An Address

ON

THE TREATMENT OF ABDOMINAL EMERGENCIES.

Delivered before the Middlesbrough and District Medical Society on Oct. 29th. 1902,

By J. RUTHERFORD MORISON, M.B.,
F.R.C.S. EDIN.,

SURGEON TO THE NEWCASTLE-ON-TYNE ROYAL INFIRMARY.

MR. PRESIDENT AND GENTLEMEN,—When your secretary, Dr. R. E. Howell, honoured me with an invitation to address you on some subject of practical interest it seemed to me that I could not do better than to offer for your consideration some of the thoughts and information inspired by my experience of abdominal emergencies.

Nothing shows more clearly the completeness of the surgical revolution, initiated by Lord Lister, than a recognition of the fact that 25 years ago we were afraid to open the abdomen lest septic infection resulted, while at the present time the most necessary and useful of abdominal operations have as their chief object its prevention. The time has come, however, when we should cease to wonder at the "marvellous advances of abdominal surgery" and begin to expend our energies on considering how future progress is to be brought about. I do not doubt that this is to be sought for in the prompt treatment of abdominal emergencies; that the most important advances depend upon your care and skill; that they are not to be expected from the ingenuity and daring of mere operators, but from more prompt diagnosis and more vigorous action on the part of the general practitioner. My aim to-night is to enforce with all the energy I can the fact that in the treatment of abdominal emergencies the prevention of septic infection of the peritoneum easily occupies a first place.

Experience in the post-mortem room and operating theatre has taught me that in at least 90 per cent. of patients dead in consequence of abdominal emergencies death has been brought about by septic infection of the peritoneum, and in the great majority of cases of general peritonitis I have found that the more elaborate and thorough the operation undertaken for its treatment the more quickly the patient dies. The following brief notes are from examples of the most tragic and appalling abdominal emergencies.

CASE 1.—A young woman, aged 26 years, employed in a shop, had tea at five o'clock one afternoon. An hour later she shut up shop and started to walk home. When not more than 100 yards from the door she was seized with such sudden severe pain that she had to support herself against the wall and was helped back. When a medical man saw her an hour later she was in a condition of collapse and complaining of agonising abdominal pain. She had never had any rest since. Three days later, when I saw her, she was obviously dying from general peritonitis and I obtained a history of previous stomach troubles of three years' duration. No operation was performed. Post-mortem examination showed a perforating ulcer on the anterior wall of the stomach and general peritonitis.

CASE 2.—A robust man, aged 30 years, was awakened at three o'clock in the morning with a "grumbling" pain in his abdomen. He fell asleep again and got up to go out at 5.30, feeling uncomfortable inside. He worked all day in discomfort. Just before leaving work a sudden severe pain seized him and doubled him up. He vomited and broke into a cold sweat and had to be taken home in a cab. A medical man saw him at once and found him collapsed, in great pain, and vomiting frequently. Repeated hypodermic doses of morphia relieved his pain but he had not slept four days later when I saw him. He was then moribund. Post-mortem examination showed a gangrenous perforated vermiform appendix and general peritonitis.

CASE 3.—A healthy woman, aged 35 years, was sitting quietly at tea with her family when she suddenly jumped up, pressed her hands on her abdomen, called out, "Oh, my God, what a pain," and fell on the floor unconscious. A quarter of an hour later she was seen by a medical man who found

her to be unconscious and in a condition of profound collapse. With energetic stimulation and treatment she had recovered consciousness next day and her pulse could be felt. Three days later, when I saw her, the abdomen was much distended and tense, there were signs of free fluid in it, and though her general condition was bad it had improved. Operation showed an enormous quantity of blood in the peritoneal cavity and a ruptured pregnant Fallopian tube. The small intestines were enormously distended; all efforts to get the bowels to act by medicines and enemata failed and the patient died three days after the operation. Post-mortem examination showed only enormously distended anæmic intestines.

CASE 4.—An athletic young man after supper in the house of a friend was reaching upwards for a book when he was suddenly seized with agonising abdominal pain. He was so ill that he had at once to be carried to bed and a medical man found him in a condition of collapse. He gave a definite history of indigestion causing pain one hour after food, but otherwise he had been exceptionally healthy. A week later, when I saw him, he was fully under the influence of opium, but had never slept for more than a few minutes at a time and he was dying. Post-mortem examination showed a perforating duodenal ulcer and general peritonitis.

In each case related the medical man in attendance formed a definite opinion at first sight that some abdominal catastrophe had occurred and that recovery was most unlikely. These may be designated the desperate cases.

The first proposition I have to offer for your discussion is, that the most acute and serious abdominal injuries and diseases give rise to such characteristic symptoms and signs that a probable diagnosis can be made. The diagnosis need not, however, be profound. It amounts to this. Some grave abdominal lesion has occurred. It is most unlikely that the patient will recover unless something can be done to prevent peritonitis or to arrest a possible internal hæmorrhage, and the probabilities are that the patient, if left alone, will die before the end of the fifth day.

