figures published in table I at their face value and calculating χ^2 for heparin versus stimulation, omitting the redundant control group, the sum is 4·924, which where D.F.=1 gives a value for P which only just reaches the 5% mark, a very different result from that published.

Staying with table I, I calculated χ^2 for the major D.V.T. only. Again comparing heparin directly with stimulation, this time the sum was 3·021, which gives a value for P of between 10% and 5% where D.F.=1. A value of that level is not regarded as significant. Therefore the published results for major D.V.T. could easily be due to chance alone.

My most serious criticism is that 22 patients were withdrawn from the trial without it being stated to which of the three groups they originally belonged. If they all, or most, belonged to the same group it would make nonsense of the trial. Furthermore, Sevitt's rule was broken. Four patients were withdrawn because they died. Not only was their original group left unrecorded but there was no necropsy to prove they had not died of a pulmonary embolus.

Lastly, four more patients were withdrawn because of haemorrhages great enough to need blood transfusions. Mr. Rosenberg and his colleagues brush these complications aside as due to chance. If they had calculated χ^2 for this complication on the basis of four haemorrhages in 273+4 patients they would have found a sum of 9·999, which where D.F.=2 gives a value for P of less than 1%. In other words, the chance that the haemorrhage was directly due to the heparin is 99%.

For 10 years I have practised electrical stimulation of the calf and no major pulmonary embolism has taken place. The trial reported by Mr. Rosenberg and his colleagues has not converted me to heparin.—I am, etc.,

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Gianotti-Crosti Syndrome and Viral Infection

SIR,—I have unfortunately only just seen the letter on this subject from Drs. Sarah C. F. Rogers and J. H. Connolly (30 November, 1974, p. 529). I should like to clarify and refute their remarks.

I feel that the six patients examined by Drs. Rogers and Connolly were affected by papular vesicular acrolocated syndrome; otherwise it would have been possible to detect the hepatitis B antigen, which is always present in papular acrodermatitis of childhood and can be detected in the child's serum for at least two months by immunodiffusion, electrosyneresis, or radioimmunodiffusion or with the electron microscope. Moreover, hepatic function tests and liver biopsy carried out during the dermatitis phase demonstrate the presence of an acute hepatitis. This has been found in all our 48 cases examined in the past few years in collaboration with Clinica Medica III of the University of Milan and in other cases in various countries.

Dermatologically it is easy to recognize papular acrodermatitis of childhood and to distinguish it from papular vesicular acrolocated syndrome. In papular acrodermatitis of childhood the cutaneous eruption (fig. 1)

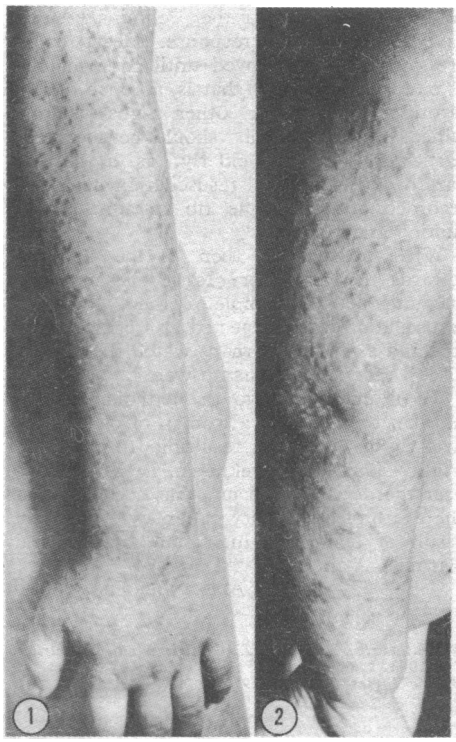

FIG. 1.—Papular acrodermatitis of childhood.
FIG. 2.—Papular vesicular acrolocated syndrome.

