(1957). They have shown in cats that section of pre- or post-ganglionic sympathetic fibres is followed by the growth of sprouts from neighbouring axons. The stimulus leading to sprout formation is probably a humoral agent released by adjacent degenerating nerve fibres (Edds, 1953). These sprouts may reinnervate the denervated structures, provided that they are of the same type (cholinergic or adrenergic) as the axon they are replacing. It was found, for example, that sprouts originating from the vagus can reinnervate ganglion cells in the cervical sympathetic that have lost their afferent (pre-ganglionic) fibres. Murray and Thompson (1957) suggest that a similar ingrowth of sprouts from the vagus could explain the gustatory sweating that may follow cervical sympathectomy in man. The pilomotion observed in this patient is an important piece of evidence, because it is mediated by adrenergic fibres, while sweating is mediated by cholinergic fibres. It is highly unlikely that the cholinergic vagus fibres could make a functional connexion with structures innervated by adrenergic fibres. It is probable, therefore, that reinnervation has occurred proximal to the ganglion, since all pre-ganglionic fibres are cholinergic. Reinnervation at this level, by sprouts from the vagus, would explain the simultaneous occurrence of sweating and pilomotion in response to gustatory stimuli. No change in the post-ganglionic fibres need then be postulated.

No satisfactory explanation has so far been found for the gustatory sweating and flushing that often follows disease or injury in the parotid or submandibular region (Goatcher, 1954; Young, 1956). The work of Murray and Thompson (1957) now suggests that in these cases denervated sweat glands and perhaps vasodilator receptors have been reinnervated by sprouts from neighbouring cholinergic fibres.

Gustatory pilomotion has not been described before, presumably because goose-skin is very difficult to detect on the face. It seems possible that the tingling which in many patients accompanies gustatory sweating results from pilomotor activity that fails to produce visible goose-skin.

Summary

A case is reported of gustatory sweating and pilomotion following operative damage to the sympathetic. It is suggested that sprouts have grown out from the vagus and made connexions with sympathetic ganglion cells whose normal pre-ganglionic afferent fibres have been divided.

I am grateful to Dr. A. C. Dornhorst for his help with the investigations and to Dr. P. J. D. Heaf for drawing my attention to this patient.

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Six American women medical scientists will leave during the second week in May for a one-month tour of medical institutions in the Soviet Union. Preliminary arrangements for the trip have been completed by the National Academy of Sciences in co-operation with the Rockefeller Foundation, the American Medical Association, and the American Medical Women's Association. The visit is the result of an exchange agreement entered into by the U.S. Department of State and the Soviet Union. Last October six women scientists from the U.S.S.R. spent a month in the United States visiting medical institutions in six cities in the East and Middle West. The delegation will be headed by Dr. HELEN B. TAUSSIG, of Johns Hopkins University Hospital, a paediatrician known specially for her work on cardiac disease in children.

VOLVULUS OF CAECUM COMPLICATING NON-ROTATION OF MIDGUT

A CASE SIMULATING REVERSED ROTATION

BY

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Non-rotation of the midgut is an uncommon anomaly in which the second stage of rotation completely fails to occur. The midgut returns to the abdomen during the tenth week of foetal life in the same relative position that it occupied while in the umbilical cord; the small intestine remains largely to the right of the midline and the large intestine to the left, with the terminal ileum entering the right aspect of the caecum. The condition has been well described by Dott (1923) in his classic paper on the anomalies of intestinal rotation.

The importance of non-rotation stems mainly from the fact that the mesentery of the midgut in these cases often fails to develop any secondary adhesion to the posterior abdominal wall, or does so in an incomplete and irregular manner. As a result of this deficient fixation, massive torsion of the midgut loop is apt to occur, particularly during the neonatal period, resulting in the well-known condition of volvulus neonatorum. A lesser degree of volvulus, confined in its effects to the caecum or ileo-caecal region, appears to be an uncommon complication of non-rotation, and reference to the readily available literature has revealed record of only four such cases (Rixford, 1920; Bulman, 1924; Carter, 1938; River and Reed, 1942). The following case is of particular interest on account of the volvulus having occurred in such a manner as to give a close simulation of the rare anomaly of reversed rotation.

