Haemorrhage during Convalescence

In the vast majority of cases haemorrhage of this delayed type occurs on the fifth night following operation and is due to factors already enumerated. As a rule it is not severe, and may consist only in the patient coughing up a small bright red clot. It usually ceases after a gargle of weak hydrogen peroxide. Sometimes the haemorrhage, although not severe, continues during the course of an hour or more, and is associated with discomfort in the throat due to the presence of clot. The patient's efforts to dislodge the clot may then prolong the bleeding, in which case the treatment becomes exactly the same as that already described for reactionary haemorrhage. It will be found, however, that it is rarely necessary to give morphine, still more rare to have to remove the clot, and extremely rare to have to take the patient back to the theatre.

True Secondary Haemorrhage

Haemorrhage of this type is much more serious than that described under the heading of haemorrhage during convalescence. Fortunately, however, it is also more uncommon. When it does occur there is frequently a small "warning" haemorrhage on one day, followed by a larger one within the next twenty-four hours. In such a case it is likely that the patient will have to be taken back to the operating theatre. The control of this type of bleeding is often difficult, for it is seldom possible to clamp and ligate a single bleeding point on account of the friability of the sloughing and oedematous tissues of the bed of the tonsillar fossa. If the more simple measures already advocated for the control of bleeding are ineffective, recourse to suturing the faucial pillars will be necessary.

Ligation of the external carotid artery and the use of snake venom are measures of questionable value in the control of post-operative haemorrhage of this type. According to the literature ligation of the external carotid artery is of doubtful efficacy in any type of tonsillar haemorrhage on account of the extensive anastomosis of this vessel through the circle of Willis. Blood transfusion may be helpful—even life-saving—in cases of severe haemorrhage, but it is desirable that effective measures for the control of any bleeding should be instituted before transfusion becomes necessary.

I wish to express my gratitude to Mr. C. P. Wilson for his helpful advice and criticism during the preparation of this paper.

REFERENCE

McNally, W. J. (1927). *Canad. med. Ass. J.*, 17, 690.

The Fifth Biennial Conference on Mental Health will be held at the Central Hall, Westminster, London, S.W.1, from January 12 to 14, 1939. The conference luncheon will take place at the Hotel Victoria, W.C.2, on January 13. The subjects for discussion at the sessions of the conference are as follows: January 12, Is our national intelligence declining? Should mental treatment be practised solely by doctors? January 13: The psychological factor in sexual delinquency; The organization and staffing of out-patient mental treatment clinics; Mental hygiene and the Press. January 14: Education and emotional needs of the child; Problems of adolescent instability and of juvenile delinquency; The place of the social worker in mental health. Tickets and information can be obtained from the Secretary, The National Council for Mental Hygiene, 76/77, Chandos House, Palmer Street, London, S.W.1.

**PERFORATED PEPTIC ULCER IN
ORGANIC NERVOUS DISEASE**

BY

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The following two cases, in which death resulted from diffuse peritonitis due to perforation of a peptic ulcer, occurred within a little while of each other at St. Bartholomew's Hospital. Both patients were suffering from an organic disease of the nervous system, which masked the symptoms and signs of perforation, so that the complete condition was recognized only at necropsy.

Case I

A woman aged 41 was admitted to a medical unit on February 21, 1938, complaining of a pain in the shoulders. Ten years previously a diagnosis of disseminated sclerosis had been made, following some loss of the use of her legs. Similar attacks and remissions had occurred since. Two months previously she again lost the use of her legs, felt unwell, and vomited for a few days. She had experienced vague abdominal discomfort since. Three days before admission she developed sudden severe pain in both shoulders. Her bowels had not been opened subsequently.

On admission she was collapsed, clammy, cyanosed, and dyspnoeic. She complained of pain and tenderness of the shoulders, and she looked moribund. Her temperature was 98° F., pulse 160, and respirations 36. She showed no nystagmus. Her tongue was furred. There were no abnormal physical findings in her chest beyond the tachycardia and the tachypnoea. Her abdomen was distended and tense, but not rigid, and there was slight tenderness on deep palpation. The percussion note was resonant all over, and the area of liver dullness was reduced. There was spasticity of the left lower limb with increased knee-jerk and extensor plantar response. The temperature and pulse showed no marked variation, and she died the following day.

Post-mortem Examination.—The typical grey translucent scars of disseminated sclerosis were found throughout the cerebral hemispheres and, to a lesser extent, the cerebellum and spinal cord. The largest was 1 cm. in diameter and occupied the greater part of the right side of the pons. The heart was moderately dilated. Both pleural cavities contained small serous effusions, and the lungs showed emphysema and terminal congestion. The greater sac of the peritoneal cavity was distended with much gas and two pints of foul turbid fluid. The lesser sac contained an abscess cavity surrounded by dense fibrous tissue, but anteriorly the abscess had perforated through the gastro-hepatic omentum into the greater sac. This perforation was 1 cm. in diameter, and was situated midway between the cardia and pylorus. In the stomach a chronic peptic ulcer was discovered on the posterior wall immediately proximal to the pylorus. Its floor was completely deficient, and this hole communicated directly with the abscess cavity in the lesser sac. Just proximal to this perforation there was also a subacute ulcer 2 cm. in diameter.

Case II

A man aged 55 was admitted to a medical unit on March 18, 1938, complaining of feeling weak. One hour previously, while walking in the street, he had suddenly "come over queer" and felt some pain in the back of the shoulders. He passed a loose motion, his vision became dim, and he collapsed, though he was never unconscious.

