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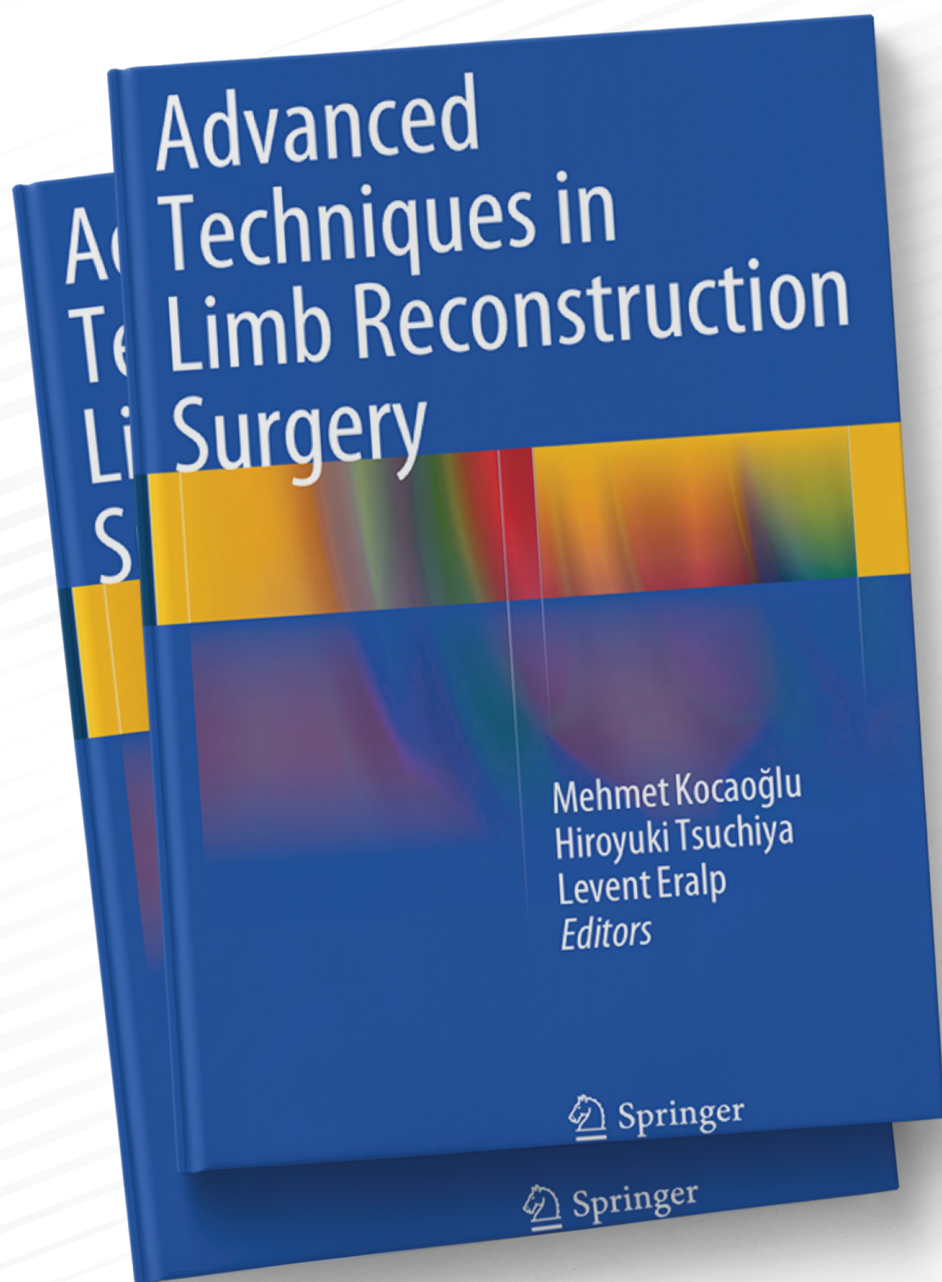
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SON 5 YILDA YAYINLANAN ULUSLARARASI KİTAP EDITÖRLÜK VE BÖLÜM YAZARLIKLARIM

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Ortopedi ve Travmatoloji Uzmanı



Advanced Techniques in Limb Reconstruction Surgery



The image shows an open book with the 'Contents' page visible. The page is divided into two columns. The left column lists chapters 1 through 10, and the right column lists chapters 11 through 21, followed by an Index. Each entry includes the chapter title, authors, and page number.