Case Report

A married woman of 37 was admitted to Kingston Hospital as an emergency case on May 6, 1956. For some 20 hours previously she had had severe central abdominal pain, colicky at first, but later becoming continuous. She had been nauseated, but had not vomited. Flatus had not been passed since the onset of the pain, and her abdomen had become noticeably distended. Since childhood she had been subject to recurrent attacks of similar pain, but these had never before lasted more than an hour or two, nor been of the severity of the present attack. She had always had a tendency to constipation.

On examination her general condition was good, though she was clearly in considerable pain. Her temperature was 99° F. (37.2° C.), pulse 60, B.P. 110/80. The abdomen was moderately distended and showed generalized tenderness but no rigidity. The bowel sounds were obstructive in character. There was some guarding in the lower abdomen, where a centrally placed, soft, ill-defined tympanitic swelling some 8 in. (20 cm.) in diameter was palpable. There were no other significant findings on physical examination. A straight x-ray film of the abdomen showed gross gaseous distension with a very large fluid level in the pelvis. A diagnosis of acute obstruction of the large intestine, probably due to volvulus, was made.

Operation.—Under general anaesthesia the previously noted swelling was readily visible and palpable, extending from the pubis to well above the umbilicus. A right paramedian incision was made, and the swelling was found to be a grossly distended but viable caecum and ascending colon. It was readily delivered out of the wound, and on following up the distended ascending colon the latter was found to

disappear beneath the tightly stretched mesentery of the bowel in the region of the hepatic flexure; collapsed transverse colon emerged at the far side of the mesentery. The wall of the colon was twisted at the point where it disappeared (Fig. 1). There was moderate distension of the lower part of the small bowel, but clearly the ileo-caecal valve had remained competent, resulting in a closed-loop obstruction of the right colon. Apart from the obvious twist in the colon at the point of obstruction, the anatomy as found at operation closely resembled that so well depicted in association with some of the case reports of reversed rotation—for example, Dodd (1923) and Donald (1927).

After delivering most of the small intestine out of the wound it was a simple matter to reduce the volvulus by rotating the caecum and ascending colon through 180 degrees in an anticlockwise direction. It was then clear that this was in fact a case of non-rotation, the colon being entirely left-sided (Fig. 2). The caecum lay low down in the left iliac fossa adjacent to the lower end of the descending

colon. In the rare anomaly of congenital reversed rotation the herniated part of the midgut loop undergoes a 180 degrees clockwise rotation at the time of its return to the abdominal cavity in the tenth week of foetal life. This results in anatomical relationships almost identical to those found at operation in the case here described, in which a very similar rotation had occurred as an acquired event. Rose (1941) pointed out that commencing volvulus of the whole midgut loop associated with non-rotation would give a condition closely simulating reversed rotation, and suggested that this was possibly the true explanation of some of the cases previously reported as examples of reversed rotation. The case described above would seem to support this contention.

From the practical point of view, when a case of apparent reversed rotation is discovered at operation, and fixation of the mesentery to the posterior abdominal wall is found to be lacking, it would appear well worth while attempting to reproduce the state of non-rotation by rotating the whole midgut through 180 degrees in an anticlockwise direction. If this could be accomplished it would be a simple, safe, and physiologically sound method of relieving compression of the transverse colon. It has been shown by Ladd and Gross (1941) that intestinal function in the non-rotated position is quite satisfactory.

Obstruction of the transverse colon supervening in cases of congenital reversed rotation is usually ascribed to compression by the overlying superior mesenteric vessels. Comparison with the case here described suggests the possibility that axial torsion of the bowel wall may on occasion be a contributory factor. Case reports by Doyle (1937) and Maxwell Ellis (1944) lend some support to this view.

