Medical Memoranda

Two Cases of Injury to the Femoral Artery

The following two cases are thought to be worthy of record on account of the rarity of injury to the femoral artery.

CASE 1

A butcher aged 35 was disarticulating a joint when the knife he was using—a short one with a sharp point—slipped and he sustained a punctured wound in the right groin. Haemorrhage was profuse. The accident happened at about 8 a.m., and he was admitted to the hospital at 8.10 a.m. On arrival he was pulseless and still bleeding from the wound, which was just below Poupart's ligament. There was swelling of the groin as a result of a large haematoma. Steps were taken to control the bleeding by pressure. He was given an immediate intravenous transfusion of dextran—4 pints (2.3 litres) rapidly in one hour. His condition improved, and he was transferred to a surgical ward at 10 a.m.

Operation (12.30 p.m.).—By this time, four and a half hours after the injury, 5 pints (2.8 litres) of dextran had been given intravenously. Blood transfusion was started. Care was taken not to remove any dressings until the patient was anaesthetized and on the operation table, and preparations had been made to deal with the condition found. On removing the dressings there was a gush of blood. The bleeding was controlled by digital pressure and the field of operation was prepared. The common femoral, the superficial femoral, and the profunda arteries were then exposed by an incision below the site of digital pressure, and the common femoral artery was controlled by means of a clamp. A perforated wound of that artery was demonstrated at the junction of the middle and lower thirds. Suture of the two wounds in the artery, without obliterating the lumen, was considered impracticable. The common femoral artery is approximately $1\frac{1}{2}$ in. (3.8 cm.) in length. A section of it, a little less than $\frac{1}{2}$ in. (1.3 cm.) in length, was removed at the junction of the middle and lower thirds. The divided ends were united by an end-to-end anastomosis. The operation wound was closed except for drainage at the lower end. The tissues were relaxed by fixing the hip in a flexed position by means of a plaster-of-paris bandage.

The wound healed and the condition of the limb remained satisfactory. The popliteal and posterior pulse could be felt 24 hours after the operation. The plaster was removed at the end of three weeks, and the local condition was satisfactory when the patient was discharged from hospital four weeks after admission.

CASE 2

A youth aged 19 was riding in a motor-cycle race on May 4, 1953, when his machine skidded. He was thrown off and rendered unconscious. There was a severe laceration of the left groin and bleeding was profuse. A tourniquet was applied. The accident happened at about 3.30 p.m. and he was admitted to hospital at 5 p.m.

On admission he was conscious but dazed, and general condition was satisfactory. Nothing amiss was detected with his cranial nerves. On removal of the tourniquet there was a little oozing but no severe bleeding from an irregular lacerated wound of the soft tissues in the upper third of the left thigh. The anterior and posterior pulse could not be felt; otherwise the limb was satisfactory.

Operation (6 p.m.).—The operation field in the left thigh was cleaned, and a vertical incision 6 in. (15 cm.) in length was made over the femoral artery. The common femoral artery was exposed and found to be undamaged. A clamp was applied to the femoral artery. The profunda artery was found to be intact. The superficial femoral artery was found to be torn across and there was a gap of about $1\frac{1}{2}$ in. (3.8 cm.) between the divided ends, which were contracted

but not bleeding. The proximal end of the divided artery just distal to the profunda was ligated. The distal end was also ligated. The wound was excised and closed except for drainage.

Progress was satisfactory, as was the condition of the limb. Five days after the injury the posterior tibial pulse was palpable. Recovery was uneventful.

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Peptic Ulcer Perforation after Phenylbutazone Therapy

Among the many complications of phenylbutazone therapy, gastric ulcer activation, or formation of fresh gastric or duodenal ulcers, is well recognized. At least 15 cases of gastro-intestinal bleeding have been reported. Peptic ulcer perforation occurring during phenylbutazone therapy is rare enough to warrant the publication of the following case.

