SEPTIC UMBILICAL ARTERITIS*

BY

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The number of cases of umbilical arteritis seen by surgeons in this country is small, but in view of the disastrous complications to which the condition may give rise, it deserves more consideration than is accorded to it in modern text-books of paediatrics and surgery. It therefore seems appropriate to review six cases of this condition seen in the last few years.

History

During the middle and later part of the last century thousands of infants in this country and on the continent died annually as a result of umbilical infection.

Two types were recognized: (*a*) Moist spreading gangrene of the umbilicus (hospital gangrene) when children died of toxaemia, septicaemia, peritonitis and occasionally rupture of the abdominal wall. (*b*) Erysipelas of the umbilicus, also called puerperal fever of the newborn, in which septicaemia was the common termination.

Both types were much commoner in institutions than in private practice and carried an appalling mortality. Both occurred in epidemic form, often but not always concurrently with epidemics of maternal puerperal infection. Outbreaks of umbilical sepsis were recorded by physicians all over Europe.

Trousseau (1844), in describing his cases, noted the occurrence of peritonitis and osteomyelitis, and pointed out that death could result from umbilical infection even when the umbilicus appeared to be normal.

Meynet (1857), in Paris, described an epidemic which affected 53 infants, 36 of whom died. Eighteen came to necropsy and of these 10 had peritonitis and in three there was septic umbilical arteritis. He treated umbilical sepsis with Canquoin's paste (zinc chloride in wheaten flour) apparently with some success. He stated that Ambroise Paré regarded the malady (umbilical sepsis) as so grave that he advised the surgeon 'not to raise a hand lest he be accused of having caused the death of the infant'.

Bergeron (1866) reported an epidemic at L'Hôpital Necker. Nine babies came to necropsy and all showed involvement of either vein or arteries or both.

Runge (1893), at La Charité Hospital, Berlin, found evidence of umbilical infection in 30 out of 36 babies dying of sepsis. He states that the infecting organism was either the streptococcus or the staphylococcus. He studied the method of separation of the cord in uninfected babies and recognized that obliteration of the umbilical vein and umbilical arteries is not brought about by exactly the same process.

At necropsy on 55 cases Runge found septic arteritis in all but one, whereas he found phlebitis only once. He gives a very full description of arteritis and periarteritis and states that in some cases the infection may reach right down to the internal iliac artery.

Lambert (1897) described a small epidemic in the Children's Hospital, New York.

Ritter von Reuss published his book *The Diseases of the New-Born* in 1914. In this interesting work there is a large section on the care and diseases of the umbilicus. By this date epidemic umbilical sepsis was rare in the hospitals in Vienna in which Reuss worked.

In 1916 Cullen produced his monumental volume on the umbilicus and its diseases, appropriately dedicated to his mother. He reviewed the literature on umbilical sepsis and describes in detail a large number of cases. He states that in some epidemics phlebitis and in others arteritis predominated.

The influence of Lister and Pasteur, improvements in the care of newborn babies and in general hygiene, had put an end to epidemics of florid umbilical sepsis by the time von Reuss and Cullen wrote, and from this time onwards references to umbilical arteritis almost disappear from the literature.

Cruickshank (1930) in his report on 'Causes of Neo-natal Death' found umbilical sepsis in seven cases in a series of 800 necropsies. He does not mention umbilical arteritis.

Henderson (1943) and Smith, Platou and Good

* A paper read at the meeting of the British Association of Paediatric Surgeons in London in July 1956.

(1956) are of the opinion that the incidence of omphalitis is low and is nearly always due to an enterococcus. They do not mention umbilical arteritis. These writers were reviewing sepsis in newborn babies.

In recent years most attention has been given to the condition by Morison, who in 1944 mentioned four instances of arteritis complicating 11 cases of umbilical phlebitis, and in 1952 illustrated the various routes of infection in his book *Foetal and Neo-natal Pathology*. Fig. 1 is reproduced from this publication.

