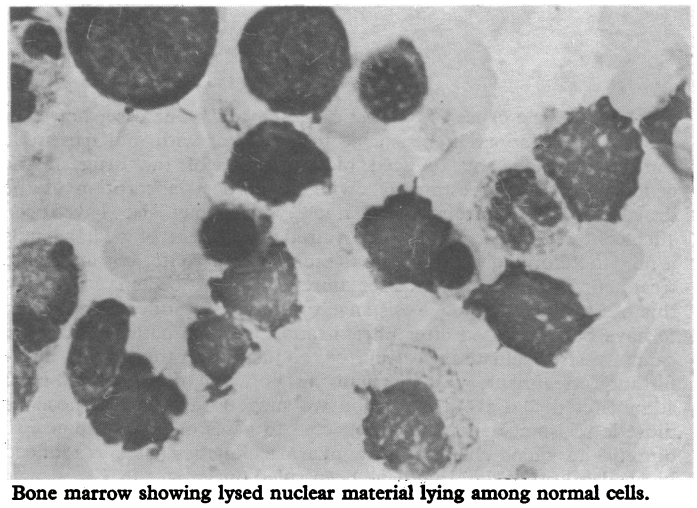

Bone marrow showing lysed nuclear material lying among normal cells.

other medication was cimetidine, which she took throughout her stay in hospital.

Her total white cell count remained between 0·8 and $1·5 \times 10^9$/l for six days with 0-10% neutrophils. The total white cell count increased on the seventh day and two weeks after admission was $17·8 \times 10^9$/l with 76% neutrophils.

A skin biopsy showed changes consistent with the diagnosis of scleroderma. Intravenous pyelography, cystoscopy, and retrograde pyelography showed nothing abnormal. Renal arteriography showed reduced cortical blood flow with irregularity and beading of the interlobular, arcuate, and interlobar arteries. A renal biopsy showed changes consistent with malignant scleroderma.

The patient remained anuric and was transferred to another renal unit for chronic haemodialysis but died soon after.

Comment

This patient with malignant scleroderma developed spontaneous neutropenia with circulating white cell antibodies and bone marrow features suggestive of an autoimmune mechanism. The neutropenia was reversed after corticosteroid treatment—a response similar to that seen in patients with autoimmune haemolytic anaemia[1] and thrombocytopenia[2] associated with scleroderma.

Neutropenia has been reported in association with cimetidine therapy.[4] In the present case the neutropenia resolved while the cimetidine treatment was continued and the bone marrow findings were not consistent with a drug-induced neutropenia.

Although antinuclear antibodies were present, they were present in only a dilution of 1/50 with a speckled pattern. Serum complement concentrations were normal and DNA antibodies were not raised. Thus the neutropenia was unlikely to have been caused by systemic lupus erythematosus.

We think, therefore, that this patient's neutropenia had an autoimmune basis associated with scleroderma. Various other autoimmune haematological disturbances have been described in patients with scleroderma,[1-3] but isolated autoimmune neutropenia has not to our knowledge been reported. Regular blood counts should be performed in patients with severe scleroderma, so that early appropriate treatment can be prescribed.

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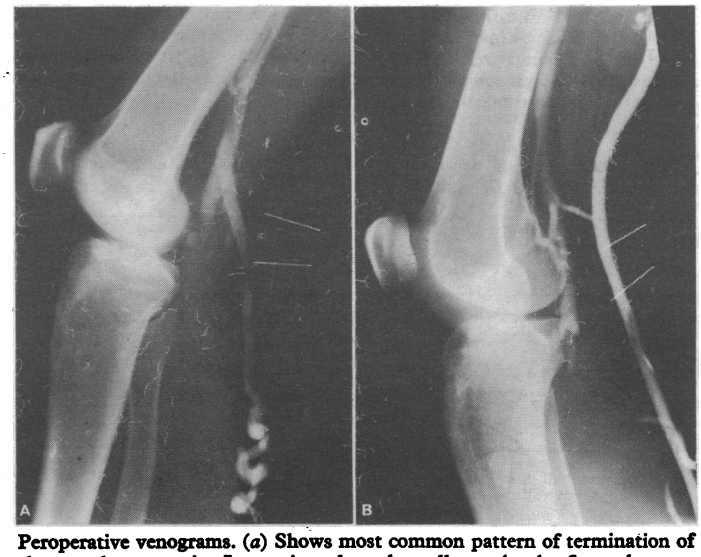
Peroperative venography to ensure accurate sapheno-popliteal vein ligation

