Nowadays other methods are found serviceable in the diagnosis of chest diseases, such as examination of the sputum and the *x* rays, the opsonic index standard and other means. No method, however, is so simple as that of auscultation, and the stethoscope remains an instrument which all medical practitioners ought to know well, for good hearing and patience is all that is required. Some patients have no sputum to test and the shadows of the *x* rays may be capable of many explanations. Auscultation, therefore, holds its own and will continue to do so to the end of time.

WHAT DOES MEDICAL SCIENCE OWE TO LAËNNEC?

By his great discovery he bequeathed an instrument which can be used, and, though modified in its construction, has been in common use to this day. What we owe most to him is the introduction of a new sense into our investigation of disease. Our eyes and the sense of touch alone being formerly used, he taught us to use our ears. Consider that the organs of the heart and lungs are packed into the bony cavity of the thorax and cannot be approached very easily. By bringing the sense of hearing to bear he unlocked the secrets of the heart and lungs. He further investigated thoroughly by his method the physiology and pathology of these organs. In *De l'Auscultation Médiate* you will find hardly any disease unnoticed. The last thing that he did was to provide a proper nomenclature for diseases of these organs. Before the invention of auscultation one could not describe the sounds of mitral stenosis, of aortic regurgitation, or of aortic obstruction. They were not known. All that was known of heart disease was that certain lesions were found after death, the diagnosis in those days being generally made on the *post-mortem* table. These were the grand legacies which Laënnec bequeathed to medicine, and remembering that he died at the age of 45, we must all agree that he died too soon. He attributed his death from phthisis to a *post-mortem* wound from a tuberculous subject, which had been neglected, and he used to cauterize this wound with butter of antimony, and he maintained that if his lungs were affected it was through this wound, which is highly interesting in the light of later experience. The nomenclature of lung and heart disease consists largely of French terms, such as "râles," "bruit," "bronchophonie," and "oëgophonie," all of which were invented by Laënnec.

The late Sir Prescott Hewett used to tell a humorous story of the use of the term "bruit" in the early days of the stethoscope. When the experiments were being carried on by Dr. Hope and Dr. C. J. B. Williams on a donkey in the St. George's Hospital dissecting room, experiments which led to the discovery of the cause of the second sound of the heart, there appears to have been on one occasion a difference of opinion. The experimenters and several medical men present could not agree as to the audibility of a bruit or murmur which one said was present, while others denied it. They bethought themselves of getting the impartial evidence of a fresh ear to decide the point, and called to their aid the dissecting room porter, who no doubt had overheard the discussion, and was beginning to get familiar with the new terminology. He applied the stethoscope to the donkey, listened attentively, and looking satisfied, said, "Yes, gentlemen, I hears the brute."

Finally, we must never forget that, in addition to the grand field of scientific work which Laënnec opened to us, he also bequeathed his own brilliant example as an investigator.

MAJOR ANDERSON, I.M.S., has described in *The Indian Medical Gazette* recently 2 cases of abscess in the spleen in malarial individuals in the Andaman Islands—a very rare condition. Of his 2 cases, 1 recovered after operation, the other died, and he quotes Catrin as stating that Grand Moursel collected particulars of 14 cases recognized during life and operated upon, 11 of them recovering. Marchiafava's and Bignami's theory that such abscesses are the result of pyogenic cocci is generally looked upon as correct, the malarial parasite *per se* never producing pus. Major Anderson also publishes some notes on the occurrence of *Amoeba coli* in Port Blair. He believes that the parasite may be placed in the category of a harmless commensal. Perhaps he has not read the recent American researches in the Philippines on this subject, researches which attribute to the amoebae a very important part in the causation of one of the forms of tropical dysentery.

WHERE PHYSICIAN AND SURGEON MEET.*

By ROBERT KIRKLAND, M.B.,

SENIOR PHYSICIAN TO THE CHELTENHAM GENERAL HOSPITAL.

WITHIN recent years the physician and surgeon have made great advances undreamt of years ago, the former in diagnostic ability and a more correct interpretation of the phenomena of disease, the latter in greater manipulative skill, boldness, and careful technique. They have now met face to face, especially in the region of the abdomen, and it is about some of the diseases, acute and chronic, of this region I wish to say a few words.

The responsibility in the acute cases should, as a rule, first rest with the physician, for it is essential to a correct carrying out of treatment that a quick and accurate diagnosis should be made and then quick and decisive action taken. In acute abdominal affections the physician must take no half-hearted action with regard to the calling in of his surgical colleague. Opposition of friends to operation must not deter him from doing his duty, and he should not wait to suggest it until the patient is too ill for operation to be successful. Lives may thus be saved or relieved which are otherwise doomed to death or prolonged suffering. But let me insist that to effect this it is necessary that a careful diagnosis should be made and the surgeon's aid sought early. It is manifestly unfair to him to ask him to operate, say, in a case of acute gastric perforation, after twenty-four hours have elapsed, or in a case of abdominal obstruction with great distension, free fluid in the abdominal cavity, and other evidences of peritonitis. Now, in these affections correct diagnosis is not always possible, but every care should be taken by eliciting the history of the onset of the illness, examining the patient, and giving due weight to the clinical symptoms, to arrive at a fair conception of the state of matters. I am no advocate of the practice of "here is an abdominal case; let us make an opening and look in." I agree with Mr. Mansell Moullin that no surgeon should have a roving commission inside the abdomen, and I am sure none of the surgeons connected with our Branch would desire it.