What should be the immediate treatment in cases such as I have described above? The answer first concerns the use of opium. Such agony as the patient suffers demands relief and you cannot and will not refuse to give it. Experience has taught us that not only relief but real temporary good, by the diminution of shock, follows the use of morphia. So much immediate good is effected by the dose that the main objection raised against it by surgeons is that the improvement in the patient is apt to lead to the serious error of a failure to recognise the gravity of the condition. The possibility of such a failure constitutes a real danger if no opinion has been formed before the administration of the morphia.

The first duty of the practitioner on seeing an abdominal emergency case is to arrive at a definite conclusion as to whether it is one of these calamitous cases or not. This will not take long to decide if a person previously in fair health is suddenly reduced by agonising abdominal pain to a condition of serious collapse. When this has been settled, but not till then, one-third of a grain of morphia should be injected hypodermically. The next step is to arrange for an operation without a moment of unnecessary delay and I hold very strongly the view that the patient should be conveyed, with every care of course, to a hospital, public or private, if the best chance of recovery is to be given. The grave symptoms have all been relieved or abolished by the dose of morphia; nevertheless, with the courage of conviction derived from previous knowledge immediate operation must be insisted upon. And what are the chances offered by early operation in such cases as those mentioned? The answer is that the mortality-rate could be very considerably reduced. Can I offer you any statistics of my operations for abdominal emergencies in support of this statement? Honestly, I cannot, and if I could I dare not; sufficient early cases are not available and the results of operations upon patients with general peritonitis are too horrible for publication. But if time allowed I could relate many cases which support my contention. Here two must suffice. They were both young women and both were healthy except for stomach troubles. Both had gastric ulcers which perforated and each had the good fortune to have as their medical attendant Dr. Robert Campbell. He made at once a diagnosis of ruptured gastric ulcer in each and I believe I am correct in saying that he never left either until he saw them into the infirmary and was assured by having seen it commenced that an operation was to be done. In both I

performed a similar operation, first making an incision in the epigastrium to verify the diagnosis, and finding gas and stomach contents in the peritoneal cavity I extended the incision from the ensiform cartilage to close above the pubes. I then turned out and thoroughly washed with hot saline solution (one teaspoonful of salt to one pint of water) the intestines and whole peritoneal cavity, taking special care to clean every pocket and in each repaired a large perforation through a stomach ulcer by sutures. In both the abdomen was closed entirely without drainage. This comprises my whole experience of perforating gastric ulcer operated on within 12 hours of the perforation and the two patients made straightforward recoveries.

Before leaving these cases let me ask to whom are the patients indebted for their recovery. All I can say is that a few hours of delay to try the effects of morphia and hot poultices would have given the operator discredit instead of appreciation. Is it too bold to prophesy that a mortality-rate of at least 90 per cent. will be reduced by early operation to one of not more than 10 per cent.

Unfortunately, the treatment of an abdominal emergency, too often from delay, resolves itself into the treatment of septic infection of the peritoneum, and a few minutes spent in giving you shortly my views on this important subject may not be wasted. The peritoneum is credited with a susceptibility above the average to suffer from septic infections. I feel confidence in stating that the reverse is the fact and that with the exception, perhaps, of the puerperal uterus it possesses a resistance greater than other portions of the human body. In previous papers¹ I have advanced evidence in favour of this and other contentions, but time does not to-night allow of more than what may appear to those who have not read my papers to be too dogmatic statements. It is now recognised that septic infection of the peritoneum may cause death by acute poisoning before time has been given for the manifestations of peritoneal reaction which we call peritonitis. For this condition we can as yet do nothing; the peritoneum and the patient are overwhelmed by a virulent poison. Fortunately the more common result of the introduction of septic contents into the abdominal cavity is that the peritoneum reacts and peritonitis results. This may be limited and local or diffuse and general, according to the virulence or amount of the infective agent, or the power of resistance in the patient's tissues, or the position of the lesion in the abdominal cavity. It is only with the treatment of general septic peritonitis that we are at present concerned and of this alone that I intend to treat.

Many pathologists and surgeons hold that every patient with general septic peritonitis dies and that recovery means a mistake in diagnosis. The majority of surgeons believe that the only chance of recovery is offered by operation. I do not agree with either view, for I have, in one of the papers previously mentioned,² offered the strongest evidence against each and could, if time permitted, add still more. There can be no doubt, for I have abundant evidence of it, that patients recover whose peritoneum in every nook and corner has been covered with purulent fluid or purulent lymph, the result of infection from a ruptured gastric ulcer or perforated appendix or similar gross septic source, and it is mere quibbling to call this anything but general peritonitis. It has been my experience that a fair percentage of patients with general peritonitis who have lived over the fourth day can recover, and I would like to draw your attention to the clinical fact that no case is hopeless in which the abdomen though distended is not tense. A patient with tense and much distended abdomen I have never, so far as my recollection goes, seen to recover. At the end of a few more days such immunity has been acquired that in the third week of an illness due to appendicitis I have opened a peritoneum charged with pus and faeces and the patient has got well.

