

agglutinins, and may indeed be lower throughout the whole infection. On the other hand, it is certainly the case that at its maximum the titre for the infecting micro-organism will usually exceed the titre of inoculation agglutinins even though these may be greatly raised as a result of the infection. Nevertheless, as the fall in titre proceeds the curve of A and B agglutinins falls more rapidly than the curve of T. inoculation agglutinins, so that the latter is cut by the former at some points in the fall.

The maximum agglutination titre for the infecting organism most frequently occurs about the eighteenth to twentieth day, and almost always lies within the limits sixteenth to twenty-fourth day of the disease.

As regards the effect of active paratyphoid infection on the typhoid inoculation on agglutinins one of three things may occur: (a) The titre may remain unchanged throughout the infection; (b) it may undergo a rise of moderate extent; (c) it may undergo a marked rise. When such rises occur it is found that they are either synchronous with or antecedent to the rise of agglutinin titre for the infecting micro-organism, and that the maximum falls somewhat earlier than the maximum for the infection agglutinins (or occasionally at about the same time). The change in T. inoculation agglutinins associated with paratyphoid infection is in general much more marked in the case of paratyphoid B than in the case of paratyphoid A.

In view of what has here been shown, it is obvious that it would be quite futile to try to fix a limit of agglutination titre for T.-inoculated individuals, and to proceed to diagnose cases which showed higher titres (even many times higher) as cases of active typhoid infection. The final guide in all these cases of enteric infection is the relative extent of the rise and fall in agglutination titre for the organisms concerned, together with period when the maximum titre is reached.

In a recent article by C. J. Martin and W. G. D. Upjohn these authors emphasise the existence of numerous difficulties in the diagnosis by agglutination tests of the enteric fevers in typhoid-inoculated persons. These difficulties might readily have been avoided had they taken cognisance of what has been repeatedly and clearly stated on this subject.^{3 4 5 6} And they would have been saved from the conclusion that since the introduction of triple inoculation (T., A., B) "the interpretation of observations upon the agglutination of enteric organism will be too difficult to possess any practical value, and the isolation of the infecting organism must be resorted to for diagnosis."

In a subsequent article it will be shown that although the difficulty of diagnosis by agglutination tests is to some extent increased by triple inoculation the validity of the tests is in no way impaired. They remain not only the most certain method of diagnosis, but form, in fact, the only method which can be relied upon if a large proportion of the cases are not to be missed. For it is well known that isolation methods fail to detect the infecting micro-organism in blood, stool, or urine in a majority of all cases of typhoid and paratyphoid fevers.

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NATIONAL ASSOCIATION FOR THE PREVENTION OF INFANT MORTALITY.—A course of advanced lectures on infant care is to be given under the auspices of this association at the College of Ambulance, 3, Vere-street, London, W., on Mondays from March 12th to June 18th inclusive. The lecture hour is 5.30 P.M. The course is in preparation for the advanced certificate given by the association, and is intended for teachers and infant welfare workers. The theoretical lectures are supplemented by tutorial courses at various training centres in London. Particulars may be obtained from the secretary, Miss Halford, at 4, Tavistock-square, London, W.C.

A SIMPLE ULCER OF THE ŒSOPHAGUS

PERFORATING THE DESCENDING PORTION OF THE AORTIC ARCH AND CAUSING FATAL HÆMATEMESIS.

BY J. B. CHRISTOPHERSON, M.A., M.D. CANTAB.,
F.R.C.P. LOND., F.R.C.S. ENG.,

DIRECTOR OF THE CIVIL HOSPITALS OF KHARTOUM AND
OMDURMAN, SUDAN.

SIMPLE ulceration of the œsophagus is, clinically, a rare condition, it is probably present more often than diagnosed, but death from the perforation of a latent ulcer of the œsophagus into the aortic arch must be very rare.¹ I beg, therefore, to take the liberty of recording this case. Of all parts of the alimentary canal in man ulceration is least commonly met with in the œsophagus.² A few cases of simple "peptic" ulcer of the œsophagus have been reported—one only is recorded which perforated the aorta. The following account is of a case of fatal hæmatemesis in an Englishman aged 36, which occurred in July, 1916, at Khartoum, and was due to the ulcer perforating the descending part of the arch of the aorta. The clinical history is as follows.

