"cigarette drain." The upper incision was closed in three layers. On his return to bed he was put in the Fowler position and continuous rectal saline infusion given. Unfortunately he developed chest symptoms and died on the tenth day after the operation.

At the *post-mortem* examination it was found that the abdominal organs were matted together by recent plastic peritonitis. There was some lymph around the track of the drain; the rupture was closed so as to be watertight. The upper lobe of the right lung was in a condition of grey hepatization, and almost the whole of the left lung was in the same condition. He had clearly died from pneumonia, and not from the abdominal condition.

These three cases have so many points in common that it is difficult to believe that coincidence is a sufficient explanation. They were all well-developed, strong men, in the prime of life; they all suffered accidents which involved them being suddenly and unexpectedly dragged by their arms fully extended above their heads; they none of them complained of violence applied to the abdomen or showed any external evidence of severe injury to that part. Two of them were absolutely free from the slightest abrasion or bruising on the trunk; the other had some superficial skin scratches; none of them showed immediate marked collapse or such symptoms as would lead me to feel certain that any severe internal injury had occurred; each of them had a rupture of the small intestine; in all the rupture was transverse to the long axis of the bowel; and in all the rupture was within a foot of the commencement of the jejunum; and unfortunately all of them died. I have been strongly impressed by these cases, and ask myself whether the mechanism of the rupture was not as follows: When each was suddenly dragged by the arms, the chest would be forced into the position of full inspiration, and the diaphragm raised, as shown by indrawing of the epigastrium; therefore the upper abdominal organs (liver, stomach, and spleen) would be drawn upwards. The pancreas and duodenum are usually considered to be fixed, but any small movement which might take place in these organs would be in an upward direction. At the same time, as one easily finds by standing with the arms extended above the head, the lumbar spine would be markedly arched forwards, and there would be, I think, a very strong contraction of the anterior abdominal muscles made by the patient in the effort to recover himself. I find by examining thin people in this position that the anterior and posterior abdominal walls actually meet. It seems to me that the small intestine would then be suddenly and violently forced down towards the pelvis, and I ask if it is possible that the only part of the small intestine which cannot go downwards, the first part of the jejunum, is so dragged upon as to rupture transversely, just as one finds that a string fixed at one end and jerked by the other, breaks near the fixed end.

The explanation of the mechanism of these cases is of more than academic interest, because, if they can be explained in this or some similar manner, surgeons would, I think, be justified in operating in such accidents on clinical evidence which falls short of that usually associated with acute abdominal conditions, and therefore they would not lose those favourable early hours whilst waiting for indications which in injuries to that part of the intestines have often not appeared until the time favourable for operation has passed. I should not have operated as early as I did in the last of these had I not had in mind the other two cases, and remembered the vagueness of the symptoms for some time after the accident.

I am indebted to Dr. Gerrard, the pathologist to the Oldham Royal Infirmary, for the particulars of the second of these cases, and for his reports upon the *post-mortem* findings in all of them.

WE learn from the *Medical Record* that a bill has recently been introduced into the New York State Legislature which would provide at least one medical judge in each county, three in the Court of Appeals, and three in each Appellate Division of the Supreme Court, to sit when any medical evidence is to be offered. Under the terms of the bill the medical judges would be appointed for five-year terms at £1,440 a year, with an additional allowance of £100 for expenses. They would sit when any evidence as to sanity was to be given, and would advise the court and would instruct the jury as to the correctness of statements and conclusions of medical witnesses.

RUPTURE OF DUODENUM CAUSED BY A BLOW.

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THIS case is of extreme interest, as, beyond diagnosing a subphrenic abscess, I could not be sure as to the original seat of the trouble.

A Hindu, male, aged 25, stated that six weeks before admission to hospital on February 8th, 1914, a wooden plank fell from the ceiling on to his right side while he was asleep. The corner of the plank hit him over the eighth rib in the mid-axillary line. For three weeks he felt a dull aching pain, and then noticed a small swelling, and the pain became severe, especially on respiration; at the same time he said he had fever every day. There was no history of dysentery. The patient was desperately ill, extremely emaciated and in great pain; the evening temperature was 101.8° F., and the pulse 100; a dry cough was very troublesome. Over the centre of the eighth rib was a fluctuating swelling about two inches in diameter; it was extremely painful on palpation. The liver dullness was diminished, reaching to one inch above the costal margin. The abdomen was moderately swollen and hard; it was evident that chronic peritonitis was present. The bowels moved freely, the tongue was coated.

On February 9th, under cocaine, I incised the swelling in the seventh intercostal space in the mid-axillary line, and at once gas and 6 oz. of pus escaped. The odour was faecal. I then introduced my finger and discovered that the eighth rib was denuded of periosteum and very rough. I could then feel an abscess cavity in the right lobe of the liver, and that the liver had fallen away from the chest wall, so that I could only just reach it. Above I could feel the diaphragm, but below nothing. I put a large-sized drainage tube just through the chest wall, and applied an antiseptic dressing and bandage.

