The child was placed in an oxygen tent and penicillin therapy was given to control the bronchopneumonia. The right lower limb was exposed to room temperature, and at 4 p.m. a right paravertebral block was performed with 10 ml. of 0.5% procaine hydrochloride in an attempt to open up the collateral vessels. At this stage the child's general condition prevented any more heroic measures. No change in the condition of the leg was noted.

At 8.30 p.m. an intense uncountable tachycardia occurred and the abdomen became distended; bowel sounds were absent. An occlusion of the superior mesenteric artery, causing a paralytic ileus, was envisaged. At 11 p.m. it was noted that the toes of the left foot had become icy cold and pale. The left femoral and popliteal arteries were palpable, but the left dorsalis pedis was impalpable. The abdominal distension gradually resolved, bowel sounds returned, and the child passed a motion mixed with blood.

At 9 a.m. on December 22 marbling of the left foot was present, extending to the ankle. The condition of the right limb remained unchanged. As the child's general condition had improved slightly it was decided to explore the aortic bifurcation despite the fact that 24 hours had elapsed since the onset of vascular inadequacy in the lower limbs.

Operation.—Under general anaesthesia the aortic bifurcation and the iliac tributaries were exposed by a midline subumbilical extraperitoneal approach. Pulsation was absent in the right common iliac but was present in the left iliac vessels. The thrombus was palpable, extending from the aortic bifurcation along the whole extent of the right common iliac and partially into the left common iliac vessels, partially obstructing the latter. The right common iliac artery and its tributaries were dissected out and rubber catheters passed round the external iliac and the common iliac arteries and aorta. A small vertical incision was made in the right common iliac artery, and the clot extruded itself and was withdrawn. Further extensions of the clot were milked and sucked out, proximally and distally, until brisk gushes of arterial bleeding occurred. The clot was smooth and not adherent to the arterial wall. The opening in the vessel was closed with five interrupted waxed-silk sutures. After suture the right common iliac artery was distinctly narrower than the left, but active pulsations were transmitted. The wound was then closed in layers.

The child's general condition remained unchanged and no appreciable improvement was noted in the limbs as compared with the pre-operative state. The blood-coagulation time, estimated at 9 p.m., was $7\frac{1}{2}$ minutes. Five thousand units of heparin was given intravenously. The child died at 3.25 a.m. on December 23.

Post-mortem Findings.—The heart disclosed no abnormality, but the lungs showed marked bronchopneumonic changes. The small intestine was covered with multiple small haemorrhagic infarcts, but no clot was found in the superior mesenteric artery. The thrombus removed from the aortic bifurcation was red in colour, and consisted of hyaline material containing red cells and granulocytes.

Discussion

All writers on arterial occlusion in infancy stress the importance of sepsis in its causation, but, as Gross correctly points out, overwhelming infection in infants may cause multiple areas of cutaneous and deep-tissue necrosis representing focal areas secondary to pyaemia or septicaemia, but only rarely will occlusion of larger vessels occur without the presence of endocardial vegetations.

Thrombosis of the umbilical and hypogastric arteries in the neonatal period is a physiological mechanism which may progress abnormally against the blood current into the common iliac artery. By progression the thrombus may extend even into the lower portion of the aorta. Arterial obstruction in the newborn should suggest this pathological sequence.

The presence or absence of a congenital heart lesion must always be ascertained; arterial embolism in such cases

carries a bad prognosis, as the likelihood of repeated episodes is ever present. In the presence of such a lesion, vascular accidents are fostered by any severe illness which may induce dehydration, haemoconcentration, and augmentation of blood coagulation.

In the above case the necropsy findings excluded a congenital heart lesion or endocardial vegetations—the latter having been considered clinically as the cause of the multiple emboli. It had been thought that the first embolus had impacted in the right common iliac artery, with retrograde extension to the aortic bifurcation, and that a second embolus involved the superior mesenteric artery. The absence, however, of a common source for the emboli strongly suggests that the arterial occlusions were due to separate thrombotic processes complicating a severe bronchopneumonia.

The collateral circulation in infancy must be extremely adaptable, with great potentiality for compensatory dilatation. Thus sympathetic nerve block is a useful measure. The administration of heparin may be difficult in infancy, but ethyl biscoumacetate ("tromexan") may prevent propagation of an established thrombus.

