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MEDICAL MEMORANDA

Phenytoin Treatment of Thalamic Pain

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Treatment of the painful, burning dysaesthesias which occasionally occur after thalamic infarction has been a particularly vexing problem. Administration of a variety of psychotropic and analgesic drugs has yielded variable but generally impersistent and ineffective results. The improvement noted in some cases after stereotaxic lesions in thalamic nuclei or other parts of the sensory pathway has often been transient. The following two cases illustrate successful treatment of thalamic pain with phenytoin.

Case Reports

Case 1.—A 56-year-old man with hypertensive cardiovascular disease abruptly developed tingling paraesthesias in his left fingers which progressed over 24 hours to involve the arm and leg. In the next few weeks he developed progressively severe, persistent, painful dysaesthesias of the left hand and foot aggravated by touch. Examination of these areas showed a modest rise in pain threshold. Strong painful dysaesthesias occurred with tactile or pin stimuli. During his second week of treatment with 300 mg of phenytoin daily there was a progressive decrease of the dysaesthesias culminating in their disappearance. Phenytoin was discontinued one month later because of toxicity. Over the next 10 days the dysaesthesias progressively recurred as the toxic signs diminished. On 150 mg of phenytoin daily he noted no toxicity and a tolerable reduction of dysaesthesias.

Case 2.—A 51-year-old man with hypertensive and atherosclerotic cardiovascular disease awoke with persistent tingling of the left face, hand, and foot. During the next few days the foot became normal. Increasingly severe burning sensations developed in the hand and face. Examination showed an increased threshold to pin

and touch stimuli on the face, and forearm. Tactile and pin stimulation evoked burning, dysaesthetic pain. Progressive improvement began on the sixth day of phenytoin treatment. One year later the dysaesthesias recurred when phenytoin was stopped. Reinstitution of treatment again caused alleviation of pain.

Comment

The abrupt onset and progression of symptoms, the lack of E.E.G. or brain scan abnormalities, and the presence of chronic hypertension are consistent with the diagnosis of a small lacunar thalamic infarction (Fisher, 1965; Caplan and Young, 1970).

Effective relief of severe, persistent pain by phenytoin has been reported in cases of trigeminal neuralgia, sphenopalatine neuralgia, tabes, and peripheral neuropathy (Green, 1961; Boshes and Arieff, 1968; Meyer, *et al.*, 1970). Experimentally, phenytoin stabilizes electrolyte transfer across neuronal membranes and increases the neuronal threshold to repetitive stimulation. Hyperexcitability is lessened by a reduction of both post-tetanic facilitation and the duration of the after-discharge occurring after supra-threshold stimulation (Domino, 1964; Schmidt and Wilder, 1968). Cortical responses to single repetitive thalamic stimulation are also reduced by phenytoin (Herman and Bignall, 1967). In light of these experimental results it is reasonable to postulate that phenytoin may be effective in thalamic pain by reducing the spread of abnormally excessive excitatory discharges resulting from the thalamic lesion.

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