CASE REPORT

A 53-year-old lorry driver was admitted to Surgical Department A of Frederiksberg Hospital on November 8, 1953, at 8 p.m. as an emergency case. He had been six months in a German concentration camp until the end of hostilities in 1945. There had never before been dyspeptic symptoms. He had been complaining of a slight feeling of oppression and dyspnoea on exercise for the past two years, relieved after nitroglycerin. For about a week before admission he had suffered from epigastric discomfort, heartburn, and nausea, relieved after food. Three hours before admission he was suddenly seized with acute abdominal pain in the epigastric region and under the right costal margin with no irradiation. The pain subsided without treatment. Two similar attacks occurred in the ambulance on his way to hospital and just after admission.

On examination his temperature was 37.5° C. and pulse rate 60. He was sweating, but showed no signs of dehydration. There was some fixation of the abdomen during respiration, with tenderness and muscular guarding on palpation under the right costal margin, falling off towards the right iliac fossa. Liver dullness was normal on percussion. A preliminary diagnosis of penetrating ulcer or cholelithiasis was made and he was kept under observation, with application of local heat.

It was then revealed that he had been taking phenylbutazone, 1 tablet (200 mg.) three times daily for the past week (a total of 4.2 g.) because of rheumatic pains in the left shoulder.

As his temperature rose and the pain grew worse, and as it was known that phenylbutazone could activate an ulcer and provoke a perforation, operation was decided upon. The abdomen was opened through a transverse epigastric incision, and yellowish fluid gushed out on opening the peritoneal cavity. After mopping up, a perforation the size of the head of a pin on the first part of the duodenum came into view. It was closed and the suture line reinforced with a wisp of omentum, followed by closure of the abdomen. The post-operative course was uneventful.

COMMENT

Four cases of peptic ulcer perforation that occurred during phenylbutazone therapy have been published (Shields *et al.*, 1953). According to Burns *et al.* (1952), Mason (1953), and others, phenylbutazone is slowly eliminated; when treatment is discontinued, it will normally be seven to eight days before a single dose is eliminated. Cases have been described in which phenylbutazone could be demonstrated in the blood two weeks after discontinuation of treatment. Accordingly, complications may occur several

days after treatment is stopped. In one case perforation actually occurred 72 hours after stopping phenylbutazone medication (Shields *et al.*). The ulcer-provoking and ulcer-activating effect of phenylbutazone has been compared with that of cortisone. Some authors have postulated that phenylbutazone acts via an adrenal cortex stimulation (Wilhelmi, 1949), but most investigators deny this (Bach, 1952; Currie, 1953; Cudkowicz and Jacobs, 1953; Domenjos, 1953).

Phenylbutazone is a dangerous drug. If it is used it should be administered, after meals, with milk or alkali. It is absolutely contraindicated in patients with peptic ulceration.

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A Fatality Due to the Use of Gold

Until the recent introduction of cortisone, A.C.T.H., and phenylbutazone, the most important drugs available for the treatment of rheumatoid arthritis were the gold salts. They are still widely used, and will continue to be, until the supply and the efficiency of the newer drugs are more satisfactory than at present. The toxic effects of gold salts are well recognized, and yet are generally supposed to be controlled if a careful watch is kept for their early manifestations, and if a certain maximum dose is not exceeded. In the present case an unusually small total dose resulted in fatal damage to the bone marrow, and the first signs of serious toxic effects developed a fortnight after the course of gold had been finished.

CASE REPORT

A married woman aged 27 was admitted to hospital on August 20 on account of menorrhagia. Her menstrual period had begun two days before admission, and she had since suffered from a continuous and very heavy loss. She gave a history of a chronic arthritis which had first shown itself immediately after the birth of her second child two and a half years before. The disease had developed very rapidly and had reached its height only nine weeks after her confinement, remaining thereafter almost stationary. It mainly affected her hands and feet, although her elbows were also involved to some extent. She had been under treatment—as an out-patient at another hospital—since the beginning, and various treatments had been tried: ascorbic acid and deoxycortone acetate, pregnenolone, and insulin. All had produced an initial improvement followed by a relapse. The first and only course of gold was begun in the middle of May.