Embolectomy or surgical evacuation of the thrombus within 6 to 10 hours is indicated if the infant's limb is in danger, but amputation, even if indicated, should be deferred for as long as possible during this time, every effort being made to augment the impaired circulation.

We are indebted to Dr. M. L. Thomson and Mr. H. M. Goldberg for permission to report this case and for their advice and encouragement.

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Medical Memoranda

Pilonidal Sinus of the Suprapubic Region

Professor Ian Aird (1952) reports a case of pilonidal sinus of the axilla and states that this makes the sixth site of pilonidal suppuration, the five sites previously recorded being the sacro-coccygeal region, the perineum, the barber's hand, the sole of the foot, and the umbilicus. The following case report is of interest in that the patient presented a lesion in the suprapubic region which looked like and behaved like a pilonidal sinus. Histologically, it was a simple dermoid. Professor Aird's article stimulated the recording of this case of pilonidal suppuration at a site which must be rare, as it does not seem to have been reported previously.

CASE REPORT

The patient, a woman aged 35, was admitted to hospital on January 30, 1952, with a history of having had, in the suprapubic region, a swelling like a boil, which pointed and burst, then leaked pus for some weeks and finally regressed, only to recur and go through the same cycle again and again. The history was of two years' duration. Fourteen years before she had had a sacro-coccygeal pilonidal sinus excised. Following this operation the wound had healed soundly and there had been no recurrence of suppuration at that site.

On examination a sinus with a pinhole orifice was observed in the middle of a shallow depression $\frac{1}{4}$ in. (6 mm.) in diameter, in the midline immediately above the symphysis pubis. On pressure a small bead of pus was expressed from the orifice.

On January 31 the sinus, along with a cyst with which it communicated, was excised through an elliptical incision. The cyst extended downwards for about $1\frac{1}{2}$ in. (3.8 cm.) in the midline, its most distal part lying superficial to the symphysis pubis. A small tuft of hair was found inside the sinus. On February 6 the patient was discharged with the wound soundly healed.

"The histological report on the excised sinus and cyst was as follows: "Sections show a simple dermoid lined with squamous epithelium and containing a few hair follicles. There is a thick fibrous wall with a little subacute inflammatory change in parts."

COMMENT

In this case, as in Professor Aird's, there was a previous history of operative treatment for a pilonidal sinus in the sacro-coccygeal region. The clinical and histological appearances of the sinus were those of a pilonidal sinus. It is probable that the origin in this case was a dermoid cyst occurring at a point of embryological fusion. Being a hair-bearing dermoid, it formed a hair-bearing sinus. It is therefore etymologically correct to define it as a pilonidal sinus.

It is suggested that to the six previously recorded sites of pilonidal suppuration may be added a seventh, the suprapubic region. It is also suggested that pilonidal suppuration could occur in any place where dermoids are apt to occur—that is, at points of embryological fusion—provided that the cyst contained hair follicles in its lining membrane. It may be that the term "pilonidal sinus" is usually reserved for the sinus occurring in the sacro-coccygeal region, and that similar sinuses occurring elsewhere are not labelled as pilonidal sinuses but given names such as suppurating dermoid cysts and so have not appeared in the literature as additional sites of pilonidal suppuration.

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Deaths of Two Patients Treated by Phenylbutazone

Agranulocytosis and haemorrhage, usually from the gastrointestinal tract, are the principal recognized toxic reactions caused by phenylbutazone ("butazolidin"). The drug is contraindicated in the senile, the hypertensive, and those with a history of peptic ulcer, oedema, or other manifestations of heart failure. Four deaths attributed to the drug were recorded recently (*British Medical Journal*, 1952; Davies and others, 1952). In one the cause was haemorrhage from multiple ulcers of the stomach, and in two agranulocytosis was responsible. The following two cases were both the subject of a coroner's inquiries.

CASE 1

A woman aged 65 had suffered from rheumatoid arthritis for 17 years. This had progressed steadily, and before the administration of phenylbutazone she had been confined to her chair and suffered much pain. After being warned of the risks the patient agreed to phenylbutazone therapy, and one 0.2-g. tablet thrice daily was prescribed. Her pain was relieved and she was able to walk better. After 15 days' continuous treatment she complained of severe pain in the right loin, with radiation to the groin, and her urine was heavily contaminated with blood. She died the following morning.

