Current Concepts of Diagnosis and Treatment of Bone and Soft Tissue Tumors

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With 143 Figures

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Preface

During the past decade, the prognosis of bone and soft tissue tumors has improved considerably. This progress has not only been due to improved therapeutics and diagnostic methods but also due to a team approach now universally adopted.

Comparison of results, possible today, were hampered in the past by lack of a clinically relevant staging system and definitive treatment protocols. The complexity of the therapeutic management of bone tumors requires a constant exposure to tumors, highly skilled diagnostic techniques, and specialized expertise. It is therefore not surprising to learn that small and medium-sized hospitals embarking on the diagnosis and treatment of bone and soft tissue tumors are generally less successful than better equipped major centers. This fact has already been recognized by most physicians and surgeons who now refer their patients to specialized oncologic centers. Nevertheless, they all have to know the present state of the art. This symposium was therefore mostly addressed to them.

This international symposium, organized by the Division of Orthopaedic Surgery, University of Ottawa, and cosponsored by the Association des Chirurgiens Orthopédistes du Québec, was held in Ottawa from May 12-14, 1983. Each of the 40 presentations clearly demonstrates that disregard for proper staging before initiation of treatment and omission of a clearly defined treatment protocol is definitely unacceptable in view of the knowledge we have acquired.

The fast publication of these Proceedings has been made possible by the diligence of all contributors, the extraordinary editorial skills of Mrs. Elvira Stahl and her team, and the efficient collaboration of Mrs. Marianne Kalow and Dr. Heinz Götze, Springer Verlag, Heidelberg.
I sincerely hope that a comprehensive review of such a vitally important topic by a distinguished international panel of investigators, pathologists, and surgeons will find acceptance by a wide readership.

Hans-K. Uhthoff
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IN MEMORIAM

Crawford Jennings Campbell, MD

Crawford Jennings Campbell, a distinguished academic physician, educator and medical investigator, died on June 24, 1983, at the age of 69, in Laconia, New Hampshire, after a prolonged illness.

Crawford Campbell, the son of James S. and Marian J. Campbell, was born in Cooperstown, New York, December 15, 1913, and lived during his early years in the Panama Canal Zone where his father was employed as a lawyer with the Canal Authority. In 1932, he entered Yale University, receiving his Bachelor of Arts degree in 1936 and his Doctorate in Medicine in 1940. He received his surgical and orthopedic education at the University of Chicago Clinics under Dallas B. Phemister and C. Howard Hatcher, two leading orthopedic scientists of the period. During the Second World War he served with distinction as a Major in the United States Army Medical Corps with the 31st Field Hospital in Hawaii, the Philippines, and in Okinawa.

After the war he returned to Chicago to complete his education in orthopedics and then joined the faculty of the Albany Medical College. He became Chairman of the Section of Orthopedics and Professor of Orthopedics in 1961, a position he held until 1975 when he joined the faculty at Harvard Medical School as Lecturer and Visiting Staff at the Massachusetts General Hospital.

Crawford Campbell became widely recognized for his special skills in musculoskeletal pathology and his ability in the management of patients with bone tumors. He was insatiably interested in all aspects of orthopedics and maintained a close relationship with a group of academic orthopedists, spread widely across the United States, who shared a profound interest in musculoskeletal pathology. Under Crawford's gentle prodding, they continuously exchanged ideas, techniques, interesting case reports and pathologic materials.

Crawford assembled a large collection of rare and unusual pathologic findings which served as the focus of his research, chiefly in the description of a large variety of disease states and their effects on the skeletal system. In all, he contributed over 50 scholarly publications to the literature. He was frequently sought out as guest lecturer, visiting professor, and special essayist in his area of expertise. Up to the time of his death, he served as director of a series of Harvard Orthopedic Basic Science review courses.

His expertise in musculoskeletal pathology was so outstanding that any bone tumor conference in North America remained incomplete without his contribution. It is therefore not surprising that in the preliminary program of the symposium published in this book his name was associated with six different topics.