On July 21st, 1916, the patient consulted a medical man because he had, he thought, "something in the throat" and, as he had first noticed it after dinner on the previous evening when he had partaken of a dish of soft fish, he thought that he might have a fish-bone lodging in his throat. There being no clinical evidence of fish-bone or foreign body he was put to bed and given expectant treatment. His symptoms were as follows. He felt as if there was "something in the chest"; he felt it when he swallowed, more especially when he swallowed "nothing" and also when he drew a deep breath. He located this sensation, which was more discomfort than pain, inside the chest behind the sternum, at a place which would correspond to the level where the ulcer was eventually found. He also had some pain behind, which he located between the shoulder-blades. He had a thickly coated tongue; it was always thickly coated, he said, and the thermometer registered 100° F. on admission. There was nothing to be seen in the throat, no physical signs of anything amiss in the chest, and, in fact, the only symptoms indicating that there was anything wrong were a rise of temperature to 100°, a coated tongue, and discomfort, sometimes amounting to a little pain, on swallowing or drawing a deep breath.

I am emphasising the total absence of serious symptoms, because seven days after the time when he first consulted a medical man about these small symptoms he had a large hæmorrhage, and one day after this the fatal hæmorrhage occurred. On July 23rd, the temperature being normal and the symptoms having almost disappeared, the pain having become diffused amongst the muscles of the back, as he said, he returned to his own house. On the 25th he was still having discomfort in swallowing and was admitted again into hospital. Again the temperature was 100.2°, and again it dropped to normal on the following day and remained so, and on the morning of the 28th, when I asked him to swallow a mouthful of water and also to draw a deep breath and tell me whether he had pain and where, he, after thinking for some time, said in both instances, "Well, I really cannot say that I feel anything at all now." I wish still to emphasise the absence of symptoms and physical signs, because we have reached the very day of the first hæmorrhage, which was nearly fatal, and the day before the fatal hæmorrhage; and yet, although the ulcer must already have attacked the aorta and was progressing and on the verge of perforating, there was nothing to indicate it but a dirty tongue and a little discomfort in the chest.

At 4 P.M., on July 28th, whilst reclining in a large chair, the patient brought up a little mucus streaked with blood. He was put back into bed. At 6 P.M. he vomited half a basinful of blood, clotted and fairly bright, which had evidently been retained in the stomach. No further visible hæmorrhage occurred for 24 hours; evidently the opening into the aorta being a small one became plugged by a clot (this was seen post mortem). The patient seemed to be doing well and he was kept under morphia and given nothing which

¹ Osler (Principles of Medicine, eighth edition, p. 473): "Liljeston collected 40 cases of peptic ulcer œsophagus (first described by Albers 1839). Six perforated the œsophagus and one perforated the aorta with fatal result."

² This immunity of the œsophagus is due to the fact that, taking very little part in digestion, it is simply a connecting tube of comparatively simple structure, vertical in position, and being accommodated in capacity it allows of the rapid transit of foodstuffs through it. It is therefore not much exposed to the danger of contracting disease from outside sources.

would tend to raise the blood pressure. The question of a gastrotomy was discussed and dismissed. In point of fact, as was subsequently found post mortem, the situation of the ulcer was inaccessible to operation. On the 29th, 24 hours after the first large hæmorrhage at 6 P.M., he had another large hæmatemesis, evidently blood which had accumulated in the stomach having trickled down from the ulcer, and he died one and a half hours afterwards.

At the necropsy it was found that the hæmorrhage came from an œsophageal ulcer which had become adherent to the descending part of the arch of the aorta, in the neighbourhood of the bifurcation of the trachea (Fig. 1), and which had

FIG. 1.

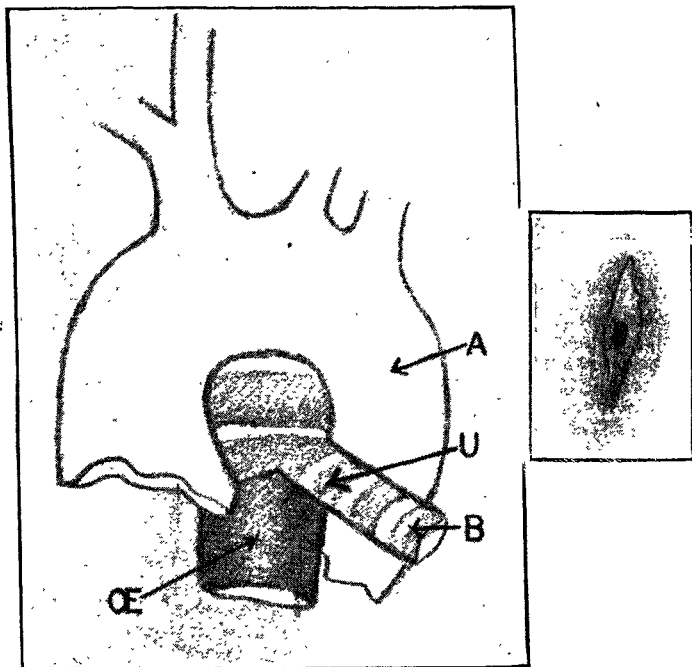


Diagram to show approximate size of ulcer (U) in œsophagus (œ) and the relation to it of the descending part of the arch of the aorta (A), B, left bronchus. A drawing of the ulcer perforating the aorta just behind the left bronchus is attached to the diagram.