Surgical treatment does not always, I believe, offer the best chance in the treatment of acute general peritonitis, and this I take to be the guide as to surgical or medical treatment. Operation when once the abdomen has become distended is too late. If operation is decided against the first step indicated is to stop the administration of all food and drink by the mouth and to give the required nutriment and fluids by enema. The next is to wash out the stomach with abundance of hot water as frequently as vomiting is troublesome. The most efficacious method of doing this is through

the stomach-tube and patients usually obtain such relief from its use that they raise no objection to a repetition of it. As a substitute a pint of hot water or a pint of champagne may be offered as a drink. The third necessity is to keep the patient perfectly quiet and for this purpose small and repeated doses of morphia are administered hypodermically. A linseed meal poultice large enough to cover the whole abdomen should be applied for an hour and repeated every eight hours, abundance of heated cotton-wool being substituted in the intervals between the poultices. Heat is the greatest promoter of cell activity and it is largely on this that reliance is placed in the natural "cure" of peritonitis—hence the recommendation. Last, but not least, a good nurse is essential to the best management of these dangerous cases.

Fortunately the majority of abdominal emergencies are not so tragic as those just described and the next most severe may be described as grave cases. They also commence suddenly with severe pain and vomiting, but shock is moderate in degree. Under this heading may be included small perforations of the stomach, perforations of the intestines and vermiform appendix, acute intestinal obstruction, ovarian cysts with twisted pedicle, and some acute gall-stone cases. The treatment of the most serious of these cases is the same as that of the first class—viz., immediate operation.

Of the less serious it is similar to that of the next class, which I designate serious cases. This class is the most important because the most ordinary. The patient has severe abdominal pain and may be seriously ill but there is no evidence of immediate danger. The general aspect and the pulse are good and the skin is warm. In these cases the keenest effort should be made to form as accurate a differential diagnosis as is possible. What could be more uncalled for than to open an abdomen for intestinal obstruction due to a femoral hernia? The best that can be said of such a proceeding is that it is not so disastrous as leaving things alone. "Examine the hernial sites" should be the first demand made by the practitioner on himself in every abdominal case attended by vomiting. I have emphasised this repeatedly in published papers but neglect of it still causes the loss of many lives. It is in other instances equally important to the patient that the surgeon should operate with a knowledge of which part of the abdomen to enter in order to gain direct access to the seat of the lesion. I must here remark that I have no sympathy with those who say that abdominal diagnosis is so difficult that it can only be made by exploratory means. This is not true. A correct diagnosis is possible in the majority of cases if due care be taken in listening to the history of the illness, in noting the physical signs, and in a careful balancing of the problems suggested by both. The greatest importance is naturally attached to physical signs, but to ignore the history, as has been advised, is sure to lead to the gravest, because avoidable, errors in diagnosis. A tumour is present in the right side of the abdomen and the temperature is raised. Is it a large suppurating gall-bladder due to a stone blocking the cystic duct or a large suppurating kidney due to stone in the ureter? Is it most important in the patient's interest to discover, not always an easy task, whether the colon lies behind or in front of this tumour or to learn that previous attacks have been followed by jaundice or by hæmaturia? Is the history of a previous hernia or of an attack of appendicitis or of tuberculous peritonitis of less value than the physical signs in determining the cause of a large number of cases of intestinal obstruction? In the female does not the history of the existence of a previously harmless tumour with sudden peritonitis, without other signs, make the diagnosis of ovarian tumour with twisted pedicle probable? And does not the history of a menstrual period missed for a week or more and followed by acute abdominal illness in a healthy woman previously never irregular suggest more clearly than any sign or combination of signs that her condition is the result of ectopic gestation? The second proposition I have to make for your discussion is this: That in all except the most acute abdominal emergencies it is possible to make a differential diagnosis sufficiently accurate to suggest the most suitable treatment.

No one in this room, I presume, doubts that the proper treatment of strangulated hernia or suppurating gall-bladder or kidney, acute intestinal obstruction, or acute appendicitis, or ovarian tumour with twisted pedicle, or ectopic gestation, is operation at the earliest convenient hour. It is when doubts arise in the diagnosis that difficulties in the treatment follow. The question must be of this nature to make the treatment other than surgical. Is the patient seriously ill or is the illness due to some trivial or temporary upset

¹ Edinburgh Medical Journal, July, 1873; Northumberland and Durham Medical Journal; THE LANCET, Feb. 23rd, 1901, p. 533.

