Case 3

A male child aged 3 was admitted to hospital on May 27, 1946. Two days before admission he had had abdominal pains and vomiting, and his last bowel motions occurred three days previously. The temperature was 101° F. (38.3° C.), pulse 100, regular. The general condition was fair. He had abdominal facies, the tongue was coated, and respirations were of the thoracic type. The abdomen was distended, diffusely very tender, with rigidity especially in the right iliac fossa. There was free fluid on percussion. Per rectum there was no appreciable disease. On the assumption of an inflammatory intestinal condition involving the peritoneum, possibly of appendicular origin, laparotomy was performed under general open-ether anaesthesia.

A right pararectal incision was made, and a fair amount of heavily blood-stained turbid fluid of slightly faeculent odour was found on opening the abdominal cavity. The caecum and appendix did not show any abnormality. A loop of ileum belonging to the middle portion of that gut was found to be distended and intensely red; its wall was thickened with inflammatory infiltration, and on the serous surface a thread-like whitish network of swollen lymph vessels was visible. The loop involved measured about 18 in. (45 cm.), a kink forming a double-barrel-shaped incomplete intestinal obstruction about 1/2 in. (1.25 cm.) long. The wedge of mesentery belonging to it was grossly thickened to approximately 1/3 in. (0.8 cm.) in diameter, and appeared white with fibrinous-purulent infiltration. The mesenteric glands were swollen to the size of a cherry or walnut. A mass of partly curled-up worms was palpable in the diseased gut, and there was worm infestation of less intensity in other sectors of the small intestine, which, however, did not look inflamed. The coil of worms was removed through a small incision; it contained about 30 ascarides of various sizes. The near-by loops were emptied by "milking" the worms in them towards the original incision; the latter was then closed. A rubber drain was inserted, and the wound closed around it. After the operation a course of 500,000 units of penicillin was given. On May 31 the drain was shortened, only sero-sanguinolent discharge being seen. There was one normal motion. The drain was removed on June 2, and next day the general condition was satisfactory. The abdomen was soft and not distended. The patient took and retained his food, and the bowel action was satisfactory. On the 5th he vomited five small ascarides. Two days later his general condition deteriorated and he vomited worms on several occasions. Next day there was further deterioration and he vomited twelve worms during the night. On June 9 the patient died, extremely cachectic, choked with ascarides creeping in masses out of his nose and mouth.

Discussion

Diseases due to roundworms play a considerable part among the abdominal troubles of the native population in the Middle East. The clinical picture they produce may mislead the observer, unless he bears in mind their relative frequency in this part of the world as compared with Europe, and the possible variety of accompanying symptoms.

In Case 1 it did not occur to us that roundworms might be the cause of the frequent abdominal spasms. The boy's mother made no mention of their presence in the faeces, and examination led us only to the conclusion that operation was indicated because of clear signs of appendicitis with peritoneal involvement. From previous experience we concluded that the masses palpable through the thin abdominal walls in Case 2 consisted of conglomerates of roundworms; the only disquieting sign was the raised temperature, which we had not found before in roundworm disease. The success of the anthelmintic treatment confirmed our assumption, and suggested that roundworms may cause inflammatory intra-abdominal changes; and the tenderness on palpation may be significant in that connexion. The intense inflammatory changes in Case 3 reminded me of Crohn's disease. However, the presence of a big mass of roundworms in the diseased intestinal loop led me to believe that they were the aetiological agent, not only grossly damaging the bowel wall but also causing the most severe inflammatory infiltration of the related mesentery. Through an unfortunate accident we lost a swollen mesenteric gland which had been removed for histological examination during the operation. Therefore, and because the relatives refused permission to perform a post-mortem examination, no histological details are available.

