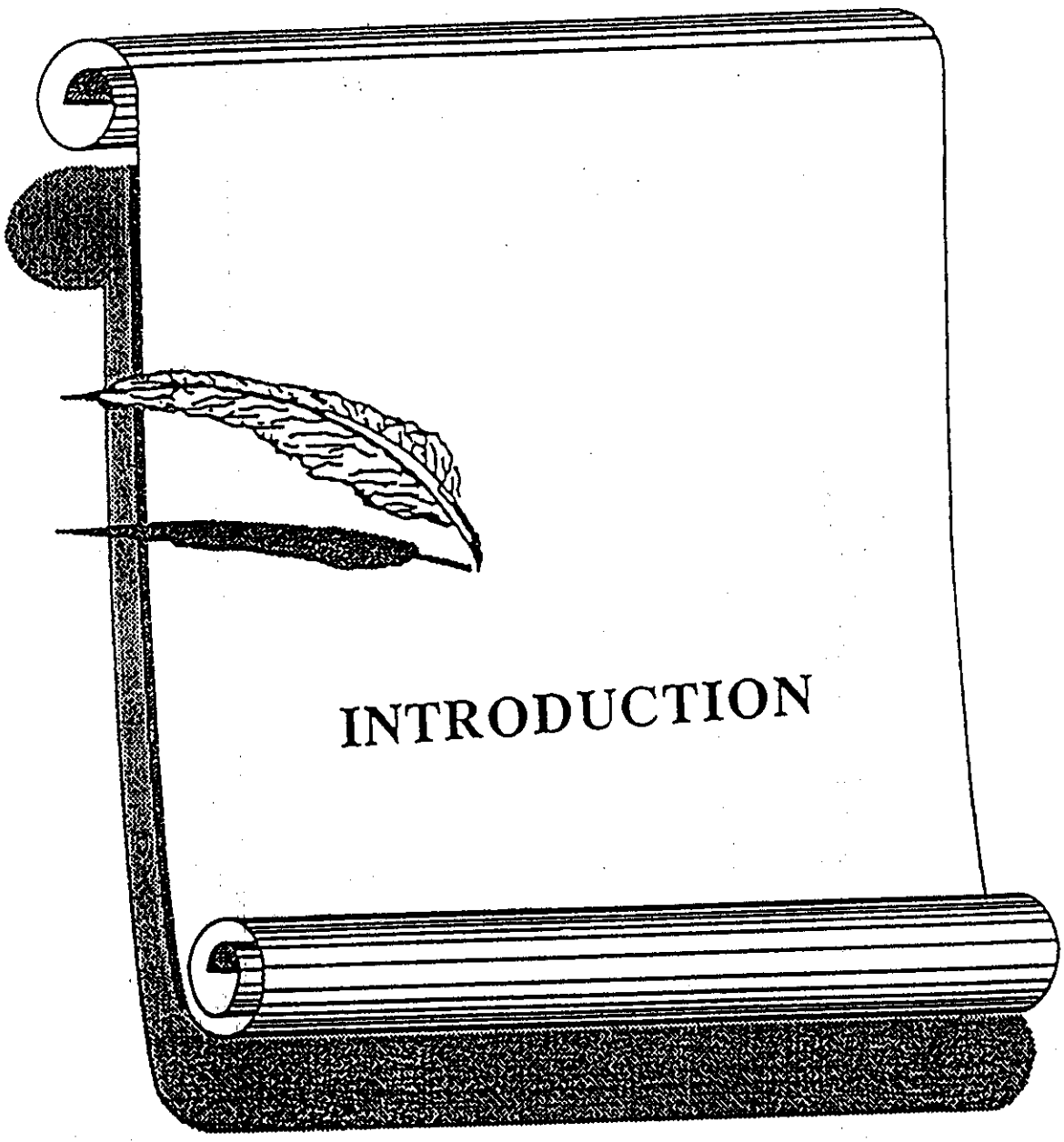

INTRODUCTION

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Glioma is one of the major brain neoplasia that occurs in the human (Hideyuki Saya et al., 1986).

Malignant gliomas account for 40% to 50% of the primary brain tumors diagnosed each year in the United States (Kimmel et al., 1987).

All intracranial gliomas are clinically malignant regardless of the degree of histological differentiation or anaplasia, because without appropriate treatment they are fatal to the host (Hoshino, 1984).

Even with aggressive therapy, the median survival time after diagnosis is only approximately 1 year (Isabelle et al., 1989).

Most gliomas are classified as glioblastoma multiforme, anaplastic astrocytoma of varying degree, or well differentiated tumors, such as fibrillary astrocytoma, oligodendroglioma or ependymoma (Hoshino, 1984). The tumors in this latter group are separated by vague diagnostic borderlines (Russell and Rubinstein, 1977).

Some clinicians assume that well differentiated or low grade gliomas are benign because they usually grow very slowly and patients survive longer than those with anaplastic gliomas or glioblastoma multiforme (Hoshino, 1984).

The degree and type of cellular abnormalities have long been known to influence the natural history of brain tumors (Tooth, 1912) and many grading systems have been devised based on this observation (Gulotta, 1981) although

widely used in diagnosis, none of these systems has gained general acceptance or has been unequivocally correlated with prognosis (Cohaden et al., 1985).

Because the prognosis for brain tumor patients depends largely on the rate of tumor growth (Isabelle et al., 1989), the need to measure the proliferative activity of tumors quantitatively in order to supplement the histopathological diagnosis has prompted the development of methods for estimating the cell cycle time, the duration of S phase and the growth fraction (Hoshino, 1984).

So, cell kinetic studies have been vigorously pursued for many years in an attempt to better understand tumor growth and to improve therapy (Hoshino, 1981).

Conventional histology has revealed lymphocytic invasion of many malignant tumors (including intracranial tumors); as an expression of the immunological response (Hitchcock and Morris, 1988).

Furthermore, the establishment of a correlation between degree of lymphocytic infiltration and prognosis, as judged by period of survival (Brooks et al., 1978), has prompted several investigators to try to define the properties of these cells in an effort to better understand the intricacies of glioma-immune interactions (Farmer et al., 1989). This approach has been greatly facilitated by the availability of monoclonal antibodies (MAb's) that can distinguish subsets of lymphoid cells mediating specific immune functions (Hitchcock and Morris, 1988).

In recent years, a possible role of natural killer (NK) cells in rejection of tumor has generated much interest (Herberman and Ortaldo, 1981).