PERSISTENT JAUNDICE.

At times, however, after every care has been taken to arrive at the nature of an acute abdominal lesion, we may be in doubt, and it may then be necessary to make an exploratory incision, for by this means only can we clear up the nature of the case, and be able to deal with it promptly. For example, if you think you have a case of acute pancreatitis you are bound to have the abdomen opened, for the condition so closely simulates acute obstruction in every particular that nothing but exploration can clear it up. Again, suppose we have not such an acute case, but a case of persistent jaundice; by this I mean jaundice which has lasted more than six weeks. Are we to wait for some more weeks until intermittent hepatic fever and cholangitis with pus in the ducts enables us to diagnose obstruction of the common duct with calculus from chronic pancreatitis or malignant disease of the larger bile ducts? If so, the prospect of healing becomes bad, and there is risk from haemorrhage and septicaemia. The diagnosis of chronic jaundice of about six weeks' duration is admittedly sometimes most difficult. Within the past two years I saw, in consultation with the same friend, Dr. Foljambe Forster, two cases of this nature. In both, the onset of the jaundice was fairly sudden, but without any pain or vomiting.

One was a lady over 55 years old; the jaundice was marked; there was a history of an isolated attack of colic ten years before, but no sudden chills with fever. The liver was not enlarged, but in the right hypochondrium could be felt an oval, even swelling continuous with the liver and with its long axis vertical. We were in doubt whether this was an enlarged gall bladder or a Riedel's lobe, the tongue-like projection of the right lobe so often felt in connexion with former attacks of acute cholecystitis and gall-stone mischief. But its junction with the liver seemed constricted or narrowed and not broad, as is the case with a Riedel's lobe. We could discover no other source of malignant disease in breast, uterus, stomach, or bowel, and the left supraclavicular gland was not enlarged, nor was there to be felt the little tell-tale gland behind the left sterno-mastoid in the posterior triangle of the neck. There was no salivation nor was there any sugar in the urine nor fat in the motions, which were clay-

* A paper read before the Gloucestershire Branch.

coloured. Believing that the tumour we felt was an enlarged gall bladder, and remembering Courvoisier's law that in jaundice due to gall stones the gall bladder is small, whereas in jaundice due to the pressure of a growth on the duct the gall bladder forms a palpable tumour, we thought the most likely diagnosis was cancer of the head of the pancreas. The patient was taken up to see Mayo Robson, and, as it was just possible it might be a calculus in the common duct on account of the attack of colic years before, an exploratory operation was advised. The tumour we felt was found to be a Riedel's lobe, with the gall bladder beneath and adherent, containing one stone. There was no disease of the head of the pancreas, but the unusual and rare condition of primary malignant disease of the hepatic duct.

The other case was that of a gentleman over 60. He had a feeling of slight cold, and in a day or two jaundice supervened which became intense, with clay-coloured stools and troublesome itching of the skin. As the jaundice had come on insidiously, it was naturally supposed to be a case of gastro-duodenal catarrh or simple catarrhal jaundice. But it had persisted for over five weeks when I saw him, and it was not lessening. Simple catarrhal jaundice was therefore now out of court. The liver was not enlarged, there was no pain on pressure over Mayo Robson's point, and so far not much wasting. He had never suffered from spasms, and had no pain or vomiting previous to the onset of jaundice and no intermittent temperature or chills. Nothing could be felt in the region of the gall bladder. We thought the condition was most likely to be chronic pancreatitis the result of the extension of the duodenal catarrh up the lumen of the pancreatic duct, or due, but this less likely, to a stone in the ampulla of Vater. The jaundice still persisting, he also consulted Mayo Robson, who was unable to give a definite opinion. An exploratory operation was performed, when, strange to say, primary malignant disease of the common hepatic duct was again found.

A few years ago I should have taken grave exception to any operation being performed on either of these cases, for the first seemed most likely to be malignant disease of the head of the pancreas and the other simple pancreatitis, which, with rest and dieting, in course of time, as a rule, gets well of itself. Recently I have seen, in consultation with Dr. Halliwell, a case somewhat similar to the two preceding cases.

