

- De la Huerga, J., and Popper, H. (1949). *J. Lab. clin. Med.*, **34**, 877.
- Duguid, J. B. (1955). *Brit. med. Bull.*, **11**, 36.
- Fearnley, G. R., Balmforth, G., and Fearnley, E. (1957). *Clin. Sci.*, **16**, 645.
- Fox, F. W., and Golberg, L. (1944). *Publ. S. Afr. Inst. med. Res.*, **9**, 123.
- Fullerton, H. W., Davie, W. J. A., and Anastasopoulos, G. (1953). *Brit. med. J.*, **2**, 250.
- Gertler, M. M., and White, P. D. (1954). *Coronary Heart Disease in Young Adults*. Harvard Univ. Press, Cambridge, Mass.
- Gilchrist, E., and Tulloch, J. A. (1952). *Edinb. med. J.*, **59**, 561.
- Gillman, T., Naidoo, S. S., and Hathorn, M. (1957). *Lancet*, **2**, 696.
- Greig, H. B. W. (1956). *Ibid.*, **2**, 16.
- Harrison, G. A. (1957). *Chemical Methods in Clinical Medicine*, 4th ed., p. 136. Churchill, London.
- Higginson, J., and Pepler, W. J. (1954). *J. clin. Invest.*, **33**, 1366.
- Hjort, P., Rapaport, S. I., and Owren, P. A. (1955). *J. Lab. clin. Med.*, **46**, 89.
- Keys, A., Buzina, R., Grande, F., and Anderson, J. T. (1957). *Circulation*, **15**, 274.
- Koller, F., Loeliger, A., and Duckert, F. (1951). *Acta haemat. (Basel)*, **6**, 1.
- Kunkel, H. G. (1947). *Proc. Soc. Exp. Biol. (N.Y.)*, **66**, 217.
- Kwaan, H. C., McFadzean, A. J. S., and Cook, J. (1956). *Lancet*, **1**, 132.
- Lackner, H., and Goosen, C. C. (1959). *Acta Haematol. (Basel)*, **22**, 58.
- Lawry, E. Y., Mann, G. V., Peterson, A., Wysocki, A. P., O'Connell, R., and Stare, F. J. (1957). *Amer. J. Med.*, **22**, 605.
- McDonald, L., and Edgill, M. (1957). *Lancet*, **2**, 457.
- Macfarlane, R. G., and Biggs, R. (1948). *Blood*, **3**, 1167.
- Maclagan, N. F. (1944). *Brit. J. exp. Path.*, **25**, 234.
- Manning, P. R., and Walford, R. L. (1954). *Amer. J. med. Sci.*, **228**, 652.
- Merskey, C. (1950). *J. clin. Path.*, **3**, 130.
- and Nossel, H. L. (1957). *Lancet*, **1**, 806.
- Meyers, L. (1948). *Arch. intern. Med.*, **82**, 419.
- Morgan, A. D. (1956). *The Pathogenesis of Coronary Occlusion*. Blackwell, Oxford.
- Oliver, M. F., and Boyd, G. S. (1953). *Brit. Heart J.*, **15**, 387.
- Owren, P. A., and Aas, K. (1951). *Scand. J. clin. Lab. Invest.*, **3**, 201.
- Pool, J. G., and Robinson, J. (1959). *Brit. J. Haematol.*, **5**, 17.
- Poole, J. C. F. (1955). *Brit. J. exp. Path.*, **36**, 248.
- (1958). *Brit. med. Bull.*, **14**, 253.
- Praetorius, E., and Poulsen, H. (1953). *Scand. J. clin. Lab. Invest.*, **5**, 273.
- Quick, A. J. (1957). *Hemorrhagic Diseases*. Kimpton, London.
- Rokitansky, C. (1841). *Lehrbuch der pathologischen Anatomie*. Vienna.
- Sohar, E., Rosenthal, M. C., and Adlersberg, D. (1957). *Amer. J. clin. Path.*, **27**, 503.
- Sougin-Mibashan, R. (1959). To be published.
- Steiner, A., Kendall, F. E., and Mathers, J. A. L. (1952). *Circulation*, **5**, 605.
- Tulloch, J. A., Overman, R. S., and Wright, I. S. (1953). *Amer. J. Med.*, **14**, 674.
- Virchow, R. (1856). *Gesammelte Abhandlungen zur wissenschaftlichen Medicin*. Frankfurt.
- Walker, A. R. P., and Arvidsson, U. B. (1954). *J. clin. Invest.*, **33**, 1358.
- Watt, B. K., and Merrill, A. L. (1950). *Composition of Foods—Raw, Processed, Prepared*. Dept. of Agric., Washington, D.C.
- Weichselbaum, T. E. (1946). *Amer. J. clin. Path.*, **16**, Techn. Bull., 7, 40.
- Wolfsen, W. Q., Cohn, C., Calvary, E., and Ichiba, F. (1948). *Amer. J. clin. Path.*, **18**, 723.

