Orthopaedic Care OF THE Geriatric Patient

Edited by

THOMAS P. SCULCO, M.D.

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FOREWORD

Diseases and disabilities of bones, muscles, and joints are the common problems in the elderly, accounting for more than one third of significant disabilities. To give some insight to the personal and socioeconomic costs, osteoporosis has an incredible annual cost: 200,000 hip fractures that account for almost 50,000 deaths a year, millions of days spent in hospitals, and billions of dollars in cost.

Genuine progress in dealing with musculoskeletal disease will depend on basic science as well as application. We must better understand bone and cartilage cell structure and function as well as bone deposition and resorption. We must learn more about the nature of inflammatory destruction of joint cartilage.

We see that there is no shortage of research needs and opportunities for those interested in the study of the common afflictions of old age. Mechanical, degenerative, biochemical, and regenerative processes need study. Collagen appears to undergo cross-linkage with aging. Proteoglycans, compounds that account for cartilage elasticity, need to be better understood. As is so often the case, methodologic developments constitute the necessary infrastructure to uncover new findings. Sensitive radioimmunoassays and computed tomography are among such technologic steps.

The study of connective tissue cells such as fibroblasts, osteoblasts, and chondrocytes can be studied in tissue culture from young or old animals or from humans. Thus we can learn more about the synthesis of collagen, proteoglycan, and enzymes in relationship to aging. Of course, we must also study, invest, and then evaluate types of medical and surgical management of the orthopaedic and musculoskeletal problems of old age.

Fortunately, the "National Plan for Research on Aging, Toward an Independant Old Age" published in 1982 by the National Institute on Aging includes consideration of musculoskeletal aspects of aging and is a useful guide to those concerned with the discovery and application of new knowledge.

I hope and expect that this fine book edited by Thomas P. Sculco will have successive editions reflecting the continued flourishing of this important field—and so enhancing further the quality of life of older persons.

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FOREWORD

There are two common tragedies of aging that humanity presently has to live with: the aging person of sound body whose mind fails and the aging person of sound mind whose body fails. With an increasingly aging population, the incidence of each is on the rise.

Dr. Sculco and his coauthors have done a great service for our aging population by writing this book, which concerns itself with the second of these two tragedies and how to deal with it. The book brings together a team concept of how to manage musculoskeletal disability in the geriatric patient. The thrust of the text is to provide to the orthopaedist and gerontologist an overview of the spectrum of the orthopaedic problems that occur in the elderly.

The first part of the text deals with the geriatric patient in general and the changes of aging in the musculoskeletal system in particular. The second section deals with specific anatomic areas and the pathologic processes that affect these areas in the geriatric age group. The authors give details of their preferred treatment for common afflictions affecting each anatomic area. The third section deals with associated disorders of the musculoskeletal systems in the elderly and includes metabolic bone disease, fracture management (including pathologic fractures), diabetic complications, amputations, and the management of musculoskeletal complications of stroke. The fourth section deals with the nursing and physical therapy considerations in these patients, as well as long-term planning of care.

Although there have been one or two monographs on this subject, no other text exists that deals as comprehensively as this book with the musculoskeletal problems of the elderly.

Orthopaedic Care of the Geriatric Patient should be on the shelf of every physician who cares for geriatric patients, regardless of specialty, as a valuable resource of information about what to expect in the way of problems in the musculoskeletal system of the aging patient and what to do about them when they occur.

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PREFACE

One must wait until evening to see how splendid the day has been.
Sophocles

When sitting down to write a preface to a book dealing with the orthopaedic care of the geriatric patient, the editor is confronted with a series of questions that reflect back to the initial stimulus to organize such a book. Why collect this information at all? Who is geriatric? Should such a text be directed to orthopaedists, to gerontologists, or to all physicians (since most will deal with musculoskeletal problems in the elderly)?

Certainly the population is aging and with increasing life expectancy will continue to do so. With advancing years the musculoskeletal system is a common cause for seeking a physician's advice, and the entire spectrum of primary care providers may be approached for such advice. This text is designed to deal with the myriad disorders affecting the geriatric patient and to emphasize the overlap of disorders from other organ systems in these patients. The goal is to discuss all elements of the musculoskeletal system and conditions that affect it so that primary diagnosis and treatment can be instituted. More comprehensive consideration of the surgical treatment of these conditions is provided for the surgeon involved in their care.