The temperature that evening was only 99° F., and after that remained normal. The differential blood count was as follows: Polymorphonuclears 86 per cent.; mononuclears 1.6 per cent.; lymphocytes 12.4 per cent.

The patient improved slightly, the pain being negligible, and he slept soundly. The discharge from the wound was only thick yellow pus. On February 13th I gently washed out with a weak iodine solution. He passed five loose motions a day, with a tinge of blood and slime. The pulse kept rapid, varying between 92 to 100. His condition remained the same until February 16th, when at 7.30 a.m., in spite of the strictest warnings, he suddenly got out of bed and fell back on to the bed in a state of collapse, from which he never recovered; he died at 11.45 a.m.

Post mortem it was found that the posterior surface of the descending portion of the duodenum had ruptured in the long axis of the bowel. The infection then spread round the chest wall, along the eighth rib, pointed, and then infected the liver, forming an abscess cavity. The lower portion of the right lobe of the liver was gangrenous. The peritoneal cavity contained serous fluid, and large masses of clear gelatinous matter.

The history was clear that the injury was the starting point of the illness, and after opening the abscess I realized that we were dealing with some intestinal injury, but the most careful examination of the discharge gave no clue as to what part of the intestinal tract was damaged, as nothing but small quantities of foul, thick pus were discharged through the drainage tube. The actual tear in the duodenum was closed by adhesions, and I think that, considering his wonderful vitality, if the patient had come to hospital earlier, there would have been a reasonable chance of his recovery.

CONDENSATION OBSTRUCTION IN RELATION TO PERICOLITIS AND INTESTINAL KINKING.

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SINCE interest has been aroused in the presence, pathology, symptomatology, and mode of formation of accessory peritoneal adhesions and bands, causing more or less definite symptoms of alimentary toxæmia, a number of explanations have been advanced. The membranes or bands associated with the names of Jackson and Lane especially have been known and noted since the advent of laparotomy. Only recently has the more correct name "appendicitis" replaced the older one of "perityphlitis";

and though actual acute inflammation arising from the appendix alone was no doubt classed under the other name, I believe that very many of the old surgeons have seen and noted a veil-like matting of the thickened peritoneal cover to the caecum, and simply regarded it as a consequence of inflammatory action. I have seen a large number of cases in which this veil was present over the intestines, and remarked on during laparotomy, without, however, any marked evidence of consequent constriction. Delore and Alamartine subscribe to a theory of repeated and mild peritonitis, which doubtless applies in many instances, as recovery from slight peritonitis is, in my opinion, very common, the complicated protective armoury of the peritoneal sac and omentum being responsible for overcoming many attacks of infection.

Lane imagines a natural reaction in the tissues, which adapt themselves to mobile or distended intestines by developing bands or adhesions as a result of crystallization of the lines of strain. Mayo considers a dislocation of embryonic arrangements of the caecum to result in a thickened pericolic membrane, and Gray and Anderson believe it to be due to excess of physiological fusion—that is, that the caecum in descending from the region of the liver carries with it a fold of omentum, which gets stretched and thinned, losing its fat, and remaining to cover the caecum. The last two theories presume the existence of a membrane throughout life, with later development, if any, of symptoms of the nature of autointoxication or stasis.

Doubtless many extraneous membranes and adhesions are due to the result of microbic action. Lane's theory also has much in its support, and accommodation of parts to altered physiological strain is but natural and reasonable; while Gray and Anderson's theory has much to commend it from anatomical grounds. When kinking occurs, symptoms appear of a gastro-intestinal, toxæmic, or coprostatic variety. Kinking may be due to gravitational pull of a loop of bowel attached by a band, it may be due to altered intra-intestinal or intra-abdominal tension, or to shrinking of the tissue of the band.

My experience is limited to about thirty-two cases of well-marked dense veil formation seen during laparotomy for appendicitis, in which all the patients were over 40 years. In volvulus, too, it is well known that young persons are not usually attacked, and in band obstruction generally, except when due to dense gross adhesion, the patient is often in middle life. I believe this age incidence is not a casual one, and that bands existing previously and without symptoms may develop to a noxious degree, as suggested by me before under the name of "condensation obstruction."¹ Old superficial scars which have been pliable in early life become puckered and hardened as years go on, especially in women after the menopause, in whom stitch marks are commonly felt as button-like prominences; a linear scar is converted into a ridge. Tissue sclerosis is a sign of age, be it in arteries, eye, bone, muscle, brain, or other organ.

In this short communication I wish to suggest, therefore, that many acute abdominal diseases, or chronic conditions, occurring in middle life, are the effect of contraction of former resilient and pliable adhesions—a form of condensation obstruction.

REFERENCE.

¹ *Lancet*, January, 1914.