First-aid boxes, cases, and cupboards for all workers in factories, at docks, or on building-sites throughout the country have had important changes made in their contents from January 1, when new regulations made by the Minister of Labour under the Factories Acts came into operation. These changes have arisen from the advances made in medical and first-aid treatment of injuries during the last quarter of a century. For example, antiseptics, special burn dressings, and cocaine eye-drops will no longer be required. A leaflet giving advice on treatment, which has been revised in the light of these advances, must be included in each first-aid box. All materials have to be of a grade and quality not lower than the standard specified by the *British Pharmaceutical Codex*, and specifications for adhesive dressings for wounds and for eye ointment, for which the regulations provide, are given in certificates of approval of the Chief Inspector of Factories (Ministry of Labour).

BLEEDING OESOPHAGEAL VARICES TREATED BY OESOPHAGEAL COMPRESSION TUBE

BY

A. E. READ, M.D., M.R.C.P.

A. M. DAWSON, M.B., M.R.C.P.

D. N. S. KERR, M.B., M.R.C.P.

M. D. TURNER, M.D., M.R.C.P.

AND

SHEILA SHERLOCK, M.D., F.R.C.P., F.R.C.P.Ed.

From the Department of Medicine, Postgraduate Medical School, University of London

Oesophageal tamponade for the arrest of haemorrhage from oesophageal varices was introduced by Westphal (1930), who applied local pressure by means of an oesophageal sound. Later an inflatable rubber balloon on a modified Miller-Abbott tube was used to compress the coronary veins in the fundus of the stomach which drain into the oesophageal varices (Rowntree *et al.*, 1947; Bixby, 1948; Tocantins, 1948; Barnett and Cohen, 1949). The introduction by Sengstaken and Blakemore (1950) of a tube with two balloons enabled local pressure on the varices by the oesophageal balloon to be added to the effect on the fundal veins of the gastric balloon (Fig. 1).

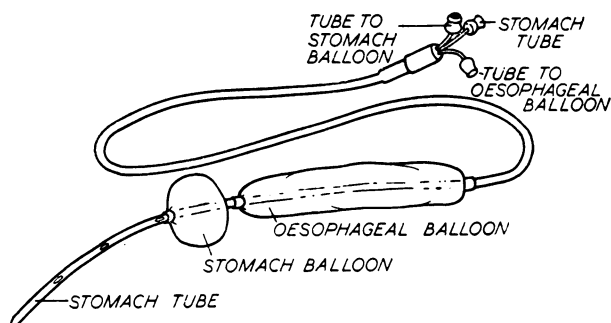


FIG. 1.—Sengstaken oesophageal compression tube.

The purpose of this paper is to describe and discuss the indications for and the results obtained with this apparatus in relation to a group of 101 patients with alimentary bleeding associated with portal hypertension seen at Hammersmith Hospital during the years 1954 to 1958.

Apparatus and Indications for Use.—The Sengstaken tube was a standard three-lumen instrument made of stiff rubber with latex rubber balloons (manufactured by J. G. Franklin and Sons Ltd., London). If possible a new apparatus was used for every second patient, and when not in use it was stored in the refrigerator to maintain its stiffness. The indication for use was haemorrhage from oesophageal varices uncontrolled by liberal blood transfusion and bed rest.

