A CASE

OF

RUPTURE OF THE ŒSOPHAGUS

CAUSED BY VOMITING

TOGETHER WITH A TABLE OF SEVENTEEN OTHER CASES

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Mrs. B—, aged 62, was suddenly attacked by agonising pain in the epigastrium after acute vomiting about 11.30 a.m., March 25th, 1895. It appeared that she had taken overnight a pill containing aloes and rhubarb. After this had acted as usual, on taking some milk, which always disagreed with her, she vomited four or five times, and still feeling sick took a tumblerful of salt and water "to clear the stomach." The vomiting produced

by this was followed by sudden onset of epigastric pain accompanied by collapse of an alarming nature, so that she was conveyed upstairs to her bedroom with great difficulty.

She was given brandy and twenty minims of chlorodyne by her maid, and complained of aggravation of her pain immediately after swallowing it. She remained in acute agony until seen by Mr. Turner about 2.30 p.m. She was then sitting propped up in bed by pillows, moaning from pain, with cold livid extremities, gasping respiration, extremely anxious expression, and with an almost imperceptible pulse. She was able, however, to help in giving the history above related, and described her pain, which she referred to the epigastrium and dorsal spine, as "not on either side," "as if I was breaking in two," and later on as "not connected with the bowels." On this latter point she was very positive, and regarded examination and palpation of the abdomen as useless.

There was retraction and some tenderness of the upper part of the abdomen with rigidity of the recti. The lower part of the belly was prominent as compared with the upper retracted portion.

There was no swelling in any of the hernial orifices, and all vomiting had ceased. She was given some brandy and fifteen minims of laudanum by the mouth. Her pain was immediately aggravated, and she complained of a burning sensation produced by swallowing.

It was thought that possibly some perforation of the stomach had occurred, and Dr. Monier Williams undertook the charge of the case while preparations were being made for a laparotomy, if on consultation with Dr. Bowles such a course should be thought necessary. In about an hour's time, when Dr. Bowles first saw her, at 4.30 p.m., she had slightly improved, the collapse was less intense, and the abdominal symptoms were decidedly modified. There were, however, urgent symptoms of dyspnœa, extreme pain and distress around the waist high up, back and front, retraction of the epigastrium,

respiration 50, pulse 50, full and tense in character, the pulse rather of suffocation than of collapse; the skin was moist. The extreme distress, the dangers of changing her position, the gasping moans, and rapid short respirations rendered auscultation difficult. The only marked symptom was the absence of cardiac sounds, which from the character of the pulse could have scarcely arisen from simple cardiac failure.

There was no dulness indicating the effusion of blood from a ruptured aneurysm, and no sign of effusion into the pericardium. It was decided that as the thoracic symptoms were now more pronounced than the abdominal, and that the collapse was not so intense, to give her more morphia and await further developments. The dose of the morphia was carefully discussed, and the amount already taken duly considered, viz. twenty minims of chlorodyne at 11.30 a.m., fifteen minims of laudanum at 2.30, and a third of a grain of morphia under the tongue at 3.30 p.m. It was finally decided to give her, an hour later, a third of a grain of morphia. The patient's distress was so great that this was hypodermically injected at 7 p.m. She went to sleep in a few minutes, and at 10 p.m. was in a state of profound coma, so profound that, although the amount taken in the twelve hours did not justify danger to life from opium, it was felt necessary to watch her, to inject coffee into the rectum, salicylate of caffeine under the skin, to rouse her from time to time, and finally to apply electricity. At 2 a.m. she was safe as regards morphia poisoning, and was left under the care of Dr. Monier Williams and Mr. Evans. At 5 a.m. Dr. Bowles was called to her again, in consequence of her old distress returning as the effects of the morphia passed away. She was now hot, perspiring profusely, had a full pulse and great dyspnœa. The cardiac sounds were still inaudible; there was percussion resonance over the whole of the front of the chest and deficient respiratory murmur on the left side. Behind, as far as one could examine her in a semi-recumbent position, there was percussion

dulness, coarse râles and fair respiration in the right lung in front.