The patient was about 60. The jaundice had come on in an insidious way with no pain or vomiting, and with no past history pointing to gall stones. The stools were clay-coloured and the urine contained bile. The jaundice, instead of clearing up after five weeks, persisted, and became more intense. Then I saw her. The liver was not enlarged, there was no tenderness or pain to be elicited over the gall bladder region even by Murphy's method, and the gall bladder was not palpable. No primary source of malignant disease could be found in breast, uterus, stomach, or bowel. Until the jaundice came on she had felt in her usual state of health. At first it was hoped that it was simple catarrhal jaundice, but this idea had to be put aside when the jaundice instead of lessening after four weeks, became more intense. It seemed to me that it must be one of four things—either malignant disease of the larger bile ducts, malignant disease of the head of the pancreas, chronic pancreatitis, or impacted stone in the common bile duct. Malignant disease of the liver could be excluded, for the liver was not enlarged, and when the liver is enlarged, as it sometimes is in either of the four maladies mentioned, it is a uniform enlargement, smooth and regular, with an even edge. Cirrhosis of the liver could also be excluded, for here the jaundice is not intense, and the stools are not typically clay-coloured. Besides, we have other symptoms, such as haematemesis, vomiting, ascites, and enlarged spleen. It was not likely to be impacted stone, as there was no past history of spasms or jaundice, and the attack of jaundice had come on without vomiting or pain in the right hypochondrium, radiating round to the lower angle of the right scapula; nor were there any periodic attacks of intermittent hepatic fever, although perhaps these had hardly time to develop. This being so, I suggested waiting for the time factor to help.

If Dr. Cammidge's pancreatic reaction urine tests were thoroughly reliable, they would be of inestimable value in this connexion, for they would, if conjoined with steatorrhoea or fat in the stools, at once separate the pancreatic affections from malignant disease of the bile ducts. Unfortunately these tests are still *sub judice*, for other competent observers state that these reactions sometimes occur in other pathological conditions and even in normal urine. I have no experience of these and can give no opinion. We are therefore thrown back on clinical observation, but at this early period of the jaundice, it does not help us much in the differentiation of the three other maladies—cancer of the head of the pancreas, malignant disease of the larger bile ducts, or chronic pancreatitis. Age over 50 and more rapid wasting would

be in favour of the two first-mentioned illnesses, and perhaps an enlarged, distended, and palpable gall bladder would point more to cancer of the head of the pancreas. Greasy white motions and sugar in the urine would also indicate pancreatic disease. If my diagnosis were uncertain I should recommend at first rest in bed, a diet of milk and chicken broth with the avoidance of everything fatty. With regard to drugs, I should prescribe sodium salicylate to keep down the infection of the bile ducts, and I should give at night a pill of iridin with a morning draft of Carlsbad salts or Cheltenham Lansdown water warmed, and with ten grains of urotropin added. This to be slowly sipped. If after six or eight weeks the jaundice remained as intense, and especially if it increased, and if any symptoms now pointed to impacted calculus in the common duct, I should advise operation—a choledochotomy or duodeno-choledochotomy. In quiet impaction the presence of a Riedel's lobe would guide me to the same decision. If the probabilities were against it being an impacted stone, and if the patient were over 50, I should then state to the patient or his friends my fear of its being malignant disease of the head of the pancreas or larger bile ducts, and the only chance of palliation by surgical means would be if the growth were situated in the common bile duct. Here resection with implantation of the hepatic duct into the duodenum, or putting the gall bladder into communication with the small intestine, might obviate cholaemia with its attendant symptoms, and thus greatly improve the patient's condition. Most of us are never likely to see primary carcinoma of the hepatic duct, as it only occurs 18 times in 80 cases of malignant disease of the larger biliary ducts; but I do think we are likely to meet, if we have not already met, with the disease in the common duct. The prevalence of the disease is doubtless commoner than is thought, since it may be easily overlooked or the jaundice ascribed to something else. At any rate, it is suggestive that within the past fifteen years the number of cases described has increased fourfold. If an operation were declined I should try Dr. Beard's treatment, the injection of trypsin into the deeper subcutaneous tissue of the buttock or back, of course with strict antiseptic precautions. I should begin with five minims of trypsin injection daily, increasing up to 20 minims by the end of the first week, and 40 minims by the end of the third week. Combined with this I should give by the mouth one capsule of holadin, which is an extract of the entire pancreatic gland, thrice daily one hour before meals with a pepule of ox gall compound at night.

GASTRIC AND DUODENAL ULCERS.

It is difficult to draw a hard-and-fast line between the acute and chronic forms. Perforation and haemorrhage are the immediate dangers in the former; in the latter there are, in addition to these, all the troubles that arise from an open sore and the production of contracting cicatrices. From the sex, age, and history, you can arrive at a fairly correct conclusion whether the ulcer be gastric or duodenal. Simple ulcer of the stomach is a common condition, more common in women, and these young and chlorotic, than in men. Pain generally comes on soon after the introduction of food into the stomach, and is relieved by vomiting. Haematemesis is common, but seldom fatal. Contrast with this ulceration of the duodenum, where the patients are usually men from 30 to 45 years of age. When pain occurs it is at some interval after a meal, caused by the food passing from the stomach into the duodenum, and the introduction of food into the stomach often relieves the pain. This is a useful point to remember. Vomiting is not usual, but haematemesis, should it occur, is often fatal. When haematemesis and melaena occur in duodenal ulcer you may sometimes detect a round soft lump just below the ribs and just outside the right rectus, although I have never seen this mentioned in the textbooks. This is blood clotted *in situ*, for large blood clots can form in the duodenum. Twice I have demonstrated this in cases seen in consultation. Another interesting point is the occurrence of transient attacks of jaundice in duodenal ulcer, and as tenderness can generally be elicited over an area just above and to the right of the umbilicus, in the same situation that tenderness is found in cholelithiasis, it is most difficult sometimes to diagnose duodenal ulcer from gall stone trouble. In a case which I saw in consultation

I made this mistake and diagnosed gall-stone trouble, until the occurrence of haematemesis and melaena cleared up its nature. Later on I shall revert to this case.

