Fig. 14.—Later stage; the vocal cords have been forced into apposition by the extrinsic muscles of the larynx and of the neck.

was again closed, I noticed that the respiratory waves on the blood-pressure tracing completely disappeared, although they were as well marked when the chest was freely open on one side, as in the normal condition.

My method of opening the pleural cavity allowed of fluid being easily injected into it, and I found that it was easy to kill the animal by injecting fluid under the pressure of a few inches of water. Moreover, the injection of a few cubic centimetres of 1 in 20 carbolic acid was rapidly fatal. These two points are of considerable practical importance. It has long been known that forcible injection of the pleura in cases of empyema has often resulted in the death of the patient. The ease with which death is produced by reflex respiratory stoppage by an irritant like carbolic acid injected into a normal pleura shows the necessity of employing only non-irritating solutions for this purpose.

I have also been able to confirm in my experiments what is commonly noted in operations on the chest, that an anaesthetic is usually more potent when only one lung is acting; and if in my experiments the anaesthetic was pushed dangerously far, I failed to produce the reflex mechanism of the glottis, which I regard as so all-important in these cases.

After the establishment of the conditions described with intact recurrent laryngeals, though the respiration is slowed, the animals breathed quite quietly, and though the air which was forced into the affected lung had been already once breathed, or at any rate had occupied the upper respiratory passages of the sound lung, there was never cyanosis or any other sign that aëration was insufficient.

If, in conclusion, we compare the typical case that I have described at the outset, and which suggested to me the present investigation, with the animals with which I sought to establish similar conditions, we see that in the main the mechanism by

which the affected lung is inflated by overflow from that on the sound side, owing to expiratory efforts taking place with the glottis closed, is the same in both cases.

REFERENCE.

¹ Article in Clifford Allbutt's *System of Medicine*, vol. v, p. 340.

TWO CASES ILLUSTRATING THE FALLACY OF CERTAIN PHYSICAL SIGNS IN DIAGNOSING ACUTE PERFORATED GASTRIC ULCER.

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THE two following cases may prove of especial interest to medical men in general practice whose lot it is to see cases of perforated gastric ulcer in the early stage, upon whose correct diagnosis so much depends.

CASE I.

History.—I was called to see Mary H., aged 23 (formerly in domestic service) at 10.15 p.m. on July 22nd, 1899. For the previous four months she had been living at home working at chicken stubbing, a form of employment which entails sitting nearly all day. There was a history of dyspepsia of some months' duration, but there had never been any hæmatemesis or melaena as far as the patient knew. She had done her work as usual that day, and had taken a meal at 6 p.m., consisting of tea with bread and butter. At 9.30 p.m. she was seized with severe abdominal pain, and she vomited twice but there was no blood in the vomit.

State on Examination.—The patient, who was not collapsed and was quite able to describe her symptoms, looked somewhat anæmic, and complained of severe pain in the upper part of the abdomen, which extended round both sides, and became worse every now and then. The abdomen was resonant, the liver dullness present, and there was tenderness on pressure in the epigastric region, with some resistance. The tongue was clean and moist, and the bowels had acted the day before, the motion being somewhat relaxed. The temperature was normal, the pulse 72, regular and of good volume. The diagnosis seemed to be between some form of colic and gastralgia, due probably to a gastric ulcer; the absence of collapse, especially the good condition of the pulse combined with the presence of liver dullness seemed to negative perforation. Morphine having been procured one-sixth of a grain was injected into the skin of the abdomen at 11 p.m., when the pulse had not altered. Hot fomentations were ordered to the abdomen, and it was arranged that word should be sent at 6 a.m. the following morning if the patient was no better. At that hour she was a little easier, so that, unfortunately, no message was sent until 9 a.m., when the pain was returning as severely as before. The condition then found was as follows: Abdomen somewhat distended and tender to touch, liver dullness abolished, tongue furred, pulse 100, temperature 100°. There had been no more vomiting, but some retching. As it was clear that the condition of peritonitis, then present, was due to a perforation which took place at 9.30 the previous evening, when the first very acute pain was felt, operation at home was suggested but declined; the patient, who assisted to dress herself, was therefore removed to the Maidstone Hospital, a distance of eight miles.

(For the further notes on this case I am indebted to Mr. Frederick Travers, Surgeon—at that time House-Surgeon—to the West Kent General Hospital, who performed the operation.)