Introduction of the Tube

The apparatus was thoroughly tested for leaks. The alert and co-operative patient was told the purpose of the manoeuvre and his co-operation obtained. The pharynx was sprayed with 4% lignocaine, the patient being propped up in bed. A bandage was placed over

his eyes so that he could not see the size of the "mouthful" he was being required to swallow. The well-lubricated tube was then passed, usually through the mouth, with relatively little difficulty. The introduction was carried out speedily and in a determined manner to avoid prolonged retching. In those who were comatose the operation proved more difficult, and in three instances the patients were so noisy and violent that the operation proved impossible.

A mixture of 20 ml. of 70% diodone and 100 ml. of water was used to fill the gastric balloon, so making it

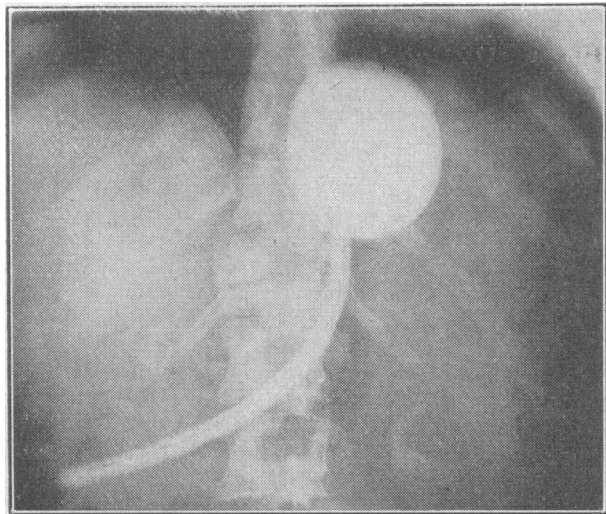


FIG. 2.—Plain x-ray film of abdomen showing the Sengstaken tube in good position. The gastric balloon is distended with 10% diodone solution.

radio-opaque (Fig. 2). Considerable pressure may be needed to distend the gastric balloon with this volume of fluid.

The oesophageal balloon is distended with air at a pressure of 30 mm. Hg, great care being taken to observe any respiratory embarrassment following its inflation. In some patients of small stature the adult-sized oesophageal balloon produced respiratory difficulty and a smaller-size tube had to be used. Sengstaken tubes of various sizes should always be available. The gastric and oesophageal balloons should be attached close together on the main tube to prevent the oesophageal balloon reaching the larynx. Several methods of fixation have been tried. The tube may be pulled up and then attached with adhesive tape to the patient's face, but this leads to ulceration of the corners of the mouth. A head-mask was devised to which the apparatus was fixed, but this led to excessive traction being applied and limited the patient's movement in bed.

The most satisfactory method has been to suspend the Sengstaken tube over a pulley-wheel on a ball-bearing race, allowing the weight of the apparatus to apply traction (Figs. 3 and 4). This enables the patient to move his head freely, and by altering the position of the suspension beam he can be easily turned in bed. The pulley-wheel is kept above the patient's mouth so that all friction is avoided. Clip-on weights (150 g. each) can be applied to the tube if further traction is necessary; not more than four should be used. The tubes leading to the oesophageal gastric balloons are clearly marked and double clamped, and the nursing staff must never touch them except in an emergency such as laryngeal obstruction.

The position of the gastric balloon is located by portable plain x-ray film of the abdomen (Fig. 2). It must fit well into the fundus of the stomach, and if it does not extra weights may be required if bleeding continues.

General Management

Once the oesophageal balloon is inflated the patient must not be left unattended, and a special nurse is necessary. The pulse and blood-pressure are recorded every 30 minutes. A mechanical suction apparatus is used to remove pharyngeal secretions from above the oesophageal balloon. A careful watch is kept for signs of respiratory difficulty due to migration of the balloons causing laryngeal obstruction. In the event of such signs occurring the nurse must release the oesophageal balloon and if necessary cut the tube across with scissors, so effecting a rapid decompression of both balloons.