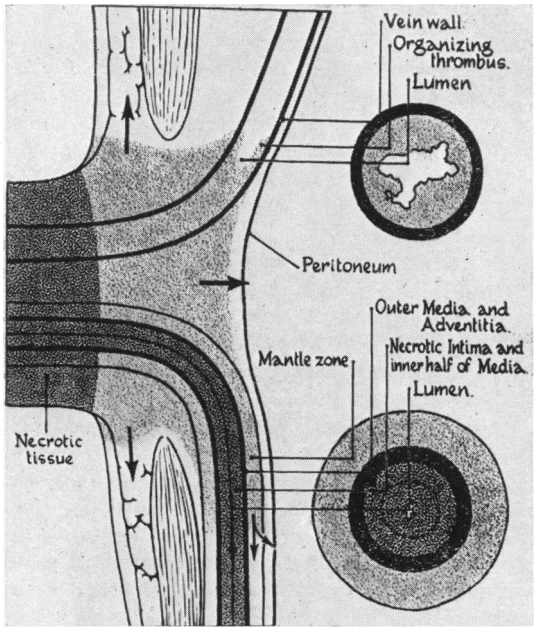

FIG. 1.—Diagram showing the routes along which infection from the umbilicus may spread (reproduced from Morison, 'Foetal and Neo-natal Pathology' by kind permission of Butterworth & Co. (Publishers) Ltd.). After birth the necrotic tissue of the umbilical stump separates. This provokes some inflammatory reaction but this is limited by fibroblastic reaction and only fibroblastic reaction should extend to the inner margin of the lightly stippled area. The inner half of the media and the intima of the umbilical arteries become necrotic, but this does not stimulate inflammatory reaction. Arrows indicate routes by which infection may spread beyond the granulation tissue barriers. Organisms invading the thrombus in the vein before this is organized may disseminate by emboli.

Pathogenesis

Soon after the child takes the first breath, the umbilical arteries contract strongly, blood flow ceases, and the arterial lumen becomes very narrow. The intima and the inner part of the media are thus deprived of blood supply and undergo aseptic necrosis.

The older writers postulated ante-natal, natal and post-natal infection. It is now considered that infection usually takes place within 48 hours of birth but that it may occur later in children with low resistance to infection, such as premature infants with large succulent cords. As separation of the cord proceeds, granulation tissue, which is resistant to the passage of microorganisms, normally develops, and epithelium quickly covers the granulation tissue.

Bacterial infection from the open umbilical wound may invade, lie latent in, or spread along, the lumen, the inner necrosing coats of the artery or the mantle zone of loose connective tissue around the artery, and suppuration in any one of these situations may spread to invade the others.

As infection delays or prevents obliteration of the vessels, umbilical bleeding is a common sequel. Indeed, bleeding from the umbilicus, if we exclude the haemorrhagic disorders, is probably always an expression of infection.

Depending upon the length of artery affected and its patency, the following sequence of events may result from infection of an umbilical artery:

(1) Sepsis in a segment of an umbilical artery, the iliac end of which is obliterated, carries the implications of any septic focus. If the umbilical end remains patent there will be chronic or intermittent purulent umbilical discharge. If the mantle zone is infected either primarily or secondarily, local or general peritonitis may result. Pus is prevented from pointing forwards by the tough fascia of the anterior abdominal wall. It may track along the course of the artery to point as an abscess in the scrotum or thigh. Infection may reach the blood stream to cause septicaemia through the small vessels of the anterior abdominal wall.

(2) If the infection involves the whole course of the vessel, the iliac end remaining patent, in addition to the possibilities already mentioned, bacteria from the lumen of the umbilical artery may be released into the blood stream and a rapidly fatal septicaemia result. Should a periarterial abscess spread the whole length of the vessel, pus would ultimately collect around the iliac vessels, where it might in time cause erosion and weakening of the wall of an iliac artery. I have not found a description of this latter happening in the literature, though it seems the most likely explanation of one of the cases detailed below.

Case Reports

Case 1. Stephen O. was born at home and admitted at the age of 7 weeks. No information is available about the condition of the umbilicus in the post-natal period. He had made good progress, apart from a probable urinary infection at 5 weeks, when the umbilicus started to discharge pus. Thirty-six hours

before admission he had become extremely fretful, refused his feeds and vomited.

He was found to be very ill with grunting respirations, pallor, dehydration and the appearance of shock.

The abdomen was distended and tympanitic. There was guarding and a possible mass on the left below the umbilicus.

Clinical and radiological findings suggested peritonitis.

At laparotomy the abdomen was full of seropurulent fluid, no cause for which was found. The child died 48 hours after the onset of the illness.

At necropsy, the umbilicus was discharging but not inflamed. A segment, 1 cm. in length at the iliac extremity of the left umbilical artery, was obliterated and appeared normal. The whole of the left umbilical artery, apart from this small lower segment, was swollen and contained blackish clot, and there was a large loculated periarterial abscess with rather fibrotic walls, containing yellow pus.

