found in the peritoneal cavity. Massive oedema and inflammation of the pelvic colon were present, probably from a perforated diverticulum. The picture appeared to be so hopeless that the temptation was to close the abdomen, but a transverse colostomy was performed. No relaxant was needed for the closing of the abdomen. During the operation 4 pints (2,270 ml.) of blood was administered, and the systolic blood pressure was steady at 100 mm. Hg. The pulse continued to be irregular, but it improved with 0.4 g. of aminophylline in a drip and 0.25 mg. of digoxin intravenously.

The patient was kept under mild hibernation for nine days, receiving chlorpromazine hydrochloride, 25 mg., and promethazine hydrochloride, 25 mg., four times in the first day, three times in the second, and only once daily for the remaining seven days. During this time he was very quiet and easily aroused; there were no technical difficulties with the intravenous therapy, the same needle being used for three days. He made an uninterrupted recovery and was discharged four weeks after operation.

Case 3

A man aged 63 was admitted to hospital complaining of severe abdominal pain, with a thready rapid pulse, tending to disappear, and a blood pressure of 90/70. There was a history of sudden onset of severe pain in the left iliac fossa six hours previous to admission. Examination of the abdomen showed a swelling in the left iliac fossa, but palpation was difficult owing to the size of the patient—17 stone (108 kg.). Intravenous therapy and general resuscitative measures were started, but his condition gradually deteriorated. A diagnosis of ruptured aneurysm was made, and it was decided to operate under hibernation.

On his arrival at the theatre 2 pints (1,140 ml.) of blood was given concurrently and 8 ml. of hibernation mixture administered. The speed of the drips increased automatically; the patient became warm and pink and the blood pressure was 80 mm. Hg. He then received a further 18 ml. of hibernation mixture, and was intubated and moved to the operating table on a N_2O-O_2 50% mixture. It was apparent that he was still bleeding, as the blood had to be run in very fast to maintain the blood pressure at 80 mm. Hg after the sympathetic block was complete.

Before opening the peritoneum thiopentone, 150 mg., and D-tubocurarine chloride, 5 mg., were given. There was a huge retroperitoneal haemorrhage, but the obesity of the patient made palpation of the aneurysm extremely difficult. The operation continued for six and a half hours, the patient receiving 9 pints (5.1 litres) of blood, but, apart from 50 mg. of suxamethonium chloride for closing the peritoneum, no further anaesthetic was needed. The technical difficulties of ligating the left common iliac artery above the level of the aneurysm were such that it was decided to insert fine stainless-steel wire into the aneurysm. While this was being done the pulse suddenly became extremely irregular. X-ray examination showed that the wire had entered the auricle via the inferior vena cava; its removal was technically very difficult, owing to tangling. A further attempt at ligation above the aneurysm stopped all bleeding and pulsation from the aneurysm. One hour after the patient was moved off the operating table he collapsed suddenly and died. It is believed that the wire had caused clot formation in the inferior vena cava, but unfortunately a post-mortem examination was not possible.

Technically the whole operation was extremely difficult owing to (1) the patient's obesity, (2) the massive retroperitoneal haemorrhage, and (3) the size of the aneurysm, which appeared to originate from the left common or internal iliac artery and completely filled the pelvis and left iliac fossa, making "landmarks" impossible to find. Though the patient died, we believe some form of treatment was essential, as he would most certainly have died without operation. His condition during the operation was excellent, and the value of the hibernation technique for such cases was clearly demonstrated.

Comment

The clinical picture presented by these three cases is of relatively common occurrence in a general hospital when the decision to operate is often influenced by the superimposed risk of general anaesthesia. This, while producing a further deterioration in the patient's hypotensive state, offers no solution to the problem of speedy and safe fluid replacement.

The dramatic improvement in the patients' condition, occurring as it did as a result of the sympathetic block and before the benefit of the restored fluid and electrolyte balance could be experienced, was most impressive. It is of the utmost importance to ensure that the replacement of fluid keeps step with the enlargement of the vascular bed resulting from the sympathetic block, and to this end the induction of hibernation must be performed very slowly and with constant supervision of the blood pressure. The speed and relative safety of fluid replacement are an important factor throughout the management of these cases.

Summary

Three cases of severely shocked patients who had operations performed under "hibernation" are described. Dramatic improvement in the patients' condition occurred before completing the induction of hibernation.