A specimen is aspirated from the stomach every hour, and these are labelled and preserved in a test-tube in a rack. If the aspirate is blood-stained the stomach should be completely emptied. Various therapeutic agents, such as 20% glucose solution, neomycin, potassium supplements, and purgatives, are administered down the gastric tube (Sherlock *et al.*, 1956). A careful record of these must be kept and also of urine passed and the nature of the stools.

The patient's position is frequently changed without disturbing the apparatus. Ideally, in order to minimize the risks of aspiration of pharyngeal secretions the foot of the bed should be blocked. In the presence of tense ascites this position is poorly tolerated because of respiratory distress, and cautious release of 1 to 2 litres of ascitic fluid may make such a position more bearable.

Control of bleeding is shown by improvement in the patient's general condition, blood-pressure, and pulse, and by obtaining a continuously clear gastric aspirate.

After bleeding has ceased for 24 hours the oesophageal balloon is deflated and traction taken off the tube.

After a further 24 hours, with the apparatus "lying free," if bleeding does not recur the fluid from the gastric balloon is aspirated and the apparatus removed. Decisions to release the traction or remove the tube are made by the attending physician and cannot be relegated to a more junior level.

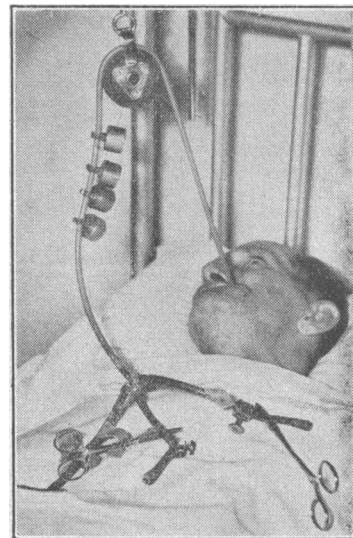


FIG. 3.—A patient with the Sengstaken tube in position. The tube passes over a pulley-wheel on a ball-bearing race. Weights are attached. The tubes leading to the oesophageal and gastric balloons are labelled and doubly clamped.

Results of Treatment

The Sengstaken tube was used in 38 patients with gastro-intestinal haemorrhage associated with portal

hypertension. In 32 the haemorrhage was initially controlled. In the other six the tube was ineffective, and subsequent examination showed the cause to be a haemorrhagic gastritis in two patients and gastric varices in two; the remaining two showed no apparent reason for failure. Only 10 of the 38 patients treated survived to leave hospital. In many of those who eventually died the tube was passed on several occasions

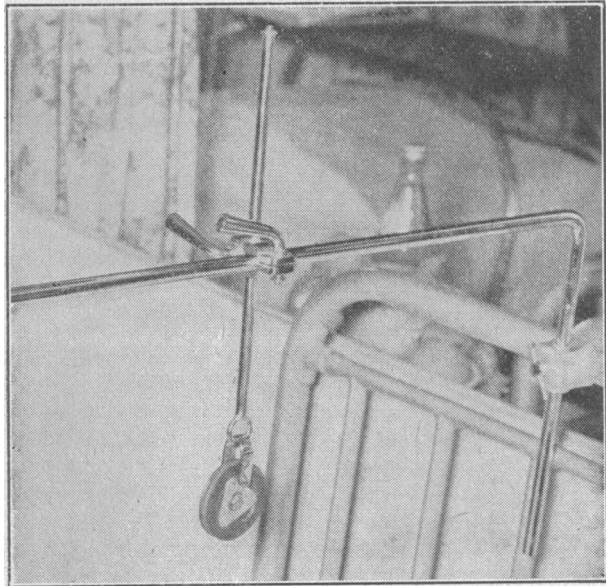


FIG. 4.—Suspension apparatus used for the Sengstaken tube. The position of the horizontal suspension beam and the height and angulation of the pulley-wheel can be varied.

in an effort to control recurrent haemorrhage. There was a marked difference between the initial effect of the tube and the response to further bleeds. The patients seemed to develop a state of "tube resistance" and finally died still bleeding. Evidence of haemorrhage was in fact noted in 23 of the 28 fatal cases. Despite the poor results obtained in this group, 5 of the 10 survivors were later fit enough to undergo porta-caval anastomosis. Further, it is unwise to generalize, as one patient, a woman of 51, despite hepatic coma, ascites, and bleeding varices, made a full recovery after use of the Sengstaken tube and, without subsequent surgery, was alive and well five years later, working as a cinema usherette.

