more closely than any other distinct dyspnoea, yet cases of heart disease are not so often confounded with asthma as are cases of bronchitis, and for these two reasons:—first, because with a great many people, especially in the lower and lower middle classes, bronchitis commonly goes by the name of asthma—an old man coughs and spits and wheezes, and tells you he is asthmatic. And secondly, because a great many cases of bronchitis are complicated with true asthma—bronchial spasm is an integral part of the condition; the patient has asthma not because he has not bronchitis but because he has bronchitis; his bronchial tubes contract because they are inflamed. The error, therefore, in such a case does not lie in the recognition of the asthma, but in the non-recognition of the bronchitis which is the immediate cause of the asthma, and the substantive disease.

The principal points of contrast between bronchitis and pure asthma are the following.

IN BRONCHITIS.

The typical sounds are moist.
Cough is always present.
The winter is the worst time of the year, often the only time.

Cause catarrhal.

Sputum more or less purulent.
Severe dyspnoea never evanescent.
Often ends in dropsy.

IN ASTHMA.

The typical sounds are dry.
Often there is no cough.
The hot weather of summer and autumn is the worst time of the year.

Cause emotional, dietetic, and various.

Sputum, if any, non-purulent.
The severest paroxysm may come and go quickly and suddenly.
If uncomplicated never produces dropsy.

If, in any case of chronic or recurrent difficult breathing, I feel a doubt as to whether the essential condition is bronchitis or asthma, the first question I put is—"Is your complaint a winter complaint?" If I get a negative answer, if I am told the season makes no difference, that it is as bad in summer as winter, the thing is settled—it is *not* bronchitis. If, on the other hand, I get an affirmative answer, I see strong reason to suspect that bronchitis is present, and if not uncomplicated, that is to say, if asthma is present as well, is at any rate the cause of the accompanying asthma. I then put these two additional questions:—"Is cough a constant and troublesome part of the attacks?" "Do you expectorate much, and is the sputum thick and yellow?" If these questions are answered in the affirmative, then, beyond all doubt, the case is one of bronchitis; if they are answered in the negative, then, I think, it is certain that it is not bronchitis; and the fact that the first question was answered in the affirmative—that the attacks of difficult breathing are confined to the winter—merely shows that atmospheric sources of irritation, such as cold air, damp, and fog, are the chief excitants of the asthma. I do not think that after these three questions any possible doubt can remain as to the presence or absence of bronchitis. As to the presence or absence of asthma (the presence of bronchitis being granted) a doubt may remain, and in the further prosecution of this inquiry I shall say a word or two presently.

As for the distinction of the difficulty of breathing due to asthma from that due to emphysema, I think (supposing each to be uncomplicated by the other) the three following points of contrast would always suffice.

IN EMPHYSEMA.

Respiration is never free.

Drumminess of chest unvarying.

Intercostal depressions effaced.

IN ASTHMA.

Respiration may be perfectly free in intervals.

Chest only drumming at paroxysms.

Intercostal depressions strongly marked, especially at each inspiring effort.

ON THE PATHOLOGY OF ILEUS.

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THE subject of the Pathology of Ileus is one on which, from time to time, a good deal of difference of opinion has prevailed. On its general nature, all are agreed; namely, that it is a mechanical obstacle to the circulation of the blood in the intestine; but, while many describe it as proving fatal in the way of inflammation, others admit that it is not necessarily associated with this condition. In the following remarks, I wish to call attention to the appearances observed in cases of pure ileus, a satisfactory explanation of which must form the foundation of the genuine pathology of this disease; and, as it is only constant appearances that ought to be taken into account, it follows that if, in fatal cases of genuine ileus, inflammation can be shown to be wanting, then we must

regard its occasional presence as non-essential, and look in some other direction for our explanation.