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Breast Disease



The Local Management of Bone Metastases

43

Levent Eralp and Halil Buldu

Abstract

Breast cancer is osteotropic, similar to prostate cancer; thus, breast cancer-related bone metastases are common. The local management of bone metastases is focused on pain control and the prevention or treatment of pathological fractures. In rare cases, the treatment may be definitive for the removal of isolated bone metastases, which may improve the patient's survival. All of these surgical treatment modalities are restricted by the patient's life expectancy and comorbidities. The major goal of the treatment is the improvement of the patient's quality of life. The treatment of bone metastases should be performed using a multidisciplinary team approach.

Keywords

Breast cancer • Bone metastases • PTH-rP • RANKL • Surgical treatment • Medical treatment • Metastases • Pathological fracture • Long bone • Vertebrae • Kyphoplasty • Vertebroplasty • M-CSF • PDGF • VEGF • Bone destruction • Bone production • Mirels score • Bisphosphonates • Hormone therapy • Tamoxifen • Polymethylmethacrylate • Impending fracture • Osteoarticular allografts • Intramedullary nail • Intercalary prosthesis • Kyphosis

Introduction

Metastatic disease is the most frequently observed malignant lesion of the bone [1]. Breast, kidney, thyroid, and lung cancers have high incidences of bone metastases [1, 2]. Approximately 70 % of patients who die of breast cancer have also had bone metastases [3]. Twenty percent of breast cancer bone metastases become symptomatic, and 17 % of these symptomatic cases require surgical

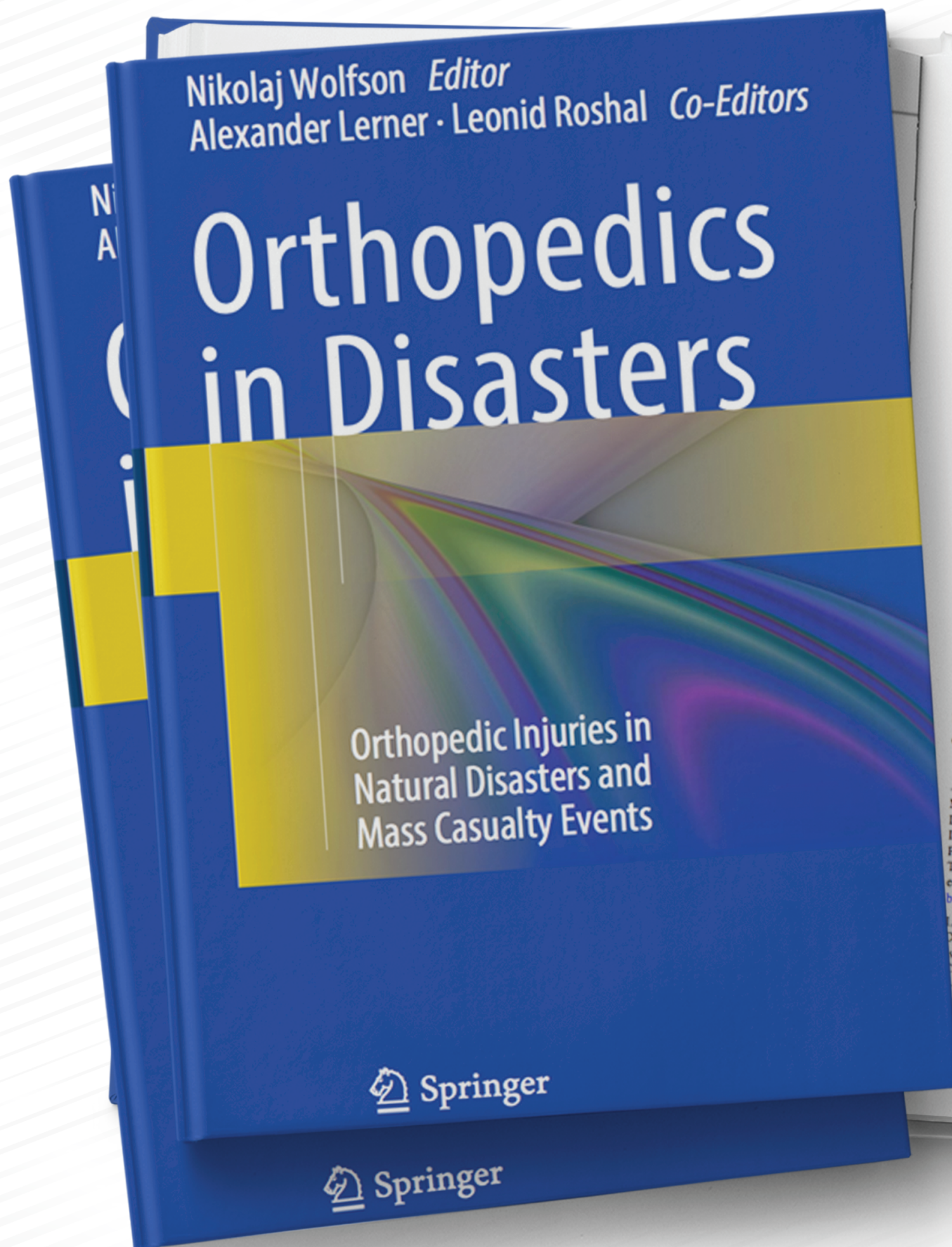
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Orthopedics in Disasters