The preparation used was "myocrisin"; the initial dose was 0.01 g. Subsequent doses at weekly intervals were 0.02, 0.03, 0.03, 0.04, and 0.05 g. This last dose was repeated four times, making a total of 0.38 g. in ten weeks. The course was finished at the end of July, and throughout it there were no toxic signs or symptoms, except a slight pruritus following the third injection. The urine was regularly tested for albumin, and the test was as regularly negative. She was seen again as an out-patient on August 10, and at that time mentioned some bruising she had on her arms. Ten days later she came into hospital.

On examination she was still losing heavily per vaginam, and in addition there were many fresh bruises and purpuric spots on her limbs, and some bleeding from the gum margins. A blood count showed: haemoglobin, 48%; white cells 3,200 per c.mm. (48% polymorphonuclears); blood platelets 2,000 per c.mm.

A transfusion of 2 pints (1,140 ml.) of fresh blood was given, raising the haemoglobin to 68%, stopping the bleeding from the gums, and diminishing the loss per vaginam.

It was considered an unlikely coincidence that she should develop an idiopathic thrombocytopenic purpura so soon after the administration of a drug known to have toxic effects on the bone marrow, and the illness was assumed to be due to gold poisoning. Two further facts supported this assumption: first, the examination of the peripheral blood (and later of the bone marrow) showed that all cellular blood elements had been affected, not only the platelets; and, secondly, the only drug that the patient had taken, apart from the gold, was aspirin—one which, although it may prolong the bleeding-time when taken in excessive doses, has never been shown to have any poisonous effect on the bone marrow. An attempt was therefore made to increase the excretion of the gold remaining in the body by using dimercaprol. A first injection of 400 mg. was given on the day after admission, and the drug was continued in a decreasing dosage for five days thereafter. The urine was tested for gold on the third and fifth days of this treatment, but none was found; and no evidence was ever obtained that dimercaprol had any effect, either at this time or when a second course was given between August 29 and September 2.

In addition to the dimercaprol and the blood transfusions she was given A.C.T.H. in a dose of 25 mg. six-hourly between August 25 and September 2. The object in giving her the hormone was only in part to relieve her arthritis, although this was achieved within the first three days of treatment, and her hands continued to improve throughout the course; it was also hoped that the A.C.T.H. might have a stimulating effect on the bone marrow. In the three or four days immediately following the first administration of A.C.T.H. there was some increase in the number of lymphocytes in the peripheral blood, but the number of polymorphonuclears showed a steady decline until the end. In view of this severe and increasing granulopenia, penicillin was given as a prophylactic measure throughout.

The patient was also given pentnucleotide, but there was no noticeable effect in the peripheral blood consequent upon it. In spite of blood transfusions she died on September 4.

At necropsy an enormous intraperitoneal haemorrhage was found which had originated from a normal ruptured ovarian follicle. This had obviously been the final event which caused her death, although haemorrhages were also present in the pelvis of the left kidney, in both pleural cavities, on the pericardium, and on the surface of the brain. Histological examination of the femoral marrow revealed merely a fatty marrow with scanty haemopoiesis, and that mainly erythroblastic.

A report on the sternal marrow, a sample of which was taken and examined on August 22, stated: "A few flecks of a very hypoplastic marrow were obtained. No megakaryocytes were seen. Erythropoiesis is normoblastic, but there is hypoplasia of the erythropoietic and myeloid tissue. There is no maturation arrest of the myeloid tissue."

Reviewing this case after an interval of time, it seems that two failures contributed to the outcome: (1) failure to appreciate the significance of the evanescent pruritus after the third injection of gold; and (2) a failure to foresee the possible danger from ovulation, which could readily have been avoided by timely administration of oestrogen.

Mild toxic symptoms of intolerance to gold occur in nearly half the patients to whom the salts are given, but it must always be remembered that these mild reactions may be the only warnings of more severe ones to come. In regard to the second failure, if ovulation had been suppressed artificially death would have been delayed at least some days, though whether this would have affected the final outcome can never be known.

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