catheter, (b) determination of platelet counts on all samples and (c) the inclusion of a group receiving placebo.

Twelve male volunteers received subcutaneous injections of either water or adrenaline tartrate (1 : 1000 solution, 1 ml/70 kg body weight). Blood samples (20 ml) were collected into a citric acid-sodium citrate-dextrose buffer (ACD; 5 ml) for platelet MAO assay and 5 ml into disodium ethylenediamine tetraacetate (EDTA) for platelet count determination. Samples were taken, from an indwelling catheter in the ante-cubital vein, immediately prior to injection and at 20, 40, 60 and 80 min post-injection. Platelet MAO activity was assayed by the method of Robinson, Lovenberg, Keiser & Sjoerdsma (1968) with benzylamine as substrate at a concentration of 1 M for all samples, and at the much lower concentration of 2.1×10^{-5}M used by Gentil *et al.* (1975) for the first two samples only. Platelet counts were determined with a Technicon Autocounter. The results of the platelet MAO assays are presented in Table 1.

There was a small but significant increase ($P < 0.05$, paired '*t*' test) in platelet MAO activity of the group receiving adrenaline in the 20 min post-injection samples only, with benzylamine as substrate at 1 mM concentration. There was a small but not significant increase in the activity of platelet MAO, in both groups of subjects, with benzylamine at a concentration of 2.1×10^{-5} M. Platelet counts were significantly increased in the 20 min ($P < 0.001$) 40 min ($P < 0.05$) and 60 min ($P < 0.05$) only in the group receiving adrenaline. As suggested by Gentil *et al.* (1975) the efflux of a different population of platelets from the spleen may account for the increase in platelet MAO activity observed in the 20 min post-injection samples.

We were unable, therefore, to fully confirm the findings of Gentil and his co-workers. The small changes in platelet MAO activity attributable to 'stress', at least in as far as it is mimicked by injections of adrenaline seem unlikely to have an important bearing on the current controversy over the activity of the enzyme in schizophrenia.

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Role of lymphocytes in accumulation of fibrin in rabbit skin homografts

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Indomethacin-like drugs relieve joint pain and stiffness in rheumatoid arthritis but do not reduce the swelling. Further insight into this problem has been obtained by investigating the mechanisms influencing the fibrin content of homografts, as suggested by Jasani, Lewis & Tweed (1974).

Fibrin accumulation was studied by administering iodinated ^{125}I-labelled human fibrinogen (100 μCi in 1 ml water, i.v.) to New Zealand White rabbits bearing six homografts (from Norfolk donors) of the right hind limb and an equal number of autografts on the left leg. One homograft and its corresponding autograft were removed daily from day four. ^{125}I-labelled fibrinogen was given on day five and a blood sample taken daily thereafter. Grafts were kept frozen (−20°C), homogenized (Jasani, 1973) and the water-insoluble radioactivity determined and expressed as a fraction of blood radioactivity according to Colvin & Dvorak (1975), thus minimizing inter-animal variations.

Figure 1a shows that the insoluble ^{125}I-labelled fibrinogen content, although similar at first in both types of graft, began to increase significantly in the homografts following the appearance of cyanosis.

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Figure 1a shows that the insoluble ^{125}I-fibrinogen content, although similar at first in both types of graft,

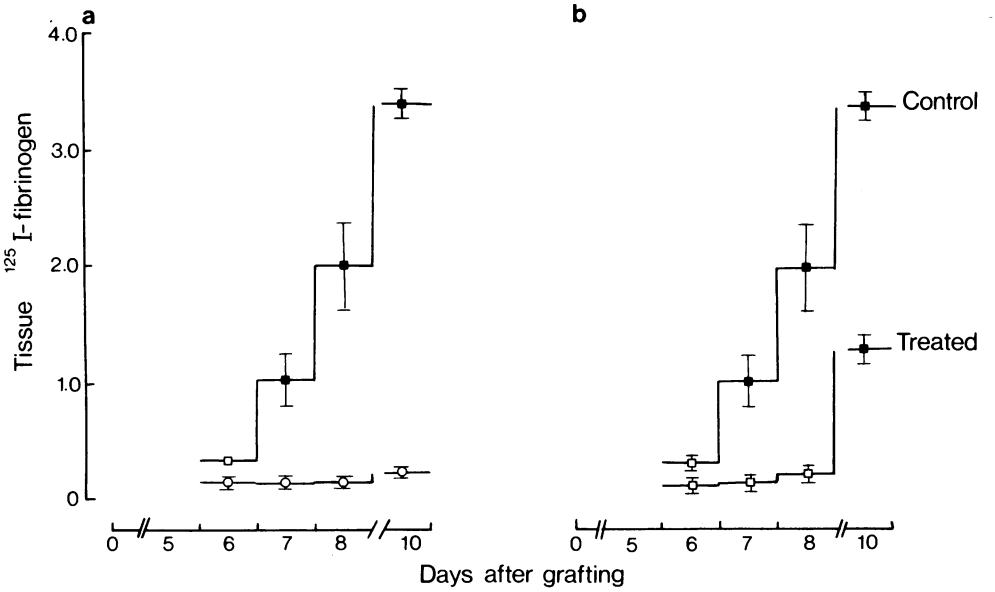

Figure 1 Relationship between fibrin accumulation and the entry of circulating sensitized lymphocytes (SLs) as judged by the appearance of cyanosis in rabbit skin homografts.

Water insoluble ^{125}I-labelled fibrinogen in (a) homografts (□) and autografts (○) and (b) control and cyclophosphamide (750 mg/animal) treated homografts. Full squares (■) denote that the homografts are cyanosed. Results are mean ± s.e. for three experiments.

began to increase significantly in the homografts following the appearance of cyanosis.

As cyanosis is initiated by the entry of circulating sensitized lymphocytes (Jasani, 1975), it is suggested that some factor released from these cells is responsible for the accumulation of fibrin in the homografts. This view is strengthened by the results of Figure 1b which show that the immunosuppressive agent cyclophosphamide, known to delay the development of circulating sensitized lymphocytes and swelling, prevented the insoluble fibrinogen content of homografts from increasing above the values found in the autografts, as long as cyanosis did not appear.

As a working hypothesis, failure of indomethacin-like drugs to arrest rheumatoid swelling may now be viewed as reflecting their inability to prevent the entry of circulating sensitized lymphocytes into the joint.

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