² THE LANCET, Feb. 23rd, 1901, p. 533.

in a hyperæsthetic individual, and is it practically certain that the condition of the patient or the nature of the disease is such that surgical treatment has more danger than the disease itself? The most difficult cases to give a decision upon are those of suspected intestinal obstruction. The pain at first may not be bad enough to raise alarm and its paroxysmal character and accompanying wind-rumbings may be placed to the credit of dyspepsia, or colic, or constipation, or some other more trifling trouble. During their stay in my wards several patients have developed symptoms of acute obstruction due to bands and adhesions and I will mention in detail the treatment which we have adopted for them. If the pain has been severe and paroxysmal and has been attended by vomiting and wind-rumbings and visible or palpable peristaltic movements of the intestine making the diagnosis definite, immediate operation has been the rule. If the diagnosis has been less certain one-quarter of a grain of morphia injected hypodermically has been ordered. If the symptoms are removed by this and do not reappear after eight hours the condition is probably due to nothing serious, but whether or not, a dose of castor oil and an enema are given to clear up the remaining difficulty. If the case is one of intestinal obstruction acute symptoms of obstruction are produced and operation is undertaken at once. The results of this treatment will carry greater weight than anything I can say.

In the absence of both of our surgical registrars I am indebted to Mr. J. W. Heslop for examining the hospital records from 1900 up to date. He has found a note of 11 patients who were attacked by acute intestinal obstruction whilst under my care and suffering from other conditions. Of these, nine recovered and two died. One death occurred in a patient who was not operated upon from mistaken diagnosis. Of 10 patients operated upon nine recovered and one died. The patient who died was a young man whose appendix had been removed two days previously and who developed a complicated volvulus, involving the small intestines.

It cannot be held of these cases that they were easy from the surgical point of view. Every one required more serious manipulation than the reduction of a recent intussusception, or the release of bowel strangulated by a band, or the reduction of a kinked or twisted loop of intestine necessitates. To prove this I need only read notes of the last case of the sort that I operated upon, to be quite fair prefacing it by the statement that the patient was an unusually robust young woman.

CASE 5.—The patient, a female, aged 35 years, was operated upon first on June 10th, 1902. She was then very ill, with an acute abdominal illness diagnosed as abscess in the pelvis and in connexion with the vermiform appendix. Her abdomen was opened over the right iliac fossa by an incision extending from the loin behind. Some clear fluid escaped on opening the peritoneum. The colon and the cæcum were adherent to the parietal peritoneum on the outer side and on gently separating them foetid pus escaped. The appendix was found lying on the outer side of the cæcum and colon with its tip pointing upwards to the hepatic pouch and was excised. The pelvic brim was filled with matted adherent intestine. The abdominal wound was covered up and the patient was placed in the lithotomy posture, the pelvic abscess, from which a large amount—about one pint—of foetid pus was evacuated, being opened and drained through an incision in each lateral vaginal fornix by drainage-tubes sewed in. The vermiform appendix contained a stercolith and was perforated at its centre. There was a free discharge of pus, especially from the vagina, for some days, but the patient made a good recovery and was about to leave when, on the night of July 14th, one month after the operation described, she was suddenly seized with symptoms of acute obstruction and became very ill. The second operation was performed a few hours later. The abdomen was opened in the middle line. The small intestines were congested and beginning to distend, the distended loop leading to the pelvis where the obstruction was located. It was due to adhesions, many of which were easily separated, but those in the depths of the pelvis became more dense. In separating a loop there a small abscess was opened and pus escaped. The traction on the inflamed and adherent loop resulted in tearing the intestine almost completely across. Six inches of torn and damaged intestine were excised and an end-to-end approximation was made by catgut sutures. The abscess in the pelvis was thoroughly mopped and the whole pelvis was washed out with saline solution. The abdomen was entirely closed. The patient recovered and is now in first-rate health with firm scars¹

These results have not depended on any special operative skill of mine, for some have come under the care of my assistants and their cases have done as well as my own. As additional proof of this I may state that my results from operations for acute intestinal obstruction involving the small intestine, performed on the third or fourth day when the small intestines are distended, have been so bad that I could count the recoveries on my fingers. There is only one explanation of these surprising results, and I want to drive the lesson this experience has given me as strongly home as I can, and it is that if patients with acute intestinal obstruction are to have the best chance they must submit to operation without delay. As further bearing on this point I may mention the results obtained at the infirmary in intussusception and strangulated hernia. From the commencement of 1900 12 cases of acute intussusception have been admitted to the surgical wards of the Royal Infirmary for operation. In four of the cases the gut was found to be gangrenous and all of the patients died. Of the eight cases in which the gut was not gangrenous six patients recovered and two died. One of the deaths was caused by a second intussusception which was not discovered till the post-mortem examination. The second death occurred in an acute case of 24 hours' duration in a child five months old. The records of strangulated hernia are even more instructive, for ten years ago the operation for the relief of strangulation was as skilfully performed as it is now, and the superadded danger of a radical cure was less frequently incurred, but from the infirmary reports I find that of 11 patients suffering from strangulated inguinal hernia five recovered, four died, and two remained under treatment in 1891, whilst in 1901, of 22 patients 18 recovered, three died, and one remained under treatment. The cause of death in two of the last mortal cases I know was gangrene of the gut owing to delayed operation, and in one case this was due to the obstinate refusal of the patient to submit to early operation.