Comparison of the present case with the four previously reported cases of caecal volvulus and non-rotation reveals interesting differences. Whereas in the latter the volvulus was in each case confined to the caecum or ileo-caecal region, in the case here described the whole of the free part

of the midgut loop had in fact rotated, though the ill effects were confined to the right colon. A closed-loop obstruction had developed between the competent ileo-caecal valve and the point of torsion and compression at the hepatic flexure.

It is, of course, just possible that the case here reported was in fact one of congenital reversed rotation. However, the view of it given above seems more likely because of the ease of reduction to the non-rotated position and the natural-looking lie of the intestines in the latter position. Furthermore, there was no hypertrophy of the caecum such as one might have expected had there been long-standing partial obstruction.

Summary

A case is described of non-rotation of the midgut complicated by volvulus of the caecum.

Close similarity between the condition found at operation and reversed rotation is noted.

Certain aspects of reversed rotation are discussed in relation to the case described.

My thanks are due to Mr. L. R. Leask for permission to publish this report and to Mr. W. H. W. Jayne for his helpful advice.

FIG. 1.—Diagram of condition found at operation, showing obstruction at hepatic flexure by compression from overlying mesentery and axial torsion of the bowel wall. FIG. 2.—Diagram of condition after reduction of the volvulus, showing basic anomaly of non-rotation.

colon, and the ileum entered it on its right aspect. The ascending colon passed upwards to join a short transverse colon, and the left colon was in the normal position. The right colon and the small bowel shared a common free mesentery, unattached posteriorly except at its root. There were a few adhesions in the region of the duodenum and upper jejunum, but when these had been divided the duodenum was seen to pass downwards entirely on the right-hand side of the superior mesenteric vessels.

Reduction of the volvulus resulted in immediate relief of the obstruction; the bowel was left in the position of non-rotation and no attempt was made to fix it. The abdominal wall was repaired in layers.

The post-operative course was uneventful, and the patient was discharged home on May 16, 10 days after operation. She has subsequently remained well, though still troubled by her lifelong complaint of constipation.

Comments

The basic anomaly in this case was non-rotation of the midgut, as depicted in Fig. 2. There was complete lack of any secondary fixation of the midgut mesentery or of the bowel itself from the level of the second part of the duodenum to the hepatic flexure. This had allowed clock-

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Medical Memorandum

Recovery after Rupture of Liver in Newborn Infant

Recovery after rupture of the liver in the neonatal period has rarely been recorded, although the condition is a surprisingly frequent post-mortem finding. In her analysis of 2,000 perinatal deaths in Chicago, Potter (1940) recorded 24 cases of intraperitoneal haemorrhage due to injury to the liver. J. L. Emery (1957, personal communication) found five such cases in a series of 1,000 perinatal deaths in Sheffield.

We have been able to find only six reported cases of survival following this injury. Of these, Arden (1946, 1951) in Brisbane described four, one was successfully treated by Rubovits and Strauss in Chicago (Rogers, 1934), and one was reported by Greaves (1955).

CASE REPORT

The infant, the fourth child of normal parents, was born at home by spontaneous vertex delivery on August 26, 1956. The second stage of labour lasted 10 minutes, and the infant, though cyanosed, cried immediately and did not require resuscitation. Birth weight was 9 lb. 4 oz. (4.2 kg.). For the first five days he behaved normally in every respect and appeared to be a healthy infant. During the fifth night he cried at 2 a.m. but appeared quite normal when fed at that time. At 7 a.m. his mother noticed that he looked pale and that he would not suck. The general practitioner, on seeing the child an hour or two later, arranged his immediate transfer to hospital.

On admission at 12 noon he was extremely pale, limp, and a little dyspnoeic. A few petechiae were seen on the forehead and there was a subconjunctival haemorrhage of the left eye. Slight abdominal distension was noted at this time. The haemoglobin was 24% (3.5 g./100 ml.).

