Journal of Clinical Densitometry: Assessment & Management of Musculoskeletal Health, vol. ■, no. ■, 1–19, 2017

1094-6950/■:1–19/$36.00
https://doi.org/10.1016/j.jocd.2017.10.041

Review Article

Proceedings of the 2017 Santa Fe Bone Symposium: Insights and Emerging Concepts in the Management of Osteoporosis

E. Michael Lewiecki,[1,] John P. Bilezikian,[2] John J. Carey,[3] Richard M. Dell,[4] Catherine M. Gordon,[5] Steven T. Harris,[6] Michael R. McClung,[7] Paul D. Miller,[8] and Michael Rosenblatt[9]*

[1]New Mexico Clinical Research & Osteoporosis Center, Albuquerque, NM, USA; [2]Columbia University College of Physicians and Surgeons, New York, NY, USA; [3]National University of Ireland, Galway, Ireland; [4]Rapid Registry Deployment, Cypress, CA, USA; [5]Cincinnati Children's Hospital Medical Center and University of Cincinnati College of Medicine, Cincinnati, OH, USA; [6]University of California San Francisco, San Francisco, CA, USA; [7]Oregon Osteoporosis Center, Portland, OR, USA; [8]University of Colorado Health Sciences Center, Denver, CO, USA; and [9]Flagship Pioneering, Cambridge, MA, USA

Abstract

The 18th Annual Santa Fe Bone Symposium was held on August 4–5, 2017, in Santa Fe, New Mexico, USA. The symposium convenes health-care providers and clinical researchers to present and discuss clinical applications of recent advances in research of skeletal diseases. The program includes lectures, oral presentations by endocrinology fellows, case-based panel discussions, and breakout sessions on topics of interest, with emphasis on participation and interaction of all participants. Topics included the evaluation and treatment of adult survivors with pediatric bone diseases, risk assessment and management of atypical femur fractures, nonpharmacologic strategies in the care of osteoporosis, and skeletal effects of parathyroid hormone with opportunities for therapeutic intervention. Management of skeletal complications of rheumatic diseases was discussed. Insights into sequential and combined use of antiresorptive agents were presented. Individualization of patient treatment decisions when clinical practice guidelines may not be applicable was covered. Challenges and opportunities with osteoporosis drug development were discussed. There was an update on progress of Bone Health TeleECHO (Bone Health Extension for Community Healthcare Outcomes),

Received 10/23/17; Accepted 10/31/17.
Funding sources: Manuscript preparation and editing was supported by the Osteoporosis Foundation of New Mexico, Albuquerque, New Mexico, USA.
Conflicts of interest: E. Michael Lewiecki receives research grant from Amgen. He is consultant at Amgen, Radius, Alexion, Shire, and Ultragenyx, and speaker at Alexion, Shire, and Radius. John P. Bilezikian is consultant at Amgen, Merck, Shire, Radius, Ultragenyx, and Regeneron. John J. Carey is speaker at Pfizer Canada and Pfizer Brazil. He receives travel grant from Amgen. Richard M. Dell has nothing to disclose. Catherine M. Gordon is consultant at Janssen Pharmaceuticals. Steven T. Harris is consultant at Merck and Radius. He is also speaker at Shire, Lilly, and Gilead. Michael R. McClung is consultant at Amgen and Radius. Paul D. Miller receives research grants from Amgen, Alexion, Merck, Merck Serrano, Boehringer Ingelheim, Novartis, Novo Nordisk, Immunodiagnostics, Roche Diagnostics, National Bone Health Alliance, Lilly, Radius, Regeneron, and Ultragenyx. He is also consultant at Amgen, Lilly, Merck, Alexion, AgNovos, Radius, Roche, and Ultragenyx, and serves as data safety committees at Allergan and Grünenthal Group. Michael Rosenblatt receives salary, shares, and stock options, and is former CMO from Merck. He also receives shares and stock options from Radius; stipend as board member at Ferring USA; and salary as CMO from Flagship Ventures.
*Address correspondence to: E. Michael Lewiecki, MD, New Mexico Clinical Research & Osteoporosis Center, 300 Oak St. NE, Albuquerque, NM 87106. E-mail: mlewiecki@gmail.com

a teleconferencing strategy for sharing knowledge and expanding capacity to deliver best-practice skeletal health care.

Key Words: Anabolic; atypical femur fracture; Bone Health ECHO; parathyroid; pediatric.

