Hyperparathyroidism

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Parathyroid hormone is among the major regulators of calcium homeostasis and bone turnover; alterations of parathyroid hormone secretion are involved in the pathogenesis of several metabolic bone diseases. Remarkably, two physicians from Vienna (Jakob Erdheim and Felix Mandl) made significant contributions to the pathophysiology and treatment of hyperparathyroidism. The American endocrinologist Fuller Albright, who—among numerous other discoveries in calcium metabolism—recognized the role of the menopause in the pathophysiology of osteoporosis, in a presidential address distinguished an “Austro-German pathological approach” and an “American physiological approach” to the elucidation of hyperparathyroidism [1]. The existence of “glandulae parathyreoideae” in humans was first described in 1880 by Ivar Sandström from Sweden. In 1891 von Recklinghausen reported on an autopsy case with osteitis fibrosa cystica. In 1906 the Viennese pathologist Jakob Erdheim induced tetany in rats by the removal of the parathyroid glands and consequently discovered the role of these glands in the metabolism of calcium. One year later in an autopsy series of patients that died with osteomalacia, Erdheim for the first time recognized compensatory parathyroid hypertrophy/hyperplasia. In the late 1920s Fuller Albright spent 1 year in Vienna with Jakob Erdheim; Albright was very much impressed by Erdheim and stated “that he knew more about disease processes than any living man” [1].

A further milestone in the history of hyperparathyroidism was accomplished in 1925. At the Vienna General Hospital the surgeon Felix Mandl for the first time in the world removed a parathyroid tumor in a patient with osteitis fibrosa cystica. After the operation, the patient rapidly improved; in X-rays bone recalcification could be demonstrated. For the second time in the world the diagnosis of (primary) hyperparathyroidism was made at the Massachusetts General Hospital (“American approach”); unfortunately, because of the ectopic location of the parathyroid adenoma, the patient underwent six unsuccessful parathyroid explorations. During the seventh operation the adenoma was removed from a mediastinal location; the patient died several weeks later from post-surgical complications [1].

In the first paper of this thematic issue of the *Wiener Medizinische Wochenschrift* Katharina Kerschan-Schindl gives an overview on the regulation of calcium homeostasis and bone turnover in hyperparathyroidism. In addition to established markers of bone formation and bone resorption also osteoprotegerin, RANKL, FGF-23 and Klotho are discussed. In the next paper, Rudolf Gasser concisely reviews clinical aspects of primary hyperparathyroidism. Quite in contrast to the historic descriptions of osteitis fibrosa cystica the vast majority of patients with primary hyperparathyroidism in Western countries present in an asymptomatic disease stage and are identified by routine blood chemistry or bone mineral density testing. Patients with chronic renal failure frequently present with significant alterations of calcium, phosphorus, vitamin D, and bone metabolism; in the final manuscript of this thematic issue Daniel Cejka describes the pathophysiology, diagnosis, and treatment of renal bone disease. In recent years a pathophysiologic link between renal osteodystrophy and vascular calcification has been recognized; renal bone disease thus has a very broad clinical impact.

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Conflict of interest

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Reference

1. Albright F. A page out of the history of hyperparathyroidism. *J Clin Endocrinol.* 1948;8:637–57.