The high mortality must be discussed in relation to the whole problem of portal hypertension. Between 1954 and 1958 101 patients with portal hypertension and haemorrhage were seen at Hammersmith Hospital (Table I). A group of 43 patients had suffered and recovered from an episode of bleeding oesophageal varices without the use of oesophageal tamponade. They were referred for consideration of definitive surgical treatment. These patients were not jaundiced, generally, had good serum albumin levels, and rarely showed fluid retention. They might be considered "good-risk" subjects. Twenty-six were suitable for some surgical treatment and 20 had porta-caval anastomoses and six local operations on the portal system. Only one died.

The other group of 58 patients consisted of those who were referred to Hammersmith Hospital because bleeding could not be controlled elsewhere or who bled while in a hospital under treatment for some other

TABLE I.—The Two Groups of Patients Studied

	Patients Admitted with or Developing Haemorrhage	Patients Admitted having Recovered from Haemorrhage
Total	58	43
Portal cirrhosis	54	39
Portal-vein block	4	4
Jaundiced	13	0
Mean serum albumin (g./100 ml.)	2.9	3.5
Ascites and/or oedema	34	3
Sengstaken tube	38	0
Survived intubation	10	—
Porta-caval shunts	5	20
Other surgery	6	6
Deaths	40	1

complication of cirrhosis such as jaundice, hepatic precoma, or ascites. These patients were therefore often jaundiced, and usually showed a low serum albumin level and also fluid retention. They might be considered "poor-risk" patients. The Sengstaken tube was passed on 38 patients, and, though bleeding was initially controlled in 32, only 10 survived to leave hospital. Only 11 were in a sufficiently good condition for surgical intervention. Five had porta-caval anastomoses, and six various operations designed to reduce the blood-flow to the lower oesophagus; all these latter procedures proved fatal. Total deaths were 40.

Complications (Table II)

Ulceration of Pharynx and Lower Oesophagus.—Ulceration of the lower oesophagus was seen in 10 of the 28 patients intubated who came to necropsy (Table III). It seemed to be related both to the duration of intubation and to the use of traction. The average duration of intubation in these 10 patients was five days. The average duration in 18 patients showing no ulceration at necropsy was 2.3 days. There were exceptions, and ulceration developed in two patients within 24 hours, and was not present in two others even after six and seven days' intubation. Fixed traction was used on four occasions, the tube being attached to a special head-piece. This resulted in deep oesophageal ulceration in one patient and superficial ulceration in the three others. Pharyngeal ulcers were noted at necropsy

TABLE II.—Complications of Intubation with the Sengstaken Tube

	No.
Ulcerative	10
Lower oesophagus	10
Pharynx	3
Respiratory	5
Obstruction	5
Aspiration of vomitus	5
Infection and collapse	4
Mechanical	3
Delirious patient	3
Tube bitten through	1
Burst gastric balloon	5
Gastric balloon migration into oesophagus	5
Infective	2
Staphylococcal enteritis	2

TABLE III.—Pharyngo-oesophageal Ulceration Following Intubation with the Sengstaken Tube

Patient	Site	Intubation (Days)	Traction by Head-mask	Ulcer	
				Type	Size (mm.)
1	Oesophagus	1	No	Superficial	2 × 2
2	"	2	Yes	"	60 × 15
3	Oesophagus; pharynx	8	"	"	5 × 10 15 × 5; 10 × 7
4	Oesophagus	7	"	Deep	60 × 20; 40 × 20
5	"	5	"	Superficial	10 × 10
6	"	3	No	"	3 × 3
7	"	3	"	"	5 × 5
8	"	4	"	"	15 × 40
9	Oesophagus; pharynx	1	"	"	15 × 10 35 × 20
10	"	16	"	"	30 × 20 5 × 7

in three patients, all of whom had accompanying oesophageal lesions (Figs. 5 and 6).