Summary

Roundworm infestation may become of surgical importance not only by causing mechanical obstruction of the small gut but by producing inflammatory changes varying in degree from trivial (Case 2) to the most serious conditions (Case 3). It is usually

difficult to distinguish this variety of inflammation from that of other origin. Correct diagnosis will be facilitated if in regions with prevalent roundworm infestation one will bear that possibility in mind, and go into the history of the case accordingly, using also the laboratory methods for investigation.

Surgical treatment will have to consider removal of as many worms as possible from the bowel, by "milking" them from adjoining loops towards an incision made over their biggest accumulation.

I would not hesitate even to make more incisions in cases where very extensive infestation renders removal through one opening impracticable.

At times the prognosis seems to depend to a considerable extent on the thoroughness with which the worms were removed during the operation. As medical anthelmintic measures are not advisable for at least two weeks after incision and suture of the gut, the remaining worms have ample time to grow and flood the organism. They may decide the fatal issue in cachectic individuals whose resistance through chronic roundworm infestation has already been lowered a long time before sudden developments compel them to seek medical advice.

MESENTERIC VASCULAR OCCLUSION

BY

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Mesenteric vascular occlusion is one of the less common abdominal emergencies. It is extremely difficult to diagnose and the mortality rate is high; consequently scant attention has been paid to it in most surgical works. In the past five years I have had two cases which recovered, and I feel that wider recognition and earlier treatment would do much to improve the gloomy prognosis.

The first case was recorded by Tiedmann in 1843, but it was not until fifty years later that the first resection was performed (Elliot, 1895). In 1913 Trotter presented 359 cases collected from the literature, with seven personal cases. Cokkinis (1926) published a monograph embodying an exhaustive description of mesenteric vascular occlusion and the mesenteric circulation. A series of cases was reported by MacLeod (1937) and by Atkins (1937), a case complicating labour was described by Grey Turner (1937), and two cases were recorded by Fallis (1940). A review of the literature was given by Brown (1940), and Moore (1941) followed with another, supported by eight cases. In 1943 Luke described a case treated with heparin.

Anatomy

For a proper appreciation of the possible consequences of mesenteric vascular occlusion an understanding of the anatomical arrangement of the blood supply of the intestine is necessary. The whole of the small intestine and the large intestine as far as the middle of the transverse colon are supplied by the superior mesenteric artery. This artery rises at an acute angle from the abdominal aorta behind the pancreas, crosses the third part of the duodenum, and passes forward into the root of the mesentery. It gives off ten to fifteen intestinal branches, and ends near the caecum by anastomosing with one of its own branches—the descending branch of the ileocolic artery. One of its early branches—the inferior pancreaticoduodenal artery—anastomoses with the superior pancreaticoduodenal artery, which has its origin in the gastro-duodenal branch of the coeliac axis, and this forms a collateral channel if the superior mesenteric artery is occluded. The branches of the superior mesenteric artery form numerous arterial arcades in the mesentery by anastomosing with each other. These are more numerous in the lower ileac than in the upper jejunal region. From the arcades the vasa recta, which are true end-arteries with no anastomoses, run to supply the gut-wall. The large bowel as far as the middle of the transverse colon is supplied by the ileocolic, right colic, and middle colic branches of the superior mesenteric artery, the last-mentioned of which anastomoses with the left colic branch of the inferior mesenteric artery, which is a separate branch of the abdominal aorta. This is therefore another important anastomosis should the superior mesenteric circulation be interfered with. Other anastomoses occur with the lumbar arteries in the retro-

peritoneal plexus. The venous drainage of the intestine follows closely the arrangement of the arterial supply, with the important difference that all the blood empties into the portal vein and not direct into the systemic circulation. This places the liver as a filter between the intestines and the systemic circulation, and any abnormality in the liver may have a profound effect upon the mesenteric venous circulation. Venous anastomoses between the portal and systemic veins occur in the haemorrhoidal and oesophageal plexuses and in the retro-peritoneal plexus; between the para-umbilical veins and the abdominal wall; between the splenic and left renal veins; and between the left colic and capsular veins. Cokkinis (1926), in his injection experiments, found that the arterial collateral circulation, excluding the vasa recta, was considerable, and that the venous circulation was of even greater volume.