Aging brings with it a subset of unique problems that are superimposed on musculoskeletal pathology. It is correct that this compendium should be available in one place, with emphasis on the pathophysiology and treatment, without subjecting the physician to multiple reference sources.

Considerable debate arises in the attempt to define what is meant by "geriatric." There is probably no absolutely correct explanation, and the societally imposed chronologic age of 65 years is inexact. In a medical sense "geriatric" refers to physiologic characteristics that may not manifest themselves until the eighth or ninth decade of life, or perhaps as early as the sixth. The intent of the text is to deal with these pathologic and age-related conditions, since often both components affect the chronologically geriatric patient.

The emphasis of this volume is on maintaining function and independence for the aged patient with a musculoskeletal disorder. Surgical treatment is feasible in these patients if careful perioperative supervision is maintained. The team approach is paramount; from the initial medical evaluation to anesthetic and surgical care to nursing, rehabilitation, and long-term care goals, all involved must develop a reasonable and coordinated plan of care if the geriatric patient is to improve his functional capabilities.

As the writer Tryon Edwards has stated, "Age does not depend upon years, but upon temperament and health." Indeed, proper care of the orthopaedic problems of the geriatric patient can improve both of these elements.

Many contributions to this text must be acknowledged, especially those of the authors who brought the idea to fruition. I would particularly like to thank Carol Tabatt and Isabel Gnau for the voluminous typing and correspondence they completed and Jean Carey of Mosby for her support and patience. Finally, I offer love and thanks to my wife and children, who persevered in my absence while this book was in progress.

Thomas P. Sculco

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part I
THE GERIATRIC PATIENT

chapter 1
AGING OF THE MUSCULOSKELETAL SYSTEM

Kenneth P. Scileppi

Discrimination against elderly patients as surgical candidates solely on the basis of age is a prejudice that has markedly diminished over the past several decades. Advancements in anesthetic and surgical techniques and their adaptation to suit the special problems of the elderly have reduced significantly the once prohibitive mortality of major surgery in this age group. The performance of such major feats of cardiac surgery as valve replacements and coronary artery bypasses in patients older than 75 years of age is no longer a reportable event, although not yet a routine one either. The field of orthopaedic surgery has more than shared in this process of the gradual extension of age eligibility limits for major procedures; even more impressive within the field of orthopaedics has been the development of what may be legitimately called "geriatric surgery." Techniques of joint replacement and fracture repair have been developed in the past 20 years that specifically address a spectrum of problems common only in the elderly and for whom the average patient—not the exceptional one—is in the eighth or ninth decade of life. Geriatric orthopaedics has revolutionized in such a short time the prognosis for death and disability from such conditions as arthritic and fractured hips that physicians frequently find elderly patients and their families in need of persuasive encouragement to abandon their outdated pessimistic fears of surgery.

The achievement of successful operative results in the elderly surgical patient requires not only technical skill but also an awareness of the aging changes in the human body and of the biologic variability inherent in them. Broadly stated, aging is characterized by an erosion of the physiologic reserve function existing in the organ systems of younger individuals with a consequent reduction in the older body's ability to compensate correctively for superimposed stress and to return to the original homeostatic balance. It is important to recognize, however, the tremendous variability among individuals of identical age in the degree to which such physiologic constraints of age are applicable. Superior genetic endowment at birth and a life-style of active body use can result in a fairly well defended functional reserve far into old age. Such individuals might well be superior surgical candidates compared to others a decade or two younger in whom marginal initial biologic endowment or subsequent disease, abuse, or disuse atrophy may have seriously narrowed the elastic limits of survivability. While a description of the "universal" changes of aging is important in developing an appropriate awareness of the potential problems of geriatric surgery, the application of such information to an individual patient is intelligible only as a guide to clinical evaluation of the patient; it must not become an automatic presumption because of the patient's age.

AGING OF THE NEUROMUSCULAR SYSTEM

The most conspicuous change in the human motor system with advancing old age is the decline in total body mass as a consequence of the loss in the size and/or numbers of specific types of muscle fibers.[42] Precise delineation of this effect has been made difficult by the exquisite sensitivity of muscle cells to minor fluctuations in their trophic milieu, so that the effects of "senile atrophy" are summed into other secondary effects upon muscle related to changes in the cardiovascular and endocrinologic systems. Even such psychosocial realities as the expectations of both society and the elderly themselves for participation in physical work and exercise may find their expression in reductions of muscle mass because of disuse atrophy. It appears clear, however, that independently of these other effects a significant degree of senile muscular atrophy results from the primary effect of age upon the muscle itself and/or its neural components, since some degree of atrophy with age is universal and observed even in exceptionally well conditioned individuals who remain active physical culture enthusiasts far into their senior years.

