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ABSTRACT BOOK



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Somatostatin in cancer therapy.

We are employing Somatostatin, associated with dl- α -bromocriptin, melatonin and cyclophosphamide in breast-, lung-, stomach-, bowel cancers, in Hodgkin and non-Hodgkin lymphomas, in malignant histiocytosis, in bone- and smooth muscle sarcomas, in neuroblastomas, in melanomas. The dosages have been moderate (250 μ g i.m./day, to the highest degree); apart from some temporary and tolerated trouble, no drawback has been met with, even after several years of unbroken therapy. It is difficult or even impossible to state the role of Somatostatin, in a compound treatment of several remedies, but we have become convinced that Somatostatin plays an important although not yet exactly defined role. Somatostatin has proved useless in the last, quickly progressive stages of malignancies.

In neuroblastoma Somatostatin has been poorly tolerated. In some case of cerebral metastasis from breast cancer a clinically complete recovery has been reached since more than four years. The goodness of the results includes both the possibility of patients continuing their normal social and family life, and a significantly longer survival. Urinary bladder irritation may lead to temporary discontinuance of cyclophosphamide.

Good results have been obtained in those cases of chronic lymphatic leukemia which cannot any longer be treated with common antileukemic remedies owing to the dangerously low blood platelet count. Malignant histiocytosis has been equally well treated by a continued treatment with Somatostatin.

The treatment of cancer with Somatostatin, melatonin, prolactin inhibition blocking agents and ACTH is probably the most physiological among the proposed and applied official therapies for cancer. It demonstrated no toxicity, is well tolerated, allows the common duties and performances of everyday social life to be undertaken. The combined remedies converge in blocking the increment of adenohipophyseal hormones that impinge on tissue growth, and probably also act on their receptive peripheral action sites, without provoking any rebound action or adaptation or counterbalancing reaction.