Pathology

The pathological manifestations of interference with the mesenteric circulation are various. When the circulatory obstruction develops slowly and gives time for the establishment of a collateral circulation, the changes in the intestine may be minimal, consisting of oedema and congestion of the gut wall, patchy or diffuse, and intramural haemorrhage. Compensated vascular occlusion may be manifest clinically by a temporary ileus, and complete recovery is possible if further extension of the damage does not occur.

More usually the vascular disturbance, whether caused by thrombosis or embolism, either arterial or venous, results in gross pathological changes. The most common of these is haemorrhagic infarction of the wall of the intestine. The gut is at first congested, swollen, and oedematous; then follow rupture of the capillaries and extravasation of blood throughout its wall. The gut becomes dull, plum-coloured, stiff, and inelastic, and gradually passes into a state of infective gangrene, becoming green or black. The changes are in fact the same as those in strangulated hernia. The extravasated blood also passes into the lumen of the intestine and into the peritoneal cavity. Some of that in the lumen may be passed on by a distal healthy portion of gut and give rise to bleeding per rectum, or it may be found in a diagnostic enema—an important point in differential diagnosis. The blood and oedema fluid in the peritoneal cavity give rise to a haemorrhagic peritoneal exudate, which is a very common finding. The affected portion of mesentery also undergoes gross changes; it becomes oedematous and friable, may be 1 in. (2.5 cm.) or more in thickness, and contains thrombosed vessels.

A very rare change is the development of anaemic gangrene in which a pale, bloodless infarct occurs, followed by putrefactive changes. The portion of intestine most commonly involved is the ileum, although in my own two cases it was the jejunum. No part of the intestine from the duodenum to the lower colon is exempt, and the extent of the affected area may be from a few inches to almost the whole of the intestine. An important point when considering treatment is that the limits of the infarcted area are not usually sharply defined but merge gradually into the normal portions.

Aetiology

The vascular occlusion may be primarily arterial or venous, the latter being the more common. As soon as the condition is developed, however, both arteries and veins become thrombosed. This is owing to the tendency of the thrombosis to spread when the circulation has been slowed down, and is responsible for some of the failures after operative treatment. Arterial occlusion is usually embolic and affects the superior mesenteric artery more often than the inferior. It may result from mitral stenosis, mural endocarditis, atheroma, pulmonary vein thrombosis, or pyaemia. Arterial thrombosis is usually associated with arterial degeneration, atheroma and arteriosclerosis being the common lesions.

Venous thrombosis may be due to a variety of causes. In many cases of mesenteric venous occlusion, however, no definite cause has been demonstrated. The number of such cases will probably diminish with a more diligent search for a causative factor. An important cause, and according to Cokkinis the most frequent, is peripheral sepsis in the portal area, arising

from appendicitis, pelvic infection, or diverticulitis. Any of these may lead to portal thrombosis or portal pyaemia. All the blood in the mesenteric venous circulation has to pass through the liver. There are many possible causes of obstruction to the portal vein by pressure from without, the most frequent and important being cirrhosis of the liver. Peripheral venous thrombosis may sometimes be a cause. Changes in the blood itself may predispose to thrombosis, and in this connexion a blood disease such as polycythaemia rubra, leukaemia, splenic anaemia, or severe primary or secondary anaemia may be the initial lesion. Predisposing factors are debility, degenerative diseases such as alcoholism, inflammatory lesions, neoplasms, and occasionally mechanical factors such as trauma.

Symptomatology

Mesenteric vascular occlusion has the reputation of being almost undiagnosable. Its recognition is certainly difficult, but in a typical case there are features which should enable a diagnosis to be made if the disease is borne in mind.