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Orthopedics in Disasters

Orthopedic Injuries in
Natural Disasters and
Mass Casualty Events

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Nonunion and Osteomyelitis Following Fractures

34

Mehmet Kocaoglu, I. Levent Eralp,
and F. Erkal Bilen

34.1 Introduction

Natural disasters generate often high-energy trauma, leading to fractures of the bones and significant damage to the surrounding soft tissues. Bone is the only tissue that can heal without scar formation; however, this prerequisites optimum conditions. The higher the energy of the trauma, the less optimum conditions for healing occur. Knowledge of normal bone healing and the reasons for nonunion and osteomyelitis may help the surgeon to better plan the management of these complications. The lack of a normal healing process during the inflammation phase leads to an atrophic nonunion, whereas it leads to a hypertrophic nonunion if it occurs during the bone healing repair phase.

A fractured bone needs mechanical stability, biological sufficiency, and contact between properly aligned fragments for healing to occur. Bone

defects or severe displacement of the fragments, infection, insufficient local blood supply, usage of steroids and nonsteroidal anti-inflammatories [1], radiotherapy, soft tissue problems, and atrophic muscles and contractures may negatively impact the healing process.

Delayed union is a term used for a fracture that has not united within a period of time that is considered adequate for bone healing; the union is slow but will eventually occur without additional surgical or nonsurgical intervention. Thus, delayed union is mainly a clinical diagnosis [2]. According to the FDA, a diagnosis of nonunion may be established "when a minimum of 9 months has elapsed since injury and the fracture shows no visible progressive signs of healing for 3 months." The time frame, however, is different for different fractures. A fracture of the tibial shaft is not considered a nonunion until at least 9 months, whereas a fracture of the femoral neck can be defined as a nonunion after just 3 months. Tibial diaphyseal fractures that do not exhibit sufficient bridging callus to achieve clinical stability by 16 weeks are considered to be delayed union fractures [3]. On the other hand, nonunion refers to a fracture that will not unite without additional surgical or nonsurgical intervention (usually by 6–9 months). Of the long bones, the tibia is the most common site for nonunion development.