Respiratory Complications.—With the complete blockage of the oesophagus it is not surprising that secretions collecting above the oesophageal tube should be aspirated into the air passages. Such complications may be prevented by pharyngeal suction, by blocking the foot of the bed, and by vigorous physiotherapy. Aspiration is particularly likely to occur while the apparatus is being passed, for this manœuvre may produce retching. If the stomach is at the same time distended by a large volume of blood this may asphyxiate the patient. Two of our subjects died in this way. When the passage of the Sengstaken tube is tolerated poorly it is wise to empty the stomach with a soft rubber tube, and suppress the gag reflex with a small intravenous dose (25 mg.) of pethidine. Epistaxis is also potentially lethal, and as it is common in cirrhotic patients it must be carefully and energetically treated to prevent aspiration. Laryngeal obstruction due to the distended oesophageal balloon occurred in four patients, and in one other case laryngeal obstruction associated with oedema and laryngeal ulceration resulted in death.

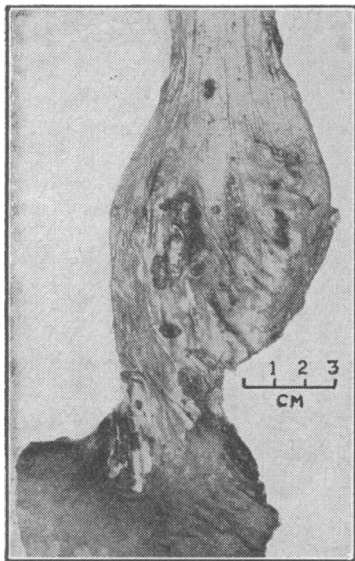


FIG. 5.—Patient 3. Necropsy specimen of oesophagus and upper part of stomach showing superficial ulceration of the lower oesophagus after eight days' intubation.

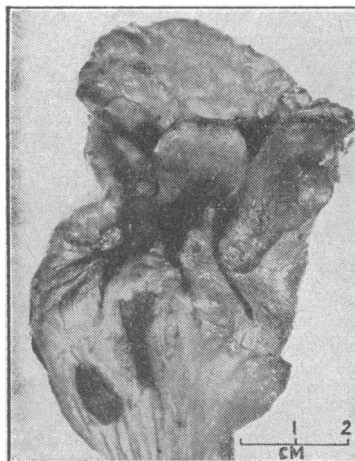


FIG. 6.—Patient 3. Two large superficial pharyngeal ulcers are seen.

intubation may lead to necrosis of soft tissues and cartilage and possibly initiate epistaxis; this route was reserved for those with some contraindication to oral intubation. The gastric balloon was the usual cause of mechanical difficulties. Bursting occurred five times, and may be noticed by the patient as a "pop" in the stomach. A puncture of the oesophageal balloon can be detected by noting collapse of a small "pilot" balloon attached to the inlet tube of the oesophageal balloon. The gastric balloon must be adequately distended both to exert a firm pressure on fundal varices and to prevent migration into the oesophagus. Such a

migration tends to occur early if the balloon is not properly distended, and later if the traction on it overcomes the resistance of the cardio-oesophageal sphincter. Migration may cause respiratory distress and also pain in the chest, felt in the back between the shoulder-blades. Though discomfort is usual, frank pain is uncommon, and its occurrence should suggest that the gastric balloon is in the oesophagus. Weights used for traction should be suspended just above the level of the bedclothes. If the apparatus moves up the oesophagus the weights will then drop on to the bed and so break the upward movement of the tube. Provided the balloons are not punctured, once displacement of the tube is diagnosed it can be replaced and the balloons redistended.