penetrated the arterial wall. The stomach was very much distended, containing two or three pints of blood which had found its way down from the ulcer into the stomach and distended it. There were two other ulcers in the œsophagus, both situated at the cardiac end just within the œsophagus, one of which had healed with a cicatrix and the other, a larger oval one, in the process of healing. (Fig. 2.) Sometimes he had complained of a little pain in the epigastrium, and the situation of these ulcers would perhaps account for this. The stomach and all the other organs were healthy. The liver weighed $2\frac{1}{2}$ lb., the spleen $2\frac{1}{2}$ oz. The liver and spleen were examined particularly because the patient was said to have had kala-azar some years ago. In appearance they presented nothing abnormal (excepting the spleen, which was unusually small).

Non-malignant ulcers in the œsophagus are rare; they may be classified as follows:—

1. *Traumatic*.—Corroding fluid (swallowing of); sharp instruments (injury by); foreign bodies (such as fish-bone) blows, violence (bruising by).
2. *Latent ulcer*.—Which may perforate just as gastric ulcer.
3. *Specific*.—Syphilitic.
4. *Secondary ulcers*.—During the course, or as sequelæ, of diseases such as typhoid, kala-azar, diphtheria, scarlet fever, or pneumonia.
5. *Aneurysmal*.—Due to an adjacent aneurysm of the aorta, or other great vessel, ulcerating into the œsophagus.

The (1), (3), and (5) classes may be excluded at once in the present case.

(a) There was no history of anything which could have produced a traumatic lesion, not even a fish-bone; the other two small ulcers (nearly healed) at the cardiac end of the œsophagus are in themselves sufficient evidence against the perforating one being traumatic—both were chronic ulcers in the process of healing, and the fatal one was also a chronic ulcer. Perforating chronic ulcers (peptic) have been described in the œsophagus. They are rare; only a few cases are described.³ Albers first

described them in 1839. Tileston collected 40 cases; six perforated and one perforated the aorta.

The same conditions do not exist in the stomach as in the stomach to give rise to ulceration. So that simple œsophageal ulcer must be a diagnosis based to a large extent on a process of exclusion. A definite diagnosis is, I think, impossible to make until hæmorrhage takes place.

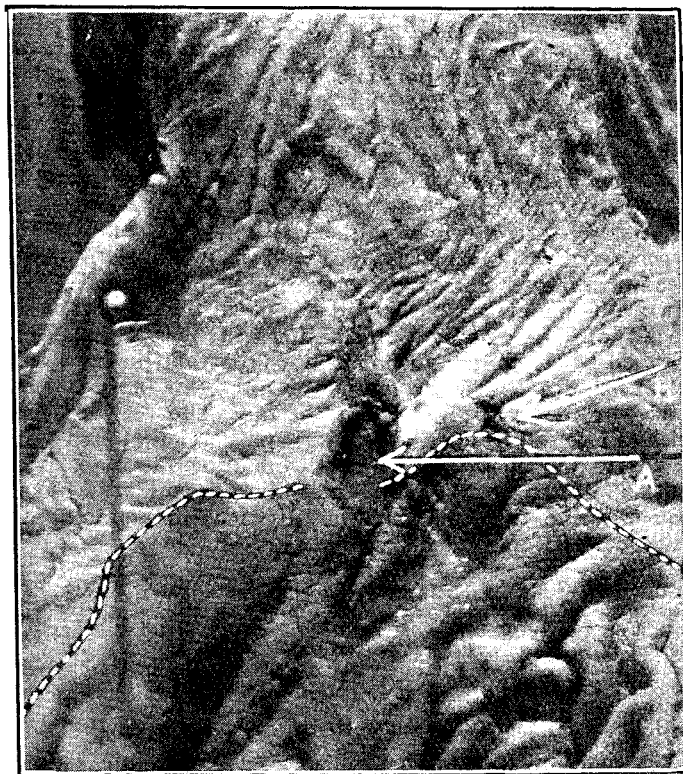
(b) Nor was it a syphilitic ulcer. There was no evidence at all to favour such a diagnosis.