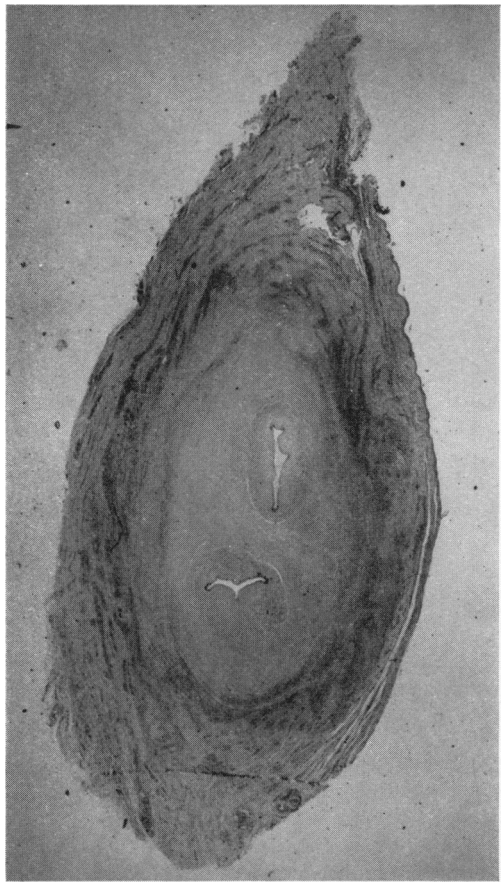

FIG. 2.—Section of umbilical arteries in Case 2 showing dense inflammatory infiltration in mantle zone reaching peritoneum, arterial walls free of inflammatory reaction, and masses of bacteria along margins of patent arterial lumina.

Staphylococcus pyogenes was grown from the peritoneal fluid, the abscess and the lumen of the artery.

In this child the mantle zone was infected within the immediate post-natal period. Infection spread inwards to involve the lumen of the artery. By 5 weeks of age a large periarterial abscess had formed from which the peritoneal cavity became infected, and from which pus discharged at the umbilicus.

Case 2. Stephen McC. was born in hospital, a normal healthy baby until the seventh day, when there was bleeding from the umbilicus. On the ninth day the child refused feeds and vomited repeatedly; the vomitus contained blood.

He was admitted on the eleventh day having been ill for 36 hours. He was pale and collapsed with a subnormal temperature and tachycardia. The limbs were cold and mottled, skin petechiae were noted, examination of the chest showed widespread fine crepitations and the abdomen was distended. The blood chemistry was fairly normal apart from a blood urea level of 216 mg. %. The white cell count was 10,000 per c.mm. with a normal differential count.

The child died in convulsions 44 hours after the onset of the illness.

At necropsy there was no discharge from the umbilicus, but the depths of the cavity were purulent and haemorrhagic.

The umbilical arteries were patent throughout, the part adjacent to the umbilicus being filled with black clot.

Microscopically (Figs. 2 and 3) the inner part of the wall of the arteries showed only the normal involutional necrosis but masses of cocci were seen here and in the lumen. There was widespread periarteritis and early fibrinous peritonitis.

The lungs were solid with haemorrhagic pneumonia. Microscopically, embolic lesions were seen in the pancreas and both kidneys.

Staphylococcus pyogenes was grown from the umbilicus and the bronchi.

The mantle zone was infected at birth and this led to a periarterial abscess, from which, as in Case 1, the peritoneum became infected. The infection in the lumen and inner part of the wall of the artery had probably been present since birth (it caused no reaction) as the organisms proliferated in avascular necrotic tissue. The lumen of the umbilical artery was patent throughout, and from it cocci were discharged into the internal iliac artery. A virulent septicaemia resulted with embolic lesions in the lungs, pancreas, kidneys and skin.

Case 3. John McC., aged 4½ years, was born in hospital.

When seen as an out-patient the umbilicus had been discharging pus for three months. *Staphylococcus pyogenes* was grown from the umbilical discharge. A probe passed from the umbilicus downwards and to the left.

At operation the left umbilical artery from the umbilicus to the level of Poupart's ligament was as thick as a cigarette. The iliac extremity was a fine, string-like structure. The peritoneum was adherent to the infected artery and posteriorly omentum was adherent to the peritoneum. The infected tissue was removed *en bloc*. Sections showed granulation tissue heavily infiltrated with plasma cells, a few polymorphs and eosinophils. Arterial structure could not be defined.

We concluded that infection at birth, either luminal, intra-mural or periarterial, had remained quiescent until the child was 4½ years old.