It may be, however, that the first indication of gastric or duodenal ulcer is sudden perforation. What are the usual symptoms of such a catastrophe? Shortly these: At the moment of rupture the patient may volunteer the statement that he or she is conscious of something having given way, and that immediately they felt a burning pain in the epigastrium as if something hot were being poured out. In the epigastric region the pain and tenderness are most marked, but I have seen in perforation of a duodenal ulcer the pain referred to the right iliac region, simulating perforation of an appendix, as I have seen the pain in perforation of the stomach referred to the left iliac region. Collapse is extreme if the leak be large, not so evident if the leak be small. The pain is constant, unlike the intermittent colicky pains of obstruction. We note that the abdomen is rigid, motionless, and retracted. After the lapse of a few hours this gives place to a tense, distended tympanitic condition. The bowels usually remain constipated. In all but the rapidly fatal cases the temperature after the collapse begins to rise and the pulse to increase in frequency; in two hours the pulse may rise from 90 to 120. Obliteration of the liver dullness due to free gas in the peritoneal cavity with retraction of the abdomen is a very important sign. These signs and symptoms, then, with or without a previous history of indigestion, pain in the epigastric region immediately or three or four hours after taking food, haematemesis or melaena, should suggest such an accident.

There can be no difference of opinion as regards the need of a surgeon in a case such as this. But he must act promptly, and the sooner the abdomen is opened the better. After 36 hours it is useless. The mortality among those operated on under 12 hours, according to Mayo Robson, is 28.5 per cent.; among those operated on from 12 to 24 hours, 53.6 per cent.; this is with reference to perforation of a gastric ulcer. As to perforated duodenal ulcer, Mr. Moynihan has collected from various sources 51 cases, 2 only of which were correctly diagnosed, and 18 were operated on under the impression that the cause of the peritonitis was appendicitis. I do not think the diagnosis should be so difficult if careful attention be paid to the previous history. Fortunately, the gastric and duodenal contents are usually at the time of perforation but slightly septic, and Mayo Robson hopes that if a skilful surgeon be obtained within twelve hours of the rupture of a duodenal ulcer, and if when the abdominal cavity is drained the ruptured intestine be closed, the mortality should be only 10 per cent. At present, when the operation is performed within 24 hours of the rupture, the mortality is 38 per cent.; after 24 hours, 82 per cent.

CHRONIC ULCER OF THE STOMACH.

Here the question—When is the physician to cease and the surgeon to take up the thread?—is not quite so simple. The diagnosis of this condition is not so easy as it seems, because the symptoms of anaemic gastralgia are those also of gastric ulcer, with the exception of the haematemesis, and it is notorious that this symptom, on which, perhaps, most reliance is placed, frequently occurs without any evidence *post mortem* of gastric ulcer. Dr. Hale White, I think, was the first to point out that many women who have severe and alarming gastric haemorrhage, and who are said to have gastric ulcer, have not got gastric ulcer at all. With this haematemesis we have gastric pain, nausea, and sickness, and this condition chiefly affects women between the ages of 20 and 40, so that its diagnosis from genuine gastric ulcer is very difficult. Of course, such cases never get any symptoms due to the mechanical effects of ulcers, such as adhesions, pyloric stenosis, or subphrenic abscess. Have I any proofs personally of this state of affairs? Well, yes I have, for I have had two such cases admitted into hospital under my care with a diagnosis of gastric ulcer, in which diagnosis I concurred. One was a woman of 40, the other a woman about 50. Both were pale but well-nourished women, who had complained for years of pain after food and nausea. One had had haematemesis before her present illness; the other had a first attack. Both died suddenly with symptoms of internal haemorrhage, followed by profuse haematemesis, and on *post-mortem* examination there was no aneurysm, no atrophic cirrhosis of the liver,

no evidence of granular kidney or heart disease, no duodenal ulcer, or other condition to account for it—simply small pin-head erosions of the mucous membrane of the stomach, from which the blood had come, but these were not ulcers.