is monomorphic, lenticular, flat, erythematopapular, and non-itching. In papular vesicular acrolocated syndrome it generally presents itching, pinhead-sized, vesicular-like papules, sometimes non-coalescent and regularly distributed (fig. 2) on the face, buttocks, and limbs, though in some cases they may be unevenly distributed and coalesce in patches, especially on the limbs. We do not know whether these varying clinical features correspond to different causes or whether they are only different cutaneous reactions. We named this condition "syndrome" because while the clinical patterns are quite similar, there may be many aetiological agents. Indeed, similar cutaneous eruptions may be observed in infectious mononucleosis, vaccinid (post-vaccinal rash appearing 10-15 days after the primary inoculation), or Schönlein-Henoch purpura. For these reasons we now classify under papular vesicular acrolocated syndrome only these papular or vesicular-like and sometimes purpuric acrolocated eruptions of unknown origin.—I am, etc.,

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Practolol-induced Pleurisy and Constrictive Pericarditis

SIR,—A recent report from this hospital (12 April, p. 68) described a patient with a subacute bowel obstruction due to fibrinous peritonitis, which was part of a polyserositis attributed to practolol. We have now seen another patient who developed polyserositis during practolol treatment, but whose symptoms suggested pleural and pericardial involvement.

The patient, aged 65 years, was commenced on practolol 100 mg three times a day in November 1971 because of intractable angina. In December 1973 he developed a generalized psoriasiform rash which continued thereafter. In May 1974 he complained of deteriorating vision; marked bilatera superficial punctate keratitis was present in both eyes. Despite treatment perforation of his right cornea occurred.

In July 1974 he first complained of difficulty in breathing, though examination and chest *x*-ray were normal. His dyspnoea progressed, wheezing developed, and practolol was stopped in October 1974 as it was thought that it might be contributing to his bronchospasm. However, his respiratory symptoms did not improve and he was admitted to Worcester Royal Infirmary in March 1975.

On examination he was a thin, dyspnoeic man with no cyanosis. Jugular venous pressure was elevated 3 cm. No pulsus paradoxus was noted. The prominent finding was a poor chest expansion with dullness to percussion at both bases. PO_2 was 8·9 kPa (67 mm Hg) and respiratory alkalosis was present. Antinuclear factor negative. E.S.R. 31 mm in 1 hour. On chest *x*-ray the heart was not enlarged, but extensive bilateral pleural thickening was present. Despite bronchodilators, antibiotics, and large doses of steroids his respiratory condition worsened and terminally he developed a subacute bowel obstruction and died.

At necropsy the prominent findings were: adhesive pericarditis with a normal heart but quite severe coronary arteriosclerosis; adhesive pleurisy over both lungs and in the interlobar fissures—histological examination showed no evidence of fibrosing alveolitis; dense adhesive peritonitis causing multiple subacute obstructions in the small and large bowel.

Post-mortem examination in this man, as in our previous case, demonstrated that serosal changes were more widespread and severe than clinical examination suggested. In spite of the presence of dense peritoneal adhesions he did not complain of any abdominal symptoms until the final 36 hours of the illness. However, the pleural changes and constrictive pericarditis were thought to be the main cause of his symptoms and death.

We had previously expressed the hope that withdrawal of the drug might result in resolution of the polyserositis because the adhesions were thin and filmy in our first case. Unfortunately, despite cessation of treatment for seven months, this man's serositis not only failed to resolve but actually advanced, as demonstrated by clinical and radiological findings.

We should also emphasize that withdrawal of practolol should be gradual because of the dangers of abrupt termination of any beta-blocker. Alternatively, another preparation could be substituted.—We are, etc.,

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γ-Glutamyl Transpeptidase in Myotonic Dystrophy

SIR,—With reference to the recent article by Dr. P. J. Martin and others (4 January, p. 17) we have determined the serum activity of γ-glutamyl transpeptidase (γ-GT) in myotonic dystrophy (M.D.) on the assumption that the metabolic defect of this disease may lie in the γ-glutamyl cycle. γ-GT activity was estimated by the kinetic method of Szasz[1] at 25°C in 12 patients (six males and six females) aged 14-43 (mean 30·9± 8·65) years and 12 healthy controls (six males and six females) aged 15-50 (mean 28±9·5).

The mean γ-GT activity was greatly increased in the patients with M.D. (24·97± 16·94 U/l) compared with the controls (8·83±1·87 U/l) (P=0·001).

γ-GT is associated with cell membranes of different tissues, particularly in cells in