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VOLUME TWO

ROCKWOOD AND GREEN'S FRACTURES IN ADULTS

SEVENTH EDITION

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EDITORS

Robert W. Bucholz

Charles M. Court-Brown

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We dedicate this Seventh Edition of *Rockwood and Green's Fractures in Adults* to Charles A. Rockwood, Jr, MD, and David P. Green, MD, who served as our inspiration and mentors for carrying on the revision and update of this textbook.

To Marybeth for her unwavering support over the years.

RWB

To Susan for her encouragement and understanding.

JDH

To my family for their help and support.

CCB

To my mother, Phyllis, who found the best in people, had compassion for all, and whose insight, guidance, and love have always made me believe that anything is possible.

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PREFACE

The 7th edition of *Rockwood and Green's Fractures in Adults* continues with the changes that were instituted in the 6th edition. In this edition there are four more chapters and 53 new authors drawn from three continents and ten different countries. Eleven new chapters focus on topics that have not been covered in separate chapters in previous editions of *Rockwood and Green*. To allow us to cope with the advances and changes in Orthopaedic Trauma, Paul Tornetta has become an Editor, and Margaret McQueen from Edinburgh and Bill Ricci from St Louis have been appointed Associate Editors. All three are accomplished orthopaedic trauma surgeons and their complementary interests and areas of expertise have greatly assisted the production of the 7th edition. In addition, many of the new authors represent the next generation of orthopaedic trauma surgeons who will be determining the direction of trauma management over the next two or three decades.

Orthopaedic trauma continues to be an expanding discipline, with change occurring more quickly than is often realized. When Drs. Rockwood and Green published the 1st edition in 1975, there were virtually no orthopaedic trauma specialists in most countries, fractures were usually treated nonoperatively, and mortality following severe trauma was considerable. In one generation the changes in orthopaedic surgery, as in the rest of medicine, have been formidable. We have worked to incorporate these changes in this edition. The continuing importance of wartime and severe civilian injuries is reflected in new chapters on gunshot and wartime injuries, the principles of mangled extremity management, bone and soft tissue reconstruction, and amputation. There is expanded coverage in this edition of the inevitable complications that all orthopaedic surgeons have to deal with, and we have included new chapters that discuss systemic complications, complex regional pain syndrome, infection, nonunion, and malunion. We have also separated distal tibial fractures into pilon and ankle fractures.

The other area of orthopaedic trauma that is expanding quickly, particularly in the developed countries, is the treatment of osteoporotic (or fragility) fractures. These fractures are assuming a greater medical and political importance, and orthopaedic implants are now being designed specifically to treat elderly patients. It is likely that this trend will continue over the next few decades; many of the chapters in this edition reflect this change in emphasis.

The changes in the 7th edition are highlighted by the altered presentation of the book. Many of the operative pictures and diagrams are now in color, as are all the tables. This edition is strengthened by the inclusion online of 20 new videos of surgical procedures done by Drs. Tornetta, Ricci, and Schmidt. Twelve additional videos will be created in the next year. The user will be able to download clips from these videos for lectures and presentations. We have also made available videos of many surgical approaches useful for trauma procedures. Two features that we have not changed are the Pearls and Pitfalls and the Authors' Preferred Treatment, these features having been present in the last edition. It is perhaps a paradox that we ask our authors to emphasize Level 1 evidence in the form of randomized double blind studies but we promote the authors' preferred methods, which is Level V evidence! However, we continue to believe that it is the function of *Rockwood and Green* to feature the world's leading orthopaedic trauma surgeons and to listen to what they say.