Crawford devoted much time to the education of medical students and residents. In recognition of his interest in and contribution to the education of foreign students, he was elected to honorary membership in the Japanese and Thai Orthopedic Associations. He was a diplomate of the American Board of Orthopedic Surgery and served as an examiner and member of the Board for many years. He was an associate editor and trustee of The Journal of Bone and Joint Surgery and an associate editor of The Journal of Trauma. He served with the National Institutes of Health, Surgical Study Section, and the National Academy of Science-National Research Council Committee on the Skeletal System.

Crawford will stand as an example of independence, integrity, and selfless service to the entire orthopedic community. All of us who were fortunate to have known Crawford Campbell will always remember him as an outstanding surgeon, a compassionate physician, a deeply respected educator, a true scientist and, above all, a warm and devoted friend.

H.J.M. W.F.E. H-K.U.

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Staging of Musculoskeletal Neoplasms

W. F. ENNEKING

Introduction

The purposes of a staging system for musculoskeletal neoplasms are to:

1) incorporate the significant prognostic factors into a system which describes progressive degrees of risk of local recurrence and distant metastases to which a patient is subject,
2) stratify the stages so they have specific implications for surgical management, and
3) provide guidelines for adjunctive therapies.

Over a number of years, staging systems for various classes of malignant tumors have been developed under the auspices of the American Joint Committee for Cancer Staging and End Results Reporting (AJC). The systems vary among cancers related to the natural course of a particular type of cancer. In 1980, a system for the surgical staging of musculoskeletal sarcoma was proposed, studied, and adopted by the Musculoskeletal Tumor Society (Enneking et al, 1980) and subsequently adopted by the AJC.

In this review, I will report the natural evolution of benign and malignant lesions of connective tissue derivation that led to the staging system, the system for both benign and malignant lesions, its articulation with surgical treatment, and early experience with its use.

Natural Evolution

The natural course in progression from the most benign to the most malignant connective tissue tumor is the same, lesion for lesion, whether the tumor arises in bone or somatic soft tissue. A fibrosarcoma behaves as a fibrosarcoma whether it arises in soft tissues and invades bone or vice versa. Aggressive benign fibrous lesions in soft tissue (fibromatosis) behave the same as their counterparts arising in bone (desmoplastic fibroma). Therefore, a common staging system was devised for bone and soft tissue as opposed to separate systems for bone and soft tissue lesions. The system, as befits the natural history, applies only to lesions of connective tissue histogenesis and not to primary lesions of round cell origin (leukemias, lymphomas, myeloma, Ewing's sarcoma) or metastatic lesions.

Current Concepts of Diagnosis and Treatment of Bone and Soft Tissue Tumors.

The significant progressive changes in the biologic behavior of musculoskeletal lesions are: 1) localized, latent or static, inactive, benign; 2) localized, active, benign; 3) aggressive, invasive, but still benign; 4) indolent, invasive, malignant, low risk of regional lymphatic or distant metastases; 5) rapidly, destructive, malignant, high risk of local, regional, and distant metastases; and 6) regional and/or distant metastases. Each of these progressions has distinctive clinical, radiographic, and histologic features that form the basis for the staging system and will be presented in some detail. The radiographic features that express these evolutionary changes in skeletal lesions have been previously studied and classified by their probability of occurrence by Lodwick et al (1980).

Inactive benign lesions are usually asymptomatic, discovered incidentally, and seldom related to pathologic fracture or mechanical dysfunction. They may slowly attain large size but eventually reach a steady state where they no longer grow. They appear quite responsive to contact inhibition and remain completely encapsulated. They remain intracompartmental and seldom deform the compartmental boundaries of cortical bone, articular cartilage, or dense fascial septae. When palpable in soft tissue, they are often small, movable, nontender, with little or no significant enlargement on subsequent clinical observation. Radiographic characteristics are lesions that are well marginated by a mature shell of cortical-like reactive bone without deformation or expansion of the encasing bone (Lodwick IA). Angiographic staging studies show little or no increase in isotope uptake and no significant reactive neoangiogenesis about the lesion or intralesional neoangiogenesis. CT scanning shows a homogenous density, well marginated, with no cortical broaching or cross-fascial extension.

The histologic characteristics of the lesion are: 1) low cell-to-matrix ratio; 2) mature, well-differentiated matrices; 3) benign cytologic characteristics (no hyperchromasia, anaplasia, or pleomorphism); 4) encapsulation by mature fibrous tissue or cortical bone; and 5) little or no reactive mesenchymal proliferation, inflammatory infiltrate, or neoangiogenesis about the lesion.