There was now observed a fulness above the suprasternal notch, soft to the touch, which had not existed earlier. This fulness increased and extended, and soon left no doubt of the supervention of emphysema. At the time of her death, at 10 a.m., March 26th, some twenty-two and a half hours from the commencement of the attack, it had extended to the face and eyes and was perfectly characteristic in all its details.

The following is a report of the post-mortem examination made by Dr. Cyril Ogle:

"Subcutaneous emphysema of left side of upper part of the chest and neck. Pneumothorax on the left side, the air in the chest being under considerable pressure. About six ounces of brownish-red opaque fluid in the left pleural cavity; a small quantity in the right cavity; the posterior mediastinum was infiltrated with similar fluid.

"The lungs were perfectly healthy on both sides, but the left one was collapsed. There was neither old nor recent tubercle anywhere, nor any adhesions, and no rupture of the visceral pleura. At about one and a half inches above the diaphragm there was a rupture of the œsophagus, a longitudinal rent in its left side measuring about five eighths of an inch. Its edges were thin, and the œsophageal wall around it was somewhat softened, but there was no peeling of the mucous membrane. Neither malignant disease nor cicatrix, nor any sign of inflammation were present.

"The wall of the stomach adjoining the œsophagus appeared quite healthy and free from softening.

"The other organs examined were normal."

The specimen of the ruptured œsophagus was taken to St. George's Hospital, and was for some time in the museum. It has now unaccountably disappeared, and so cannot be shown to the Society.

The diagnosis of these cases is not only difficult during life, but even after death they have led to much differ-

ence of opinion. During life the diagnosis would be between rupture of the œsophagus, perforation of a gastric or duodenal ulcer, rupture of an aneurysm, angina pectoris, a severe attack of colic, irritant poisoning, and possibly other conditions attended with pain and collapse. After death, ulceration and post-mortem softening are the conditions most likely to be confused with the effects of rupture. We believe that the symptoms of rupture of the œsophagus are fairly clear, and think that the rarity of this condition is the reason of the difficulty of diagnosis. Should any case of a similar nature come before us in the future we think that it would be at once recognised. The sudden onset of the pain, which is not abdominal, but referred to the back and front of the lower part of the thorax; the cessation of vomiting when once the vomiting has done its mischief; the agonising pain that follows any attempt at swallowing the stimulant which the occasion seems urgently to demand, should make the case clear to any one who has once seen this rare injury. A slow tense pulse indicating irritation of the vagus may, perhaps, too, be noted. Later on when morphia and other appropriate remedies have relieved the distress, the absence of abdominal symptoms and the supervention of emphysema of the neck and face would corroborate the initial diagnosis.

With reference to possible surgical treatment, it may be remarked that the œsophagus can be reached by an incision to the left or right of the vertebral column, the resection of portions of three or four ribs, and stripping up the pleura.

The 'British Medical Journal' of January 7th, 1899, page 34, has a *résumé* of work done in this direction by Nasiloff, Quenu, Hartman, Potarca, Ziembicki, Bryant, Rehn, and Obalinski. The latter reports thirteen cases of suppurative posterior mediastinitis treated by drainage. Ten of these were dependent on tubercular disease of the dorsal vertebræ. Two cases of œsophageal stricture are on record in which the œsophagus has thus been exposed

without injury to any important viscus. It would seem, therefore, that if the injury were diagnosed, the immediate treatment of rupture of the œsophagus should be drainage of the posterior mediastinum, and, if possible, suture of the rent.

Nothing, of course, should be given by the mouth; pain and collapse must be relieved by hypodermic morphia injection, and stimulants given by the rectum. Later on, if the condition of the ruptured œsophagus demanded it, or rectal feeding failed, a gastrostomy might well be required.

The chief difficulty must always be to tide the patient over the alarming collapse which invariably follows this injury. In the case recorded, the rent being to the left side, suture of the rupture, although difficult, would not, perhaps, have been impossible. The general state of the patient, however, was such that any operation would probable have proved speedily fatal.

The literature of the subject extends back more than a century, but is very limited and confused. The emphysema that sooner or later shows itself has been put down to some rupture of the lungs or air-passages, which is not always found, however, after death, and other symptoms have been attributed to angina pectoris and post-mortem softening.

