and questioned, but it is none the less capable of proof, direct, continuous and complete.

Glasgow.

LONDON AND COUNTIES MEDICAL PROTECTION SOCIETY, LIMITED.—A meeting of the members of the medical profession in Hampstead and the neighbourhood was held on June 25th at 127, Fellowes-road, South Hampstead, to establish a division of this Society for the north-western district of London. The Council of the Association had, under their articles, large powers, of which they only retained such as were absolutely necessary to secure the stability of the Society; all other powers were transferred to the Branch Councils, which were thoroughly representative of their respective districts.

WEST RIDING MEDICAL CHARITABLE SOCIETY.—The annual meeting of this Society was held at Wakefield on the 14th inst. After a lengthy conference of the officers, the chair was taken by Dr. Wright, president for the year. The report stated the Society to be in a prosperous condition, with about 450 members, whilst the invested capital amounted to upwards of £20,000. The claims of twenty-four applicants were investigated and awards were granted for the year amounting to £830. The Society was originated in 1829 in Wakefield.

A Mirror OF HOSPITAL PRACTICE, BRITISH AND FOREIGN.

Nulla autem est alia pro certo noscendi via, nisi quamplurimas et morborum et dissectionum historias, tum aliorum tum proprias collectas habere, et inter se compare.—MORGAGNI *De Sed. et Caus. Morb.* lib. iv. Proœmium.

MIDDLESEX HOSPITAL.

TWO SURGICAL CASES.

(Under the care of Mr. HULKE.)

SURGEONS are, it is believed, without exception agreed that lacerations of the urinary bladder, implicating those parts of the viscus where a complete rent of all its coats involves direct outflow of the urine into the peritoneal sac, should be immediately closed by suture. Here every hour’s delay adds to the already great danger of the injury, and were it possible to diagnose a *partial* laceration, implicating the mucosa and muscular walls, but leaving the peritoneum untorn, it is suggested that the safest course would be to treat this similarly, since with the infiltration and softening of the injured coats which will certainly ensue there must be a great risk of the peritoneal coat giving way, and thus of a *partial* becoming a *complete* laceration. Unfortunately such early precise recognition of the true nature of the injury as would warrant instant surgical interposition is not in every case possible. The initial shock is not always great and where present is not pathognomonic, and it may soon lessen. The presence of urine, as is well known, is not in all instances so poisonously irritating to the peritoneum as to immediately excite inflammation of the serous membrane. Doubtless much depends on the quality of the urine. The analogy of cellular urinary extravasation proves this; thus a familiar instance is the rapid sequence of gangrene of the perineal tissues and scrotum upon the extravasation into them of urine contaminated with inflammatory substances and with products of decomposition, such as is not uncommon where the urethra gives way behind a stricture in concurrence with a chronic cystitis, whereas extravasation of normal urine—particularly the limpid urine of young persons—such as occurs with fractures of pelvis, occasions swelling, which for some time may differ but slightly from that of a simple œdema. Of course, if on introducing a catheter no, or very little, urine is found in the bladder, whereas on gently pushing the instrument a little further in a larger quantity escapes, this affords a strong presumption of a complete laceration; and this presumption becomes a certainty if on depressing the handle the vesical end of the catheter mounts and projects the abdominal wall over the pubis. These signs are, however, not in every case patent and obviously recognisable, even where a rent is complete. That this is a fair statement of the difficulties surrounding the diagnosis is illustrated by the following case. Here the bloody tinge of the urine raised a strong presumption of injury of the bladder, but the quantity of urine that the bladder apparently held seemed to contraindicate a complete rent; the early subsidence of the symptoms of great shock and the retardation of symptoms of peritonitis conspired to mislead and so led to the postponement of laparotomy, which, had the true nature of the accident been immediately perceived, would have been undertaken with a reasonable prospect of the patient’s recovery.

CASE 1. *Extensive laceration of the urinary bladder; laparotomy and suture of rent; death from peritonitis.*—C. E—, aged thirty-three, in the evening of Jan. 14th, 1892, in rough horse-play with others with whom he had been drinking, was butted sharply in the abdomen. He at once felt great pain and he was shortly after this brought to the hospital at 9 P.M. He had, he said, passed urine two hours previously. On reception into Founder ward the house surgeon noticed that he had an anxious expression, coldness of surface, a small, weak, slow pulse. He complained of severe pain in the lower part of the abdomen and of an urgent desire to micturate. The front of the abdomen was everywhere resonant. A catheter was passed without difficulty and some urine (roughly judged by the ward sister to be six to eight ounces), very slightly tinged with blood, was drawn