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Otosclerosis is a multifactorial disease. There is a number of theories on the pathogenesis of this disease during the last two decades. According to existed recent data on the pathogenesis of the disease, otosclerosis is considered as a severe inner ear disease leading to deafness in the majority of cases.

The presence and evolution of otosclerosis depends on genetic, infective, immunological, inflammatory factors, as well as the impaired bone metabolism underlying the pathogenesis of

the disease. It is likely that genetic predisposition associated with morbilli infection may lead to bone resorption in the stapes and cochlea followed by spongiosis, fibrosis and sclerosis. It has been suggested that immunological mechanisms play a central role in the development of the disease.

Some authors consider otosclerosis as autoimmune disorder based on the presence of several autoantibodies. Apart from classical diagnostic methods, such as audiometry and X-ray, novel radiological techniques including CT, MRI or radionuclide scan are helpful in the localization of otosclerosis **[1]**.

Computed tomographic (CT) scanning with slices of 1 mm or more has not been sufficient to demonstrate otosclerotic foci in most cases to date. High-resolution CT scans are a valid tool that can be used to confirm, localize, and determine the size of clinically suspected otosclerotic foci **[2].**

In many cases the hearing loss that follows otosclerosis can be corrected surgically. But in many other cases surgical treatment is not always successful or feasible.. Sometimes, surgery is contraindicated or it is unsuccessful **[1]**.

Sodium fluoride has now been used for 45 years in an effort to slow down or arrest sensorineural hearing nerve deterioration in patients with stapedial otosclerosis or after stapedectomy, as well as in patients with pure cochlear otosclerosis. Extensive clinical

experience in thousands of patients with this therapy has demonstrated its value in arresting previously progressive sensorineural hearing loss.

For a long time there were those who objected to this therapy on the basis that it had not been adequately proven by double-blind, placebo-controlled studies. They have been answered by Bretlau's study in Denmark and Fisch's from Switzerland; both investigators confirmed on small groups the value of sodium fluoride by double-blind, placebo-controlled studies. Extensive research by Professor Petrovic of Strasbourg while at our tissue culture laboratory at Northwestern University demonstrated the action of sodium fluoride on bone.

A nicely designed study with radioactive strontium by Linthicum, House, and Althaus demonstrated its value in promoting maturation of a spongiotic focus**[3].**

Sodium Fluoride (NaF) is the only medication so far clinically available with a bone formation stimulating property, through its peculiar mitogenic dose-dependent action on the osteoblast cell line. Bone strength is commensurate to bone mass, and in a condition with fragility fractures, like osteoporosis, it seems logical to restore bone mass without weakening bone strength. However, as with any active drug NaF therapy requires adhesion to elementary rules if drawbacks are to be prevented. A first mandatory rule is not to prescribe NaF without calcium supplementation, if bone loss at the appendicular skeleton is to be avoided; to prevent this, the availability of sodium fluoride , containing the fluoride and calcium salts (calcium carbonate as oyster shells) in the same preparation has enhanced the compliance to calcium supplementation**[4].**

Florigkel , the currently available food supplement, has showed better absorption than any enteric coated tablets and only slightly less than sodium fluoride alone. For active cochlear

otospongiosis one- two capsules three times a day should be prescribed **[5, 6].** **Florigkel** contains also Magnesium citrate, Vitamin D3 and vitamin K2.

Florigkel promotes maturation of otospongiotic lesions, and thus slows down or arrests progression in sensorineural hearing loss **[3, 6].**

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