tumours failed to respond because prolactin was not abolished; (5) 2/2 oestrogen dependent tumours got worse on oestrogens; (6) 2/2 androgen dependent tumours got worse on testosterone; (7) 10/11 *in vitro* independent tumours failed to respond. The *in vitro* test thus gave a correct prediction in 37 of 40 patients.

CHANGES IN RESPONSE TO CHEMOTHERAPEUTIC AGENTS DURING THE LIFE HISTORY OF MONOLAYER CULTURES OF A MOUSE TUMOUR CELL LINE. P. R. TWENTYMAN and N. M. BLEEHEN. Academic Department of Radiotherapy, Middlesex Hospital Medical School, London.

At the previous meeting of the Association, we described how EMT6 mouse tumour cells become less sensitive to bleomycin as they pass from exponential growth into plateau phase. This result was the opposite of that reported by other workers using Chinese hamster cells. Detailed investigation of the proliferation kinetics of our EMT6 cell line has revealed that the plateau phase may be subdivided into early plateau (with a pulse labelling index of 25% and considerable cell loss) and late plateau (with a labelling index of $<5\%$ and little cell loss). Sensitivity to bleomycin is indeed reduced during early plateau (compared with exponentially growing cells), but in late plateau the sensitivity becomes greater than that in exponential cells. Sensitivity to a number of other chemotherapeutic agents has also been investigated in cultures of various ages.

THE EFFECT OF WHOLE BODY HYPERTHERMIA IN ADVANCED CANCER. R. T. PETTIGREW, C. M. LUDGATE and A. N. SMITH. Department of Anaesthetics and Department of Clinical Surgery, Western General Hospital and University of Edinburgh.

The anaesthetized patient is immersed in molten wax at 50°C. This reverses the normal physiological processes of heat loss. A 5°C rise is achieved in one hour and maintained for 3–4 h. Fifty-five patients with advanced cancer have been heated to 41·8°C and the tumour response assessed by criteria which include relief of pain, weight gain, serial biopsy changes and evidence of tumour regression. One group (45 patients) was treated by hyperthermia alone; sarcomata and gastrointestinal tract tumours were the most responsive (8 in 11); an intermediate group, skin and lung tumours, was less so (6 in 16); a third (mainly genito-urinary), the least (0 in 14). The addition of chemotherapy to hyperthermia in 10 patients raised the proportion regressing from 11 in 23 (48%) to 7 in 10 (70%). Occasional complications were mild superficial burns, tracheitis, ventricular fibrillation and disseminated intravascular coagulation.

ANALYSIS OF THE ANTIMETASTATIC ACTION OF THE ANTIMITOTIC AGENT ICRF 159. K. HELLMANN, S. E. JAMES and A. J. SALSBURY. Imperial Cancer Research Fund, London.

ICRF 159 inhibits metastases from the spontaneously metastasizing Lewis lung carcinoma (3LL) without markedly impeding the growth of the primary implant. It has previously been proposed that this antimetastic action of ICRF 159 is due to the inhibition of malignant cell release from the primary tumour consequent upon normalization of the tumour blood vessels by the drug. Lung "metastases" due to intravenous injection of 3LL cells should therefore be unaffected by ICRF 159 administration. This was not, however, found to be the case.

When primary tumours were excised up to 6 days following implantation, secondary growths were not apparent in the lungs at 21 days. Treatment of primary tumours for the first 6 days by 30 mg/kg ICRF 159, at a time therefore when no circulating malignant cells would have been present, produced an almost complete inhibition of metastases. Thus, under these conditions the antimetastatic action cannot be ascribed to an effect of the drug on 3LL cells in the blood stream.

A COMPARISON OF THE CELL KILLING IN THE MOUSE AFTER EXPOSURE TO FTORAFUR AND TO 5-FLUOROURACIL. L. M. VAN PUTTEN, L. K. J. KRAM-IDSENGA and M. PIJPERS-DE BRUIN. Radiobiological Institute TNO, Rijswijk, Holland.

Ftorafur (N-1-(furanidyl)-5 fluorouracil) was compared with 5-fluorouracil (5-FU) in mice. The LD_{50} and the slopes of the dose-effect curves for killing of L1210 leukaemia