Fitz has written an admirable paper in the 'American Journal of Medical Science,' 1877, on a case on which he made a post-mortem examination. He analyses the cases up to then recorded. He is inclined to throw doubt on Boerhaave's case of Wassenaer, but from its clinical symptoms and post-mortem appearances it would seem to have been one of transverse rupture of the œsophagus near the diaphragm. Dryden's case, too, would seem to us to have been a typical one; it occurred after drinking warm water to induce vomiting. As in our case, there was emphysema and a longitudinal rent in the œsophagus just above the diaphragm. Guersent's case of a child, seven years old, would again seem to us to be a

genuine one of rupture after vomiting rather than one of scarlet fever and post-mortem softening. It is not the only case recorded in early life. Munro speaks of having seen a preparation in which there was a considerable longitudinal rupture of the œsophagus in a child. We see no reason for rejecting Oppolzer's case; indeed, his description, brief though it is, would appear to indicate personal acquaintance with clinical symptoms. Charles's case, too, would appear to us to be typical. We must differ from Fitz in rejecting these six cases, and cannot but think that he draws his conclusions more from a post-mortem than a clinical point of view. The description of some of his rejected cases exactly tallies with the one we had the opportunity of closely observing. The majority of cases seem to be caused by the act of vomiting, which act then ceases, it being, according to one authority, "no longer possible." Some expulsion of blood is noted in those cases where the gullet has been obstructed by the impaction of food.

In all the recorded cases, eighteen in number, a list of which we append, the rupture has been a longitudinal one, except in the celebrated one of Admiral Wessenaer and another of Tändler. Experiments made on the healthy œsophagus show that it can bear, without tearing, weights up to five kilogrammes. This has induced some authors to assume that an "intra-vital œsophago-malacia" —a softening attended often with hæmorrhagic extravasations—is a necessary antecedent of rupture from such a cause as vomiting.

It was our intention to have made an experimental inquiry by forcible distension of the œsophagus on its lacerations by force from within, and to see whether such laceration would be vertical rather than horizontal, but so far we have not been able to carry out more than a few such experiments.

In those that have been done, both on the œsophagus of the adult and child, the laceration has been of the longitudinal variety, and the same occurs on forcible

distension of the small intestine. Specimens of such artificially-made ruptures of œsophagus and bowel are on the table. The rupture was produced in these by attaching the œsophagus to an oxygen gas cylinder.

Table of 18 *Cases.*

Name and reference.	Cause.	Description of rupture.	Emphysema.	Result.	Remarks
Dr. Allen, of Boston, Amer. Journ. Med. Sci., Jan., 1879. Male, aged 31, drunkard	Obstruction by food 1 hour	Long rent 2 inches at and below bifurcation of trachea	Present immediately after accident; ? rupture of air-passages	Died 7½ days	—
Boerhaave, (Lisabaud, Hist. Anatom. Medica, vol. ii, p. 311, 1767). Admiral Wassenaer, aged 50	Vomiting self-induced	Transverse near the diaphragm	A large amount of gas in pleural cavity; emphysema present	Died 24 hours (suffocated)	Main symptom pain within chest near diaphragm extending to back.
John Dryden, Med. Commentaries, Edinburgh, 1788, p. 308. "Strong healthy man"	Vomiting self-induced	Longitudinal, large enough to admit 2 fingers above the diaphragm	Emphysema of neck; fluid in both pleural cavities	Died 12 hours	Pain referred to stomach; no vomiting after rupture.
Guersent, Bulletin de la Faculté de Médécine de Paris, 1812, vol. i, p. 73. Child, aged 7	Vomiting caused by emetic	Rent 2 centimetres long, 5 centimetres above diaphragm; edges and œsophagus healthy	Rent communicated with right pleural cavity, which contained a brown fluid	Died 24 hours after emetic; previously ill with diarrhœa, etc.	Longitudinal rupture in a child is mentioned by Monro, The Morbid Anatomy of Human Gullet, Stomach, and Intestines, Edin., 1811, p. 311.
Oppolzer, Wiener Medizinische Wochenschrift, 1851, p. 65. Woman	Vomiting; "violent pain on vomiting"	No details given; healthy œsophagus	Hæmorrhage into mediastinum	Died	No vomiting after accident.
Dr. Bowles and Mr. Turner Woman, aged 62	Vomiting after an emetic	Longitudinal, 1½ inches above the diaphragm	Very marked	Died 22½ hours	