Bone necrosis, damage to adjacent tissue, and penetration of bacteria are the prominent

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Pediatric Lower Limb Deformities

Sanjeev Sabharwal
Editor

Pediatric Lower Limb Deformities

Principles and Techniques
of Management

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Metabolic Disorders

15

Mehmet Kocaoglu, I. Levent Eralp, and F. Erkal Bilen

General

Deficiency in the shape, strength and structure of bone tissue due to altered bone mineral homeostasis is called *metabolic bone disease* (1). The major factors affecting this homeostasis can be thought of as a three 3's: the intracellular and extracellular levels of *three* ions (calcium, phosphorus, and magnesium), which are controlled by *three* hormones (parathyroid hormone, calcitonin, and 1, 25-dihydroxyvitamin D) and act upon *three* tissues (bone, gut, and kidney) [1, 2]. Common clinical manifestations of metabolic bone disease in children include electrolyte disturbances, fractures, bone deformity, abnormal gait, and short stature.

The most commonly encountered forms of metabolic bone disease in children are the various types of rickets and renal osteodystrophy. Other less common but important pediatric metabolic conditions include osteoporosis, malabsorption syndromes, inherited diseases such as hypophosphatasia, X-linked hypophosphatemia, and various forms of vitamin D-dependent rickets [1, 3] (Box 15.1).

Pathophysiology

Although there are several types of *rickets*, the basic pathogenesis is a relative decline in calcium or phosphorus (or both) of large enough amount so that it interferes with phy-

Box 15.1

- Metabolic problems related to vitamin D must be addressed prior, during, and after the surgical treatment.
- There are various forms of metabolic bone diseases. Thus, collaboration with the pediatric/internal medicine department is essential.

seal growth and mineralization of the bone matrix in the growing child [1, 3, 4]. This decrease in the mentioned serum ion levels may result from inadequate intake or diminished absorption of phosphorus, calcium or vitamin D, reduced conversion of vitamin D to its active form, end-organ insensitivity, impaired release of calcium from bone, or phosphate wasting. In addition, there is evidence that insufficient vitamin D may interfere with mineralization independent of calcium or phosphate levels [1, 3].

In renal osteodystrophy, glomerular damage leads to phosphate retention, and tubular damage causes decreased production of the active form of vitamin D (i.e., 1,25-dihydroxyvitamin D) due to absence of 1-hydroxylase activity. These two factors severely impede intestinal calcium absorption and reduce plasma ionized calcium. The subsequent hypocalcemia generates secondary hyperparathyroidism, which remains ineffective in increasing intestinal absorption of calcium. Consequently, the body's only means of increasing serum calcium levels is by bone resorption. Metabolic acidemia may further deteriorate this condition (Figs. 15.1 and 15.2) [1, 3, 4].

In patients with *osteoporosis*, the bone is structurally normal but is reduced in overall amount. In children, osteoporosis may be idiopathic, as in juvenile osteoporosis, or may be due to disuse or chronic corticosteroid administration. The mechanism is uncertain, but numerous theories include increased bone resorption versus decreased bone formation, possibly due to deficient 1,25-dihydroxyvitamin D or calcitonin