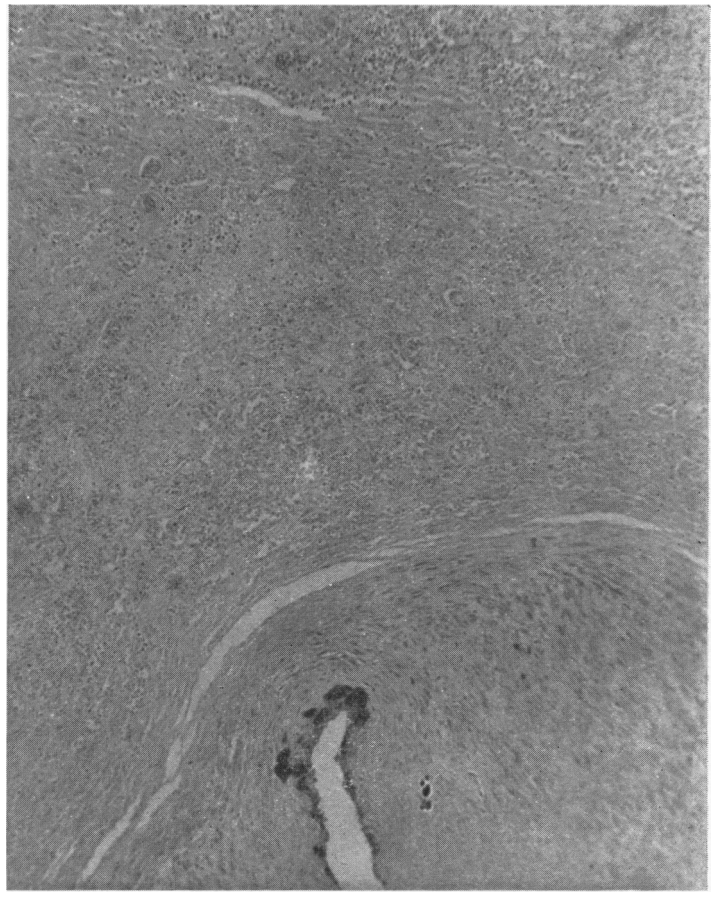

FIG. 3.—Higher power view of same section as Fig. 2 above.

Case 4. Carol S. was born at home. The cord was a little late in separating, but the umbilicus dried up normally. On the 12th day there was a transient discharge of yellowish brown material from the umbilicus. On the 21st day she was admitted to Noble's Hospital, Isle of Man, because of severe bleeding from the umbilicus. Blood continued to seep for a week and was followed by a purulent discharge. The temperature rose to 101-103° F., and the haemoglobin fell to 52 %. The upper abdomen was distended and superficial veins were dilated, the liver being enlarged. A blood transfusion was given and she was transferred to our care at the age of 8 weeks.

The above findings were confirmed and a tender mass was felt in the right iliac fossa which could just be touched from the rectum. A plain film showed nothing abnormal but a radiograph after iodized oil had been injected through the umbilicus showed pooling of the opaque medium below and to the right of the umbilicus (Fig. 4).

The discharge from the umbilicus grew *Staphylococcus pyogenes* and diphtheroids.

At operation the peritoneal cavity was almost obliterated by adhesions. The mass was extra-peritoneal and was seen to pulsate. During the enlargement of the incision to obtain better exposure, torrential arterial bleeding started which was controlled with some difficulty by applying digital pressure to the aorta.

The swelling, which was about the size of a tangerine, was incised and found to contain a cavity into which a large artery (either the internal or common iliac) opened. The opening was oversewn, a portion of the wall of the cavity was removed and the cavity obliterated by several layers of silk sutures. A mass of inflammatory tissue was seen in the line of the umbilical artery, and was left undisturbed. The child lost 2 pints of blood at operation, which was replaced, the haemoglobin being 76 % before and 66 % after operation.

Post-operatively the abdominal distension and superficial veins disappeared, the haemoglobin level rose to a normal figure, but the umbilicus continued to discharge and a second operation was required to drain the umbilical end of the artery.

Section of the tissue removed at the first operation showed it to consist of laminated fibrin in the interstices of which were erythrocytes and numerous polymorphs. In the inner layers there was some early calcification.