(c) There was also not a single particle of evidence in favour of a diagnosis of aneurysm. Seen post mortem, the wall of the aorta was perfectly normal and healthy, except where the perforation was, and the patient had not had syphilis. His urine and kidneys were perfectly normal, and he had no aneurysm-producing disease.

There remains Class (4)—ulceration due to complications and sequelæ of former illness. Two possibilities suggest themselves in this case. The patient is said—I am bound to say on unconvincing evidence—to have contracted kala-azar in 1907. Now, ulceration of the intestines is a common complication of kala-azar, so it is worth while pausing just for a moment to consider this possibility. Suppose, for the sake of argument, that he did contract kala-azar in 1907. After his return to the Sudan in 1908 up to the time of his death in 1916 he appeared perfectly free from any suspicion of it, and at his death the spleen weighed only $2\frac{1}{2}$ oz. and the liver $2\frac{1}{2}$ lb. This, in addition to the fact that ulceration of the bowels is a late complication in kala-azar when the patient is very thin and weak, sufficiently, I think, disposes of the kala-azar theory.

There remains the theory of typhoid ulceration and sequelæ, and I must say at once that the evidence appears

FIG. 2.



Photograph of junction of œsophagus (above the line) and stomach (below the line) showing the two ulcers, one healed (H), the other active (A), situated just within the œsophagus.

to me in favour of this diagnosis—sequelæ after typhoid, although it was six years after the attack of typhoid fever. The two lower ulcers exactly corresponded to healing and healed typhoid ulceration (Fig. 2). The ulcers were chronic, and the symptoms were insidious and probably had lasted over a long period, giving time for the aorta to become adherent to the œsophagus. The appearance of the ulcers was oval in the long axis, base granular, surface shallow, edges smooth and overhanging, margins level, and not irregular and heaped up. (Fig. 1.)

The evidence—partly exclusive, partly of local appearance, partly of history—and the circumstances point to old typhoid ulceration. This is the opinion I myself hold, for it is the

³ Osler, eighth edition, 1912, p. 473.

only opinion which is, I think, consistent with the facts. Still an ulcer, of whatever origin, of the œsophagus, situated at the part which is in relation to the aorta so that it perforated, causing fatal hæmorrhage, is rare. It might fittingly be placed in the category of "accidents of medicine." Six years had passed since the patient had had the typhoid fever, but the attack had been a severe one, and although the ulcers at the time of death may not have been specific in the sense that they were infected with Eberth's bacillus, still an old ulcer whose base had become fixed to the aorta might, owing to the strain of gymnastics or polo, resume activity, and this is how I view the case. A few days previous to his illness the patient had complained of some discomfort in the chest after gymnastics, and I think that probably this lighted up into activity an old ulcer which was adhering to the aorta—a sequela of typhoid six years previously. The original attack of typhoid, I am informed by the medical man who attended, was typical and serious. The patient had hæmorrhage and he was carefully watched for perforation, and convalescence was long.

From general and microscopical appearances the ulcer was an old one. The aorta was with difficulty separated by dissection from the œsophagus in the neighbourhood of the ulcer, showing chronicity. The inflammation from the base of the ulcer had fixed the aorta to the œsophagus, and under the microscope there was an arteritis round the perforation in the aorta. The aorta having become fixed to the base of the ulcer as inflammation proceeded, it became in its turn the base and was involved in the processes going on. As ulceration slowly proceeded the artery became the floor of the ulcer; then softening of the arterial wall occurred and perhaps a little bulging (the arterial wall round the perforation was funnel-shaped and thinned, as if it had been bulging at this point). The bulging occurred, perhaps, when the patient had been doing gymnastic exercises on the day previous to admission. Then came ulceration of the aortic wall forming the floor of the ulcer, and the occurrence of the streaks of blood in the brought-up secretion, and two hours later perforation and the large hæmorrhage on July 28th. Then the cessation of the hæmorrhage caused by plugging of the small perforation by blood clot, and, finally, after an interval of 24 hours, the displacement of the cork of blood clot and fatal hæmorrhage.

The above appears to have been the pathological sequence of events. There remains, however, the cause of the œsophageal ulcer. The general appearance of the ulcer itself, the presence of the two other ulcers, one quite healed and the other healing, at the cardiac end of the œsophagus, the noticeably coated condition of the tongue which the patient said he always had, would suggest that these ulcers had been present for a long while, and it is surprising that they gave rise to practically no symptoms. Perhaps they owed their origin to the attack of typhoid fever in 1910. This is likely, but there is no direct evidence. Perhaps it was a simple streptococcal ulcer, such as one meets frequently in the stomach, and much less frequently in the œsophagus.⁴

The case is, I think, remarkable not only for its rarity, but also on account of the total absence of serious symptoms up to 26 hours previous to death, when some streaks of blood were noticed in some secretion which the patient spat out.