The immediate causes of ileus are various, although they all act mechanically, and produce the same symptoms and *post mortem* appearances. The most common are: strangulated hernia; internal strangulation of a loop of gut by its being constricted in an opening—as, for instance, one in the omentum, or in the mesentery, in the foramen of Winslow, etc.; a band tightly drawn across a portion of gut not in a loop; invagination, or intussusception. The following cases will show that the constant appearances in all these point to mechanical stoppage of the circulation; and that, therefore, inflammation, which, when it occurs, has generally some other apparent cause, is to be regarded as a mere casual accessory, and by no means as an essential feature of the disease. The distinctions between these two conditions would be the following. In inflammation, the gorging of the vessels would be greatest at the point where the mischief arose (*i.e.*, at the point of obstruction); and from that, as a centre, would radiate off in all directions, gradually passing into healthy parts, and it would be followed very speedily by effusion of inflammatory products, such as lymph and exudation corpuscles, or, if time permitted, of pus. Mechanical stoppage of the circulation, on the other hand, would produce much more intense loading of the vessels, a gorging, which would not radiate, but would pass in one direction only; namely, in that in which venous return was obstructed; and the effused materials would consist not of lymph, but of bloody serum, or blood itself; the condition terminating in gangrene, if time permitted. The presence of pain must not mislead us into supposing that, of necessity, inflammation is present; for, if the pressure of loaded vessels in the inflammatory state be sufficient to produce pain, much more ought the greater loading of them in mechanical stoppage of the circulation to give rise to the same symptom.

OBSERVATION I.—*Sudden Attack of Ileus; Death in Ten Hours; Band across the Ileum.*—J. C., male, aged 25, a stout, healthy-looking farm-servant, was towards evening, and some hours after having partaken of a heavy dinner, attacked with severe twisting pain of the abdomen, attended with vomiting, at first bilious, but finally stercoraceous. He had occasionally had similar attacks previously, which, however, had always passed speedily away, leaving him in good health. On this occasion, however, the symptoms persevered in spite of treatment; and he sank and died next morning, about ten hours after the attack.

On *post mortem* examination, the following appearances presented themselves. The abdomen was much distended, tympanitic; on opening it, the small intestines were seen to be much inflated and much congested. This congestion began about the middle of the jejunum; it was slight at first, but gradually increased in intensity in passing down the gut, until, near the end of the ileum, the intestinal coats were almost black. Across the ileum, a few inches above the cæcum, was tightly stretched a narrow firm band, attached above to the mesentery near its termination, and below to the inner side of the right iliac fossa close to the brim of the pelvis. This band was perfectly organised; in structure it was like the adjacent peritoneum; and presented no appearances indicative of recent formation. Immediately above it, the gut was abruptly bent upon itself, so as to double over the band and conceal it from view; and the loop of gut so formed presented the greatest appearance of congestion. At the point where the band crossed, the deep black injection of the gut, so conspicuous above, ceased abruptly, and the remainder of the intestine was empty, contracted, pale, and healthy. A little bloody serum was effused into the peritoneum; and the intestine, for some little distance above the band, contained clots of blood, but no trace of lymph or inflammatory exudation, either on its serous or mucous surface.

This case may be taken as an example of pure intense ileus, altogether uncomplicated; and its typical appearances may be put down as intense congestion, gradually increasing from above downwards to the point of obstruction, and there ceasing abruptly. The origin of such a band as here noted might be matter of interesting speculation. It could hardly have been an old inflammatory result, as, in such a case, a band of lymph would have been adherent throughout, or at any rate must have adhered to the intestine across which it passed; whereas, in this case, it was quite free, except at the ends. It is, in fact, difficult to understand its formation, unless it had been congenital—an explanation rendered very probable by the circumstance that symptoms similar to those of the fatal attack, but passing quickly away, had repeatedly shewn themselves.

The following may be taken as a specimen of strangulation.

