Winship, 1950) and cerebral perfusion (Woodhall *et al*, 1959), it was not felt to be justified to give Endoxana by either of these two techniques unless it was found effective when given systemically. We, therefore, tried its systemic use in 14 cases of cerebral tumour. An earlier report (Simon, 1959) on the use of Endoxana in cerebral neoplasms stated that the drug 'was invariably well tolerated' and that if it was given 'following surgical excision, the prognosis for survival is better'. Our results do not substantiate either of these statements. The only patient surviving longer than eight months without any signs of increased intracranial pressure had had a decompressive lobectomy for an astrocytoma (grade II malignancy), and the result is comparable to the usual progress of a glioma of this grade of malignancy surgically treated. The necropsied specimens of the tumour tissue have not shown any significant change which could be attributed to the Endoxana therapy. The drug is, however, safer than other alkylating agents in that the tendency to leucopenia is more easily reversible.

SUMMARY

Endoxana, a nitrogen mustard derivative, was given systemically in 14 cases of intracerebral tumour; 12 of these tumours were gliomas and two cerebral metastases.

All the patients have died from the continued growth of the tumour. Only one patient survived longer than 12 months, and in this case the tumour was histologically of low malignancy.

Side-effects included nausea, vomiting, marked alopecia, iron-deficiency anaemia, and severe leucopenia; the last disappeared when the drug was discontinued. In one case, transient obstructive jaundice occurred.

In this series the systemic administration of Endoxana had no therapeutic effect.

We are grateful to Ward Blenkinsop Ltd. for supplying Endoxana.

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