FIG. 2—Close-up view of "skin tunnel" transformer in patient. (For scale, toroid has external diameter of 4 cm.)

FIG. 3—Total external component of system.

The patient has had the system for six months. The original capsule had to be replaced because of an insulation fault. Otherwise the system has continued to function well.

Discussion

In devising a pacing system suitable for use in developing countries the main considerations were safety and low cost. An induction system seemed most promising as the external generator could be built from locally available components at low cost, and by building in redundancy wherever possible a high degree of safety could be achieved. Nevertheless, the problem of having to use adhesive plaster to fix the primary coil closely over the secondary coil appeared a major one for warm countries, where perspiration would tend to loosen the adhesive. This problem appears to have been overcome by using the skin tunnel transformer. In addition this system allows very efficient transfer of energy and, therefore, low battery drain.

It remains to be seen just how robust the skin tube of such a system will prove to be. If satisfactory it may have wide possibilities in the delivery of power to implanted artificial organs.

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MEDICAL MEMORANDA

Postpartum Purpura Fulminans: Successful Management with Streptokinase

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Purpura fulminans is an acute condition characterized by extensive bleeding into the skin, resulting in haemorrhagic bullae with large areas of skin necrosis and gangrene of extremities. The bleeding leads to severe anaemia and the untreated condition is usually fatal (Henoch, 1887).

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We present a case of postpartum purpura fulminans associated with bilateral deep vein thrombosis. Successful management was achieved only by using streptokinase in addition to heparin.

Case Report

The patient, a 19-year-old primigravida, was delivered of a normal male child by lower segment caesarean section. The pregnancy had been uneventful apart from a failed forceps delivery due to fetomaternal disproportion.

There was nothing significant in the past medical history and in particular there had been no previous thrombotic episode.

On the ninth postpartum day she became pyrexial (temperature, 38.5°C) and the next day developed right sided pleuritic pain. Radiography at the time showed changes consistent with collapse and consolidation at the right lower zone. There was no clinical evidence of deep vein thrombosis and she was treated with ampicillin 250 mg six-hourly.

The next day she noticed pain and tenderness in the right calf which was slightly swollen. A deep vein thrombosis was diagnosed clinically and anticoagulation begun with heparin, then phenindione. Adequate anticoagulation with phenindione as judged by prothrombin time was readily achieved but there was no change in her condition until eight days after the onset, when the calf circumference rapidly increased. This was thought to be due to intramuscular haemorrhage. Phenindione was, therefore, discontinued and she was given vitamin

K₁ 10 mg intramuscularly. The next day extensive bleeding into the skin of the upper lateral aspect of the right thigh was observed progressing to a haemorrhagic bullus in 18 hours (see fig.). Similar smaller lesions occurred over the right calf. All of the haemorrhagic lesions possessed an erythematous margin about 0.5 cm in width, and all were tender to light touch. At this point she was transferred from the maternity hospital.

Appearances of haemorrhagic bullus.

Venography on admission showed complete occlusion of the iliac and femoral veins on the right with loose thrombus in the veins of the right calf. Moreover, there was thrombus in the left iliac veins.

Results of haematological investigations performed on admission were: haemoglobin 13.2 g/100 ml, W.B.C. 7,000/mm³, platelets 250,000/mm³, the prothrombin and thrombin times were normal, plasma fibrinogen 250 mg/100 ml, plasminogen 3.5 units/ml, euglobulin clot lysis time 150 min (normal), serum fibrin degradation products (F.D.P.s) 80 µg/ml (normal 0-20 µg/ml) measured on two occasions before therapy. Though the haemoglobin concentration was normal some fragmented erythrocytes were noted on the peripheral blood film. The blood urea was normal.

Initially, intravenous heparin 10,000 units four-hourly was given but there was extension of the haemorrhagic lesions and further rapid swelling of the leg. Because of the uncontrolled progression, heparin was discontinued and thrombolytic therapy instituted. The titrated initial dose of streptokinase was 250,000 units, given over 30 min, and this was followed by a continuous infusion of 100,000 units hourly. Within 24 hours there was noticeable improvement, and after two days the subcutaneous haemorrhages and swelling began to subside. After five days the streptokinase was replaced by continuous heparin infusion, 42,000 units in 24 hours for three weeks. During this time there was continuous improvement: the bullae ruptured spontaneously but only required treatment with non-adherent dressings.

The heparin was then replaced by oral warfarin. Six weeks after the onset a skin biopsy specimen taken from the affected area showed only a resolving vasculitis with perivascular cuffing. Repeat venography at the same time was unfortunately unsuccessful for technical reasons.

Comment

Purpura fulminans is an acute disorder in which there is rapid and progressive bleeding into the skin (Henoch, 1887). Tissue

necrosis rapidly ensues and those patients who survive usually require amputation or extensive skin grafting. Children are much more commonly affected than adults and there is often a preceding history of bacterial infection (Hjort *et al.*, 1964; Chambers *et al.*, 1952; Crawford and Ridder, 1952). There are very few recorded cases of purpura fulminans complicating pregnancy and the puerperium.

In a comprehensive review of the literature Hjort *et al.* (1964) showed that the sites of skin necrosis show a predilection for the face, buttocks, the dorsal surfaces of the calves, and lateral surfaces of the thighs. The affected areas are often painful and tender and are characteristically surrounded by a narrow erythematous border which sharply demarcates the lesions from normal skin (Hjort *et al.*, 1964). These features were present in the patient reported on here.

Though disturbances of coagulation have been frequently recorded in patients with purpura fulminans, they are not an invariable feature. Thus platelet counts were normal in 13 of the 31 patients reviewed by Hjort, and plasma fibrinogen concentrations were normal in one-third. Nevertheless, intravascular coagulation has often been confirmed by histological examination of biopsy and necropsy material (Turin *et al.*, 1959).

In the case described here, skin haemorrhages occurred nine days after institution of therapy for a probable deep vein thrombosis and were thought to be a consequence of oral anticoagulation. However, after reversal of phenindione by vitamin K₁, there was alarming progression of the haemorrhage.

At this stage disseminated intravascular coagulation was suggested by the pronounced rise of the serum F.D.P.s and the presence of fragmented erythrocytes on the peripheral blood film. It is possible, of course, that the raised F.D.P.s were derived from the demonstrable venous thromboses (Ruckley *et al.*, 1970; Wood *et al.*, 1972). Persistent raised levels such as we observed in this patient are, however, unusual, and fragmented erythrocytes are not usually found in association with uncomplicated large vessel thrombosis.

Reports of the treatment of purpura fulminans with heparin have been disappointing and in this case there was rapid progression of oedema and haemorrhage during the 12 hours the patient was receiving this drug. Replacement of heparin by streptokinase was rewarded by rapid improvement in the patient's clinical condition and there was no further progression of the subcutaneous haemorrhages. After completion of the thrombolytic therapy there was satisfactory wound healing without loss of skin.

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