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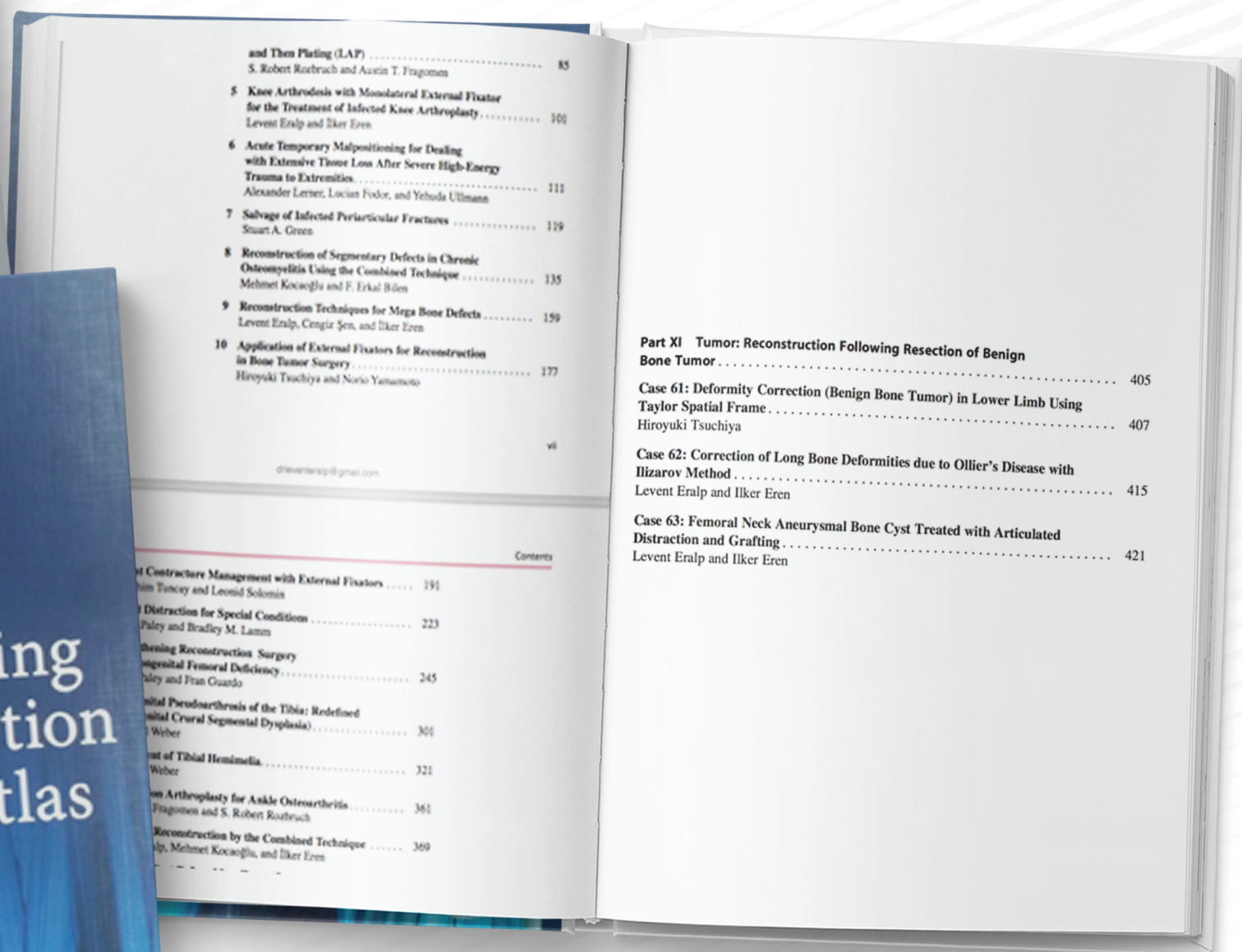
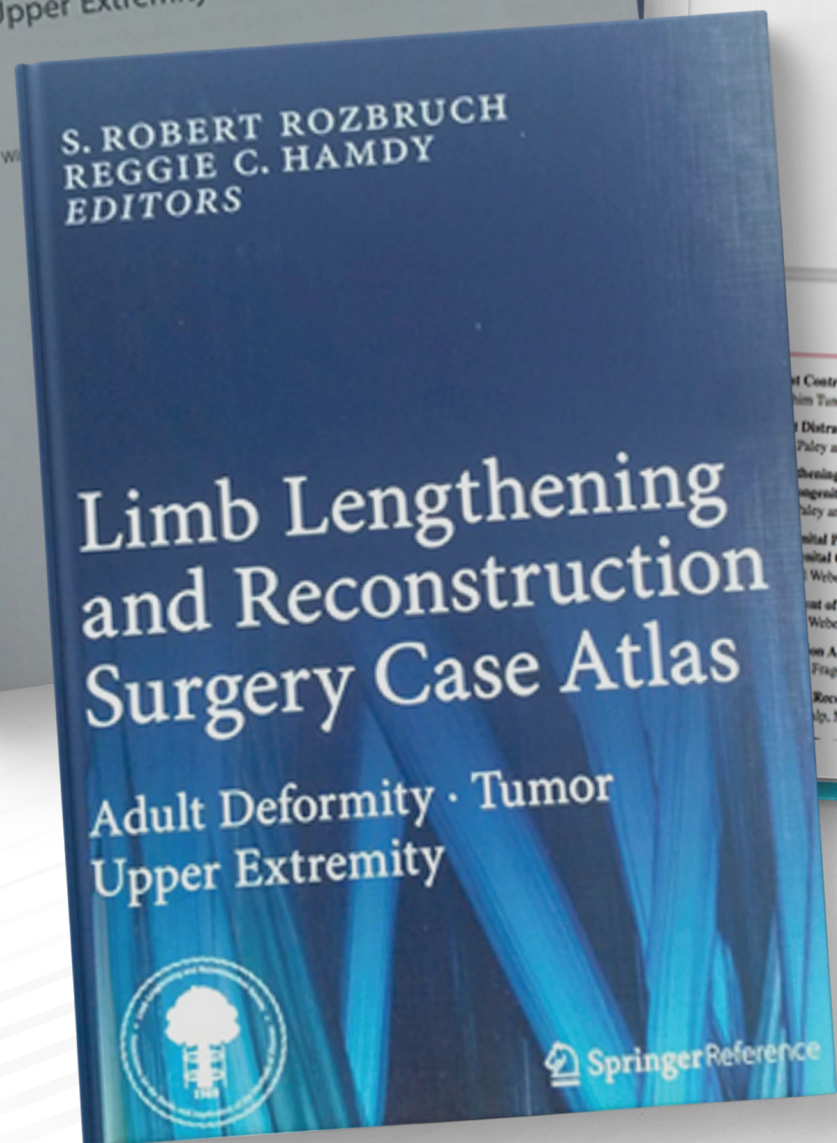
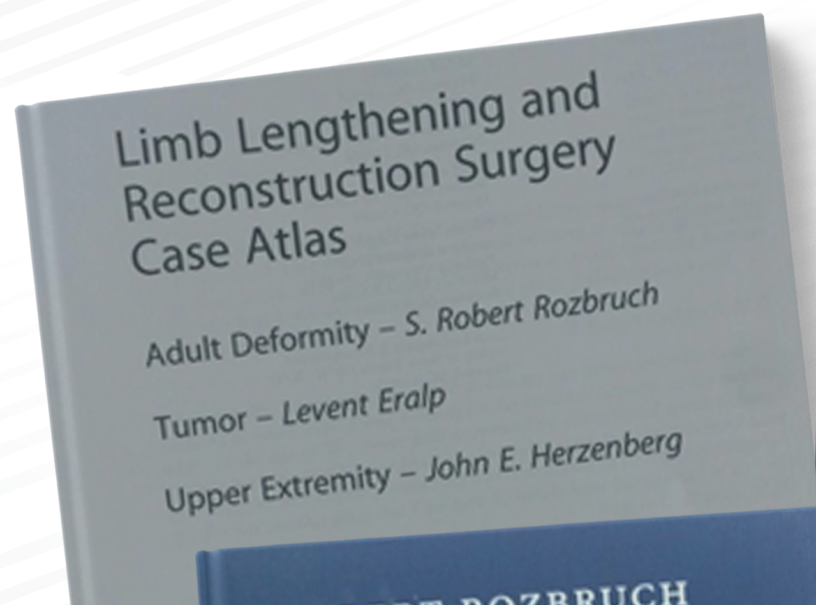
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Limb Lengthening and Reconstruction Surgery Case Atlas



Armed Conflict Injuries to the Extremities



Basic Techniques for Extremity Reconstruction