The gross pattern of senile muscle atrophy may vary somewhat between individuals inasmuch as patterns of lifelong use or disuse of particular muscle groups may have varied. In general, the muscles of the lower extremities show more significant degrees of atrophy with age than do those of the upper extremities or back.[30] Some contradictory observations have been generated regarding the effects of active muscle use in modifying the tempo of senile atrophy. Respiratory muscles kept in constant involuntary use show very little atrophy, supporting the contention that active use of a muscle perpetuates its strength.[14] Similar evidence suggests that athletic persons who remain active during their senior years maintain a differential margin of strength superiority over their peers and that their strength might be considered to be appropriate for individuals years younger.[40] In contrast, however, is the observed development of an accelerated rate of atrophy in hypertrophied muscles developed by a lifetime of occupational use in males. These individuals, who usually performed specific manual tasks during their working careers may develop lower extremity strength performance levels in their postretirement years that ultimately fall below the age-corrected norm for the general population.

Such similarities and differences as appear in studies of senile muscle atrophy may be partly caused by the heterogenous composition of muscle itself. Individual muscle fibers are composed of variable combinations of several types of fibers whose enzymatic composition and functional properties differ.[10] Type I fibers, for example, contract slowly and with a low susceptibility to fatigue because of their enrichment with oxidative enzymes, while type II fibers, in contrast, are of the fast-twitch variety containing higher levels of adenosine triphosphatase (ATPase) than found in type I fibers. The generally lower resistance to fatigue of type II fibers varies directly with their relative levels of oxidative enzyme activity, levels that are moderate in the IIA subtype but low in the IIB. Muscle fibers appear to exist in a state of active neurotrophic determination with respect to size and functional type rather than as products of terminal pathways of cellular differentiation. Experimental studies with cross-innervation and extrinsic electrical stimulation have demonstrated the capability of one fiber type to develop some of the histochemical and functional properties of another as well as the ability to hypertrophy with continued use.[8,37]

Although individual neuromotor units are uniform with respect to fiber types, different muscles may be composed of various proportions of type I, IIA, or IIB neuromotor units, with the ultimate functional characteristics of a muscle being determined by the weighted contributions of the inherent capabilities and limitations of each fiber type. The consequent effects of exercise, disuse, and aging upon muscle can be traced in some degree to their specific effects upon the individual classes of muscle fibers. Exercise, for example, is characterized not only by the commonly recognized process

of hypertrophy but also by a shift toward a greater proportion of type IIA fibers compared to type IIB.[3] Disuse atrophy is marked by a maintenance of the ratio of IIA to IIB fibers but with an overall reduction in fiber size.[16] Aging appears to result in its own differential effect upon these two fast-twitch subgroups. Type IIB fibers appear to undergo reductions in size while maintaining their numbers; type IIA fibers appear to maintain their size but undergo numerical attrition.[41] Type I fibers likewise appear to maintain their size; their numerical fate is as yet unclear. Overall there appears to be a significant reversal of the relative ratios of type II compared with type I fibers during the aging process. Cross-sectional analysis of the quadriceps muscle at the peak of adult maturity shows a 15% to 20% preponderance of type II fibers compared to type I, while by the ninth decade the summated cross-sectional area of the type I fibers at the same site is more than twice that of the type II fibers.[6]

Several ultrastructural changes within muscle fibers have been reported in aging, including patchy myofibrillar degeneration, mitochondrial enzyme depletion, and lipofuscin accumulation, but there is no evidence to date that because of these changes individual muscle fibers from elderly subjects ultimately possess any decreased capacity for the development of contractile tension compared to those of younger persons.[15,27,36] Of greater potential significance to the aging motor system than these ultrastructural changes are the observed effects of age upon the nervous system. A loss of neurons with age has been documented at various locations within the central nervous system, including the cerebellum (Purkinje's cell), brainstem (nucleus ceruleus), cortex, and hippocampus.[12] Not all areas of the brain undergo such neuronal dropouts; cell counts at a variety of other brainstem nuclear locations (e.g., the facial nucleus) reveal no appreciable changes with age. However, important age effects at other locations may nevertheless be present without an overt reduction in actual neuronal numbers; a suggestive example observed is the reduction with time of the dopamine content of the basal ganglia.[25] This muted parallel between normal aging and similar though more severe changes characteristic of Parkinson's disease demonstrates the motor system's sensitivity not only to myopathic mechanisms of aging changes but also to the aging and disease vulnerabilities of the integrative centers of the central nervous system.