As I have already said, many cases of simple anaemic gastralgia are said to have gastric ulcer. But I think that pain after food—and only then—is not so marked in anaemic gastralgia. It may occur apart from food, and especially on exertion. In gastric ulcer, too, the skin tenderness is more localized; in simple anaemic vomiting there is a large area of superficial tenderness. The vomiting, too, in the latter condition is often dependent on heart stretching. But the two significant signs of gastric ulcer are defined deep-seated pain, and the presence even of small quantities of blood in the faeces or vomit. Then, last of all, there is the therapeutic test. If you put girls with anaemic vomiting on to arsenic, it will, as a rule, stop their vomiting very soon if combined with rest in bed. Two minims of the liquor arsenicalis are quite sufficient given thrice daily. Again, before diagnosing chronic gastric ulcer try the knee-jerks and look at the gums. This may seem superfluous advice, but I remember a young man about 30 coming to my outpatient room complaining of severe gastric pain, at times with vomiting, which had troubled him for several months. He had been an in-patient at a large hospital, where his stomach had been washed out, and test meals administered and withdrawn for chemical examination. In a routine way I tried his knee-jerks and found they were absent, but this was the only symptom pointing to locomotor ataxy with the exception of rather small sluggish pupils. Later on he developed the distinctive Argyll-Robertson eye phenomena with Romberg's sign and ataxic gait. I have read of a case of lead poisoning with colic in a barmaid diagnosed as probable perforation of a chronic gastric ulcer. The blue line on the gums led to a correct interpretation of the condition.

Cases of anaemic gastralgia, locomotor ataxy, and lead poisoning are entirely within the physician's province, but what about the cases of profuse haematemesis which have not gastric ulcer? Physicians and surgeons disagree as to the treatment. Dr. Hale White implores us not to operate on such people, because, as he says all experience has shown that, if you do, they may die soon after the operation, and obviously if the stomach is oozing blood you cannot stop it by surgery. Whenever, therefore, you think you have got one of these oozing cases, do not open the stomach, but give adrenalin chloride. In marked contrast to this, Mr. Mansell Moullin says that in every case of his own in which there was general oozing, the haemorrhage has been arrested, without the least difficulty, as soon as the stomach has been opened and the mucous membrane exposed to the air. I am hardly qualified to give a decision in this matter, but I should be inclined, first, to try rest in bed, and the internal administration of adrenalin chloride with rectal feeding. If it be a case of haematemesis with ulcer, this is certainly the right course to adopt, because it is very exceptional for a patient to die from actual haematemesis due to gastric ulcer. In an ordinary case of gastric ulcer the ideal treatment would be rest for two or three months, the first four or five weeks being absolutely in bed. If there be haematemesis, I should give by the mouth sips of warm water only, and 15 minims of a 1 in 1,000 solution of adrenalin chloride every three hours for twenty-four hours. I should give rectal enemata consisting of 4 oz. of beef tea and milk, half a tube of Fairchild's peptonizing powder, a dessertspoonful of glucose, and a little salt every four hours, first cleansing out the bowels by a simple enema. The nutrient enemata should be administered at a temperature of 99° F. through a funnel with rubber tubing and glass nozzle, held about 1 ft. above the patient's hips. In the intervals between the rectal feeds give a 4 oz. enema of a normal saline solution at 99° F. Wash the mouth out several times daily with a solution of hydrogen peroxide or izal. While, theoretically, it would seem better to give the stomach a complete rest for a week, practically this strict limitation does not seem necessary. So, after four or five days, if the haemorrhage does not recur, I give 2 oz. of an equal mixture of milk and lime water every hour. When milk is not well borne, Valentine or Leube's beef solution or albumen water may be substituted.

This may be continued for five or six days, then we may give 7 oz. of beef solution at 8 a.m., 10 oz. of peptonized milk at noon, the beef solution again at 4 p.m., and the peptonized milk at 8 p.m. At the end of a month, if the patient has improved, she may be allowed scraped beef, pounded chicken, and farinaceous or custard puddings. In the way of medicine I give, if an aperient be necessary, one teaspoonful of Carlsbad salts in a tumblerful of warm water in the morning, and if there be pain and nausea, a mixture of bismuth carbonate, soda bicarbonate, and hydrocyanic acid, with or without a little cocaine hydrochloride. When convalescing, I give two or three 3-grain tablets of alginoid iron thrice daily after food, or 1 drachm liq. ferri manago-peptonati in water in the same way.

The majority of cases will get well by these measures, but if after this prolonged treatment the symptoms persist, or they return as soon as the patient begins to lead his or her ordinary life, then I should unhesitatingly call in a surgeon. For this may mean slow perforation of an ulcer with adhesions, or beginning cicatrization of an ulcer near the pylorus, or perigastritis. You may be astonished to hear that among cases severe enough to be admitted into hospital, 18 per cent. die and 42 per cent. relapse. This is based on the statistics of all the cases of gastric ulcer admitted into the London Hospital from January, 1897, to August, 1902. They were just 500 in number—98 men and 402 women. Of these 89 died, and 210 had been treated before for similar attacks. Bad as these figures are they are not the worst, for no account is taken here of the late sequelae or complications. In these cases are not included patients admitted for pyloric stenosis, hourglass contraction of the stomach, gastric dilatation, subphrenic abscess, or perigastric adhesions, nor is cancer of the stomach alluded to, although, according to Schlesinger, this follows in 5 per cent. of the cases of chronic ulcer, developing on the site of the old ulcer. In the absence of these we may still have grave anaemia, and a deteriorated condition of health ending in tubercle as the result of this disease. Let me repeat, I should call in a surgeon for the purpose of doing a posterior gastro-jejunostomy if after treatment for two or three months the symptoms persisted or if the patient relapsed again as soon as he began to lead his ordinary life, and was thus unable to earn his living. If this practice were more universal we should not see so many cases of pyloric stenosis with dilated stomach, or hopelessly distorted stomachs with ulcers eating into the liver or pancreas, representing years of suffering, and chronic misery often ending in death by haemorrhage, as in the following case:

On August 1st, 1905, my late friend and colleague, Dr. G. Bagot Ferguson, asked me to see a patient of his, a lady aged 60, who had complained off and on for many years of severe abdominal pain, indigestion, and nausea. No history of haematemesis or jaundice. During the past two months she had become much emaciated and paler, and her weakness was extreme. She now suffered agonizing pain in the left epigastric region, which could only be controlled by large doses of morphine. The abdomen was a little full in the upper part, but no peristalsis was visible. On palpation the left epigastric region was occupied by a mass with a somewhat oval irregular surface. Its long diameter was transverse, and extended almost to the middle line. Its anterior margin was not sharp or notched, and the fingers could be slipped between its posterior margin and the left lower ribs. Splenic dullness normal. Neither the left supraclavicular nor groin glands were enlarged. In spite of this I gave a definite opinion that it was malignant disease of the body of the stomach.

The profound anaemia, rapid loss of flesh and strength, with a hard tumour mass, seemed to me to warrant this conclusion. I think Dr. Ferguson himself thought there was something more than this in it. Be that as it may, two months afterwards he told me the mass had almost disappeared, and asked me again to see her. Certainly little could now be felt, but shortly afterwards she suddenly died with symptoms of internal haemorrhage. We obtained a *post-mortem* examination, and found the stomach full of blood. There was an hourglass contraction of the stomach, with part of the posterior wall adherent to the pancreas. Fresh ulcerations had occurred at the site of the old ulcer, and had eaten into the pancreas, laying open some of its vessels. This eroded part of the pancreas was lying bare in the stomach. No doubt what we felt at first was the inflammatory exudation thrown out on active ulceration occurring again at the site of the old adhesion, and which as time went on had become to a great extent absorbed.

DUODENAL ULCER.

Before discussing the treatment of duodenal ulcer may

I refer to its frequency? Andral gives the proportion of duodenal to gastric ulcer as 1 in 40; some textbooks give it as 1 in 50, and this until lately has seemed to be the prevalent opinion. I have long thought that this was an error due partly to many cases of anaemic gastralgia being labelled gastric ulcer, and partly to cases of primary duodenal ulcer being designated gastric ulcer. In our own hospital for the past year we had five duodenal ulcers to eighteen gastric. We must remember that duodenal ulcer occurs in that part of the duodenum near the pylorus; 90 per cent. of all cases, according to Mayo Robson, are situated within 1 in. of the pylorus, so that the symptoms and complications of both are very similar. It is to the surgeon that we owe a clear and explicit statement on this point, for his examinations *in vivo* are infinitely more instructive than *post-mortem* records. Here pathology based on autopsy has been corrected by the study of disease in the living body. To Drs. Charles and William Mayo of Rochester, U.S.A., in particular we owe the correction of this error, for in their last 200 cases of operation for peptic ulcer, 86 were found to be duodenal, a proportion, roughly, of about 2 duodenal to 5 gastric. In the preoperative diagnosis of duodenal ulcer from gastric ulcer, they lay great stress on the seat of pain and rigidity, to the right of the middle line in duodenal ulcer, to the left in gastric ulcer.

I can recall to mind 10 cases of duodenal ulcer I have seen within the past two or three years; 6 died from haemorrhage—an unusually large proportion—2 I lost sight of, 1 recovered after an operation for subphrenic abscess, and the tenth was the most interesting of all, for he had typical signs and symptoms of the disease, with its complications and catastrophes, and although reduced to a skeleton, and several times on the brink of death, recovered after a posterior gastro-jejunostomy, and is now, nearly four years after the operation, in good health. I shall give you this case *in extenso*, as it illustrates so well the phases of the malady, its diagnostic points and complications.