It appeared that the right umbilical artery had been infected at birth. Severe bleeding at 21 days suggested that the whole length of the artery was patent. It is not possible to say if the infection which caused it to remain patent was primarily in the lumen, or secondary to a periarteritis which also caused peritonitis, probably at the time of the febrile illness in Noble's Hospital. The periarteritis spread

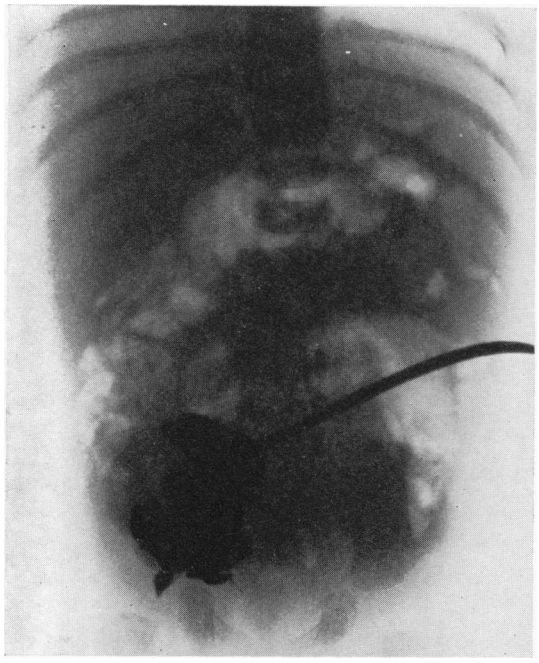

FIG. 4.—Injection through umbilicus in Case 4 showing large collection of iodized oil in right iliac fossa.

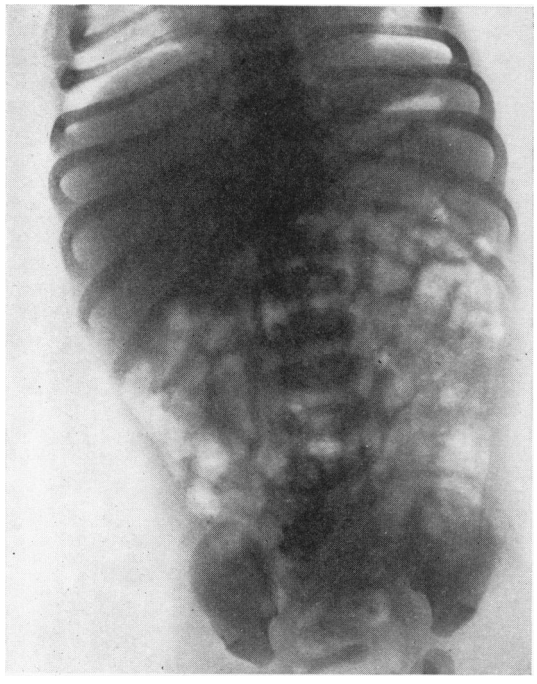

FIG. 5.—Radiograph of Case 5 after injection of iodized oil. Right periarterial abscess.

to the internal iliac artery where an abscess formed, weakening the arterial wall and ultimately causing the formation of an aneurysm. There was no evidence of bacterial embolism, and it is unlikely that the aneurysm was of embolic origin. This case is probably unique.

Case 5. Catherine M.D. was seen as an out-patient at the age of 4 weeks. She had been delivered by a midwife at home. The umbilicus had discharged since separation of the cord.

A probe could be passed downwards and to the left from the umbilicus. There was profuse purulent discharge from which *Staphylococcus pyogenes* was cultured.

Radiographs after the injection of iodized oil (Fig. 5) showed the typical picture of pooling round the left umbilical artery.

Following the injection the discharge gradually diminished and six weeks later the umbilicus was dry; the white cell count and the erythrocyte sedimentation rate were normal.

This case is probably one of periarteritis involving a short segment of the artery, drainage being improved by the injection. It is probable, but not certain, that this baby will have no further trouble.

Case 6. David S. was born in hospital by internal

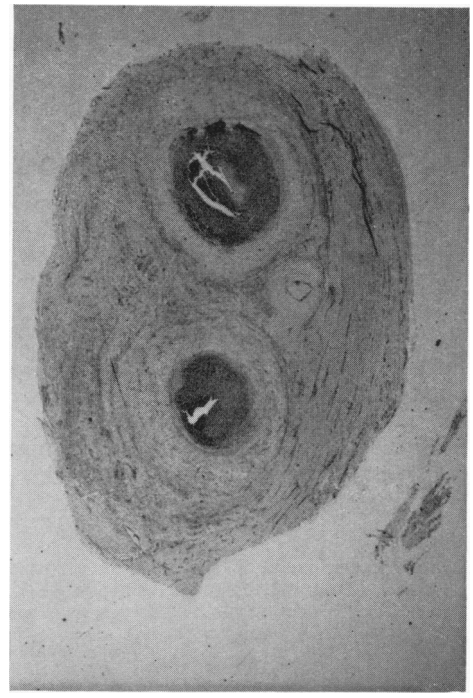

FIG. 6.—Section of umbilical arteries in Case 6 showing minimal infiltration in mantle zone, with inflammatory destruction of inner portion of arterial walls.

version and forceps delivery after 37 weeks' gestation. Five pounds in weight, he was considered a normal infant.