Effects of aging upon the peripheral nervous system have long been implicated as being contributory to the senile changes witnessed in the motor system. It has been proposed that with age there is a reduction either in the impulse activity level of the neuron or in its release of unidentified neurotrophic substances.[17] Certain histologic and functional characteristics of aging muscle,—for example, the tendency of muscle fiber types to be found grouped together rather than dispersed throughout the muscle, with some groups in uniform states of atrophy—are so suggestive of known cases of denervation/renervation neuropathy that some degree of "functional" denervation of muscle with age has been postulated to explain them.[4] This histologic impression is reinforced by electrophysiologic evidence to suggest that there may be a reduction in the number of functioning motor neuron units in the elderly, wherein fewer surviving motor neurons activate a larger cohort of muscle fibers brought under their influence by collateral propagation of axonal endings to recruit fibers whose original source of innervation has been lost.[9,31] Degenerative changes in peripheral nerves principally involve the loss of larger, fast-conducting alpha nerve fibers, with the relative preservation of the smaller-diameter fibers. Not coincidentally it is primarily these same large-diameter, fast-conducting nerve fibers that innervate the fast-twitch type II muscle fibers whose populations likewise show the greatest effect of senescence.

The clinical effect of these combined neural and motor senescent changes is reflected in altered strength, speed, endurance, and coordinative capabilities of the older person. Reduction in the size and/or number of muscle fibers finds direct expres-

sion in the reduction of isometric strength in the elderly. This decline in strength is proportionately greater in the proximal lower extremities than in the back or upper limbs, including hand grip.[20] Although the disuse of hypertrophied muscles may lead to their accelerated atrophy to levels below that seen in the overall population (e.g., a retired manual worker), continued vigorous exercise can probably retard muscle strength decline by the equivalent of perhaps a decade; it cannot arrest the process.[40]

Whereas the decline in muscle strength parallels the decline in bulk mass and cell numbers, the disproportionate loss of type II and the preservation of type I fibers is reflected in a reduction in the speed of contraction but with the relative enhancement of endurance (corrected for strength and the capabilities of the cardiopulmonary system). A pronounced falloff in dynamic performance at high speed in the elderly has been attributed to the preferential atrophy of type II fibers. Endurance for isotonic exercise is maintained, however, for work loads within the permissible bounds of cardiovascular function by the functional properties of type I fibers. Indeed, although endurance for isometric exercise declines in absolute terms, it may actually increase with age when corrected for the reduction in contractile muscle strength.[21,22,35] Aging athletes with good cardiopulmonary reserve are noticeably more suited for events that place a premium upon endurance (e.g., marathon running) rather then upon bursts of speed (sprinting). Likewise, reduction of the functional contribution of type II fibers to overall muscle performance may be a significant contributory cause to the slowing of movement and loss of fine motor coordination observed with age when it occurs in muscles responsible for digital dexterity and postural stability.

Although the loss of fast-conducting peripheral nerve fibers and type II muscle fibers results in a slight decrease in conduction velocity in the monosynaptic reflex time in the elderly, this reflex appears to contribute only minimally to the gross deterioration of automatic postural control seen in the elderly. Degradation of reflex postural control in the motor system appears to be more significantly linked to adverse changes in the long-latency polysynaptic reflex system that comes under brainstem and cortical influence.[45] Age-associated impairment of central nervous system processing mechanisms, possibly because of the numerical reduction of neurons in key areas (e.g., cerebellum) or a reduction of transmitter levels (basal ganglion), may produce visible degradation in the antigravitational stability of the skeletal-muscular system. Clinical experience suggests that further reductions in the quality of visual, vestibular, or proprioceptive sensory input to the elderly brain are usually the critical elements compounding existing central delays (spinal, brainstem, and cortical) that produce failures of automatic corrections in posture. Studies show that postural sway, which is very effectively suppressed in the final stages of adolescent development by cortical maturation, reemerges in the elderly, presumably because of a loss of higher cortical control.[39] The result is a serious enhancement of the risk of falls in the elderly as a result of delayed and uncoordinated neural activation of muscle groups that have themselves suffered inherent age-associated reductions in their capacity for strong and speedy contraction.

