ear) over a period of 2 weeks. The doses were increased from 0·25 ml. to 2·00 ml. of venom solution. The injected rabbits were able to survive amounts at least as great as five times the lethal dose of venom solution. Precipitin assays in test-tubes and on agar plates were positive for the whole venom as well as for the proteolytic and unidentified electrophoretic fractions.

The venom did not show any activity for coagulant or anticoagulant factor, histamine, histamine-liberating substances or any ileum contracting agent, acetylcholine, acetylcholinesterase, acetylcholinesterase inhibitor, phospholipase *A*, ATP phosphohydrolase, ribonuclease, NAD nucleosidase, D- and L-amino-acid oxidase.

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Failure of Methylcellulose to alter the Growth of Walker Tumour 256 in Rats

Methylcellulose is a long-chained methyl ether of cellulose. Recent reports have associated regression of the transplantable Murphy–Sturm lymphosarcoma in rats with treatment by intraperitoneal injections of methylcellulose[1–4]. In the present investigation the same type of rats and treatment were used with the Walker tumour 256. As will be shown, no beneficial effect was noted in the parameters studied, namely, size of the tumour and longevity of the rats.

Adult female Sprague–Dawley rats averaging 140 g were divided into three groups of twenty each; size and age were similar. The methylcellulose was prepared as a 2·5 per cent aqueous suspension of 400 CPS methylcellulose. Intraperitoneal injections of 2 c.c. of the suspension were made three times per week (Monday, Wednesday, Friday)[1], starting 10 days before the subcutaneous transplantation of the tumour as a cell-suspension. Injections of methylcellulose continued so long as the animals remained alive in Group *A*, and for only two injections after tumour transplantation in group *B*. Group *C* received tumour but no methylcellulose. The tumour was measured every second day and the product of the longest and shortest dimension recorded.

Fig. 1 summarizes the findings; each point represents the average of the size of the tumours in the group.

In our laboratory, the Sprague–Dawley host has shown some degree of natural resistance to the Murphy–Sturm lymphosarcoma, but not to the Walker tumour. We have observed 10–20 per cent complete spontaneous regression of the Murphy–Sturm tumour, in various experiments; however, complete spontaneous regression of the Walker tumour has not been seen. We feel that methylcellulose may enhance a natural resistance already present in the host and which is sufficient in the case of the Murphy–Sturm lymphosarcoma to tip the balance in favour of the host. However, in the case of the Walker tumour any natural resistance which may exist is not strong enough to be expressed spontaneously and cannot be potentiated to that point by methylcellulose. The mechanism of action of methylcellulose is not clear, but, the Murphy–Sturm tumour being a homograft, an enhancement of the homograft rejection mechanism may be involved. The Walker tumour 256 is also a homograft.

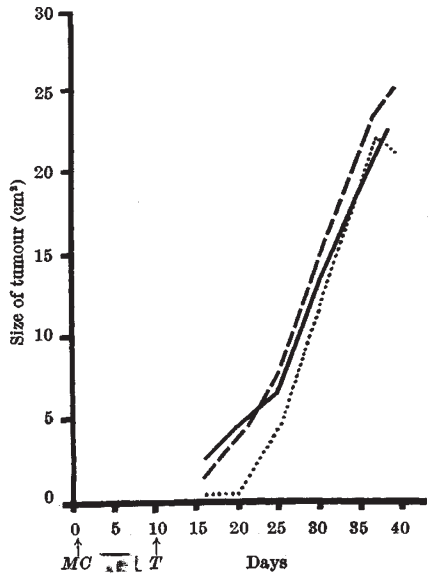

Fig. 1. Comparison of average size of tumours in methylcellulose-treated and non-treated groups. ——, Continuous treatment with methylcellulose (Group *A*). - - - -, Treatment with methylcellulose stopped after two injections post tumour transplantation (Group *B*). . . . ., Control, no methylcellulose (Group *C*). Arrow *MC* indicates start of methylcellulose injections. Arrow *T* indicates transplantation of Walker tumour

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HAEMATOLOGY

Influence of the Erythroid Activity of the Bone Marrow on the Plasma Disappearance of Injected Erythropoietin in Dogs

The finding that plasma erythropoietin titre in patients with an aplastic or hypoplastic bone marrow was higher than in patients with haemolytic anaemias despite similar haemoglobin concentration led Stohlman to postulate that the plasma erythropoietin level is related not only to the severity of the stimulus but also to the degree of erythroid activity of the bone marrow[1]. Jacobson *et al.*[2] and Hammond *et al.*[3] have suggested that erythropoietin may be consumed or degraded by actively proliferating erythrocytic elements.

This report presents data on the rate of disappearance of erythropoietin from plasma of normal, erythropoietically stimulated (acute hypoxia) and erythropoietically de-