Infective Enteritis.—Two patients developed staphylococcal enteritis; both of them had been treated with tetracycline. This complication has not been seen since neomycin has been used for sterilizing the bowel.

Discussion

The Sengstaken oesophageal compression tube will usually arrest bleeding from oesophageal varices. The method has even been used diagnostically to distinguish bleeding peptic ulcer from bleeding varix (Nachlas, 1955). It is so successful that no patient with bleeding oesophageal varices should be denied its benefit if blood transfusion fails.

The tube was effective in the initial control of haemorrhage, but the ultimate mortality was high—in 32 of 38 patients the bleeding was initially controlled, but only 10 survived to leave hospital. This can be related to the type of patient being intubated. In our series the tube was used on those with very poor hepatic function. The haemorrhage was a manifestation not only of portal hypertension but of the bleeding tendency of advanced hepatic disease. It therefore recurred and the Sengstaken tube was introduced over and over again, the patients finally dying, often with further massive haemorrhage not only from the oesophagus but also from the stomach generally (Fig. 7). Loss of tone in the lower oesophagus consequent upon the prolonged presence of the gastric and oesophageal balloons may have added to the later failures to control bleeding. These results contrast with the group of 43 patients in whom bleeding from oesophageal varices had ceased without use of the Sengstaken tube. In these patients blood coagulation was presumably adequate and the haemorrhage was a manifestation of portal hypertension and not of liver failure. These patients were often treated in hospitals where facilities for the use of the Sengstaken tube were not available. It seems probable that in many of them bleeding would have ceased sooner had the tube been used. Certainly the amount of blood necessary for transfusion would have been much less.

The use of the Sengstaken tube must be considered in relation to later definitive portal surgery. If hepatocellular function permits, a porta-caval anastomosis should be considered as soon as possible after the first haemorrhage. In the patient with poor hepatic function (ascites, jaundice, hepatic precoma, or coma) major surgery is unlikely to be tolerated. In this group the bleeding may be repeated and the decision to use the Sengstaken tube again will have to be made. In borderline cases it is justifiable to continue if there is a chance that bleeding will be controlled, hepatic function

improve, and surgery be possible. Often, however, the recurrent bleeding produces a worsening of hepatic function and it becomes clear that the major feature is hepatocellular failure and surgery will be impossible. At this stage the apparatus should be withdrawn, as its further use increases the patient's misery and complications are likely. In some of these patients local surgical attack on the varices has been suggested, porta-caval anastomosis being performed later (Linton, 1953; Welch, 1957). This was attempted in six of our patients, but all died after the first operation, a reflection of their inability to support any form of surgical treatment.

Complications of intubation are common, and in one series of 50 patients treated with the Sengstaken tube

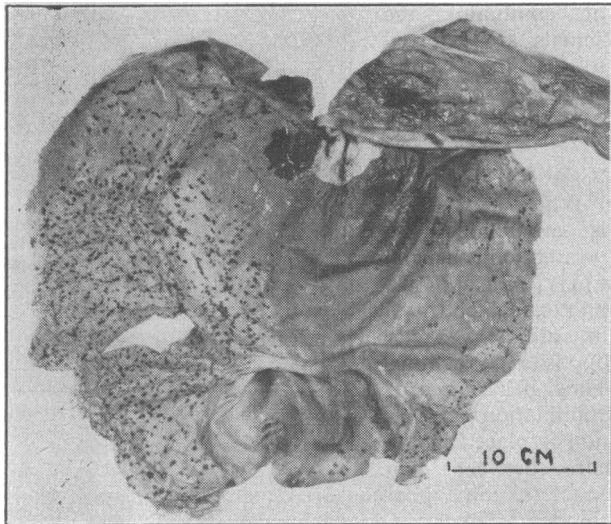


FIG. 7.—A cause of failure: diffuse haemorrhagic gastritis.

nine died as a consequence of its use (Conn, 1958). The most serious complication in this series was obstruction to the airway and regurgitation of blood and gastric secretions into the lungs. Our commonest complication was oesophageal ulceration. This was related to the duration of intubation and to the amount of traction. Nachlas (1955) believes that ulceration may be decreased by omitting the oesophageal tube and using a triple-lumen single gastric balloon tube. In our experience, however, this tube required more traction to keep it in position and the gastro-oesophageal ulceration rate was increased.