AGING OF SKELETAL BONE

Despite its inert appearance, bone is a living organ system whose cellular elements must submit to the predictable effects of passing years as well as to the numerous daily fluctuations in their physiologic milieu. Throughout life osteoclasts and osteoblasts remain active in their respective roles—osteoclasts resorb bone matrix, which results in the release of its contained minerals and the subsequent formation by osteoblasts of new collagen-proteoglycan matrix that may become mineralized into mature bone. The linkage between these simultaneous but antagonistic metabolic activities with respect to determination of relative rates, directions,

and locations is complex and only partly understood, but it includes both hormonal factors and stress-induced piezoelectric fields. More clearly understood from clinical experience, however, is the fact that bone's function as a supportive frame for the body is secondary in priority to its availability as a mineral depot for the correction of declines in the serum ionized calcium level. Differences among individuals in the bioavailability of dietary calcium to meet the primary mission of a stable serum calcium level may cause considerable variation in the degree to which skeletal calcium stores become depleted.[18]

Although the aging of any tissue may be considered to begin at birth, the practical clinical effects of age upon bone begin in young adulthood. Early childhood and adolescent years are marked by vigorous rates of osteoblast and osteoclast activity. The marked predominance of osteoblastic activity manifests itself in the growth of the skeleton during these years. Net bone formation continues for several years beyond the cessation of linear growth by epiphyseal closure but at progressively slower rates. As the metabolic activity of both osteoblasts and osteoclasts decline, they do so not only in absolute terms but also with respect to the relative difference between them. The loss of this preexisting differential advantage favoring osteoblastic bone formation brings the active process of bone remodeling into a steady-state equilibrium at approximately the fourth decade. The skeletal mass achieved at this point in life represents the peak for the individual's life span. Beyond this decade the metabolic rates of osteoblastic and osteoclastic cellular activity continue to decline further in absolute terms but with a new and reversed differential developing between them in favor of net bone resorption. The remaining decades of life are marked by a progressive and inexorable ebbing of skeletal mineral content.[13]

This loss of skeletal bone with age is marked by the appearance of several architectural variations in old bone that have important clinical consequences. Although only 20% of skeletal bone at maturity is trabecular, this compartment is the preferential site of virtually all bone lost before age 50 to 55, possibly because of its disproportionately high surface:volume ratio and its good vascular accessibility.[18] The loss of trabecular bone is neither generalized nor random; rather, trabecular struts of least structural importance within a given region are preferentially resorbed, sparing as long as possible those trabeculae carrying the major compressive or tensile forces through the bone area. Beyond age 50 to 55 years the preferential site for bone resorption shifts to the cortices. Again structural considerations appear to dictate the endosteal surface to be the preferential site of bone resorption, thereby preserving the contribution to mechanical stability of the bone's normal outer diameter even though wall thickness is unavoidably sacrificed.[18]

The universality of these patterns of growth, maturation, and senile demineralization of skeletal bone argues strongly that the skeleton is "programmed" for senescence either at the level of the cellular elements themselves or by the endocrinologic systems that modulate them. Since the resistance of skeletal bone to fracture at any site is directly proportionate to its mineral density, the generalized loss of minerals with aging confers upon the elderly an overall increased risk of fracture. This risk becomes greatest for bones enriched in trabecular content, such as femoral and radial heads and thoracolumbar vertebral bodies, since these areas are disproportionately depleted of bone mineral, having been sites of preferential resorption before age 50. Modifying this universal pattern, however, are several major genetic and physiologic factors that interact with aging and materially influence the likelihood of senile skeletal failure. Conceptually, these factors may be considered to exert their effect via changes in either the peak skeletal mass at maturity and/or the relative intensity of bone resorption in later years. These determinants are considered as part of the aging of the skeletal system since they are among the normal experiences of aging and do not properly belong with the long list of identifiable diseases and disorders that can induce changes in bone mass.