Name and reference.	Cause.	Description of rupture.	Emphysema.	Result.	Remarks.
Meyer, Medicinische verein Zeitung in Preussen, 1858, Nos. 39—41. Male, aged 38	Obstruction by food; attempts at vomiting; emetic	1¼ inches long, ⅜ths of an inch wide, 3 inches above the cardia	Emphysema of right side of face, neck, and chest	Died 50 hours	Gangrenous cavity; posterior mediastinum containing dead tissue and food; "upright position, body bent forwards."
Leyden, Grammatzki, Ueber die Rupturen der Speiseröhre. Male, aged 35	Vomiting after a debauch	Longitudinal, 5 centimetres just above diaphragm; a second rent opposite extending only to the submucous tissue	Emphysema of neck immediately followed accident; double pneumothorax; cavity above cardia communicating with pleural cavity	Died 6½ hours	Groaning, bent forward in an easy chair.
Charles, Dublin Quart. Journ. of Med. Science, 1870, vol. i, p. 311. Man, aged 35	Vomiting; been drinking to excess	Longitudinal, 1½ inches to left side, immediately above cardia.	Communicated with a space in post mediastinum, which communicated with left pleural cavity by a round opening	Died 7½ hours	—
Dr. John Griffin, Lancet, 1869, vol. ii, p. 337. Healthy man, aged 49	Vomiting	Longitudinal rent 1 inch long immediately above diaphragm	Present on both sides of neck	Died 17 hours	Typical symptoms; no vomiting after the accident though emetics were given.
Mr. Howse, Lancet, 1884, vol. i, p. 113. Child, 3 years old	Vomiting following fractured base of skull	Longitudinal rent 1½ inches on left side, and behind ½ an inch above cardiac orifice	Not noted	Died 2 days after original accident, 27 hours after vomiting	"Quite unlike the ho'e which would result from post-mortem digestion."
Dr. Habershon, Diseases of Abdomen, 2nd edit., 1862. Man, aged 24, intemperate	Vomiting	Longitudinal rent above diaphragm extending into stomach; stomach-pump was used	Emphysema of face and throat	Died about 10 hours	Specimen in Guy's Museum, No. 1799, 46.

Mr. Stanley Boyd (Mr. Godlee), Trans. Path. Soc., vol. xxxiii, 1882. Child, 4 months old	Vomiting	Longitudinal rent; circular fibres exposed between two separated longitudinal bands, midway between bifurcation of trachea and opening in the diaphragm	Sudden dyspnœa 2 hours before death	Died 2 hours	Frequent vomiting night before death, none the last night; clinical symptoms suggestive; child previously exhausted by erysipelas; reported on by a sub-committee as being probably due to post-mortem softening and digestion.
Mr. Heath, reported by Mr. Stanley Boyd. Young woman, aged 18	Vomiting; empty stomach; Addison's disease	Longitudinal rent 5 centimetres long on left side immediately above cardia	No emphysema; no dyspnœa	Died 10—12 hours	Collapse; "food returned before it could have reached stomach;" pain in lower part of sternum.
Drs. West and Andrews, Trans. Path. Soc., vol. xlviii. Man, aged 55	Vomiting	Longitudinal rent on left side and behind an inch from cardiac, ¾ of an inch long	Dyspnœa; cyanosis; apparently no emphysema	Died 24 hours	Œsophagus healthy; no signs of post-mortem digestion.
William Adams, Trans. Path. Soc., vol. xxix, p. 113. Man, aged 53	Vomiting after a meal	Longitudinal slit 1½ inches immediately above the diaphragm	Not noted	Died 7 hours	—
Dr. C. J. B. Williams, Trans. Path. Soc., vol. i, p. 151. Man, age not given	Vomiting after a meal	Extending into and immediately above the diaphragm	Not noted	Died 13 hours	—
Tandler, D. Zeitsch f. prakkt. Med., No. 52, 1878. Man, aged 17	Vomiting; considerable hæmatemesis	Transverse tear 1½ cm. long just above cardia	—	Died	Gullet softened by septic process
Drs. S. West and F. W. Andrewes, Brit. Med. Journ., April 10th, 1897. Man, aged 55	Vomiting	Longitudinal rent left side 1 inch from cardia, an inch in extent	Not noted; fluid in pleural cavity	Died	—

