New Physical Sign of Acute Deep Vein Thrombosis

Str-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 to be slightly swollen. When he returned a few days later the swelling of the left leg was more marked, and the swelling confined to the thigh was present. The flexion of the knee was to 20° short of full extension and the patient was unable to extend his knee to that 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 iliac vein. There was no evidence of venous thrombosis in the external iliac vein. Pulmonary perfusion scan and venography showed that there was no evidence of bone or joint pathology on x-ray of the knee. The patient was treated with intravenous heparin and physiotherapy. 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 pain and swelling in his left leg. He noted that within hours of the onset of pain in the calf he was unable fully to extend the knee. The leg was massively swollen 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 purple color. 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 examination 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. The leg was massively swollen. The appearance of 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 and physiotherapy. 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 that this might be due to a muscular-skeletal condition. In both cases subsequent phlebography showed extensive occlusive thrombosis of the calf, popliteal, and femoral veins. Both were surgical cases. In neither case was there any occlusive thrombosis of both saphenous veins and the iliac veins. 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 loss 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. Muscle spasm, the irritability of the adductors and gastrocnemii have been recorded, as has sciatic neuritis from pelvic vein thrombosis. This sign would appear to be a rare presentation of deep vein thrombosis with or without superficial thrombophlebitis and should be kept in mind. The presence of a flexed knee of recent onset where no joint lesion is found. We are, etc.

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Preserving Urinary Glucose for Estimation

Str.—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 an obvious reaction of a solution of toluene, which was then in vogue, as a preserving agent. It also interfered with pipetting. We therefore studied the procedure 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 Clinitest reaction deoxyribose 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 (Hibitan) 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 of the next few weeks, using the Harding modification of the Schaffer-Hartmann method.

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 estimation used, but sometimes precipitated in the presence of urinary phosphates. This did not appear to affect the sensitivity of the test. The results of this study, which 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 tolune nor benzoic acid was effective as a preservative of glucose in infected urine kept at room temperatures, but chlorhexidine was satisfactory. However, information received us has indicated that tolune 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.

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Autoimmunity in Childhood Pulmonary Alveolar Proteinosis

Str.—In your leading article on pulmonary alveolar proteinosis (12 February 1972, p. 395) you drew attention to Gough's suggestion, based on animal experiments, that the production of the characteristic alveolar proteinosis might be found in a patient with lung disease on an altered state of immunity. Clinical support for this theory was shown by Colom et al. who, in a review of 23 cases of childhood alveolar proteinosis, found a high incidence of lymphomas, low immunoglobulin levels, and thymic aplasia. 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...