On May 17th, 1902, I was asked by Dr. Moore of Churchdown to see a patient of his, a gentleman of about 50. He had suffered at intervals for many years from severe pains, a little above and to the right of the umbilicus, with flatulency and heartburn. The pain often occurred late at night. Between these attacks he enjoyed good health. When I saw him he had a slightly icteric tinge after a few days of severe paroxysmal pain at the usual site. He felt sick and had great discomfort from flatulency and acidity. Bowels were constipated. Over the situation of the pain I could detect a somewhat indefinite swelling which I thought might be gall bladder. No tumour could be felt near the pylorus, the stomach was not dilated, and there was no haematemesis. The recurrence of these attacks for years, the site of pain, and the slight jaundice, led me to conclude he had gall stones with probably adhesions round the gall bladder. This is a pardonable error, for these recurring attacks of regional peritonitis in duodenal ulcer much resemble prolonged gall-stone colic. A stone, too, impacted in the cystic duct with a contracted gall bladder closely simulates chronic duodenal ulcer. Of course, a haematemesis or the passage of even a minute quantity of blood per rectum would clear up the diagnosis. He recovered from this attack, and some months later came to reside in Cheltenham.

On November 14th of the same year, about 1 a.m., I was sent for, as he had been suddenly seized with very severe pain in the right upper quadrant of the abdomen, with vomiting and collapse. There was rigidity of the right rectus and localized tenderness below the ribs on the right side. Subsequently he had a slight rise of temperature. It was only now that it occurred to me that it might be a case of duodenal ulcer with perforation, and local peritonitis due to limiting adhesions. This attack subsided without the formation of an abscess. As time went on he had retention of the stomach contents with haematemesis and melaena, making the diagnosis certain. I now urged him to have an operation done, a posterior gastro-jejunostomy, for the haemorrhage had been severe, and he was pale, ill-looking, and much emaciated. He refused, however, to have anything done. On June 24th, 1903, he had another attack of localized perforative peritonitis, and recovered again without the formation of an abscess. He was a Scotchman. To show the never-say-die character of the man, when convalescing from this attack a friend called to inquire for him. When ushered into his bedroom the sick and sorely tried patient raised himself on his elbow, and with a dry twinkle in his eye exclaimed, "Well, M—, have you brought the wreath?" His cheery determination inspired us all. Eventually, however, he had to deliver himself up into the hands of the Phillistines, and in August, 1903, he was operated on by Mr. Swain, of Bristol. Most extensive adhesions were found, and the parts were so

matted together as to be almost unrecognizable, but a posterior gastro-jejunostomy was performed, with a result as splendid as can be found anywhere in the annals of surgery.

By a curious coincidence, while I was writing this I was sent for by Dr. Moore to see him again. But his present illness has nothing to do with his old duodenal trouble. I found him to be suffering from paroxysmal dyspnoea and dyspnoea on exertion due to granular kidney, arterial sclerosis, and dilated heart. The apex beat is diffuse and weak, and epigastric pulsation is marked. The heart's action is very arrhythmic, in fact, the condition is that of cardiac delirium. The liver is much enlarged, smooth and even, its lower border extending below the border of the umbilicus. There is congestion of both bases of the lungs and beginning oedema, so that I fear his fight is nearly over.

If I do not weary you I should like to read you the following case, as illustrative of the difficulty in diagnosing ruptured appendicular abscess from ruptured duodenal abscess, the rupture taking place into the general peritoneal cavity:

On February 5th, 1907, I was called to see a case the history of which was as follows: Patient, male, aged 65, had been in service in India, where he had several attacks of malarial fever, and an indefinite history of an abdominal attack which necessitated his staying in bed for over a week. Since returning home he had dyspepsia with great flatulency, and had been subject to frequent seizures of pain underneath the liver edge, which pain was so severe as to double him up and required the application of hot bottles. He did not vomit, nor were any of the attacks followed by jaundice. The pain radiated slightly downwards, but not to the groin or testicle, and he never passed any blood or pus in the urine. No history of haematemesis or melaena. He saw several medical men on account of this trouble, both in Cheltenham and in London, but no definite opinion was given. Five days before I saw him—that is, on January 31st—he sent for his medical attendant, as he had a recurrence of pain, paroxysmal in character, at the old site, with a temperature varying from 100° F. to 102° F. The pain was quite limited to the right side. There was no vomiting or jaundice, and no pain or frequency in micturition. The tongue was coated and bowels constipated. He improved, and on the morning of the day I saw him seemed much better, his temperature being normal. But in the afternoon, when rising up in bed, he was suddenly seized with agonizing pain in the abdomen, and became collapsed, but still no vomiting. At the moment of the seizure he felt as if his right testicle were being pulled up into the abdomen. I saw him about 7 p.m. He had the typical ominous, anxious, abdominal face—by far the best criterion of the extent and severity of an abdominal lesion. His breathing was short and quick—60 respirations per minute—and entirely thoracic. His brow was cold and damp with perspiration. Pulse only 85, small and weak; his normal pulse I was told was 60; temperature, 100.5° F. As the pulse respiration ratio (pulse 85, respirations 60) was so abnormal a very careful examination of the bases of both lungs was imperative, especially on the right side, to see if there were any complicating pneumonia, pleurisy, or subphrenic mischief. Nothing, however, was discovered. The abdominal muscles were on guard, hard, rigid, contracted, quite immobile, and the legs drawn up. There was tenderness all over the retracted abdomen, if anything perhaps greater on the right side high up. No diminution of hepatic dullness. On examination per rectum nothing could be detected on the right side, but certainly there was more tenderness here on pushing the fingers towards the right iliac fossa.