The ease of fluid replacement during the operation and in the post-operative period was a vital factor.

The smooth course of anaesthesia in all cases was very impressive.

The post-operative course of these severely ill patients was practically without incident. They were restful and free from worry, requiring little nursing care and no sedative or analgesic drugs apart from small doses of chlorpromazine hydrochloride and pethidine.

I should like to thank Mr. Eric Fenelon for allowing me to publish these three cases and for his helpful co-operation, and the resident and nursing staffs of the Adelaide Hospital for their unremitting care and interest during the conduct of the cases.

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Medical Memoranda

Benign Multilocular Intraperitoneal Air Cysts Simulating Carcinoma of the Colon

Air cysts of the abdominal cavity and parietes are apparently rare. A comprehensive account and discussion of some is presented by Koss (1952). They are normally chance findings at operation or necropsy, and the correct pre-operative diagnosis is seldom made. The following case is an example.

CASE REPORT

A married woman aged 61 was admitted to hospital as an emergency on July 7, 1954, complaining of bouts of colicky upper abdominal pain, vomiting, and a feeling of fullness in the epigastrium for a month before admission. The pain occurred in sudden attacks, four or five times a day, each attack lasting about ten minutes. She was symptom-free between attacks. There was no shoulder pain or any history of jaundice. She had complained of dysuria for three weeks before admission but no longer suffered from it, and had been constipated for as long as she could remember and was addicted to aperients. There was no recent change in bowel habit or any history of recent loss in weight.

The patient was grey-haired, but on examination looked fit and well nourished, with a tendency to obesity. She was not in obvious pain and there was no clinical jaundice. Her tongue was furred and she had herpes labialis. The pulse rate was 100 and the temperature 99.8° F. (37.7° C.). The B.P. was 200/100 mm. Hg. On abdominal and rectal examination no abnormal physical signs were detected apart from slight tenderness in the epigastrium and right hypochondrium.

A straight x-ray film of the abdomen revealed an opacity in the region of the gall-bladder. The blood picture and urinalysis were within normal limits. A presumptive diagnosis of cholelithiasis was made, and on July 16 a cholecystogram revealed that the opacity, previously seen on the straight x-ray film, resided within the gall-bladder. Six days later cholecystectomy was performed through a Kocher incision. On general examination of the abdominal viscera a hard, freely mobile mass about the size of a small orange was felt in the pelvis, arising from the sigmoid

General appearance of the specimen, cut transversely, to show the relation of the cysts to the normal structures of the colon. The continuity of the muscle coat is interrupted by the cysts encroaching on the lumen of the bowel.

colon. No regional lymphatic spread or lymphadenopathy was noted and no secondary deposits of growth were discovered either in the liver or on the abdominal parietes. A diagnosis of carcinoma of the colon was made; the growth was regarded as operable. On August 3 the abdomen was explored through a left paramedian incision. The sigmoid growth was resected and an end-to-end anastomosis performed. Recovery was uneventful and the patient was discharged home three weeks later.

The pathologist's report on the specimen was as follows.

General Appearance.—"The mass excised at operation was roughly spherical, included a portion of colon, and measured 7 cm. across (see Fig.). The appendices epiploicae were enlarged and their peritoneal covering was engorged. On cutting the mass across it was seen that the mucosa of the colon was invaginated by a smooth dome-shaped cyst, 2 cm. in diameter, which, extending upwards from the

retroperitoneal fat through the muscle layer, lay immediately below the mucosa; it was 3.5 cm. in depth. There were several other cysts lying at the side of this one in the adventitial layer, some extending into the appendices and all intercommunicating. They ranged in size from 2 cm. across to mere slits. They were held out stiffly by the general framework of the colon and were lined with a friable creamy tissue, less than a millimetre thick; the wall beneath was in some places of a stiff fibrous consistency, but in general very fragile. No gap was found in the mucosa. The lumen of the gut was only partially obstructed. No oedema, acute inflammation, or abscess formation was visible. There were no hypertrophic changes in the musculature of the gut to indicate that the cyst had been in any way functionally obstructive; and no signs of diverticulitis, perforation, or any type of injury to the mucosa."

