Abstract

Cellular Patterns and Irregularities in Human Perilabyrinthine Bone

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The pathological bone remodeling around the inner ear known as otosclerosis affects around ½- 1% of the population. Although common and thoroughly studied, the pathogenesis of the disease remains unknown. Based on contemporary concepts of bone biology, dynamic studies of temporal bone turnover anticipated the presence of inner ear osteoprotegerin (OPG). OPG is a potent inhibitor of bone remodeling and the bone immediately surrounding the inner ear, the otic capsule, is rarely replenished. Consequently, degenerative changes accumulate in a pattern similar to the spatial distribution of otosclerosis. Furthermore, clusters of dead osteocytes, so called cellular voids, have been identified in the otic capsule, and may represent a starting point for otosclerotic remodeling. 8 This thesis further evolves the theory that otosclerotic remodeling is a result of the unique bony dynamics around the inner ear. By combining undecalcified temporal bone histology and unbiased stereology the spatial distribution of microcracks and cellular voids in the human temporal bone is studied and compared to that previously established for otosclerosis. The thesis is divided into three sections. In the first section a method for quantifying microcrack surface density is developed and applied. In the second section, cellular voids are characterized and quantified. In the third and final section, the cellular compositions of cellular voids and otosclerotic lesions are described and compared.