No age is exempt, but the condition occurs most frequently between the ages of 20 and 50 (Cokkinis, 1926). Brown (1940), however, in an analysis of cases of venous occlusion found that the most common age incidence was between 50 and 60. The onset is associated with severe abdominal pain, colicky in character and centrally situated. This is often accompanied by considerable shock, and is followed by periodical recurrence of the colicky pain and vomiting. Borborygmi are often marked. The pain usually overshadows the vomiting, and this distinguishes the condition from most other types of intestinal obstruction. There is in some cases absolute constipation without passage of flatus; in others a motion may be passed containing altered blood. Melaena is an important diagnostic point, and if blood is not passed spontaneously it may be found on the examining finger or be obtained from a high enema. Abdominal distension becomes apparent, and the abdomen is tender and rigid, often more so on the left side. The affected coils of gut may be felt as a palpable lump. Free fluid can usually be demonstrated. Leucocytosis is usually present and x-ray examination may show multiple fluid levels. As time goes on the patient becomes severely ill with marked shock, subnormal temperature, rapid pulse, and low blood pressure; in fact he resembles a late case of intestinal obstruction with peritonitis. In cases of massive occlusion the element of obstruction may be overshadowed by the signs of internal haemorrhage due to the large amount of blood which may be lost into the infarcted intestine. Some of this haemorrhage may be external and become evident as haematemesis or melaena.

Diagnosis, Prognosis, and Treatment

In Trotter's large series the correct diagnosis was made in less than 5%. In the cases described by Cokkinis the diagnosis was established in approximately 15%, but many others in this group presented symptoms which should have made a diagnosis possible.

Important points to note in attempting to establish a diagnosis are: (1) a history or signs of a possible causative lesion—for example, endocarditis or cirrhosis; (2) the presence of blood in the bowel, either passed by the patient or found on examination by the enema test; (3) the presence or previous history of vascular occlusion elsewhere; (4) the presence of a blood dyscrasia; (5) severe pain, often greater on the left side, associated with signs of intestinal obstruction, free fluid, and sometimes a palpable lump. The differential diagnosis from other causes of intestinal obstruction, perforated peptic ulcers, and acute pancreatitis has to be made.

Of untreated patients more than 95% die; only those with a mild attack in whom an adequate collateral circulation is established will survive. With treatment the general mortality in cases of venous occlusion is approximately 68% (Brown, 1940), with a higher figure obtaining for arterial embolism. The mortality depends in part upon the amount of gut involved, and the prognosis improves if the length infarcted is less than 3 ft. (0.9 m.), although recovery with normal function has followed the removal of half of the small intestine.

Exploratory laparotomy should be undertaken early. The time factor is as important as in cases of perforated ulcer, and

more important than in most cases of obstruction. The patient should be rendered as fit as possible by intravenous therapy before operation. As soon as the abdomen is opened free blood-stained fluid will be encountered in considerable quantity. The affected portion of intestine is usually quite obvious, and if it does not actually present in the wound it can easily be found. Wide resection of the bowel should be performed, leaving a good margin of healthy tissue on each side of the infarcted segment, together with similar treatment of the affected portion of the mesentery. The presence of a second infarct in some other segment must not be overlooked. Continuity can be re-established by means of end-to-end or lateral anastomosis. If the large intestine is involved resection, with external drainage, should be performed, and continuity established at a later date. Exteriorization is not good treatment for the small gut; in one such case described by Atkins the complications were many and troublesome, although the patient finally recovered. Even the most desperate cases may be given a slender chance by the establishment of an enterostomy.

