negative. In such a situation E_{in_1} and E_{in_2} would summate so that $E_{out} = (R_f/R_s)(E_{in_1} + E_{in_2})$.

Instead of recording a slow wave of 500 μv generated from one source, the potentials from the frequency-coupled sources would add, giving Vanderwolf's gargantuan (1000 μv) rhythmic slow pattern that cannot be blocked by any one specific neurotransmitter antagonist or blocking agent (*4*, *7*).

Of particular relevance are the recent findings of Bird and Aghajanian (*9*) showing that microiontophoretic application of scopolamine and quinuclidinyl benzilate (QNB) blocked the excitation of hippocampal pyramidal cells by acetylcholine in the rat. We have shown that the dorsal movement-related theta generator is muscarinic (*1*), and Winson (*5*) provides evidence that the ventral theta generator may be nicotinic.

We suggest that Vanderwolf consider changing his electrode configuration so that he can record from one neurochemically distinct system at a time.

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Immunosurveillance of Naturally Occurring Feline Leukemia

Essex *et al.* (*1*) have advanced epidemiological data "supporting the concept of immunosurveillance in an outbred species of mammal," the cat.

That their results "clearly indicate that a low FOCMA [feline oncornavirus-associated cell membrane antigen] antibody titer in the presence of FeLV [feline leukemia virus] positivity is a risk indicator for leukemia, while high antibody titers are associated with resistance to leukemia development" seems established by their data. I also agree that the data are consistent with the immunological surveillance concept; however, the data are also consistent with alternative concepts which, in my opinion, need more emphasis.

The prospective study does not, of course, rigorously rule out the possibility that the tumor was the cause rather than the result of low antibody titers since it is impossible to know how long before overt disease the pathological process may have started. Perhaps more likely, however, is the hypothesis that the relation between antibody level and neoplasia need not be etiological, regardless of which came first; rather, both may be the result of an underlying immunological abnormality. The scenario could be something like the following: Some cats, for unknown reasons, do not respond to virus with normal levels of antibody to virus. Leukocytes of the antibody-producing mechanism are therefore stimulated by the feedback controls that regulate antibody production. This high level of stimulation eventually results in neoplasia in the stimulated leukocytes (*2*). The mechanism would, in principle, be analogous to the production of ovarian tumors by excess gonadotrophin in the rodent ovary transplanted into the portal circulation of an ovariectomized recipient (*3*).

The above possibility makes it extremely difficult, in my opinion, to draw conclusions concerning the immunological surveillance concept from studies of tumors derived from cells of the immune mechanism itself.

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