Active benign lesions are mildly symptomatic, discovered because of discomfort, and occasionally associated with pathologic fracture or mechanical dysfunction. They grow steadily and continue to enlarge during observation. They appear responsive to contact inhibition but not at normal levels as they can expand by deformation of overlying cortical bone, articular cartilage, or fascial septae. They remain encapsulated and have only a thin layer of filmy areolar tissue forming the reactive zone between the lesion and surrounding normal tissue. When palpable in soft tissue, they are usually small, movable, moderately tender, and grow slowly during clinical observation. Radiographic characteristics of active lesions in bone are well, but irregularly, marginated defects. The margin is a mature cancellous ring rather than a cortical shell, and the inner aspect is often irregular or corrugated giving a septated appearance. Expansion, bulging, or deformation of the combination of overlying cortex/reactive bone (Lodwick IB) is frequently observed.

Staging studies show increased isotope uptake that conforms closely to the limits of the radiographic defect and reactive changes. A thin but discernible rim of reactive neoangiogenesis about the lesion is frequently

observed but seldom any significant intralesional neoplastic neoangiogenesis. CT scanning shows a homogenous density, irregular but intact reactive bone, "expansion" of the overlying cortex, and intracompartmental containment by bone or fascia.

The histologic characteristics of active benign lesions are: 1) a relatively balanced cell-to-matrix ratio with homogenous distribution of the matrix; 2) well-differentiated matrices; 3) benign cytologic characteristics; 4) an intact capsule of mature fibrous tissue and/or cancellous bone; 5) a narrow zone of mesenchymal, inflammatory, and vascular reactive tissue between the capsule and the surrounding normal tissue; 6) resorption of preexisting bone by osteoclasts rather than by neoplastic cells as the mechanism of expansion. Intermittent areas of resorption often produce an irregular, serrated, sometimes corrugated interface between the capsule and the adjacent reactive bone.

Aggressive benign lesions are often symptomatic, discovered because of discomfort and/or a growing mass, and when in a stress-bearing bone associated with a pathologic fracture. When palpable in the soft tissues, they grow rapidly, sometimes alarmingly. These lesions are frequently tender and may have an inflammatory-like appearance. They are little affected by contact inhibition and readily penetrate or permeate the natural barriers to tumor growth: cortical bone, fascial septae, and in some cases, articular cartilage or joint capsules. They penetrate the capsule with finger-like extensions protruding directly into the surrounding zone. The reactive zone is thick, edematous, and often appears inflammatory. These aggressive lesions invade by destroying or resorbing the restraining bone or fascia and permeate into adjacent tissues or compartments rather than expanding by concomitant endosteal resorption and subperiosteal apposition.

When aggressive benign lesions involve unrestrained areas (medullary canal, cancellous bone, advential intermuscular planes within muscle bellies, pararticular tissues), they extend rapidly although usually preceded by a pseudocapsule of reactive tissue. Aggressive soft tissue lesions are usually firm, fixed, tender, and have a rapid growth history. The radiographic features of aggressive benign lesions are a ragged permeative interface with adjacent bone, incomplete attempts at containment by reactive bone, cortical destruction, endosteal buttresses and periosteal Codman's triangles, and rapid soft tissue extension (Lodwick IC). Staging studies often reflect the aggressive nature and behavior of these lesions. Isotope scans show increased uptake in both the early vascular phase and the late bone phase. The extent of the increased uptake is often well beyond the apparent radiographic limits. Angiograms show a distinct reactive zone of neovasculature on the early arterial phase and an intralesional hypervascular blush on the late venous phase of the study. CT scans show nonhomogenous mottled densities with defects in attempts at reactive containment, early extracompartmental extension from bone, and indistinct margins in soft tissues. Otherwise, occult involvement of major neurovascular bundles is often shown by either angiography or CT scans in aggressive soft tissue lesions.

Histologic features of aggressive lesions are: 1) high cell-to-matrix ratio; 2) clearly differentiated matrices of varying maturity; 3) predominantly benign cytologic characteristics without anaplasia or pleomor-