Tinnitus

Halmos, Molnar and Kormos, HNO-Praxis (1982), 2567 compared the effects of PHT and carbamazepine (CBZ) in the treatment of 138 patients with tinnitus and sensorineural hearing loss. Seventy-two of the patients received PHT, 100 mg t.i.d., and seventy-five received CBZ, starting with 200 mg b.i.d. and gradually increasing to 400 mg t.i.d. The severity of tinnitus diminished in thirty-eight of the seventy-two patients treated with PHT. Their hearing loss improved by 5 to 25 decibels. Classified according to etiology, thirty-two of fifty tinnitus patients with eighth-nerve lesions improved, as did two of three cases with otologic defects. Four of fifteen patients with presbycusis improved. There was no improvement in the four patients who developed tinnitus after head injury. No adverse effects were observed in the PHT group. Improvement was observed in only twelve of the sixty-six carbamazepine-treated patients. It had to be stopped in nine patients because of side effects. The authors state that they found PHT superior to CBZ for tinnitus. PHT had the additional advantage of being better tolerated. (See also Ref. 2500.)


Wertzberger, <Chirurgie de> (1991), 3414 reports the results of oral phenytoin treatment of eighty cases of low-perception hearing accompanied by tinnitus. More than half the patients (forty-two) responded favorably to PHT treatment (300 - 400 mg/day). Twenty-eight patients did not improve, and ten worsened. The author comments that phenytoin was well tolerated and that many other treatments have failed to be effective for tinnitus.


Tatemoto, Nippon Jibiinkoka Gakkai Kaiho (1990), 3415 successfully treated 100 patients with tinnitus with intravenous phenytoin. More than 70% of these patients responded well to phenytoin treatment. Only 5% had mild side effects. The author also evaluated the effects of intravenous phenytoin (20-70 mg/kg) on auditory system electrical activity evoked by sound stimulation. Phenytoin decreased the latency and amplitude of each waveform peak of the averaged brainstem evoked response (ABR), with the latency of waves III and IV most affected. PHT also decreased the N1 component of the cochlear nerve action potential, but did not affect cochlear microphonics (CM) or the endocochlear potential (EP), except when perilymphatic perfusion of PHT was used. The author concludes that intravenous PHT acts predominantly on the upper brain stem and also the cochlear nucleus and nerve to suppress abnormal hyperexcitability.