On admission he was cyanosed and sweating, but not in pain. He appeared very shocked, but answered questions normally. His temperature was 95° F., pulse 88, and respirations 22. His pupils reacted to accommodation but not to light. His tongue was furled. The cardiac impulse was feeble and the heart sounds were faint but regular. The blood pressure was 58/30 mm. Hg. Some rhonchi and medium rales were heard at the lung bases, but his chest appeared otherwise normal except for a band of anaesthesia over the upper part. His abdomen was soft and not distended, with some tenderness in the right hypochondrium. The right flank was dull on percussion and the area of liver dullness was diminished. The knee-jerks were absent and the plantar responses were flexor. There was a patchy anaesthesia on the outer sides of the legs. The following day he was still cyanosed and weak, but free from pain. The most striking change was the increasing distension of his abdomen. The percussion note had become drum-like, and the liver dullness was now obliterated. No flatus was passed on the insertion of a rectal tube. His temperature in the morning was 98.4° F., the pulse 80, and the respirations 17. The absence of pain or much tenderness, and the steadiness of the temperature and pulse rate, were outstanding features. He became progressively weaker, and died the same day.

Post-mortem Examination.—The air passages contained regurgitated stomach contents. There was a small serous effusion in each pleural cavity and the lungs showed terminal congestion and oedema. All chambers of the heart were dilated and the muscle was soft and friable; the valves were normal. There was atheroma but no other abnormality of the aorta and coronary arteries. The peritoneal cavity was greatly distended with gas and foul turbid fluid. In the stomach, just proximal to but also involving the pylorus, was a perforated chronic peptic ulcer with a crater measuring 2 cm. in diameter. The whole of the floor of the ulcer was deficient.

Discussion

In neither of these cases is the history or physical examination at first suggestive of perforated peptic ulcer. The existence of a disease of the nervous system was known (disseminated sclerosis and tabes dorsalis respectively), but the reason for the collapse was discovered only at necropsy. In the first case medullary failure from a plaque of sclerosis was considered a possible cause; in the second, tabetic crisis, coronary thrombosis, and perforated peptic ulcer were suggested, but no conclusion was arrived at. Both patients were collapsed, but neither of them gave a history of severe abdominal pain. The only pain of any consequence that they had experienced had been in the shoulders. The temperature and pulse rates showed no variation, and, although in the first case the pulse was 160, in the second it was only 88 on admission and fell to 80 subsequently. In neither instance was there any abdominal rigidity, nor was there any tenderness except on deep palpation. The most important positive finding, which was common to both cases, was the development of abdominal distension with a very resonant note on percussion and diminution of the area of liver dullness. This became particularly noticeable in Case II.

A review of all the cases of perforated peptic ulcer that have come to necropsy at St. Bartholomew's Hospital during the past decade revealed that during this period there have been two others with organic disease of the nervous system comparable to the two cases here described. The one, a diabetic woman of 62, was suffering from severe peripheral neuritis. She suddenly developed pain in the left side of the abdomen, with rigidity and tenderness only on the right side. She passed no urine, became incontinent of faeces, and died the next morning. At necropsy a large chronic gastric ulcer was found to have perforated, resulting in diffuse peritonitis. Degeneration

of the posterior root ganglia and posterior columns of the spinal cord, more on the left side than on the right, was demonstrated. The other case was that of a tabetic woman aged 49 who had been admitted to hospital for investigation and treatment of intermittent headaches. One night her pulse suddenly became rapid and weak. Her abdomen was found to be tender and rigid and tympanitic on percussion, but she did not complain of anything. She died the next morning, and at necropsy peritonitis due to a perforated peptic ulcer was discovered.

It is recognized that a peptic ulcer may rarely perforate in an otherwise normal patient without producing the typical syndrome, but it is difficult to assess the proportion in which this occurs. Most of the cases reviewed, in which a perforated peptic ulcer had been found only at necropsy, were admitted in a moribund condition and no clear history had been obtained.

The four cases here described are the only instances during the past ten years in which perforation and organic nervous disease were both present, and in all four the history, symptoms, and signs were atypical. In certain organic diseases of the nervous system it is thus evident that a peptic ulcer may perforate without producing abdominal pain or rigidity and without causing an increasing pulse rate. Presumably this is the result of interference with the normal pathway of pain impulses. In such instances, however, progressive distension of the abdomen with a tympanitic percussion note and diminution of the area of liver dullness are in themselves signs of the utmost significance in arriving at the diagnosis.

We are indebted to Dr. Geoffrey Evans and Dr. James Maxwell for permission to report the cases under their care.

ADMINISTRATION OF PITUITARY EXTRACT IN THE THIRD STAGE OF LABOUR

BY

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It is commonly taught that administration of any oxytocic drug during the third stage of labour is dangerous and inadvisable, in that hour-glass contraction with retention of the placenta and all its attendant risks is apt to result. Here are some conflicting views taken from standard textbooks.

Gibbon Fitzgibbon (1937) says: "There is no place for the drug while the placenta is in the uterus. Its use always entails the risk of imprisoning the placenta by closure of the cervix." Tweedy (1937) also advises against this treatment. Eden and Holland (1937) say that except for experts its use should be limited to the third stage of labour after the placenta has left the uterus. Gibberd (1938) points out the danger of giving any oxytocic drug before the placenta is delivered, on account of the risk of hour-glass contraction. Most American writers, however, are in favour of giving pituitary extract during the third stage. E. A. Schumann (1937) takes the third stage of labour as an indication for administering pituitary extract. He says that 0.5 c.cm. may be given with safety, and adds, "Incarceration of the placenta by a cervix contracted by pituitrin has been reported, but must be quite rare." DeLee (1933) holds that when pituitary extract is administered, which is very often done nowadays after the delivery of the child, the third stage is much altered, and