The most interesting point was the pain in and drawing up of the right testicle at the moment of seizure. But this was no renal or hepatic colic. My reasoning was as follows: Was there from the history of pain in the past near the gall bladder region, with flatulent dyspepsia, and oft-recurring pains some time after food an old duodenal ulcer with fresh inflammatory mischief and abscess? Had this abscess then burst into the peritoneal cavity? Although there had never been jaundice or vomiting, could there be an acute empyema of the gall bladder which had likewise burst? or the third alternative, could it be a ruptured appendicular abscess? His abdominal trouble in India might have been a previous attack of appendicitis, and there was this fact, that on rectal examination more tenderness was felt towards the right iliac region than the left. But the collapse was greater than is usually the case in perforation or rupture of an appendicular abscess, for I never saw a man so hard hit. The most likely hypothesis, at any rate, was the bursting of an abscess either connected with a duodenal ulcer or appendix trouble.

Without delay I summoned my surgical colleague, Mr. Buckell, for immediate operation. An incision was made high up, a little to the right of the middle line, and on opening the peritoneal cavity foul-smelling pus welled up, but it seemed to come from below the transverse colon, and on enlarging the incision downwards, a ragged abscess cavity in connexion with the appendix was found. The pus was gently mopped up, and, after a difficult search for the appendix, it was removed. He had normal saline injection by the bowel as

recommended by Murphy of Chicago, and afterwards subcutaneously. He was maintained in the Fowler position—that is, the semi-sitting position—to encourage drainage towards the pelvis, and away from the dangerous absorbing subdiaphragmatic peritoneal area. He had profound toxæmia for some days after the operation, then diarrhoea set in. This was favourable so far, but the motions contained varying quantities of blood, and we know how unfavourable vomiting of blood is in septic peritonitis. He became delirious, and on several occasions his condition seemed almost hopeless. But Mr. Buckell stuck tenaciously to him, and we persisted for days with subcutaneous injections of normal saline solution and strychnine. He was thus kept going until the toxins were washed out of his system. He is now well, and able to go out.

With regard to the treatment of duodenal ulcer, do you wonder that, with an experience of 6 deaths among the last 10 cases I have seen, whenever I make a definite diagnosis I urge operation—a posterior gastro-jejunostomy? I am afraid to wait, for in the first 2 cases I was quite unprepared for the sudden and fatal haemorrhage; the next 2 cases I advised operation, which was refused; and in the last 2, after haematemesis and melaena, we arranged for an operation, but one died during the night from recurring haemorrhage, and the other on the morning of the day of the proposed operation from the same cause. I am inclined to believe that whenever duodenal ulcer has reached the stage of being recognized by definite symptoms it is not amenable to medical treatment.

Well, gentlemen, I fear I have trespassed on your good nature. Incidentally I have pointed out what I have considered the duty of the physician to the surgeon. To the surgeon may I say, when in doubt do not hastily operate, for that way lies the tendency to neglect clinical observation or to shirk the trouble of accurate diagnosis? Also, with whatever class of patient we may be dealing let us all remember the warning on the little French mechanical toys: "*Quelqu'elle soit très solidement montée il ne faut pas brutaliser la machine.*"

In the compilation of this paper I am indebted to the writings of Mr. Mansell Moullin, Dr. Hale White, Mr. Mayo Robson, Dr. Wm. J. Mayo, and Mr. George Heaton.

NOTES OF AN EXPERIENCE OF STOVAIN AS A SPINAL ANALGESIC IN 100 CASES.

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THE very admirable paper of Mr. Barker which recently appeared in the JOURNAL on the subject of spinal anaesthesia was doubtless read by many with great interest, and probably with as great satisfaction, for a careful perusal of the facts given by him must to any unprejudiced mind, justify the statement he makes that there is a place for spinal anaesthesia. With this statement I entirely agree, for, while my experience is not so extended as Mr. Barker's, I have used the method in over 100 cases; and, although I have had a good many failures, in the successful cases there has been produced a most admirable analgesia. Nearly all of the patients whose operations have been done under the influence of the stovain have been greatly pleased with the complete absence of pain, and much satisfied with the fact that their consciousness was not abolished—a factor of very great importance to a very large number of people.

Some of my patients have had experience of the effects of chloroform or ether, either before or after the stovain, but all of these unhesitatingly give the preference to stovain. One patient has had stovain four times, and has elected every time after the first to have it rather than chloroform or ether. Previous to using stovain, I had tried cocaine alone, and cocaine along with adrenalin, but every time with entirely unsatisfactory results, probably largely due to defective technique; the cocaine injections were all made through the third lumbar space, and in several of them there was a defective flow of the cerebro-spinal fluid. After these failures I used Chaput's solution of stovain in two or three cases, but all were unsuccessful; quite recently, however, I have twice used Chaput's solution with complete success in two patients, and believe