Operation.—Upon arrival, at 11.45 a.m., patient was very blue, cold, and collapsed, with hardly perceptible pulse. Hot bottles were applied, and the operation commenced at 12.45, fifteen and a-quarter hours after perforation, the abdomen being considerably distended and tympanitic up to the fifth costal interspace on either side. Very little chloroform was required. On opening the abdomen a quantity of gas escaped, followed by opalescent fluid, sour smelling, and containing flocculi of lymph. The perforation was about $\frac{1}{4}$ in. in diameter, cleanly punched out, and situated on the anterior surface of the stomach near the lesser curvature at the junction of the pyloric and middle thirds of the viscus; around it were several layers of thick lymph. Thick flakes of lymph were adherent loosely to the upper and lower surfaces of the liver, the under surface of the diaphragm and peritoneal surface generally. The lymph round the ulcer was cleared off, and the wall of the stomach folded and secured by a row of Lembert's sutures, a flap of the great omentum was turned up, and fastened over this by interrupted sutures. About two pints of fluid were removed from the peritoneal cavity, which was washed out with a solution of chinosol, 1 in 2,000, and afterwards with sterilized water. A glass drainage tube was left in the lower angle of the wound. The patient had no more pain or vomiting after the operation. Enemata of hot water and brandy and hypodermic injections of strychnine were given, but she died at 3 a.m. the following morning.

CASE II.

History.—On July 26th, 1901, I was called to see Miss T., aged 23. She had suffered from dyspepsia for two or three years, characterized by pain and distension after meals, and often had to unfasten her clothes to obtain relief. Some time previously I had treated her for this at my surgery, and as she had improved so quickly and markedly with dieting and bismuth I did not consider her case one of gastric ulcer. Five days before she had sustained a great shock and since then had eaten very little. At 8 a.m. on the morning of July 26th,

when going downstairs, she was suddenly seized with violent epigastric pain above and to the left of the umbilicus, which obliged her to sit down and afterwards return to bed; the pain was continuous, but from time to time increased in severity and was described as like knives going through to the back. She stated that it was similar to the dyspeptic pain from which she had formerly suffered, but was worse than it had ever been before. There had been no vomiting.

State on Examination.—I saw her at 2 p.m., when she was complaining of great pain although she was not collapsed, and her lips were red but dry. Her temperature was 102.4°, pulse 104, and respirations 32; tongue clean. The abdomen moved a little with respiration, and was resonant and supple except from the umbilicus upwards, when there was resistance and tenderness to pressure and percussion, especially 3½ in. above and to the left of the umbilicus. The liver dullness was absent and replaced by a tympanitic note. The chest was normal. Hot fomentations were ordered but no morphine or opium were given.

Progress.—At 5 p.m. patient was in the same condition, but just about this time there was a forcible eructation of gas, and succussion splash was obtained. A diagnosis of dilated stomach was made. Milk was allowed by the mouth and a bismuth mixture given. At 12 p.m. the temperature was 100.4°, pulse 100, respirations 36, and the other physical signs as before.

July 27th. Patient had not had much pain since the last visit. Tongue clean, temperature 99°, pulse 96, respirations 36. The abdomen moved with respiration, and the tenderness was the same as the day before; liver dullness absent; her period had come on; the bowels had not been moved so an enema was ordered.

July 28th. Had had a good night. Temperature 96.8°, pulse 72, respirations 22. There was a tender spot over the abdominal aorta 3½ in. above the umbilicus, and also up under the ribs opposite the tip of the ninth left costal cartilage: still no liver dullness.

July 29th. Temperature 97.2°, pulse 80, respirations 24. Tenderness as yesterday; liver dullness ends 1 in. above edge of ribs. To try and clear up the diagnosis as to whether there was a gastric ulcer or not, a dose of salt and water was given with no subsequent smarting.

August 1st. Liver dullness normal: the tenderness up under the edge of the left ribs still remains. The patient continued to improve until the morning of August 5th, when she was dreaming and awoke in a fright. At 4 p.m., when I saw her, the temperature was 103° and pulse 120, but there was no pain or tenderness in the abdomen, and the liver dullness and chest were normal. At 10 p.m. that night the temperature was 103.2°, pulse 124. She had vomited once some milk and a green fluid, and complained of pain running up the spine to the back of the head. Her tongue was clean and moist, and there were no abnormal physical signs.

