

Differential Effects of Endocrine Dysfunction on the Axial and the Appendicular Skeleton

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ABSTRACT In 100 patients with various types of endocrine dysfunction, we measured bone mineral density (BMD) at the midradius (>95% cortical bone) and distal radius (75% cortical and 25% trabecular bone) by single photon absorptiometry and at the lumbar spine (>66% trabecular bone) using the new technique of dual photon absorptiometry. BMD in each endocrine disorder deviated in at least one site from the sex-specific age regression of 187 normal subjects. For patients with primary hyperparathyroidism, hypercortisolism, and hyperthyroidism this deviation was negative (suggesting bone loss), whereas for patients with secondary hyperparathyroidism due to chronic renal failure, acromegaly, and postsurgical hypoparathyroidism it was positive (suggesting bone gain). When all six states of endocrine dysfunction were compared concomitantly by multivariate analysis of variance, the profile of the changes in BMD differed significantly ($P < 0.001$), indicating a nonuniform response of bone to the various hormonal alterations. When values for BMD at each of the three scanning sites were compared, the midradius and distal radius did not differ significantly; either of the radius measurements, however, differed significantly ($P < 0.001$) from the lumbar spine. Thus, the BMD of the axial skeleton cannot be reliably predicted from measurements made in the appendicular skeleton. We conclude that the effects of endocrine dysfunction on bone density are complex and are both disease and site specific.

INTRODUCTION

Parathyroid hormone, cortisol, thyroxine, and growth hormone affect bone remodeling and, thereby, can

alter skeletal mass. Alterations in bone mineral density (BMD)¹ resulting from states of hormonal dysfunction have previously been studied only in the appendicular skeleton. In a few instances, total body calcium has been measured by neutron-activation analysis; these results, in general, have agreed with those of appendicular measurements. Both appendicular and total body calcium measurements, however, are relatively insensitive to changes in trabecular bone, especially when these changes occur in the axial skeleton. The appendicular skeleton is composed predominantly of cortical bone, whereas the axial skeleton contains large amounts of trabecular bone (1). Moreover, because the whole skeleton contains 85% cortical but only 15% trabecular bone (2), changes in trabecular bone would have a relatively small effect on total body calcium measurements. If changes in density of cortical and trabecular bone and changes in circulating hormones occurred *pari passu* in all regions of the skeleton, BMD measurements of the appendicular skeleton would suffice. There are several reasons for believing, however, that BMD measurements of the axial skeleton will be required to characterize definitively the skeletal effects of states of endocrine dysfunction. First, the metabolic activity of the two bone types differs considerably: cortical bone has a turnover rate of only 3%/yr (3), whereas trabecular bone has a turnover rate of up to 30%/yr (4). Second, autopsy studies have shown good correlation for BMD among sites containing large amounts of cortical bone but a poorer correlation between BMD of these sites and that of the vertebrae (5). Third, in most metabolic bone diseases, fractures occur almost exclusively at skeletal sites that contain substantial amounts of trabecular bone.

These considerations led us to examine the possi-

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¹Abbreviations used in this paper: BMC, bone mineral content; BMD, bone mineral density; iPTH, immunoreactive parathyroid hormone.

Differential Effects Of Antidepressants

M Walker



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Table of Contents Differential Effects Of Antideprebants

1. Understanding the eBook Differential Effects Of Antideprebants
 - The Rise of Digital Reading Differential Effects Of Antideprebants
 - Advantages of eBooks Over Traditional Books
2. Identifying Differential Effects Of Antideprebants
 - Exploring Different Genres
 - Considering Fiction vs. Non-Fiction
 - Determining Your Reading Goals
3. Choosing the Right eBook Platform
 - Popular eBook Platforms
 - Features to Look for in an Differential Effects Of Antideprebants
 - User-Friendly Interface
4. Exploring eBook Recommendations from Differential Effects Of Antideprebants
 - Personalized Recommendations
 - Differential Effects Of Antideprebants User Reviews and Ratings
 - Differential Effects Of Antideprebants and Bestseller Lists

5. Accessing Differential Effects Of Antidepressants Free and Paid eBooks
 - Differential Effects Of Antidepressants Public Domain eBooks
 - Differential Effects Of Antidepressants eBook Subscription Services
 - Differential Effects Of Antidepressants Budget-Friendly Options
6. Navigating Differential Effects Of Antidepressants eBook Formats
 - ePub, PDF, MOBI, and More
 - Differential Effects Of Antidepressants Compatibility with Devices
 - Differential Effects Of Antidepressants Enhanced eBook Features
7. Enhancing Your Reading Experience
 - Adjustable Fonts and Text Sizes of Differential Effects Of Antidepressants
 - Highlighting and Note-Taking Differential Effects Of Antidepressants
 - Interactive Elements Differential Effects Of Antidepressants
8. Staying Engaged with Differential Effects Of Antidepressants
 - Joining Online Reading Communities
 - Participating in Virtual Book Clubs
 - Following Authors and Publishers Differential Effects Of Antidepressants
9. Balancing eBooks and Physical Books Differential Effects Of Antidepressants
 - Benefits of a Digital Library
 - Creating a Diverse Reading Collection Differential Effects Of Antidepressants
10. Overcoming Reading Challenges
 - Dealing with Digital Eye Strain
 - Minimizing Distractions
 - Managing Screen Time
11. Cultivating a Reading Routine Differential Effects Of Antidepressants
 - Setting Reading Goals Differential Effects Of Antidepressants
 - Carving Out Dedicated Reading Time
12. Sourcing Reliable Information of Differential Effects Of Antidepressants
 - Fact-Checking eBook Content of Differential Effects Of Antidepressants
 - Distinguishing Credible Sources
13. Promoting Lifelong Learning

- Utilizing eBooks for Skill Development
 - Exploring Educational eBooks
14. Embracing eBook Trends
- Integration of Multimedia Elements
 - Interactive and Gamified eBooks

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