OBSERVATION II.—*Sudden Attack of Ileus; Death in Twenty-two Hours; Internal Strangulation of a loop of Gut.*—A female, aged 43, was admitted into the Royal Infirmary, under Dr. Reith, June 22nd, 1865. She had been in good health till between two and three o'clock on the morning of admission, when she was suddenly attacked with

pain and vomiting. The symptoms became more urgent as the day advanced; and, in spite of treatment, she sank and died at one o'clock on the morning of the 23rd, after an illness not exceeding twenty-two hours.

The following were the *post mortem* appearances. The abdomen was distended; there were four pints of bloody serum in the cavity of the peritoneum, but no lymph nor any appearance of inflammatory exudation. The stomach and small intestine, to within a foot of its termination, were distended with flatus. There was congestion of the small intestine, beginning about the end of the jejunum, and extending to within a foot of the cæcum, gradually becoming more and more intense until the point named, where it ceased abruptly; immediately above which point the colour was a deep purple. This was found to be caused by a narrow cord-like band, apparently a part of the lower right corner to the omentum, encircling tightly a loop of gut and mesentery, comprising a large part of the ileum; the last turn involved being abruptly twisted upon the constricting band. The last foot of the ileum (being the part beyond the stricture) and the great intestine were pale, healthy, and contracted.

Here, also, the same essential features presented themselves as in the last case. I have selected, as a specimen of this condition, a case of internal strangulation instead of strangulated hernia; because, in the former case, the symptoms pass on unchecked and unaltered to their fatal termination; whereas, in the latter, the appearances are materially modified by the operation, which may remove the condition of ileus to substitute (in fatal cases) peritonitis in its stead.

As a contrast to these, may be cited the following case of intussusception where, in addition to ileus, inflammation was present, depending, however, on another cause.

OBSERVATION III.—*Attack of Vomiting and Constipation, with Abdominal Tenderness; Death in Four Days; Intussusception, Perforation, Peritonitis.*—A male, aged 34, labouring under phthisis in its last stage, was attacked with vomiting and constipation; the abdomen was tender on pressure, but none of the symptoms were very urgent. He had been labouring under diarrhoea for some time previously, with occasional attacks of vomiting; the vomited matters being of a decidedly green colour, and containing sarcine in abundance. He gradually sank, and died in four days.

The following were the *post mortem* appearances. The abdomen was distended. The intestines were coated with freshly effused lymph, lying chiefly between the convolutions; the peritoneum was somewhat reddened. The jejunum and upper half of the ileum were much distended. About the middle of the ileum, was an invagination of about two feet of intestine, which was intensely congested, and contained coagulated grumous blood, which was also found in the lower part of the ileum. The congestion extended but a very short way up the gut, and not at all below; the lower part being pale and flaccid. In the jejunum, were numerous rather large ulcers scattered widely apart; the same appearance extending into the upper half of the ileum, but not into the lower. In the portion of gut forming the sheath to the invaginated part, a large ulcer had given way, causing perforation; but, from the blocking up of the interior by the invaginated gut, very little effusion of the contents had taken place.

Here the peculiar symptoms of ileus were by no means urgent, which corresponded to the small extent in length along the gut of the congestion found after death; and so, notwithstanding the access of peritonitis, doubtless set up by the perforation, and notwithstanding the weak condition of the patient, already broken down by phthisis and diarrhoea, death did not occur for four days.

From these cases—and others might be cited—it would appear that inflammation is no necessary or even frequent concomitant of ileus, certainly no necessary prelude to a fatal result; while the *post mortem* appearances point clearly to the mechanical stoppage of the circulation as the cause of death. These appearances are precisely identical with the phenomena produced by tight ligature of a limb, or of any portion of the body; the venous circulation is brought first and completely to a stand; thus throwing the blood back upon the capillaries and arteries, which last, not being so easily completely compressed, may continue for a short time to carry a little blood into the part. The gorged vessels endeavour to free themselves of their contents by the effusion of serum and blood, or of blood alone; but the stagnation still continuing, the whole circulation of the part comes permanently to a stand, and gangrene results. One point, however, in the state of parts in ileus, deserves especial notice. It is not alone the part actually strangulated that is loaded with blood; but this loading extends for a considerable distance upwards, as if the stoppage of the circulation affected not only the part directly implicated, but caused also an impediment to the circulation in the intestine for some distance above. When pressure is put upon a vein, we find that the blood is thrown back only on those parts