Necropsy.—This was made 53 hours after death. The body was that of a small, frail old woman with blue, swollen ankles. The lower end of the oesophagus was oedematous and contained a subacute ulcer $\frac{1}{2}$ in. (1.3 cm.) in diameter. The stomach was normal, but on the anterior wall of the duodenum, 2 in. (5 cm.) distal to the pylorus, there was a perforation caused by the rupture of a small subacute ulcer $\frac{1}{4}$ in. (0.6 cm.) in diameter. There was no sclerosis in relation to either ulcer, and no evidence of present or past ulceration in the remainder of the alimentary canal. The left pleural cavity was obliterated by old fibrous adhesions, and the right cavity contained about 1 pint (570 ml.) of clear serous fluid. The upper lobes of the lungs were normal, but both lower lobes were partly collapsed and very oedematous. The pericardium was bound to the heart by old

fibrous adhesions in several places, and there was slight thickening of the muscular walls of both ventricles. The kidney substance was pale and the cortex was notably reduced. The capsules stripped with some difficulty, leaving a coarsely scarred surface. The adrenal glands showed complete loss of cortical lipid, and a diffuse fibrino-purulent peritonitis originating from the duodenal perforation caused matting of coils of intestine. Death was due to peritonitis following perforation of the duodenum due to peptic ulcer associated with phenylbutazone therapy.

CASE 2

A woman aged 71 suffered from rheumatoid arthritis, and, after being warned of the risks, agreed to treatment by phenylbutazone. One 0.2-g. tablet was taken by mouth each day for a week, after which she felt much better, and the dosage was doubled to 0.4 g. a day for a second week. At the end of this fortnight's treatment she did not feel well and stayed in bed. The following day she telephoned to her doctor at 6 a.m. and complained of severe pain in the left hip. Her doctor visited her immediately, gave her an injection of morphine, and requested an enema to be given by the district nurse. At 9.15 a.m. she was found on the floor of her bedroom; she said that she was unable to get back to bed, and again complained of pain in her left hip and down her left side. She died at 2.15 p.m. the same day.

Necropsy.—This was made 26 hours after death. The body was that of an obese edentulous old woman with swollen finger-joints. The left flank and thigh were swollen and discoloured and the overlying skin was peeling or raised in large blebs. The superficial vessels of the neck presented a "marbled" appearance, and the abdomen was distended. No break in the skin was observed in the putrid areas or elsewhere. The heart muscle was pale and flabby, but the valves and chambers were normal. The main vessels were filled with frothing blood, and the intima showed intense haemolytic staining. The lungs were uniformly engorged. The peritoneal cavity was distended by foul-smelling gas, and the muscle between the spine and the left kidney was soft, black, crepitant, and friable. This gangrene involved the whole of the left psoas and iliacus muscles and extended half-way into the quadratus femoris muscle group of the left thigh. The stomach and both small and large intestines were normal and showed no naked-eye evidence of ulceration in the mucous lining, and the loose connective tissue behind the descending colon was apparently normal. The kidneys showed marked toxic pallor of the cut surface, and the bladder was empty and normal. The cut surface of the spleen was pale, soft, and putrid, and that of the liver very pale and dotted with small gas bubbles. The adrenal glands showed cortical pallor, and the remaining abdominal organs were normal. Death was due to gas gangrene.

DISCUSSION

There can be no doubt that death in Case 1 was directly contributed to by phenylbutazone. Shortly before death there was a haemorrhage from the genito-urinary tract for which no organic cause existed, and the two alimentary ulcers had acute characteristics. There was no history of indigestion referable to the stomach or duodenum, though six months before death this patient complained of pain at the lower end of the sternum about two seconds after swallowing. Radiological examination of the oesophagus was negative, and the symptoms subsequently passed off. The oesophageal ulcer found at necropsy was too recent to have caused these symptoms, though there may well have been a previous oesophagitis, with or without superficial transitory ulceration.

The mechanism leading to death in Case 2 is obscure, since the disease was obviously established before the injection of morphine into the right upper arm. Cases of gas gangrene in which the infecting organism was presumably derived from the bowel have been reported, but they are