Every practitioner of experience has seen recovery ensue in the most formidable-looking cases of obstruction under the ordinary routine treatment by opium, enemata, &c., but it must be remembered that in acute cases the mortality without operation is not less than 80 per cent., and I believe that when the small intestines have been allowed time to undergo considerable distension the best chance for the patient is not often given by operation. In this class the most common forms of appendicitis and gall-stone cases may be placed. They resemble each other in many ways but chiefly in the uncertainty of prognosis. The most serious-looking cases may get well without operation and the less serious, to all appearance, end somewhat suddenly in disaster. Hence the endless discussions as to when to operate upon cases of appendicitis and cases of gall-stone. Examples of both occur in which immediate operation offers the only probability of recovery, but fortunately such desperate cases are rare. Never a month passes, however, but I see a fine young man or young woman dying from general peritonitis due to appendicitis after an illness of four or five days' duration, and it is difficult then to think patiently of the advocates of an indiscriminate medical treatment of appendicitis, knowing that with practical certainty an operation performed on the second day would have saved this life. My view is that every acute case should be operated upon without unnecessary delay if after a single dose of morphia, and at the end of eight hours, every symptom and sign of danger have not disappeared. The risks of removing the acutely inflamed appendix have been exaggerated. I do it in every early acute case I see, and expect the patient to recover unless a diffuse peritonitis, already present, has secured too strong a hold to be remedied by the removal of its cause. The presence of a definite tender tumour following an acute attack, as I have elsewhere pointed out, denotes with certainty the formation of an abscess in connexion with the vermiform appendix and the only satisfactory treatment for this is to remove the appendix and to drain the abscess. By earlier operation a serious illness, a difficult operation, and a prolonged convalescence would have been avoided, so that I have come to regard abscess as evidence of an opportunity lost. In many of these cases of abscess the patients get well if left alone, the pus usually finding its way into an adjoining adherent coil of intestine; but operation is safer (in our last 100 hospital cases there was no death) as rupture of the abscess into the peritoneal cavity is not impossible. Operations on cases of gall-stone during the past 18 months have more

than ever impressed upon me the need for more frequent and earlier operations, and this because I have had the misfortune to lose a considerable number of patients after operation from conditions due to prolonged delay. Some cases of gall-stone come under the heading of desperate conditions, more than can be known, unless operations are performed or post-mortem examinations are obtained. As an example of this I offer the notes of my most recent case of this sort.

CASE 6.—The patient was an exceptionally healthy robust man, aged 50 years. Until his fatal illness he had never been ill a day in his life. His only complaint was of temporary attacks of severe "stomach cramp" to which he had been subject for years. He had never been jaundiced. On Sunday night he had a sudden severe attack of pain. On Monday his distended gall-bladder was felt. On Tuesday he had signs of peritonitis round the gall-bladder and a cough. On Wednesday there were symptoms and signs of pneumonia. On Thursday very acute abdominal pain and signs of spreading peritonitis were present. On Friday the abdominal conditions were worse. Operation showed diffuse purulent peritonitis in the upper half of the abdomen and a gangrenous perforated gall-bladder with three gall-stones in it, one of which was firmly impacted in the cystic duct. Death occurred on Saturday. A post-mortem examination showed double pneumonia. The peritoneal cavity was clean.

You will note that the diagnosis in this case was based upon a history of "spasms," a tender, swollen gall-bladder, a high temperature, and that there was no jaundice. Unless a stone has got into one of the liver ducts, jaundice, of course, is not a symptom and this is apt to be forgotten and so mistakes arise. A confident diagnosis of a stone, or stones, impacted in the neck of the gall-bladder or in the cystic duct, with cholecystitis, can be made in similar cases. The safest and most satisfactory treatment of them is by early operation, though it must be remembered that spontaneous recovery by rupture of the gall-bladder into the peritoneal cavity with absorption of the fluid contents of the gall-bladder and encystment of the stones by adhesions, or by rupture into a hollow viscus to which it has become adherent, or by rupture externally, may occur. If time permitted I could relate cases demonstrating each of these methods. If gall-stone surgery is to show better results in the future than in the past patients must be operated upon whilst the stones are still confined to the gall-bladder, from which they can be removed by a simple and safe operation.