Summary

The Sengstaken oesophageal compression tube controlled the haemorrhage initially in 32 of 38 patients with portal hypertension and oesophageal varices. Only 10 survived to leave hospital, and only five of these were suitable for porta-caval anastomosis. The other 28 suffered recurrent bleeds and ultimately died in hepatic failure. Results are contrasted with a group of patients with good hepatic function and portal hypertension who also suffered haemorrhage but who recovered without the use of the Sengstaken tube.

Practical details of the intubation and management of the patient are described. Complications included ulceration of the oesophagus and/or pharynx (10 cases), respiratory complications (14 cases), mechanical difficulties with the apparatus (14 cases), and infective enteritis (2 cases).

The method is a useful one for controlling bleeding from oesophageal varices. Intubation should not be continued if bleeding recurs repeatedly and liver failure is obvious. Under these circumstances the patient is made miserable and complications are many.

We would like to pay tribute to the nursing staff of Hammersmith Hospital for their meticulous and sympathetic care of the patients reported.

REFERENCES

- Barnett, C. B., and Cohen, S. (1949). *Gastroenterology*, 13, 144.
 Bixby, E. W. (1948). *J. Amer. med. Ass.*, 138, 908.
 Conn, H. O. (1958). *New Engl. J. Med.*, 259, 701.
 Linton, R. R. (1953). *Gastroenterology*, 24, 1.
 Nachlas, M. M. (1955). *New Engl. J. Med.*, 252, 720.
 Rowntree, L. G., Zimmerman, E. F., Todd, M. H., and Ajac, J. (1947). *J. Amer. med. Ass.*, 135, 630.
 Sengstaken, R. W., and Blakemore, A. H. (1950). *Ann. Surg.*, 131, 781.
 Sherlock, S., Summerskill, W. H. J., and Dawson, A. M. (1956). *Lancet*, 2, 689.
 Tocantins, L. M. (1948). *J. Amer. med. Ass.*, 136, 616.
 Welch, C. S. (1957). *Surgery*, 41, 1029.
 Westphal, K. (1930). *Dtsch. med. Wschr.*, 56, 1135.

STUDIES OF REGIONAL LUNG FUNCTION USING RADIOACTIVE OXYGEN

BY

N. A. DYSON,* M.A., Ph.D.

P. HUGH-JONES,† M.A., M.D., F.R.C.P.

G. R. NEWBERY,* B.Sc., F.Inst.P.

J. D. SINCLAIR,† M.D., B.Med.Sc., M.R.A.C.P.

AND

J. B. WEST,† M.D.

The use of a radioactive isotope of oxygen provides a method of assessing regional variations of ventilation and blood-flow in the lungs without intubation of the patient.

This method has advantages over bronchspirometry, which has hitherto been used to compare the function of the two lungs, because in the latter procedure the passage of the catheter into the bronchial tree is unpleasant, the conditions are far from physiological, and it is not usually possible to measure local variations of function within either lung. Kipping *et al.* (1957) overcame some of these problems by using the radioisotope xenon-133. They measured local variations in ventilation, in the detection of bronchial tumours, by observing the distribution of radioactivity over different parts of the chest during normal breathing of air containing the radioisotope.

If oxygen is used instead of xenon, local blood-flow as well as ventilation can be assessed. This has not been practicable until now because of the rapidity with which radioisotopes of oxygen decay; the longest-lived, oxygen-15, has a half-life of only two minutes. However, the cyclotron built by the Medical Research Council (Gallop *et al.*, 1957) was sited within a hospital for a number of reasons, one of which was to make available

*M.R.C. Radiotherapeutic Research Unit, Hammersmith Hospital, London.

†M.R.C. External Scientific Staff and Department of Medicine, Postgraduate Medical School of London, Hammersmith Hospital, London.