August 6th. The temperature and pulse were the same, and she had vomited some milk once, but from that time onwards the temperature and pulse gradually came down, and by the 11th were normal and the patient well. She has since taken up hospital nursing.

My first case might be classed under the heading of latent or masked peritonitis, but from the pulse condition an hour and a half after perforation, combined with the presence of liver dullness, the escape of gastric contents and gas into the peritoneal cavity must have been very limited at first, and therefore there could have been very little peritonitis.

Absence of Shock and Collapse with Regular Pulse and Normal Temperature.—When first seen it was not an example of the "period of repose," a term introduced by Mr. Charters Symonds before the Hunterian Society in 1899¹ to describe the fallacious calm which supervenes soon after a perforation and in which the patient feels better, the pulse falls, and the pain ceases, for in this case the pain had not ceased from the onset, and probably the pulse had not altered from the normal; in fact, the stage of shock and collapse had not been reached in which the textbooks describe the condition, for Osler² says "pulse is rapid, 110-150," Fagge and Pye-Smith³ say "pulse is frequent, 110-150," Clifford Allbutt⁴ says "pulse small, quick, thready," and Sidney Martin⁵ "the pulse is extremely rapid."

When later on the patient felt a little easier this was no doubt an example of the "period of repose." The temperature in these cases comes on as part of the peritonitis, and if collapse is great it is often subnormal.

Mr. John Campbell reported a case before the Royal Academy of Medicine of Ireland,⁶ in which the temperature was normal, pulse 80, and liver dullness present two hours and a half after perforation.

Mr. Rhys Griffiths⁷ records another case in which the pulse was about normal, and there was no pyrexia four hours and a half after perforation. The presence or absence of liver dullness is not mentioned.

Mr. Lachlan Fraser reports⁸ another in which the pulse was 80, temperature normal, and liver dullness was present six hours after perforation, and believes that the abdominal muscles held the ulcer (which was on the anterior surface, as were also those recorded above) so firmly against the immovable diaphragm that no escape of gas or stomach contents had taken place.

In all these cases there was severe pain, so that there was no question of the "period of repose."

Mr. Mitchell, in a paper read before the Ulster Medical Society in 1900,⁹ stated that he considered shock, when present in these cases, was due to (1) the presence of gas in the peritoneal cavity, pressing up the diaphragm and irritating the heart; and (2) to the disturbance of the great sympathetic ganglia and peritoneum by stomach contents, and quoted in support some of his cases, in one of which a pulse of 140 and weak dropped to 96 and became fairly strong when the abdomen was opened and gas allowed to escape; in another there was little free gas, and opening the peritoneum made little difference to the pulse; and a third in which the pulse fell from 108 to 88, and distinctly improved in tone.

Mr. Maunsell, in a paper read before the Royal Academy of Medicine in Ireland in 1901,¹⁰ reports two cases. In one the pulse was 78, and temperature normal half an hour after perforation, and the liver dullness was decreased but not lost, and in the other nine and a half hours after perforation the pulse was 84 and temperature 98°, but in this one the liver dullness was absent and the upper part of the abdomen very rigid.

Liver Dullness.—The presence or absence of liver dullness, which would seem from my first and most of the above-mentioned cases to have some causal connexion with the pulse-rate, is now generally looked upon as an uncertain sign, and absence of the dullness is considered to be often caused by colon resonance, but in my second case would seem to have been due to a dilated stomach, and Sir William Bennett, in a clinical lecture delivered at St. George's Hospital in 1900,¹¹ mentioned three cases of dilated stomach upon which he had operated; two of these he considered due to pyloric spasm; in one the liver dullness was greatly modified, and in the other two was entirely absent. In one of these latter, however, there were adhesions tying the stomach to the left lobe of the liver and under-surface of the diaphragm.

Pain of a Paroxysmal Character.—This occurred in both my cases, and the fact that the pain may be of this character in perforated gastric ulcer seems by no means universally recognized, although Clifford Allbutt¹² says, "Pain may be continuous, or may have the character of a severe colic," and Mr. Rhys Griffiths mentions¹³ two cases upon which he operated. In both of these the pain was paroxysmal in character; one of these is the case quoted above, in which the pulse was of about normal character four and a half hours after perforation.