Histology.—"Sections confirmed that there was a gap in the muscle through which the cyst was protruding; the mucosal layer was continuous over the cyst; below it lay the muscularis mucosae and, lining the cyst, a mass of granulation tissue continuous with the retroperitoneal fat. All the cysts intercommunicated and they were all of the same nature—namely, spaces in subacute or chronic granulation tissue of the retroperitoneal fat. Some cysts lay in a mass of polymorphs; others were lined by delicate fibrous tissue in the meshes of which were endothelial cells swollen with foamy contents, giant cells, eosinophils, and plasma cells. This inflammatory reaction continued up the areolar tissue spaces of the muscle layer. Some of the cysts were lined with giant cells, many of which contained foreign bodies. These and the foamy endothelial cells were a prominent feature of the granulation tissue, which, in general, resembled that found in connexion with fat necrosis. It was not clear how the cysts arose; they seemed to appear first as clefts near giant cells in the centre of a mass of granulation tissue."

COMMENT

Eight cases of subserosal cysts of the peritoneum were reported by Heinz (1952-3). These cysts were incidental findings at necropsy. They measured 1 mm. in diameter, were of uniform size, and looked like "sago grains." They were lined with cubical epithelium and contained mucinoid material with some amorphous debris. It was suggested that they might have arisen either as dilatation of vascular channels lying in the external muscular coat of the bowel or spaces occurring in local proliferations of the serosal cells.

Koss (1952) described numerous types of air-containing cysts of the abdominal cavity. They were found in various situations on the serosal surface of the bowel, singly or in clusters, sessile or pedunculated, and varying from a few millimetres to several centimetres in diameter. Other cysts lay in the submucosa, had a honeycomb appearance, were thin-walled, and contained gas under pressure. Of the large number of cysts which he reported, only four were confined to the colon. Their lining included giant cells as a characteristic histological feature. He was of the opinion that these cysts were actually lymphatic vessels distended with gas which might have been produced either by bacterial agents or by purely mechanical causes; other writers believed that the gas was formed locally within the lymphatics by expiration of the cell linings of the lymphatic channels.

An interesting solitary air cyst of the peritoneal cavity, believed to be unique, was reported by Hughes and Greene (1953). It arose from the mid-point of the sigmoid colon on its antimesenteric border, was 10 cm. in diameter, felt like an inflated balloon, and had a smooth greyish pink lining. Microscopy revealed that this inner layer was infiltrated with lymphocytes, histiocytes, polymorphs, and plasma cells, with many fibroblasts and capillary blood vessels, closely resembling the picture presented by the cyst discussed in this paper. They suggested that its origin may have been due to air distension of an infected and obstructed diverticulum of the colon, or, alternatively, that the

cyst was derived from a congenital duplication of the large intestine.

The cyst described in this paper and the retroperitoneal cyst of Hughes and Greene bear a close resemblance histologically, the endothelial foam cells and giant cells found in both suggesting a breaking down of fat cells following infection; but, whereas theirs was unilocular and lay on the antimesenteric border of the gut, the one now reported was multilocular and invaded the appendices epiploicae. It is possible that the two cysts may have a similar origin, which may be explained in the way suggested by Hughes and Greene.

The importance of this type of cyst is its clinical resemblance to carcinoma of the colon.

I am indebted to Dr. Mary E. Sharp for the pathological report and to Mr. J. R. Hughes for permission to publish the case. I would also like to thank Professor Lambert Rogers for his help and criticism with the preparation of the paper.

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Leprosy Diagnosed as Syringomyelia

Leprosy is one of the oldest diseases of mankind, and its heaviest incidence is in tropical and subtropical zones. Indigenous cases do not occur in Great Britain (*Memoranda on Medical Diseases in Tropical and Sub-tropical Areas*, 1946), although they are recorded in Scandinavia, the Baltic States, and Mediterranean countries. Thus its recognition may be delayed when it does occur in immigrants to this country, more especially if its manifestations are unusual—for example, neurological. It may therefore be worth while reporting two cases which have been recently seen in this hospital, to which they were referred as cases of syringomyelia.

CASE REPORTS

Case 1.—An Anglo-Indian aged 20 was first seen in the out-patients department in November, 1948, when he complained of numbness of the right hand for two years and weakness of that hand for one year. He had previously lived in India, and first noticed these symptoms after a painful whitlow which took a month to heal. His index finger and thumb at that time began to feel numb and clumsy, and a cigarette burn of the finger was quite painless. Subsequently the whole hand became similarly affected and he noticed impairment of touch sensation in addition to insensitivity to pain. These symptoms lasted a year before he was aware of any weakness. His past history was not relevant and there was no family history of leprosy.