The accompanying list made for me by Mr. G. Grey Turner, surgical registrar of the Royal Infirmary, is a complete record of our fatal cases of gall-stone for the past 18 months.

1.—A male, aged 50 years. Condition: very ill; high temperature. Gall-stone in the cystic duct; gangrene and rupture of the gall-bladder; septic peritonitis. Operation: laparotomy; cholecystotomy. Result: death. Remarks: died 16 hours after operation. Necropsy: gall-bladder showed patches of gangrene with a perforation; no further stones; septic peritonitis in the upper abdomen, principally on right side; broncho-pneumonia of both lungs.

2.—A male, aged 50 years. Condition: very ill; high temperature; slight jaundice. Stone impacted in the cystic duct. Gangrene of gall-bladder; localised peritonitis. Operation: laparotomy; cholecystotomy. Remarks: died six hours after operation. Necropsy limited to operation area. No other stones found. Liver very fatty.

3.—A female, aged 54 years. Condition: very ill; high temperature; slight jaundice. Stones in gall-bladder. No pus found. Patient too ill to explore ducts. Operation: cholecystotomy. Remarks: died seven days later after a few hours' illness with high temperature and jaundice. Necropsy showed a sloughing gall-bladder. One stone found impacted in lower end of common duct and projecting into duodenum. Many stones higher in duct. Two stones in hepatic ducts. Septic cholangitis. Other stones in small hepatic ducts and one free in a liver abscess.

4.—A female, aged 38 years. Condition: very ill; high temperature. Removed stones from gall-bladder and liver substances. Multiple abscesses in liver round stones. Operation: laparotomy; cholecystotomy. Remarks: recovered sufficiently to leave hospital with sinus. Died at home three months after operation.

5.—A male, aged 62 years. Condition: deep jaundice. Fistula over gall-bladder the result of old suppuration. Stone felt at bottom of sinus. Operation: stones removed by enlarging sinus in abdominal wall. Remarks: died ten months after operation. Cause could not be ascertained. No necropsy.

6.—A female, aged 33 years. Condition: slight jaundice; high temperature. Large stone removed from pus cavity behind common duct. Gall-bladder small and contracted. No stones in gall-bladder or ducts. Operation: laparotomy. Remarks: died ten days after operation. Post-mortem examination; no further stones found. Stricture of pylorus. Nothing surgical found. Death accounted for by syncope.

7.—A female, aged 67 years. Condition: intense jaundice; very ill; high temperature. Large number of stones removed from gall-bladder; one from cystic duct; none from common duct. Operation: cholecystotomy. Remarks: died 16 days after operation after an acute abdominal attack. Necropsy showed small stone impacted in duodenal orifice of common duct. *Bacillus coli communis* from fluid of ducts. Liver bile-logged. Heart fatty.

8.—A male, aged 55 years. Condition: very ill. Stones removed

from gall-bladder and cystic duct. Operation: cholecystotomy. Remarks: died ten days later. Necropsy showed an abscess around the appendix with suppurative phlebitis. Operated on by mistake on the supposition that the illness was due to gall-stones. Bile-ducts healthy. No stones left.

9. A female, aged 23 years. Condition: very ill; high temperature. Removal of stones from gall-bladder, latter inflamed; omentum contained ten drachms of pus and was excised along with gall-bladder. Operation: Cholecystectomy. Remarks: died seven days later, quite suddenly, when convalescent. At necropsy nothing surgical found to explain death; no further stones; syncope.

10.—A female, aged 54 years. Condition: good, except for chronic bronchitis and asthma. Stones removed from gall-bladder; there had been an old perforation. Mucopus in gall-bladder. Operation: cholecystotomy. Remarks: died six days later from acute double lobar pneumonia. No other stones found. Abdomen and wound healthy.

11.—A female, aged 22 years. Condition: good. Pus in gall-bladder from which stones were removed. Operation: cholecystotomy. Remarks: died 24 hours after operation from broncho-pneumonia. No other stones found at necropsy. Wound and abdomen healthy.

12.—A male, aged 60 years. Condition: good. Stones removed from gall-bladder. Operation: cholecystotomy. Remarks: died three days after operation. Acute pneumonia of right base. No further stones found at necropsy. Wound and abdomen healthy.

You may ask, Are there no disadvantages to be advanced as reasons against such surgical activity as I have suggested? Of course there are, and the dangers and difficulties must be fairly admitted. I am not one of the optimists who hold that after opening the abdomen, if it is found that nothing serviceable has been accomplished, "no harm has been done." It is poor consolation to offer for this view that the patient recovered. The recovery must be recognised as having occurred in spite of, and not in consequence of, surgical interference, and the cause of the error must be fully studied as a guide to the avoidance of similar mistakes in the future. Some mistakes are unavoidable, and it is for such that the old saying, "He who makes no mistakes makes nothing," must be offered as an excuse. Intestinal obstruction due to general firm matting of the intestines, usually the result of healed tuberculosis, acute hæmorrhagic pancreatitis, acute malignant peritonitis, and other incurable conditions, may be offered as examples of my meaning, for by our present means of diagnosis they cannot be excluded.