Early Vomiting.—About this symptom, which occurred in my first case there is a diversity of opinion. Osler says,¹⁴ "Vomiting is an early and prominent feature." Fagge and Pye-Smith say,¹⁵ "Vomiting is sometimes the earliest symptom of acute peritonitis."

Robson and Moynihan¹⁶ say "Vomiting, with or without bleeding, is an inconstant symptom." Finny finds it recorded in 40 per cent., Fenwick in 29 per cent." Clifford Allbutt says¹⁷ "Vomiting is generally absent or soon ceases if it occurs at first," and Sidney Martin says¹⁸ "Sometimes there is nausea and vomiting."

My second case, when first seen, seemed very like a perforated gastric ulcer with resulting peritonitis; but had the absence of liver dullness and succussion splash been due to gas and gastric contents free in the peritoneal cavity and the temperature and pulse condition due to subsequent peritonitis, one cannot imagine the case having such a favourable termination without operation. I consider the case was an example of dilated stomach, due probably to a gastric ulcer, causing reflex spasm of the pylorus, but not necessarily at or near that orifice, the presence of spots tender to deep pressure was suggestive of that condition, in spite of the fact that salt and water caused no smarting. The dilated stomach by itself was not enough to account for the very acute pain at the onset, and Hemmeter says¹⁹ "The pain of dilatation is not marked. If cancer or ulcer coexists pain is a prominent symptom." I think the acute pain was gastralgia, due to ulcer and was accentuated by a hyper-sensitive condition of the nervous system caused by insufficient food, coupled with the shock from which the patient was suffering, and which was of a severe character due to her missing a nephew, a small boy, and finding him drowned in a pond. As the second rise of pulse and temperature seemed clearly to have a nervous origin, I can only ascribe the first to the same condition. The proximity of the menstrual period may have accentuated the nervous symptoms. The forcible eructation of gas in a condition of perforated gastric ulcer must be rare, except when the perforation has become closed by adhesions.

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A PRELIMINARY REPORT ON THE TREATMENT OF ADVANCED PULMONARY TUBERCULOSIS BY INTRAVENOUS INJECTIONS OF IODOFORM.

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In submitting this short preliminary report of an incomplete research on which I have been engaged for some time I shall confine myself chiefly to a description of the method. The results obtained have been so striking that several of my medical friends who have examined patients before treatment was begun and after are of opinion that it warrants publication.

All the patients treated have had tubercle bacilli, cocci and elastic tissue in the sputum, and in all both lungs were affected. In every instance there has been a great and sometimes rapid improvement in the physical signs, temperatures have become normal, weight and vigour increased, and expectation and organisms diminished. This has been achieved in patients with cavitation, whose condition was regarded as practically hopeless by independent physicians of the highest repute. It was my original desire to publish completed accounts of a number of cases with charts and diagrams in which the arrest had stood the test of time, and to which the term "cure" could have been applied with some justice, to record others where it had only stayed the advance of the disease, and to admit the residuum in which it had exercised no beneficial effect whatever. Such a report, accompanied by the independent certification of other medical men will alone now carry conviction to the profession should a claim be put forward that a cure has been found. This is necessary when we consider the pathology of the disease, its varied modes of dissemination (after implantation), the extreme range of age in which it is found, the frequent complexity of the infection, its latency and power of insidious advance. An analysis of even a hundred cases would be an imperfect gauge of any therapeutic measure. Here in a country district the number of phthisical patients is small, and as most of those who are in a position to leave the towns for treatment gravitate to the various sanatoria, it would be a work of years before I was in a position to tabulate even such a small number. The impossibility, therefore, of getting material on which to work will, I trust, extenuate the imperfections and shortcomings of premature publication. While fresh air is a desirable adjunct, I have recently treated one of my worst cases without it. In fact the nurse had difficulty in getting even imperfect ventilation.

It has been my lot, as it is the lot of most general practitioners, to have had to attend a good many patients suffering from advanced tuberculous disease of the lungs. Some of these had wintered abroad, some being in sanatoria; others had had neither of those advantages. All, however, had been under treatment of some kind or other, in the face of which the mischief had advanced with greater or less rapidity. The partial success which of late years has attended efforts at combating the mischief by direct attack in situ by way of the veins led me to make some experiments in that direction. In this search the much-discussed iodoform seemed to possess distinct qualifications. Despite the difference of opinion which exists as to its antiseptic qualities and how these are effected, and that it has a sickly penetrating odour most offensive to many, it is still one of the most trusted antiseptics with the operating surgeon. In selecting it as the bactericidal agent in an active attack on tubercle in the lung there were many points in its favour.

