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# Letters to the Editor

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## Letter N°. 1

During the past several years I have encountered a few patients who had tinnitus that I thought might be a side effect of the administration of Ritalin (methylphenidate hydrochloride). As I recall, the patients were pre-adolescents ranging in age from nine to twelve years. There were both males and females that have presented with this finding and none of them had associated hearing loss or an apparent cause for the tinnitus. In none of the cases was the tinnitus so severe as to require cessation of the Ritalin that was being administered to treat attention deficit disorder. Although there have been a few reports of Tourette's syndrome developing in association with administration of Ritalin and there certainly are other side effects, I have not seen in writing or heard anecdotal reports of children developing tinnitus as a side effect or adverse reaction related to therapy with Ritalin.

My review of the Physicians' Desk Reference has not identified tinnitus associated with the administration of Ritalin. Ciba Pharmaceutical, the manufacturer of Ritalin, reviewed records of adverse drug reactions and found mention of only one report suggesting that there might be an association between the administration of Ritalin and the development of tinnitus. Certainly there is a possibility that the children who remember having had tinnitus while taking Ritalin may also have been taking other medications or, as many teenagers do, they might even have been using small earphones with portable radios, cassette, or CD players that could have caused noise induced trauma with resulting tinnitus.

I wonder if other physicians have ever encountered young patients with tinnitus who are taking Ritalin and if anyone has considered tinnitus as a possible side effect of Ritalin.

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## Letter N°. 2

I did review Dr. Claussen's work on vestibular evoked response with a great deal of interest. The area of brain mapping is an area that I have followed with great interest largely as it pertains to brain perfusion but not so much to evoked response. The specific application he uses here is somewhat new to me, and I will be talking to one of my neurology colleagues to see whether the methodology is comparable. My work, as you know, has been primarily in the animal laboratory where we used vestibular evoked response as a measure to check for pharmacologic changes in the vestibular system. In our studies the response was evoked by a rapid pressure pulse on the horizontal canal which should have discharged all sensitive units in a matter of 5 to 10 msec. If he is using a rotational impulsive stimulus, unless he uses extremely giant motors in my experience it has taken close to a second to produce a significant impulsive stimulus. A second is a very long time for invoking electrical responses of a sense organ such as a vestibular labyrinth. The other problem I have with rotational stimulation is that although one labyrinth is stimulated, the opposite is inhibited and I'm not quite sure how this is going to result in coherent electrical changes in the brain stem. Also the actual duration of the response in his article is somewhat long even if we consider cortical responses. Most of the information I have on electrical activity originating in the inner ear and going to cortex would suggest a somewhat shorter duration than is shown in Dr. Claussen's article.

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## Reply from C.-F. Claussen, M.D.

I appreciated the remarks of Prof. Dr. Konrad from December 10th, 1996, concerning our article on vestibular evoked responses in the *International Tinnitus Journal*. Starting with visually or acoustically evoked responses, it is known that the stimuli can be of very short duration without endangering the patient. Prof. Dr. Konrad reports that he has produced in animal investigations evoked vestibular responses to shortlasting pressure impulses of 5 to 10 msec. upon the inner ear receptors. If we would

transfer this test design to living humans, sitting on a rotatory chair, we would need, as he writes, extremely giant motors. I agree that this test design theoretically should be of great importance for vestibular brainstem evoked potentials. However, the unusually high accelerative forces endanger the brain vessels and the brain tissue as well as several other structures in humans. Therefore, we have refrained from this type of experimental design.

We have applied the vestibular late or cortically evoked potentials, as is performed in audiometry, for the acoustic late evoked potentials (ALEP) to human volunteers as well as to patients.

We speculated that if a human, for instance, in cupulometry could perceive a rotational threshold acceleration of  $0.15^\circ/\text{sec}^2$ , we should at least be able to find reproducible cortical responses to stimuli 10 to 100 times stronger. We have experimentally proven this hypothesis.

In patients with complete inner ear losses such typical

responses are not present, which proves our hypothesis. Concerning the latency of the cortically evoked vestibular responses, we find the first typical waves between 70 and 100 msec. In visually evoked potentials the main complex is called "wave P100". Also the typical wave complex in the cortically evoked acoustic potentials exhibits this latency duration.

Relating to psychoacoustics, acoustic as well as visual late evoked potentials demonstrate a later appearing typical wave complex around 300 msec after the onset of the stimulus. In this time window we also find the most prominent DC-shift complex of wave III/IV of the cortically evoked vestibular responses.

Thus the latencies of the vestibular evoked potentials of the late evoked or cortical type are in accordance with findings from other sensorially evoked cortical potentials.

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