New Physical Sign of Acute Deep Vein Thrombosis

SIR,—We would like to draw the attention of your readers to the following two cases with an unusual and hitherto unreported sign of extensive deep vein thrombosis.

Case 1. A 54-year-old man presented to his doctor in January 1973 with pain in his left lower limb associated with inability to extend the knees fully. An analgesic was prescribed since the leg appeared normal apart from a 5° flexion deformity of the knee. After two days erythema and tenderness developed along the medial aspect of the thigh. He failed to respond to an antibiotic and bed rest, the thigh became swollen, and he was referred to hospital. On examination the pulse rate was 72/min and temperature 36.7°C. There was erythema along the medial aspect of the left thigh and marked swelling confined to the thigh was present. The knee was flexed to 20° short of full extension and the patient was unable to extend it beyond this point. The peripheral pulses were normal. An ascending phlebogram showed complete occlusion of the deep veins of the leg from the calf to the common femoral. The long saphenous vein was patent and there was a partly occlusive thrombus in the external iliac vein. Pulmonary perfusion scan and chest *x*-ray were both normal. There was no evidence of bone or joint pathology on *x*-ray of the knee. The patient was treated with intravenous heparin and leg elevation. The swelling and erythema subsided rapidly and within 48 hours he was able to extend the knee fully.

Case 2. A 46-year-old man was admitted with a five-day history of severe pain and swelling in the left leg. He noted that within hours of the onset of pain in the calf he was unable fully to extend the knee. He was treated at home with analgesics but when he failed to improve after a few days he was referred to hospital. On examination his pulse was 90/min and temperature 37°C. The left leg was a dusky colour and was very swollen. The knee was flexed to 25° short of full extension and the patient was unable to extend it beyond this. The long and short saphenous veins were both palpably thrombosed. Abdominal and rectal examintion revealed no abnormality and there was no lymphadenopathy. Ascending phlebography showed extensive venous occlusion involving all the deep veins of the lower limb, the long and short saphenous vein, and the external iliac vein. Chest *x*-ray showed a raised left hemidiaphragm and pleural effusion. Pulmonary perfusion scan showed a segmental perfusion defect in the left mid-zone compatible with a diagnosis of pulmonary embolism. There was no evidence of bone or joint pathology on *x*-ray examination of the knee. The patient was treated with intravenous heparin infusion and leg elevation. The swelling subsided gradually during the next five days, but it was two weeks before the patient could fully extend the knee.

Loss of full extension of the knee, coming on rapidly, is almost always due to disease in the joint or adjacent bone. Both patients were treated initially with analgesics because it was thought to be due to a musculoskeletal condition. In both cases subsequent phlebography showed extensive occlusive thrombosis of the calf, popliteal, and femoral veins. In addition, case 2 had occlusive thrombosis of both saphenous veins and the iliac vein. The reason for loss of full extension is not certain, but it may be due to reflex muscle spasm. It is of interest that in both cases the limitation of extension was present when the patient was first seen by his doctor.

The clinical signs and tests used in the diagnosis of deep vein thrombosis have recently been reviewed.[1] Muscle spasm and irritability of the adductors and gastrocnemii have been recorded,[2] as has sciatic neuritis from pelvic vein thrombosis.[3] This sign would appear to be a rare presentation of extensive deep vein thrombosis with or without superficial thrombosis and should be kept in mind in the presence of a flexed knee of recent onset where no joint lesion is found.—We are, etc.,

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3 Aird, I., *Companion in Surgical Studies*, 2nd edn. Edinburgh and London, Livingstone, 1957.

Preserving Urinary Glucose for Estimation

SIR,—When involved in research in the endocrine unit at this hospital some years ago we became interested in the preservation of glucose in urine for estimation. Urine samples from patients received in the laboratory both within 12 hours of voiding and, in error, after several days of waiting at ambient temperatures, occasionally showed obvious infection. We doubted the efficacy of toluene, which was then in vogue, as a preserving agent. It also interfered with pipetting. We therefore studied the problem as follows.