When there is proximal incompetence of either the long or short saphenous vein surgery is indicated. Sclerotherapy is ineffective at these sites.[1] Sapheno-femoral ligation is relatively simple because this junction is fairly constant, although the position and number of tributaries vary. The short saphenous vein, however, varies widely in the level and pattern of its termination.[2 3] Ligating it may be difficult and hazardous. Wide incisions have been recommended to expose the contents of the popliteal fossa and, since vertical incisions are apt to fibrose or form keloid, generous transverse incisions are now usual. If too low a separate, higher incision can be made (step-ladder technique).[4] A stripper may be passed proximally and an incision made at the highest point where the tip of the stripper ceases to be palpable.[5] Sapheno-popliteal ligation need not be flush on the popliteal vein but all the tributaries must be ligated to prevent recurrence. Doppler ultrasound has also been used to locate the termination of the short saphenous vein but is inaccurate. For the past 10 years peroperative venography has been used when dealing with atypical or recurrent vein problems. It has proved particularly helpful in dealing with the short saphenous vein and has enabled an accurately positioned, small transverse incision to be used. For the past year venography has been a standard procedure in all operations on this vein. As expected, its termination has been shown to vary widely.

Method and results

Initially a sterile procedure was used and the *x*-ray picture taken after the leg had been cleaned and towelled. This was cumbersome and wasted time, so now the *x*-ray picture is taken before preparing the patient. The patient is placed in a lateral position with the lower leg flexed and the upper leg straight—the leg for operation being uppermost. A cassette containing a 30 mm × 40 mm *x*-ray film is placed under the upper leg and the *x*-ray tube positioned over the leg and centred on the knee joint. Two sterile 21 gauge hypodermic needles are inserted intradermally as markers at the flexural crease and at the highest point where the short saphenous vein is palpable. The film is then exposed after injecting 5 ml contrast media (45% Hypaque or similar material) directly through a 15 gauge needle if the vein is large or a 23 gauge butterfly cannula if the vein is tortuous. Before taking the *x*-ray picture patients under epidural anaesthesia are instructed to perform a valsalva manoeuvre. When patients are under general anaesthesia the expiratory valve is closed. The *x*-ray film is then processed while the surgeon scrubs-up and prepares the patient. Guided by the radiograph and the marker needles a small transverse incision is made over the sapheno-popliteal junction.

During 1979 125 short saphenous veins (24 bilateral, 37 left, 40 right) were operated on by this method. The actual junction was often higher than



Peroperative venograms. (*a*) Shows most common pattern of termination of short saphenous vein. Lower intradermal needle marks the flexural crease and upper needle the highest level at which vein was palpable. Note sapheno-popliteal junction higher than demonstrated clinically. (*b*) Shows short saphenous vein continuing in thigh with only very small communication to popliteal vein. Note upper marker needle over femoral condyle: valves in popliteal vein clearly shown.

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indicated by the marker (fig (*a*)). The upper level at which the short saphenous vein was palpated was over the femoral condyles and the vein continued several centimetres higher before joining the popliteal vein. By using positive intrathoracic pressure the junction was shown more clearly and valves were demonstrated in the popliteal vein. Sometimes the short saphenous vein continued into the thigh and there was only a very small communicating vein joining the popliteal vein (fig (*b*)). These high short saphenous veins commonly joined the long saphenous vein via the posteromedial tributary. Occasionally the short saphenous vein communicated with tributaries of the internal iliac vein at the upper thigh, and in one case the proximal communication was with the deep femoral vein.

Comment

The clear definition of the termination of the short saphenous vein made surgery simple and the sapheno-popliteal junction was easily located through a small transverse incision. Operations on recurrent short saphenous veins were simplified by the radiographic demonstration of the anatomy. The junction was often higher than clinically demonstrated and the peroperative venogram was particularly helpful when dealing with an atypical or recurrent vein.