MITZMAIN (*Philippine Journal of Science*, vol. viii, Section B, No. 3, June, 1913) states that surra may be mechanically transmitted by *Tabanus striatus* (Fabricius). For his experiments he used bred flies so as to eliminate fallacies which might have resulted from natural infections. He was successful three times with direct or mechanical transmission by "interrupted" feeding when only a short interval was allowed between the bites on infected and healthy animals. In sixteen experiments the minimum number of flies with which the infection could be transmitted was two. Hereditary transmission was not observed. The maximum length of time for which *Trypanosoma evansi* could be demonstrated microscopically in the gut of this species of fly after feeding on infected blood was thirty hours. The organisms were found in the fly's dejecta two and a half hours after biting the infected animal, and suspensions of flies when injected subcutaneously were found infective for animals from a period of ten hours after the flies had fed on infected blood.

FATAL CASE OF VERONAL POISONING:

RECOVERY OF THE DRUG FROM THE TISSUES AND URINE.

BY

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THE subject of the following notes was a patient of Dr. G. H. Barker, who has kindly allowed us to publish the case. One of us (G. P.) saw the patient in consultation with Dr. Barker, and the other (E. R.) is responsible for the analysis.

A. B., 50, a restaurant manager, had suffered for some time from mental depression and insomnia, and had on the advice of a friend previously taken veronal, but was warned by Dr. Barker not to do so. He had improved in health considerably. On November 11th he went to bed about 10 p.m., but was heard snoring loudly during the night. Next morning he was found to be deeply comatose and breathing heavily. The pupils were small, but reacted to light, and later on were moderately dilated. There was no evidence of paralysis, but all the limbs were flaccid, except for some little power of resistance in the left arm. No plantar or other reflexes could be obtained. The face was of a natural colour, the skin moist, the temperature raised by about a degree, the pulse was quickened but regular, and the blood pressure was not abnormally high. There was no rigidity of the neck, and no squint. The lungs were fairly normal, but the breathing was rattling from the accumulation of mucus in the upper air passages, and whenever the head was depressed over the side of the bed this ran out in a turbid stream to the amount of an ounce or two, with great relief to the respiration. A stomach tube was introduced, several ounces of clear fluid were drawn off, and the stomach washed out with potassium permanganate. The urine had to be drawn off; it contained neither albumin nor sugar. There was no return of consciousness, and since the patient was unable to swallow he was fed during November 12th and 13th by nutrient enemata and salines, which also contained coffee and strychnine. The bowels failed to act. Very early in the morning of November 14th he became worse, and the temperature rose to the remarkable level of 107.2°. Cold packs were employed, as a bath was not at hand, and the temperature was reduced to 104.5°. The heart acted fairly well, but one lung was now full of moist râles, and after a few hours he died.

A *post-mortem* examination was made next day; rigor mortis and hypostatic congestion were well marked. The brain was normal, and contained no trace of hæmorrhage or tumour. The lungs were dark, oedematous, and congested, but no pneumonic consolidation could be found. The kidneys were normal; the liver weighed 60 oz., and was slightly fatty. The heart weighed 17 oz.; there were deposits of fat on the surface, and the muscle showed some degenerative changes; the cavities were dilated, and all contained liquid blood and *post-mortem* clots. The intestines and stomach were normal, except for a little *post-mortem* congestion on the posterior wall of the latter, near the cardiac end. The stomach contained a little grumous mucus only. A culture was made from the cerebrospinal fluid, but no pathogenic organisms were obtained. There was no trace of injury or violence in any part of the body.

Portions of the brain, viscera, and of the urine drawn off before death were sent for analysis.

It is remarkable how little the decision of the Privy Council to include veronal under Schedule II of the Pharmacy Act as a poison is known to pharmacists.

THE SEARCH FOR VERONAL.

Of the urine procured from the patient 175 c.cm. were examined by Willcox's method, as follows:

Lead acetate added in excess, filtered, and excess of lead removed by H₂S; the solution remaining feebly acid to acetic acid, was concentrated at a low temperature to 60 c.cm., a little blood charcoal added and allowed to stand for a few hours, filtered, and evaporated to about 30 c.cm. in a water bath at a temperature below 60° C., extracted with two volumes of ether three times, separated, and ethereal fluid filtered into a tared flask, evaporated off either at laboratory temperature, and finally under air pump; failing to remove last traces of water by these means, evaporated in water bath below 60° C. until crystals appear.

Weight of crystals, 0.31 gram.

Recrystallized from ether, melting point 183° C.

Recrystallized from water, melting point 186° C.

Mixed with crystals of hippuric acid and succinic acid to obtain cross melting point, melting took place between 150–175° C.

Mixed with crystals of veronal, melting point 186° C., melted at 186° C.

Crystallographic appearances, both rhombic and fan-shaped, were obtained of similar character to those obtained from veronal.