Three specimens of urine, two turbid and one clear, known to be infected (*Escherichia coli*, *Proteus mirabilis*, and *P. rettgeri*) were obtained. After each sample was shown to give a negative Clinistix reaction dextrose was added to a concentration of about 100 mg/100 ml urine. Each sample was then split into four 50-ml parts, which were treated as follows: (1) control urine without added preservative; (2) urine + solid benzoic acid; (3) urine + a complete or incomplete layer of toluene; and (4) urine + chlorhexidine diacetate (Hibitane) to produce a final concentration of 1/2,000. The twelve urine samples were left on the bench and serial estimations of reducing substance ("glucose") were carried out on each over the next few weeks, using the Harding modification of the Schaffer-Hartmann method.[1]

It was found that in all but the three chlorhexidine-preserved samples the concentration of urinary glucose fell, reaching about 30 mg/100 ml within 24-72 hours. Chlorhexidine did not appear to interfere with the method of glucose estimation but sometimes precipitated in the presence of urinary phosphates. This did not appear to affect its antibacterial properties. Since this study, wards have been supplied with bottles for urinary glucose collections containing 0·25 g of chlorhexidine diacetate powder per expected 500 ml urine.

We concluded that neither toluene nor benzoic acid was effective as a preservative of glucose in infected urine kept at room temperatures, but chlorhexidine was satisfactory. However, information reaching us has indicated that toluene continues to be used as an agent to prevent degradation of glucose and other substrates such as uric acid. It may be useful to remind people that this cannot be relied upon.

We thank Dr. Dutton of the bacteriology department, Hammersmith Hospital, for advice and Dr. D. G. Higgins, of Imperial Chemical Industries Ltd., for the generous supply of chlorhexidine diacetate used in this study.

—We are, etc.,

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Prevention of Deep Vein Thrombosis

SIR,—Deep venous thrombosis with consequent massive pulmonary embolism still remains a potent cause of postoperative death. It is alleged that the majority of these thromboses are initiated during the operative period, when Virchow's triad of venous stasis, damage to the vein wall, and increased blood coagulability are maximal. The leg is the most usual site for the thrombosis of the deep veins to occur, and there is evidence that the soleal plexus of veins is especially liable as both venous stasis and damage to the vein wall will occur if the calves of the unconscious patient are compressed by the weight of his legs during a lengthy operation. Thrombosis in this site may propagate into the larger leg veins and eventually result in massive embolism. In order to reduce the risk of soleal vein thrombosis it is current practice to place a rubber pad under both Achilles tendons while the patient is on the operating table, so relieving pressure on the calves.

However, after major surgery these days the patient is removed from the operating table on to a firm bed, the pad under his heels is removed, and he is transferred to the recovery room, where he may remain for some hours before regaining full consciousness and even then be reluctant to move his legs because of pain. During this period his calves are pressed firmly down on to the mattress not only by the weight of his legs but also by the weight of the bedclothes.

It is currently my practice to insert a plastic foam pad 18×3×3 in (46×7·6×7·6 cm) in size beneath the Achilles tendons of patients who are deeply unconscious or who have had major surgery on transfer from the operating table to their bed. This pad travels with the patient to the ward and remains in place until he kicks it away. The pad can be cut from a sheet of plastic foam, may be used many times, and costs about 10p. Its use is being adopted by other surgeons in this hospital and also in the intensive care unit. Much has been written recently on prophylactic measures against deep vein thrombosis, but little consideration has been given to the immediate postoperative period and I hope the simple, and possibly effective, technique advocated here will merit adoption.—I am, etc.,

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Autoimmunity in Childhood Pulmonary Alveolar Proteinosis

SIR,—In your leading article on pulmonary alveolar proteinosis (12 February 1972, p. 395) you drew attention to Gough's[1] suggestion, based on animal experiments, that the production of the characteristic alveolar reaction found in this disease might depend on an altered state of immunity. Clinical support for this theory was shown by Colón *et al.*[2] who, in a review of 23 cases of childhood alveolar proteinosis, found a high incidence of lymphopenia, low immunoglobulin levels, and thymic alymphoplasia. I wish to present the 24th case of alveolar proteinosis in childhood, in which there was not only a low serum IgA level, but also