drained by that vein; and it might, therefore, have been concluded *à priori* that, considering the large number of veins returning blood from the intestinal coats to the trunk of the superior mesenteric, the blocking of one of them would not necessarily have interfered with the circulation in its neighbours; but the above observations show that it does do so to a very considerable extent. This can scarcely be produced simply by the spreading of irritation, for, in that case, the parts below, as well as those above, should have shared in it; and the only reasonable explanation that suggests itself is that, besides the current of venous blood returned direct, by the various small veins in the mesentery, into the trunk of the superior mesenteric, the very free anastomosis by the side of the gut permits a steady downward flow of blood parallel to the intestine; the circulation being only finally balanced, as it were, when we near the termination of the great intestine. On such a supposition, it is easy to understand the appearances produced, as the blood would thus, in all cases, be thrown back on the parts above the stricture, gorging these, and leaving those below pale. If, however, this supposition be not admitted, why should not the congestion in Case I have been limited to the one or two veins actually compressed, instead of extending many feet upwards? Or why, in Case II, should the lowest part of the loop have been the most congested, instead of having this condition equally spread over the whole part included in the stricture? In this last case, the idea of the gravitation of the blood in the congested part having any share in the result is totally inadmissible, as the beginning and the end of the loop involved (being the parts least and most affected respectively) were on the same horizontal level; and the most dependent part, being the middle of the long loop, was not nearly so much affected as the part farther down the gut, although this last was above it in level. The great length of the loop in this case afforded a much better opportunity of tracing the actual condition of parts at different points, than does the short loop usually found in a hernial sac.

The idea of some such peculiarity in the intestinal circulation as that here supposed is, to a certain extent, borne out by what is seen in animals. If a small animal be killed, and the abdomen immediately opened, before the blood in the smaller vessels has ceased to circulate, and the mesentery be spread out under the microscope, the blood will be found still flowing there, although somewhat slowly, and the current will be found setting in all the smaller veins parallel to and down the line of the gut. From the foregoing observations in ileus, I consider it probable that this may also be the case in man; and, should this supposition prove correct, it would also explain the absence of valves in the veins of the alimentary canal.

OBSERVATIONS SUGGESTED BY THE STUDY OF CASES OF CATARACT EXTRACTION.*

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GRANTING that all the precautions which I have indicated be taken, there are, nevertheless, many cases where the eye would doubtless be lost through the attempt to secure too much; and there is abundant evidence to prove that the indiscriminate adoption of the most carefully managed flap would be fatal to many cases that are saved by either a combined or a preliminary iridectomy. Thus, Mooren, in his excellent paper on Extraction by a New Method (portions of which I translated in a former essay) demonstrated, at least to his own satisfaction, that the small portion of iris which corresponds to the flap, whichever section be employed, is the portion which first takes on inflammatory action, and is not only the cause of iritis and the minor sequelæ of such disaster, but is really the initiation of that series of changes which cause supuration of the flap, and end in panophthalmitis and total loss of the eye-ball. He illustrates this observation by several interesting cases, which space will not permit my now quoting to you; but which tend to show that the bruising of the small segment of iris corresponding to the flap, caused by the passage of the lens, is the proximate cause of iritis, and that the inflammation so caused is followed by supuration of the flap as a direct consequence. This important observation induced Mooren to excise a small portion of iris upwards or downwards, according to the position of the section, some weeks before extracting in the ordinary way. He was thus enabled to save several most unpromising cases; and I have verified the truth of his doctrine by the result of my own practice, as may be seen by several cases which I reported in the *Ophthalmic Review*. Unfortunately, the division of the operation of extraction into two parts was always a serious objection to Mooren's pro-

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