Case I

A printer aged 50 experienced a sudden attack of abdominal pain after working all night. The pain was colicky in nature and became worse during next day; he vomited three times, but his bowels had not opened. Two high wash-outs were given by his doctor without any result, there being absolute constipation from 9 a.m. No blood was obtained with the wash-out. On admission to hospital he was doubled up with pain; the temperature was 97.6° F. (36.4° C.), pulse 84. His general condition was healthy and there was no pulmonary or cardiac disease, sepsis, or thrombosis. The abdomen was moderately distended and very tender all over. An ill-defined tumour measuring several inches across was present in the centre of the abdomen. It was dull to percussion; there was also dullness in both flanks. Borborygmi were heard. The severe colicky pain was relieved by morphine. A diagnosis of intestinal obstruction was made and a laparotomy performed under spinal anaesthesia. On opening the abdomen blood-stained fluid escaped and a large loop of engorged gangrenous small intestine was seen. Further examination showed that 24 ft. (0.76 m.) of the lower part of the jejunum was involved with its mesentery, which was much thickened and oedematous. There was a gradual transition to healthy intestine on both sides of the infarcted portion. The affected portion, including its mesentery, was resected, blind ends were made, and continuity was re-established by side-to-side anastomosis. The abdomen was closed without drainage, and convalescence after operation was uneventful. The patient was discharged from hospital seventeen days after operation, and at the time of writing was quite well and leading a normal life.

Case II

A regimental sergeant-major aged 38 was admitted to hospital complaining of abdominal pain. He first experienced pain across the lower abdomen and in the left flank two years previously; it was sudden in onset and shooting in character. The abdomen became very distended and the patient vomited. His bowels had not opened, and the pain was so severe that he fainted in bed. The attack subsided spontaneously. Later he had another severe attack and several milder ones of the same nature, and he was admitted to hospital. As the pain was mainly in the left flank he was subjected to a complete renal investigation, with negative results. The day after his discharge from hospital he had his most severe attack, and then came under my care.

On examination his general condition was fairly good. The tongue was coated and dry and the heart normal; blood pressure 150/90. The lungs were healthy; the haemoglobin was 70%, and the Wassermann reaction negative. The abdomen was moderately distended, rigid, and tender, the maximum tenderness being to the left of the umbilicus, where a vague mass could be felt but not accurately defined. There was dullness in the flanks, and borborygmi were marked. The rectum was empty and ballooned, and there was no blood on the examining finger. An enema produced no blood, faeces, or flatus. A diagnosis of intestinal obstruction was made.

Laparotomy was performed under general anaesthesia. On opening the abdomen a considerable quantity of blood-stained fluid escaped. The vague tumour which had been felt on the left side of the abdomen was found to be two coils of deep-plum-coloured jejunum matted together and covered with flakes of fibrin. The mesentery was more than 1 in. (2.5 cm.) in thickness, and its vessels were seen to be thrombosed. Some 3 ft. (0.9 m.) of the upper jejunum was affected and this portion was resected, blind ends were made, and a side-to-side anastomosis was performed, keeping well clear of the infarcted region. After division of the mesentery, clot could be picked out of the vessels. The anastomosis was found to lie 1 ft. 6 in. (45 cm.) distal to the duodeno-jejunal flexure. A

pint (568 ml.) of plasma was given during the operation, followed by intravenous saline and glucose. Convalescence was complicated by a post-operative bronchitis, but the patient passed flatus on the third day and the bowels acted spontaneously on the fourth. The chest soon cleared, and thereafter convalescence was smooth. It was a considerable time, however, before this man regained his appetite and began to put on weight. After a period at a convalescent home he was regraded category C (temporary) and returned to his Army duties free from symptoms. Histological section of the tissue removed showed a primary venous thrombosis, the cause of which could not be determined.