The only subjective signs were a certain degree of discomfort, not amounting to pain, in the chest on swallowing, especially on performing the act of swallowing without swallowing anything in particular, and also in drawing a deep breath.

The objective symptoms were a noticeably foul tongue (which the patient said he always had) and a small rise of temperature, never over 100°, which twice descended to normal after 24 hours in bed.

From this case one may learn that a quiescent chronic œsophageal ulcer may cause no symptoms at all if we except the coated tongue. When the process becomes active there will be discomfort or pain located indefinitely about the level of the ulcer back and front. It is only when perforation into a vessel, organ, or cavity occurs that a definite diagnosis can be made. Perforation into a vessel large or small, causes obvious symptoms. Perforation into the left pleural cavity, which, I take it, is the commonest site of perforation for an

œsophageal ulcer, causes symptoms (subjective) very similar to those produced by perforating gastric or duodenal ulcers; but the physical signs in the one case are those of acute respiratory trouble (pleuritis), in the other acute abdominal (peritonitis). In the account of such a case of perforating œsophageal ulcer into the left pleural cavity by Dr. Graham W. Christie⁵ two clinical points stand out and seem worthy of note: (1) The sudden pain and subjective symptoms caused by the perforation of an œsophageal ulcer into the left pleura suggested peritonitis, though in reality they were due to pleuritis, as was shown by absence of signs of general peritonitis; and (2) in perforating œsophageal ulcer the patient can sit up more comfortably than he can lie down, the opposite being the case in perforating gastric ulcer.

I have to thank Mr. Newlove, pathological assistant, Khartoum Civil Hospital, for helping me to put this little paper together.

Khartoum.

CHRONIC EMPYEMA: THE VALUE OF DECORTICATION OF THE LUNG.¹

BY W. H. BATTLE, F.R.C.S. ENG.,

LIEUT.-COL., R.A.M.C. (T.); SENIOR SURGEON TO ST. THOMAS'S HOSPITAL.

IN this war there have of necessity been a large number of cases of penetrating wounds of the chest. In many of these there has been an accumulation of blood in the pleura of the side wounded which has cleared up after simple rest in bed. When dyspnoea, cough, or pyrexia has developed paracentesis has been most useful in effecting relief in the symptoms and a quick convalescence. There have been several excellent communications from the medical side which bear out this statement. Sometimes septic infection of the wound has been such that suppuration has taken place in the pleura, and the decomposing clot (and pus) has required evacuation through an incision into the pleura followed by drainage. When this operation has included resection of part of a rib, thus securing a larger opening, the result has been more uniformly satisfactory. In a smaller number closure of the empyema has been incomplete, and a sinus has remained which has proved a source of much discomfort and disability to the patient. Here there will usually be found a cavity of varying size in the pleura, the dimensions of which can be defined by means of a bullet probe, auscultation, and the X rays. This requires exploration under anaesthesia, with enlargement of the track, possibly combined with excision of overlying rib to ensure good drainage. The injection of bismuth paste has succeeded in some instances of moderate sinuses in civilian practice in effecting a cure, but until exploration has proved that no foreign body is there, and no fragment of bone is concealed behind the lowermost rib, its employment is not satisfactory after gunshot wounds. The cases in which the lung has not expanded after the first operation, remains collapsed and bound down to the side of the spine, are luckily few and far between. They are the most serious.

For the cure of an empyema of the most intractable kind, one in which the whole lung is bound down to the inner aspect of the chest cavity, and cannot expand, we have a difficult problem to solve. We are confronted by a cavity the walls of which are rigid and cannot come together to close the space, the ribs are unyielding and the lung so fixed that it cannot expand. It occupies a small part of the chest cavity and is quite functionless. With this local condition there is often a serious general state of exhaustion and emaciation owing to the effects of a prolonged discharge on a system already weakened by an attack of acute disease, or the severe shock of the wound of an important region. The state of the patient will naturally vary according to the duration of the mischief, the intensity of the infection, amount of purulent discharge, and accompanying pyrexia.

The fact that a lung might be released by removal of the covering membrane was not realised until recent years. We had to rely on operations calculated to permit an area of chest wall, which was adapted to the size of the suppurating cavity, to fall in to the level of the lung, from which no aid

⁵ THE LANCET, 1915, ii., 17. Case of Perforation into Left Pleural Cavity (male, aged 60).

¹ Abstract of a Clinical Lecture delivered on Jan. 10th, 1917.

⁴ Osler: Loc. cit.