At 2 days of age he refused to feed, looked ill, showed deepening jaundice and para-umbilical redness, and died the same day.

At necropsy, the cord was still attached. The umbilicus was moist and yellow. The umbilical vein appeared normal.

The arteries contained purulent material and were patent throughout. Microscopy showed that the mantle zone was uninfected (Figs. 6 and 7). Infection was limited to the inner zone of the arterial wall and the lumen, which were heavily infiltrated with pus cells. In one artery the infection was most severe in the intramural zone, but was leaking through into the lumen. In the other artery the inner wall consisted only of pus cells.

There was a tentorial tear, without subdural bleeding, but extensive central cerebral softening.

A paracolon bacillus was isolated from the umbilicus, the umbilical arteries, the brain and the blood stream.

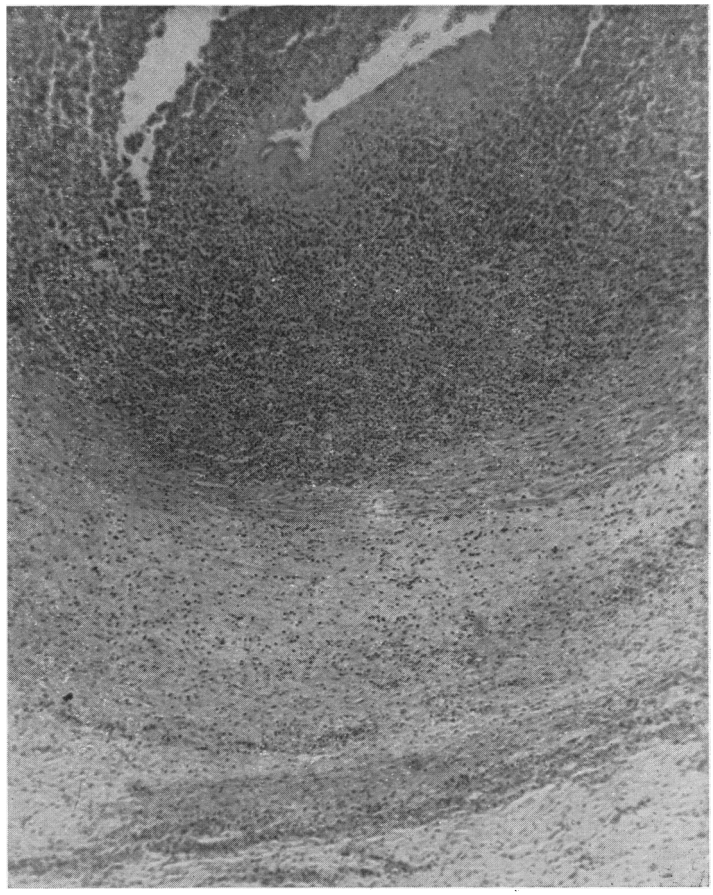

FIG. 7.—Higher power view of same section as Fig. 6.

Conclusions

The florid umbilical sepsis of the last century is now extremely rare in the western world, but infection of the open umbilical wound still occurs. One of its manifestations is septic umbilical arteritis. This condition may, if unrecognized, lead to various serious complications and cause death from septicaemia. Umbilical arteritis should be considered a possible source of infection when a child's abdomen is opened for peritonitis and no obvious cause is found. It seems possible that as the incidence of resistant strains of bacteria increases, umbilical arteritis and its complications will be seen more often in the future. In this small series *Staphylococcus pyogenes* was probably the infecting organism in five out of the six cases.

I should like to thank Dr. Edward Hall, Pathologist to Alder Hey Children's Hospital, for his help, Mr. P. P. Rickham for allowing me to include his cases and Dr. Barbara Ockenden for necropsy reports.

My thanks are also due to Dr. J. E. Morison for permission to reproduce Fig. 1 from his book.

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