Discussion

Both of the above cases had a primary venous thrombosis of limited extent. Consequently they responded well to early operation and resection of the affected segment of the bowel. Fortunately, no further spread of the thrombosis occurred after operation, as sometimes happens. An important point in preventing this is to make the resection through healthy bowel and to remove all the affected portion of mesentery. In neither case was the diagnosis made before operation, although it was immediately obvious on opening the abdomen. Both cases presented the appearance of an early intestinal obstruction, but in neither was there a key sign such as haemorrhage from the bowel, evidence of cardiac disease, or venous thrombosis elsewhere, to suggest mesenteric occlusion. In the second case the only possible predisposing cause that could be found was a moderate degree of secondary anaemia. The pain in the left flank was a pronounced feature and had led to a complete renal investigation at another hospital before the pre-operative thrombosis occurred. Loin pain was also found by Fallis. It is possible that it occurs fairly constantly on the left side when the upper part of the jejunum is involved. It was noted during convalescence that a considerable time was required for the re-establishment of the appetite and a gain in weight, and this was thought to be due to the removal of a large portion of the active absorption area of the jejunum. The presence of a palpable lump is stated by Cockkinis and others to be unusual, and most of the statistics substantiate this. Its occurrence in both my cases was fortunate, but it is in no way diagnostic since there are so many possible causes of intestinal obstruction associated with a palpable mass. However, if it is associated with evidence of bleeding this sign is of greater value. The occurrence of previous similar but less severe attacks of pain in Case II was thought to be due to a small venous thrombosis in the mesentery resulting in a temporary ileus, with recovery on establishment of an adequate collateral circulation.

Treatment in each case was early operation with resection: in an established case this gives the patient his only chance of survival. Both these patients were in quite good condition, and the length of the intestine involved was not great. This contributed in no small measure to the favourable results. If the large intestine is involved, resection with drainage of both ends is the treatment of choice, but if this method is applied to the small bowel it is liable to be followed by troublesome complications (Atkins, 1937). What can be accomplished by bold surgery is shown by a consideration of Grey Turner's (1937) remarkable case complicating pregnancy in which 10 ft. (3 m.) of small intestine was resected and a Caesarean section performed at the same time. The patient survived, led a normal life, and had another pregnancy. The possibility that an anticoagulant such as heparin may be of use in the treatment of this condition has been considered, and Luke (1943) published a case treated by this means. The case was an early one of venous thrombosis involving the jejunum, and was proved by laparotomy. The wound was then closed, and 21,000 units of heparin were given in 21 litres of saline during the succeeding 10 days. The coagulation time was estimated six-hourly and was found rather difficult to control owing to variations in the rate of drip. A complete recovery ensued. Any case in which this treatment is adopted must necessarily be an early one, before serious infarction has occurred, otherwise there is a danger of profuse bleeding into the infarcted bowel or peritoneal cavity. Haematoma formation may occur in the laparotomy wound. It is doubtful whether cases can be obtained early enough for this treatment. However, it does seem that there is a possible field of usefulness for heparin when combined with operation, with a view to preventing any extension of the thrombosis. Earlier diagnosis and prompt

treatment are the keynotes to success in this very serious abdominal emergency.

Summary

The anatomy, pathology, and aetiology of mesenteric vascular occlusion are described, and some of the literature is reviewed.

Symptomatology and diagnosis are considered, and a plea is made for the earlier recognition of the disease. The importance of signs of cardiac, vascular, or blood disease, or of any evidence of haemorrhage from the bowel, is stressed.

Two personal cases, with recovery, are presented.

Treatment is discussed and the possibilities of the use of heparin are considered.

I wish to express my thanks to Surg. Lieut. C. J. Bintliffe and Dr. J. G. Winternitz for their help in the treatment of the patients, to Dr. S. J. Scurlock, medical superintendent of Ronkswood Hospital, for his kind assistance in the preparation of the paper; and to the Director-General of Medical Services, Ministry of Pensions, for permission to publish.

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MYOPATHY WITH SPINA BIFIDA

BY

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The following is an unusual and interesting case of myopathy associated with spina bifida.

Case History

The patient was a soldier, aged 30, formerly a country bus-driver. His paternal grandfather lived to a healthy old age. His father died of ? meningitis at 28; his mother at 45 from "tumour" of the uterus. He had no brothers, but there were two half-sisters by the same mother, both healthy. The patient had no knowledge of any familial disease.