DISCUSSION

Dr. CYRIL OGLE.—There can be no doubt, I think, that in this case rupture of the œsophagus took place before death, for subcutaneous emphysema of the neck and left side of the chest was a striking feature, and obviously not due to decomposition, so that I was led to open the left pleural cavity under water, and thus found air in that cavity also, which had escaped from the posterior mediastinum. The air tubes and lung were sound. I examined the œsophagus very carefully. There was no stricture, no cicatrix, nor any evidence of ulceration, but the tube in its lower third was much softened, and in such a state as one might expect from digestion with hydrochloric acid and pepsin; through this part was a longitudinal rent. The rupture, therefore, was clearly ante-mortem, and was found to be associated with much softening; and the question arises whether any operation involving suture could be of much service in presence of such a condition of semi-gelatinous softening of the tube, whether the condition preceded or followed the rupture.

Dr. ROLLESTON.—Rupture of the œsophagus is an extremely rare accident, the only example I have seen being published in vol. xlv of the Pathological Society's 'Transactions,' p. 58, under the heading of "? Rupture of the Œsophagus; Abscess in the Posterior Mediastinum bursting into the left Pleura, Pyo-pneumothorax: Death." The patient was a coachman aged fifty, who was seized with sudden pain while making a great muscular effort. The rupture was not preceded, as in Dr. Bowles and Mr. Turner's cases, by vomiting. The man died forty-eight hours after the onset of symptoms. The rent was in the œsophagus just above the diaghragm, and led into a ragged abscess cavity, which again communicated with the left pleura. Examination of the collected cases shows that rupture occurs quite constantly in the lower third of the œsophagus. This is somewhat remarkable, inasmuch as the thinnest part of the œsophagus is its upper end, at its junction with the pharynx, where pharyngoceles or pulsion diverticula are met with. What are the factors that account for rupture occurring on the lower third? There are two possible explanations; (1) that the lower third of the œsophagus is composed of involuntary muscular fibres, while the upper part has voluntary muscular fibre, and the gullet is firmly fixed by the diaphragm and therefore cannot "give" when vomiting occurs; (2) that when the rare condition of simple ulceration of the œsophagus occurs the usual situation is in the lower third. It is a sug-

gestive fact that simple ulceration and rupture are met with in the same part of the œsophagus. If rupture takes place at the site of a small ulcer, all trace of the pre-existing ulcer may become obliterated. The œsophagus is the thickest part of the alimentary canal, and yet it appears to be practically the only part where rupture (apart from traumatism) occurs without pre-existing ulceration.

Mr. STANLEY BOYD.—I wish in the first place to add a case to the author's list. In the 'Zeitschrift für Prakt. Med.,' No. 52, 1878, Tändler reports a case of a man aged seventeen who had a contused wound of the arm and developed gangrene followed by septicæmia. On the fourth day he vomited much blood. Post mortem the left lung was found much compressed by blood, and there was a rupture of the œsophagus on that side. Referring to the cases which I have myself recorded, and which are referred to by the authors, I am rather surprised to find that in the first case the symptoms are alluded to as being typical. I can only say that we had no idea whatever that the child was suffering from any lesion of the kind. There was no sudden shock or pain, no emphysema, and no pneumothorax. A strong sub-committee of the Pathological Society decided that the rupture was due to post-mortem digestion, but I still feel very doubtful whether the committee were right. They seemed to have overlooked the first thing that struck me on opening the œsophagus, viz. that between the longitudinal rugæ of the œsophagus there were small clots about an inch long, which could hardly have been produced by post-mortem digestion. My own view was that there had been a small ulcer of the œsophagus which had been burst by some slight effort shortly before death. That perforation occurred during life was supported by the facts that the left lung was more vascular than the right, and had numerous petechiæ on it opposite the perforation. In the second case, that of a girl who had been vomiting very frequently for two months, I got a pretty clear history of Addison's disease. Here again no typical symptoms developed, and, in spite of the fact that there was a tear in the œsophagus 5 cm. long, there was no pneumothorax or emphysema. In neither of these cases, therefore, can we say that the typical symptoms or post-mortem appearances were found.