I will conclude by mentioning two more common causes of error which have been forcibly impressed upon me by personal experience of them. Some three years ago I opened the abdomen of a young woman sent into the infirmary as a case of ruptured gastric ulcer a few hours after the onset of sudden agonising pain in the upper abdomen, attended by collapse, preceded by a history of stomach trouble and followed by rigidity and tenderness of the abdominal wall. Nothing was found to account for the attack, but next day the signs of pneumonia at the left base were present and the disease ran its usual course, the patient recovering. Since then I have more than once escaped a similar disappointment by remembering that the pain attending chest inflammations may be referred to the abdomen and produce symptoms closely simulating those of acute peritonitis. This fact should have been sufficiently impressed upon me previously, for my brother, Mr. A. E. Morison of Hartlepool, in a paper on *Empyema* published in the *Northumberland and Durham Medical Journal*, had pointed out how the abdominal pain and rigidity of muscles and vomiting accompanying empyema might simulate abdominal disease and lead to error, and Dr. David Drummond has taught for years that chest inflammations in their early stages can be readily mistaken for peritonitis.

In THE LANCET of August 2nd, 1902, p. 280, Mr. H. L. Barnard of the London Hospital published an excellent paper recording in detail several cases of pneumonia mistaken for abdominal disease and treated by abdominal section, so that the possibility of forgetting this in the future may be lessened by this reminder.

The second cause of error has, so far as I am aware, not previously been noted. The subject is, however, so important that I hope shortly to have a series of cases fully recorded and must ask you, in the limited time at my disposal, to accept a bald statement of the facts.

A patient usually at or past middle age is suddenly seized with all the symptoms of an acute abdominal illness, so definite that a diagnosis of ruptured gastric ulcer, intestinal obstruction, strangulated hernia, or some such lesion, followed by peritonitis, is the diagnosis made. The abdomen is more or less rigid and more or less distended, and operation if it be undertaken reveals no abdominal lesion. Post-mortem examination in typical cases shows only an increase of fibrous tissue in the kidneys, which are granular on their surface and adherent to their capsule. In at least six of such cases I have seen the abdomen opened—by myself or by

my colleagues—in the belief that the condition was due to some surgical calamity, and guided by past experience in several cases recently the true state of affairs has been realised by Mr. G. G. Turner and Mr. Heslop who are associated with me in the infirmary, and myself in time to avert an operation on cases sent in for this purpose. The curious grey, apathetic look of the patient, with urine of low specific gravity and sometimes containing blood and albumin, has suggested to us to exclude the form of kidney mentioned before making a definite diagnosis of abdominal disease.

The last conclusion that I have to offer for your discussion is the proposition with which I introduced my paper; that future advances in abdominal surgery depend more on the general practitioner than on anyone else and that the treatment of abdominal emergencies is early operation.

FURTHER INVESTIGATIONS REGARDING THE INFECTIVE NATURE AND ETIO- LOGY OF PERNICIOUS ANÆMIA. (25 CASES.)

(SYN.: "IDIOPATHIC ANÆMIA OF ADDISON.")
ITS NON-IDENTITY WITH THE "PROGRESSIVE PERNICIOUS
ANÆMIA" OF BIERMER.¹

By WILLIAM HUNTER, M.D. EDIN., F.R.C.P. LOND.,
PHYSICIAN TO THE LONDON FEVER HOSPITAL; EXAMINER IN MEDICINE
IN THE UNIVERSITY OF GLASGOW.

I.—INTRODUCTORY.

AMONG the conclusions arrived at in the investigations recently recorded² there is one on the subjects raised by the title of this paper which has not merely a special but a more general interest alike for physicians and for pathologists.

As is well known, the severe condition of anæmia above referred to was first made known to English physicians by Dr. Addison in 1855 in a masterly account. He gave it the title of "idiopathic anæmia" and under that name it excited the attentive interest not only of the best English physicians but also of some of the best Continental observers. A distinguished reviewer has recently testified³ from his own experience as far back as 1860 to the interest taken at that time in the disease by Trousseau in Paris.