In childhood he had no serious illness, but at school he noticed weakness of his legs. He became a gardener, but found the work too hard. In February, 1941, he had pneumonia and was in bed five weeks. During a protracted convalescence there was increased weakness, with wasting of the muscles of the chest and abdomen. For the last two and a half years the condition had steadily progressed. At no time had his muscles been bulky.

His height was 65½ in. (1.66 m.). His face was thin and expressionless, and wasting of the muscles of the shoulder-girdle and winging of the scapulae were present. There was no wasting of the forearm or small muscles of the hand. Active and passive movements were free and power good. Marked wasting of the erector spinae was obvious (see figs.). The abdomen was scaphoid, with wasting of all muscles except the erector abdominis. The muscles of the pelvic girdle all showed pronounced wasting; those of the lower leg and foot were not wasted, but there was a mild pes planus. Movements were full and power only slightly reduced. No muscular fibrillation was observed.

The spine showed scoliosis and a depression in the region of dorsal vertebrae 11 and 12, with a palpable gap. In this depression there was a non-hairy, non-pigmented mole. Radiographs confirmed non-union of the posterior arches of these two vertebrae.

The blood W.R. was negative, the C.S.F. normal and W.R. negative, and the urine normal. No electrical reactions were taken. Although the cardiovascular system was normal, there was moderate tachycardia. The blood pressure was 120/80. Chest expansion was poor—only half an inch (1.25 cm.) in the nipple line. The breathing was almost entirely abdomino-diaphragmatic. Radiographs showed "some evidence of chronic bronchitis at the bases: diaphragmatic movements normal." All the cranial nerves were normal and all reflexes were present, although the abdominal reflexes were very weak. There was no sensory disturbance. No vasomotor or trophic changes were present.

Discussion

Exhaustive search through the literature of the last 25 years revealed only one other case of this association of conditions. Urechia and Dragomir (1942) described a case of myopathy

with a spina bifida occulta of the first sacral vertebra. With respect to the regions of muscular atrophy and the absence of sensory disturbance their case resembled the one now reported. In certain features it was different: there was a heredo-familial trait, one sister and a nephew suffering from myopathy. A hypertrophic stage had occurred. The progress of the disease was more rapid, and the patient was bed-ridden at the age of 25. Their case was remarkable for a large pigmented hairy mole covering the right half of the abdomen.

The location of the bifida in the thoracic spine is not common. Wheeler (1920) found no evidence of spina bifida in 3,000

FIG. 1.

FIG. 2.

thoracic vertebrae seen at the National Museum, Washington. Hadley (1941), examining a series of 1,500 consecutive spinal cases, found 3 instances of spina bifida in the thoracic region.

It is interesting to note that in the literature over the same period there are two recorded cases of spina bifida associated with a different form of muscular atrophy. In 1925 Guillaing, Mathieu, and Garcin described a case of Charcot-Marie muscular atrophy with spina bifida of the first two sacral vertebrae. Heuyer, Hurez, and Feld (1942) reported a similar case.

It is difficult, if not impossible, to decide whether the occurrence in the same patient of two uncommon pathological conditions (myopathy and spina bifida) is due to chance concurrence or to some common aetiological factor—e.g., genetic make-up. Urechia and Dragomir (1942) quote numerous authors who have described various osseous deformities in cases of myopathy. Nevertheless during the last 25 years this particular association has been noted only once before in the literature on myopathies. This suggests that chance concurrence is the more likely explanation.

Summary

A case of myopathy with spina bifida is reported. The literature has been examined. One other reported case has been discussed and compared. The comparative rarity of spina bifida in the thoracic region has been noted. The association of the two conditions is thought to be a coincidence.

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The Hospital Survey and Construction Act of the United States, which authorizes the nation-wide building of hospitals, requires that standards for the construction of all those built with Federal assistance under this scheme shall be defined by the U.S. Public Health Service. These standards, which are minimal, were recently drafted; the Federal Hospital Council has now accepted them. They fall into five classes: site, architectural, structural, mechanical (e.g., ventilation, plumbing, electrical fittings), and drawing specifications.