As it was apparent that a severe haemorrhage had occurred a blood transfusion was started immediately, 130 ml. of blood being given via the umbilical vein during the afternoon. Vitamin K₁, 1 mg., was injected intramuscularly. The baby's colour and general condition improved greatly, but the abdominal distension became more marked. There were definite signs of free fluid in the abdomen; shifting dullness could be demonstrated, although, in the right flank, the percussion note remained dull constantly. Rectal examination showed fullness of the rectovesical pouch. It was now obvious that the bleeding was into the peritoneal cavity, and a diagnosis was made of haemo-peritoneum due to rupture of the liver.

At 7 p.m. laparotomy, under general anaesthesia, was performed by Mr. N. F. Kirkman. A transverse epigastric incision was made. Approximately 180 ml. of blood, partly fluid and partly clotted, was present in the peritoneal cavity, mainly in the right subhepatic region. There were two tears of the liver capsule on the inferior surface of the right lobe, from which a persistent ooze occurred. There also appeared to be some bleeding from the posterior surface. Owing to the friability of the liver, several catgut sutures which were inserted failed to control the bleeding. Eventually the gall-bladder was sutured to the anterior edge of the liver in order to compress the bleeding area on the

inferior surface, and the posterior surface of the liver was covered with gelatin foam. The abdomen was closed without drainage.

A further 300 ml. of blood was given over a period of eight hours during and after the operation, and 1 ml. of 10% calcium gluconate and 2 mg. of vitamin K₁ were administered. A course of penicillin was given. Distension of the abdomen was noticeable for four days, but gradually subsided. The infant made a good recovery and was discharged from hospital 14 days after operation. At that time the haemoglobin was 117% (17.3 g./100 ml.), and he appeared well.

When last seen, aged 6 months, he was a healthy-looking baby weighing 20 lb. (9 kg.). The liver was normal in size and the haemoglobin was 86% (12.7 g./100 ml.).

COMMENT

Rupture of the liver occurs most frequently in large babies and premature infants. When injury to the liver is severe enough to tear the capsule immediately, death during or very shortly after delivery is the rule. Nearly all the premature infants affected die at this stage. Less severe trauma may cause a subcapsular haematoma, and in such a case there is normally a period of one to seven days after birth during which the baby progresses normally. Rupture of the capsule overlying the haematoma may then occur, allowing escape of blood into the peritoneal cavity and, with the release of pressure, further bleeding from the surface of the liver.

Henderson (1941) analysed the principal features in 22 cases from the literature in which the clinical signs had been described. The most characteristic finding was the sudden onset of severe anaemia, which was recorded in 19 instances. Dyspnoea, crying, signs of shock, and abdominal distension were each noted in some of the cases. In Arden's series (1951) sudden onset of pallor was observed in all six cases. In four abdominal distension was noted, and in two of these definite signs of free fluid in the abdomen could be elicited, as occurred in our case.

The mechanism of the injury may be apparent as in the cases occurring after attempts at external version (McNitt, 1932), injuries to the mother's abdomen (Henderson, 1941), difficult labour with breech delivery or contracted pelvis (Genell, 1930), and violent resuscitative measures (Nussbaum, 1932). In a high proportion of the cases reported the only abnormal factor has been the large size of the infant, where compression of the thorax and abdomen may be inevitable during a normal delivery. Gruenwald (1948) emphasizes the danger of compression of the thorax, leading to downward displacement of liver and tearing of the supporting ligaments.

Although a few untreated cases have survived 24 hours or more after the appearance of signs of bleeding (Pim and Barlow, 1932; Henderson, 1941) two of Arden's patients died within 10 minutes of the onset of symptoms. This emphasizes the urgency of blood transfusion as soon as the diagnosis is suspected. In some instances this may be the only treatment necessary, but arrangements for operation should be made lest bleeding continue.

We thank Dr. Duncan Macaulay and Mr. N. F. Kirkman for permission to publish this case and for their helpful advice.

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