Mr. BRYANT.—I have notes of three or four cases of rupture of the œsophagus from injury, but these have no bearing upon the class of cases before us. I remember, however, one which has been a worry to me for years. Some years ago I was called to see a patient in a lunatic asylum. She was a woman about forty years of age, of the imbecile type, who, previously healthy, had been suddenly seized with vomiting, which lasted for some hours. She was comparatively sensible, and on ques-

tioning her as to the cause of the vomiting she pointed to a corner of the room where there were some pieces of firewood, and she led the resident medical officer to believe that she had swallowed one of them. She was very collapsed, cold and sweating. Her vomit was blood-stained mucus. I examined her very carefully, but could find nothing to support the view that a foreign body was in her œsophagus. I then passed, for diagnostic purposes, a soft instrument down the œsophagus, and as it made its way quite easily there was evidently no obstruction. The instrument brought away a little of the stomach contents. The patient died, and at the post-mortem examination a large vertical rupture of the œsophagus in its lower third was the only thing found. I now believe this case to be an example of rupture of the œsophagus from vomiting, similar in its nature to the case brought before us to-night.

Dr. Bowles.—There is a point in the diagnosis of the case which I would like to emphasise, namely, the state of the pulse —fifty per minute and very tense. An accident in such a part would be very likely to affect the pulse through injury to, or irritation of, the pneumogastric. In reference to the position of the rupture, we must remember that the lower end is the narrowest part of the œsophagus and the part to receive the first shock in the act of violent vomiting. The vertical position of the rupture is also what we might expect, seeing that the circular fibres are more prepared to receive the shock, while the longitudinal fibres are easily separated from each other and the force from within is only there resisted by fibro-cellular membrane. I am told that leaden pipes do not burst horizontally, but vertically. I am inclined to think that this accident is more common than is suspected, and that the symptoms have been attributed to other causes, the œsophagus being rarely examined at post-mortems, at least not in private practice. Pneumothorax would only occur if there were rupture of the pleural membrane. We hope to make further experiments which may throw some light on this interesting subject.

Mr. Turner.—I thank Dr. Ogle for having borne testimony to this being an ante-mortem rupture. There did not seem to me post mortem to be the slightest trace of ulceration or post-mortem digestion. With regard to Dr. Rolleston's case, the query which he had inserted after the title of rupture, in the 'Transactions' of the Pathological Society, might, I think, now be taken away. I have been trying to keep to the subject of the effects of vomiting on the more or less healthy œsophagus, so that I did not avail myself of the reference to this case furnished to me by Dr. Rolleston. I cannot think that it is altogether necessary to assume that ulceration must be an antecedent condition of rupture of the œsophagus. Yesterday, when I was making a few experiments in this direction, I was

struck with the great ease with which the œsophagus ruptured. Even a healthy œsophagus can be readily distended, and it is but a step from this distension to rupture. I am indebted to Mr. Stanley Boyd for referring me to a case which we had overlooked. I shall ask permission to add it to my list of cases. I had spoken to him about these two cases of his some time ago. Even when I saw that a committee had decided that they were due to post-mortem digestion, I must confess the clinical history of the cases did not convey that impression to me. I gather, too, that he agrees with me that the cases in question were rupture of the œsophagus rather than post-mortem digestion. I think that the case of the child who up to two hours before death was able to swallow everything comfortably, and then became very ill, with sudden collapse and dyspnœa, and could swallow nothing without pain afterwards, was one of rupture of the œsophagus. I gather that Mr. Boyd, too, thinks there was a rupture, although it may have been preceded by a small ulcer. I think this injury is more common than is believed, and that many cases of death with agonising pain, where no post-mortem has been made, may have been due to this cause. In regard to the surgical treatment, I ventured to suggest opening the posterior mediastinum, or even suturing the œsophagus. When we are already dealing with a condition of pneumothorax and emphysema rapidly going on, there is no necessity to avoid injury to the pleura, and this would enable the operation to be done much more easily and quickly.