In 1871 a condition which in many of its features seemed the same as that already known to us as idiopathic anæmia was described by Professor Biermer of Zürich under the name "progressive pernicious anæmia." This account was hailed in German literature as that of a new disease, and under that name or that of "Biermer's anæmia" it is commonly spoken and written about by German observers up to the present time, although it was clearly established by Dr. P. H. Pye-Smith (1875 and 1883), one of Addison's own pupils, that the disease had been known to English observers for more than 15 years. This fact was fully admitted by Professor Eichhorst, the author of the most important German monograph on the disease: "We come to the result that this [the "primary," "idiopathic," "essential," "proto-pathic," "cryptogenetic"] form of pernicious anæmia was first described in England, both in the form of single cases and as a definite clinical disease."⁴ This just recognition came, however, too late (1878). With the stamp upon it of "a new disease" the new title of "progressive pernicious anæmia" rapidly made its home in English literature—the term "idiopathic" being either given a generic significance as including both chlorosis and pernicious anæmia,⁵ or being reserved for only one form of "progressive pernicious anæmia," that termed by German workers the "primary" form.

For the last 20 years, as already stated, it is under the name of "progressive pernicious anæmia" or the shorter title of "pernicious anæmia" that the disease has been known. The title "Addison's anæmia" is only rarely

applied to it in English literature although most English observers have Addison's account in their mind when they speak or write of "pernicious anæmia." On the other hand, the title "Biermer's anæmia" is one of the commonest names given to it in German literature, the latest modification of this, suggested as a tribute to Professor Ehrlich's work upon it, being that of "Biermer-Ehrlich anæmia" (1900).

II.—NON-IDENTITY OF ADDISON'S ANÆMIA AND BIERMER'S ANÆMIA.

The general assumption has been that Addison's and Biermer's accounts refer to one and the same condition. In German literature the account given by Addison is regarded as of a subordinate interest—relating merely to one form (so-called "primary") of what they consider should be included under the designation of "progressive pernicious anæmia." This position has been maintained consistently throughout in German literature as one quite unassailable and in many cases in English literature as well. (See Scheme.)

Now, the particular name a disease is known by may be a matter of relatively little importance provided that all are agreed as to the unity of the disease itself. But the matter is otherwise if any question exists from the very outset whether we are really dealing with one and the same disease—whether Addison's conception of "idiopathic anæmia" and Biermer's conception of "progressive pernicious anæmia" really relate to the same diseases, and it becomes one of real importance if it turns out as I propose now to show—that the two conditions are not identical.

1. The anæmia which Addison described is really a remarkable infective disease with definite antecedents, mode of origin, infective and hæmolytic lesions, and clinical course.

2. On the other hand, the investigations which have made this clear have also made clear that the condition described by Biermer is, as its supporters truly maintain, "not a disease *sui generis* but a frequently recurring group of symptoms met with in very different conditions of disease" (1900), "not an etiological unity but only a symptomatic group which can be produced by causes of all kinds" (1900), the most common cause being, as I hope to show, septic infection, this being the feature that has led them to be confounded with Addison's anæmia.

With investigations that have led up to these very definite conclusions I now propose to deal—not in any narrow spirit as if it were simply a question of international priority of observation, but in a broader spirit commensurate with the importance of the clinical and pathological facts involved. As regards the spirit in which I approach the international aspect of the subject I cannot do better than quote the judgment of a recent reviewer: "In his history of the observation and interpretation of pernicious anæmia, from the date of its discovery down to the present time, the author seems to us perhaps to err on the side of generosity to Biermer."⁶ To this I can add that from first to last during the progress of my investigations of this disease since 1885 the international aspect of the subject is the one that has interested me least. It was under the name "progressive pernicious anæmia" that I learnt of the disease from my teachers in the University of Edinburgh (1878–83). When I learnt later (1887–88) of the question of priority between Addison and Biermer this seemed to me to be of quite subordinate interest in view of the fact that all observers were equally agreed about the totally mysterious nature of the condition or conditions they severally described.

III.—FUNDAMENTAL DIFFERENCES BETWEEN ADDISON'S AND BIERMER'S CONCEPTIONS OF THE ETIOLOGY.

While in regard to the clinical features and absence of morbid changes described Addison's and Biermer's accounts showed no essential differences, in regard to etiology they displayed the most fundamental differences. Addison described the anæmia he had in view as "occurring without any recognisable cause whatever cases where there had been no previous loss of blood, no exhausting diarrhoea, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease. Accordingly, in speaking of this form in clinical lectures I, perhaps with little propriety, applied to it the term idiopathic to distinguish it from cases in which there existed more or less evidence of some of the usual causes, or

¹ A paper read before the Cambridge Medical Society on Nov. 9th, 1902.

² THE LANCET, Jan. 27th (p. 221) and Feb. 3rd (p. 296) and 10th (p. 371), 1900. "Pernicious Anæmia," 1901.

³ Brit. Med. Jour., Nov. 16th, 1901.

⁴ Eichhorst, 1878, p. 38.

⁵ Dr. Coupland, 1881; Dr. J. S. Bristowe, 1886.

⁶ Brit. Med. Jour., Nov. 16th, 1901, p. 1474.