1. Its purity and known safety as an antiseptic agent.
2. Its ready elimination.
3. Its slow volatility and therefore sustained action.
4. It seems to have an affinity for dead or dying tissues, that is, the situations where the trouble is most active.

Could it be conveyed to the lung capillaries and chiefly deposited there, it seemed reasonable to expect amelioration in the condition and possibly such an emphatic arrest that

auto-immunization, increased tissue resistance, or whatever other forces ensure the occasional unaided recovery in advanced cases of this disease, might have their opportunity to act. To effect this it required to be in solution, and its great solubility in ether was taken advantage of. At first this seemed an ideal solvent. The ethereal solution would be rapidly conveyed to the lung capillaries. There the ether might be expected to volatilize and leave some solid iodoform in situ. Having satisfied myself by injection that this solution was safe, the remedy was applied to several patients.

The breath and mouth at once smell and taste of the solution. Soon the ether is dissipated, but for several hours after (depending on the dose) the breath smells of iodoform and the patient is conscious of the smell and taste. This stability of iodoform after injection is very interesting and desirable. The liberation of free iodine to even a small extent would rapidly irritate the coating of the veins, and assist in the production of thrombosis, besides causing severe coughing. Further, this persistence seems to point to the iodoform itself as iodoform exerting the beneficial influence rather than to the decomposition of the compound, freeing pure iodine. I have repeatedly examined the urine with the starch test at varying intervals after injecting, but have failed to get the iodine reaction. On this point, however, I do not desire to be dogmatic. This slow sustained action of the drug may be counted upon, while at the same time it diminishes the risk of cumulative action. Patients soon become tolerant of the taste, and unless the injections are pressed I have met with no bad effects whatever while employing small doses. Pushing the drug in large daily doses positively seems to saturate the system. I have, after discontinuing it for several days, detected the smell in the palms of the hands and the skin of the body. The patients are then so constantly conscious of it that the appetite is affected. It is undesirable and necessary to push the injections to this extent. In the course of this work I have observed that soon after the injections are begun evidence of disease is revealed, sometimes where none was suspected at first. This diagnostic quality of the injections is valuable. The productions of fine crepitations or *râles* at an apex formerly dry, resonant, and apparently healthy is important. It proves how subtle the malady is, and how untrustworthy the most careful physical examinations occasionally may be.

Briefly, then, these are the good points, but it has important drawbacks, referable both to the ether and the iodoform. The chilling effects of the ether on the vein walls, diminishing their calibre and temporarily stiffening, if not partially freezing, them, has to be guarded against as much as possible. In a case (five years' duration) with extensive cavitation in the upper lobes of both lungs and consolidation at both bases anteriorly, with absence of breath sounds and little or no chest action, it occasioned such sudden persistent breathlessness and palpitation that I had to discontinue the treatment. As the patient could take the ether alone without any discomfort, the natural conclusion is that the iodoform was the offending cause. This did not occur once but several times, and even with such small injections as 5 minims. It is possible that this embarrassment may be found a constant characteristic and contraindication in very advanced disease where the margin of available lung tissue is only equal to continuing life under the most passive conditions and no more. To save such cases is of course the dream of the physician, but we might reasonably expect that the paralyzing influence which iodoform exerts on the motor ganglia of the heart would be intensified when it has been debilitated by unduly rapid action and the wasting effects of toxins.

Before beginning the treatment of any case, we must first examine carefully the available superficial venous system both in the arms and legs. The more extensive this is, the greater is the probability that we shall arrest the progress of the disease. Therefore men are more favourable subjects for treatment than women, and women than children. I have never had occasion to use it in children, and I fear from their natural timidity it would not be possible to carry it out. Nothing can be more disconcerting than a request to undertake the treatment of a plump or even fat young woman of 18 or 20 with extensively diseased lungs, whose total available venous system is represented by two dim blue areas $\frac{1}{2}$ in. long at the bends of the elbows. It will demand the most scrupulous care and dexterity to administer injections regularly for six months or a year. While nothing is simpler than to give an intravenous injection once in a large vessel, it is a matter of some difficulty to give a hundred through a small restricted venous supply to avoid thrombosis and have