Introduction

The Santa Fe Bone Symposium is an annual meeting of scientists, researchers, physicians, and other health-care professionals to share information about the latest advances in basic bone science and clinical research. All discussions are focused on clinical implications and relevance for patient care. The 18th Santa Bone Symposium was held on August 4–5, 2017, in Santa Fe, New Mexico, USA. This is a highly interactive meeting with numerous opportunities for participants to collaborate with colleagues. Plenary presentations are followed by lively discussions of a broad range of issues. There is a session with oral presentations of abstracts by endocrinology fellows, as well as case-based open-topic panel discussions. Progress of Bone Health TeleECHO (Extension for Community Healthcare Outcomes; http://echo.unm.edu/) program was also presented.

Highlights of past Santa Fe Bone Symposia have been presented in peer-reviewed journals *(1–11)*, monographs in print and electronic formats *(12–16)*, online slide presentations *(17–19)*, and audiovisual webcasts. This is a summary of clinical insights from presentations and discussions at the 18th Santa Fe Bone Symposium.

Skeletal Effects of Parathyroid Hormone and Opportunities for Therapeutic Intervention

John P. Bilezikian, MD, PhD(Hon)

Disorders of parathyroid function are simply categorized as parathyroid gland hyperfunction leading to hyperparathyroidism or hypofunction leading to hypoparathyroidism. This presentation focuses on new insights into both disorders and how therapeutic opportunities for the use of parathyroid hormone (PTH) evolved into clinically important options. The premise is that understanding the disorders of PTH excess or deficiency has led to therapeutic opportunities in the management of disorders of calcium homeostasis.

Primary Hyperparathyroidism

Primary hyperparathyroidism (PHPT) is a disorder of excessive secretion of PTH from one or more of the 4 parathyroid glands. Hypercalcemia and elevated or inappropriately normal PTH levels are biochemical hallmarks of the disease. PHPT is used to be characterized as a disease of "bones, stones and groans" in the days of Fuller Albright, but over the past 50 years, 2 other clinical phenotypes have been recognized. The most widely recognized form of PHPT is described as asymptomatic PHPT *(20)*. It is discovered in countries where biochemical screening testing is routine and in which the serum calcium is an integral component. These patients do not have the classical clinical features of PHPT. The third and most recent presentation of PHPT is seen in individuals whose serum calcium is virtually always normal but in whom the PTH is consistently elevated, in the absence of a secondary cause *(21)*. Although these 3 clinical presentations of PHPT evolved chronologically over time, with symptomatic disease being the first, it is now appreciated that all 3 forms of PHPT are seen concurrently in the world today. Several factors determine which of these disorders is more likely to be present in a given region of the world. If severe vitamin D deficiency is present, the disease is likely to be symptomatic. In countries where routine biochemical screening is employed, the asymptomatic form predominates. In metabolic bone diseases units where PTH is commonly measured, even if the serum calcium is normal, normocalcemic PHPT is seen. The point is that although chronology defined these 3 presentations of PHPT, over a period of 75 years, these 3 forms are concurrent throughout the world today.

Advances in noninvasive imaging technology, such as high-resolution peripheral computed tomography (HRpQCT), have given insights that were not readily discerned by routine skeletal imaging such as dual-energy X-ray absorptiometry (DXA). By DXA, a characteristic densitometric pattern is recognized in which the lumbar spine, a site of mostly trabecular bone, is relatively well preserved, whereas the distal 1/3 radius, a site of mostly cortical bone, is preferentially reduced *(22)*. Because DXA is a powerful risk factor for fracture, this observation gave rise to the notion that the nonvertebral skeleton would be at greater risk of fracture, whereas the vertebral spine would not. This observation, however, was at odds with epidemiologic observations that demonstrated as early as the late 1990s that in PHPT, both nonvertebral and vertebral fracture risk are increased *(23,24)*. By HRpQCT and trabecular bone score, it is now evident that in PHPT microstructural abnormalities can be demonstrated in trabecular bone and cortical bone *(25–29)*. These and other advances have led to revised guidelines for the management of PHPT *(30)*.

PTH for the Treatment of Osteoporosis

Although it seemed paradoxical that a hormone known for its devastating potential to destroy the skeleton could be used as a therapeutic agent to build bone, the historical roots for this concept are not new but extend to the days of Fuller Albright. However, the insight that low-dose, intermittent administration of PTH could lead to an osteoanabolic effect in rats *(31)* paved the way to the development of teriparatide [PTH *(1–34)*] as an osteoanabolic therapy for osteoporosis *(32)*. The time course of the