He was an apparently healthy man without abnormalities other than those in the right arm. The skin of the hand was thickened and showed the scars of many wounds. There was global wasting of the intrinsic hand muscles as well as impairment of all forms of sensitivity below the middle forearm. The reflexes in the arms and legs were normal. There was no palpable thickening of any peripheral nerve and the skin was completely normal. Radiological examination of the cervical spine and examination of the cerebrospinal fluid revealed no abnormality. Although the possibility of leprosy was considered at that time, nasal scrapings were negative, and, despite the atypical nature of the sensory loss, a diagnosis of syringomyelia was made, for which he was given a course of deep x-ray therapy.

Five months after his discharge he noticed numbness of the ulnar border and little finger of his left hand and a reddened area on his left thigh. A month later the right foot became numb, and soon afterwards he experienced shooting pains in the right forearm and foot. Meanwhile the sensory loss had been gradually extending up his right arm. He was readmitted in November, 1949, by which time the wasting of the right hand had produced a well-marked claw deformity. The sensory loss extended higher up the

forearm, and there was wasting of the left hand with slight loss of all forms of sensitivity along its ulnar border. There was a reddened area of skin 2 in. (5 cm.) across on the front of the left thigh, also two small areas on the back, over which sensitivity to all forms of stimulus was impaired. The cranial nerves and reflexes were unchanged. Acid-fast bacilli were found in scrapings from the left thigh and a diagnosis of leprosy was made.

Case 2.—An Anglo-Indian aged 26 came to England in 1950 and subsequently joined the Army, from which he was discharged in January, 1955, with a diagnosis of syringomyelia. A month later he was referred to the out-patients department. Weakness and wasting of the right hand had first been noticed four months previously. These symptoms rapidly became worse, and he soon noticed loss of appreciation of touch, heat, and cold. For two months he had a patch of similar sensory loss over the right knee. Ten years earlier he had noticed a small patch of anaesthesia over his right elbow; this had remained unchanged. There was a family history of leprosy, his father suffering from the disease.

The patient was a healthy-looking man. Neurological examination showed the cranial nerves to be normal; a thickened posterior auricular nerve was palpable in the left side of the neck. There was severe weakness of the right hand, with global wasting and some wasting of the flexors and extensors of the forearm. All forms of sensitivity were impaired in the arm up to midway between the elbow and the shoulder. The reflexes were brisk and symmetrical. The right ulnar nerve was thickened to the size of a pencil at the elbow, and could thence be traced continuously up to the axilla. The left ulnar nerve was also thickened, but to a less extent. There was anaesthesia to touch and pin-prick over the right knee and upper third of the leg, but no other abnormality. On these clinical grounds leprosy was diagnosed, and skin biopsy at the Hospital for Tropical Diseases confirmed this diagnosis.

DISCUSSION

Although in leprosy early diagnosis is important (Manson-Bahr, 1950), in this country the disease is liable to be overlooked owing to its rarity. The occasional resemblance of neural leprosy to syringomyelia is of interest, and it is noteworthy that, whereas accounts of syringomyelia rarely include leprosy in discussing differential diagnosis, almost all accounts of leprosy refer to syringomyelia. Since the neurological changes of leprosy are due to involvement of peripheral nerves alone, there is no evidence of a spinal-cord lesion, such as is provided by long-tract disturbances or by a Horner's syndrome, and which is almost invariably present in syringomyelia by the time wasting of the hand is pronounced. In leprosy the sensory loss usually includes all forms of sensitivity, although, as discrimination between heat and cold is lost first, at an early stage the anaesthesia may appear to be dissociated (*Memoranda on Medical Diseases in Tropical and Sub-tropical Areas*, 1946). It is often of "glove" distribution, but in syringomyelia this type of distribution of sensory loss is uncommon (Brain, 1951). A further significant point is that in syringomyelia there is almost invariably depression of one or more of the reflexes in the upper limbs by the time wasting is pronounced, but in leprosy the reflexes are preserved and may even be exaggerated (Cochrane, 1947). The most valuable confirmatory sign of neural leprosy is palpable thickening of peripheral nerves.

I wish to thank Dr. E. A. Blake Pritchard for permission to publish these cases and for his advice and encouragement.

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