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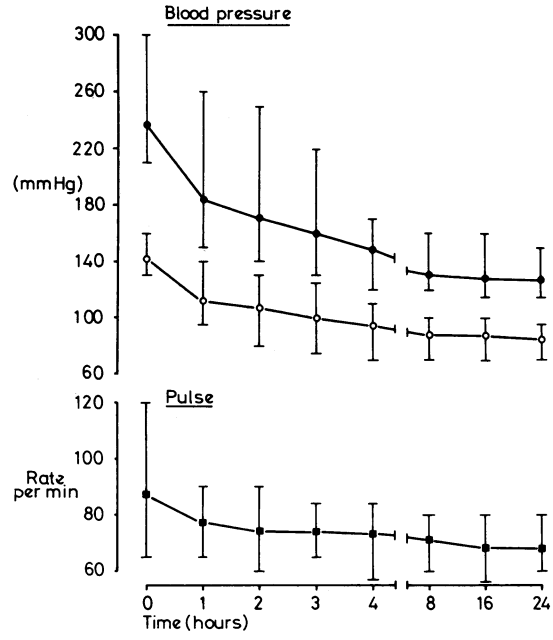
Treatment of severe hypertension with chlorpromazine and frusemide

Rapid reduction of blood pressure in severe hypertension has recently been the subject of controversy because cerebrovascular or coronary insufficiency are occasional complications.[1] Nevertheless, it is generally agreed that it is still appropriate treatment for severe hypertension associated with encephalopathy, cerebrovascular accident, cardiac failure, aortic dissection, subarachnoid haemorrhage, prolonged epistaxis, or eclampsia.[2] But all methods hitherto described are restricted in application by their complexity or their adverse effects. Hence there is scope for an effective alternative that does not have these limitations. Chlorpromazine is a major tranquilliser with a complex action on the cardiovascular system resulting in hypotension.[3] Frusemide, a loop diuretic, has an initial acute volume-depleting effect on the circulation.[3] That these drugs in combination reduce severely raised blood pressure satisfactorily and safely does not seem to have been recorded. We report a prospective study of the use of this regimen in nine cases.

Patients, methods, and results

We studied all patients aged under 70 years admitted to our unit with severe hypertension (≥210/130 mm Hg supine after over one hour's bed rest) during one year. There were five men and four women with a mean age of 53 years (range 43-61). Presenting features were cerebrovascular episodes (3), left ventricular failure (2), uncontrolled epistaxis (1), myocardial ischaemia (1), headaches (2), and uncontrolled hypertension (2). The electrocardiograms of eight patients fulfilled the voltage criteria for left ventricular hypertrophy, seven having associated ST/T wave changes ("strain pattern"). The electrocardiogram of the ninth showed changes of myocardial ischaemia. In seven the cardiothoracic ratio was >0·5. Grade IV hypertensive retinopathy was present in two, grade III in two, and grade II in four. Creatinine clearance was >100 ml/min in four and <60 ml/min in the remainder (mean 36, range 15-53 ml/min). Three patients had been taking antihypertensive drugs before admission.

Treatment was with 50 mg chlorpromazine intramuscularly, 50 mg frusemide intravenously, and bedrest. In one case the combination was repeated after three hours (initial blood pressure 300/150 mm Hg). Maintenance oral treatment with a diuretic and beta-blocker was begun from four to eight hours after parenteral treatment. Pulse rate and blood pressure (right arm supine, mercury sphygmonanometer) were recorded hourly for four hours and four-hourly thereafter. A gradual and adequate reduction in blood pressure and pulse rate resulted in every case (figure). Mean arterial pressure fell by an average of 79 mm Hg (42% of initial value, range 31-50%). There were no side effects.



Effect of chlorpromazine 50 mg intramuscularly and frusemide 50 mg intravenously on supine systolic blood pressure (●), diastolic blood pressure (○), and pulse rate (■) in nine patients with severe hypertension, showing mean values and range during 24 hours after administration. Oral maintenance treatment was begun 4 to 8 hours after parenteral therapy.

Comment

This method commends itself to routine use for the following reasons. It is simple to administer, unlike continuous intravenous infusions of nitroprusside or labetalol. The rate of action is gradual and predictable compared with the variable and precipitate hypotension induced by diazoxide or hydrallazine, and in contrast to these there is no reflex tachycardia. While being slightly more effective than oral labetalol,[4] unlike this drug it is not contraindicated in cardiac failure or asthma and a pressor response has not been observed. The only caveat applies uniformly to all methods of rapidly lowering blood pressure—namely, the risk of inducing cerebral or myocardial infarction, especially the former. In our series, however, the maximum fall in mean arterial pressure was considerably less than that at which cerebral hypoperfusion has been shown to lead to cerebral ischaemia and a risk of permanent neurological deficit.[1]

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