Robert W. Bucholz
James D. Heckman
Charles M. Court-Brown
Paul Tornetta, III

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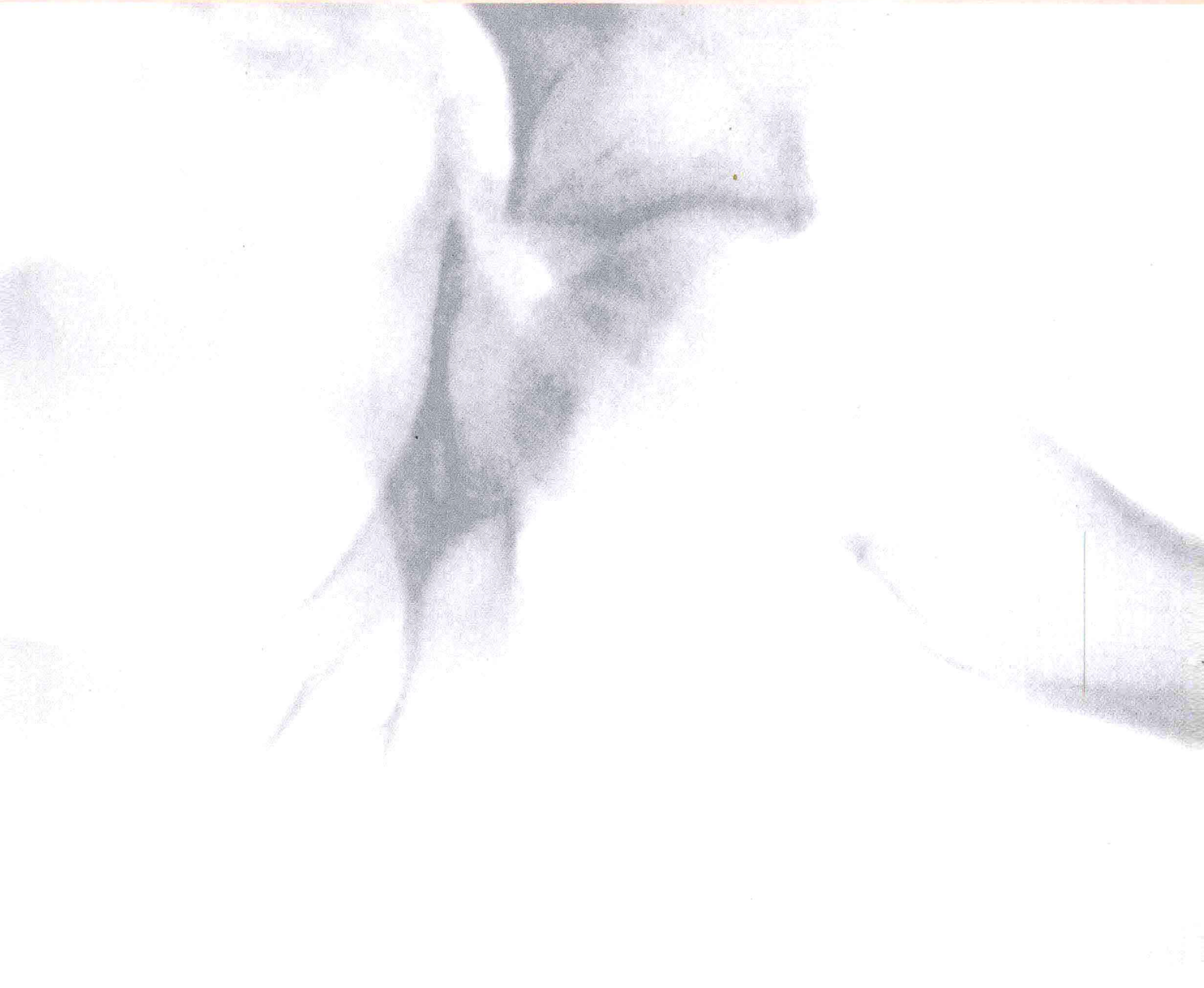

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PRINCIPLES OF SPINE TRAUMA CARE

Christina V. Oleson, Nathan E. Simmons, and Sohail K. Mirza

INTRODUCTION

An undiagnosed or suboptimally managed spine injury can result in a neurologic deficit and permanently impair a patient's function and quality of life, and in some cases may lead to death. Additionally, the legal context of working in emergency rooms makes the difficult work of evaluating trauma patients for a potential spine injury even more stressful for training and practicing physicians. Making this work a little less stressful is the goal of this chapter.

Archeological records from over 45,000 years ago are noted to forewarn that paralysis is incurable[137] and this remains true today, but that does not that nothing can be done for patients who sustain severe neurologic deficits. Patients with spinal cord injury today regain mobility, improve their quality of life, and achieve prolonged survival.[19] Research at the cellular and ge-

netic level continues to improve our understanding of the fundamental processes,[222] and clinical research methods to study spinal cord injury in real patient populations have improved, renewing optimism for novel spinal cord injury treatments.[278] This chapter focuses on general principles of spinal injury care. Subsequent chapters in this section discuss specific injury patterns in greater detail.

MECHANISMS OF VERTEBRAL COLUMN INJURY

Spinal Cord Injury Mechanisms

Terminology of Spinal Cord Injury

The meaning of terms commonly used to describe spinal cord injury depends on the context of the discussion.[184] Although precise definitions for many of these terms are lacking, broad interpretations are nevertheless useful for conveying a general meaning when discussing related processes.

Neural tissue injuries are divided into two broad etiology-based categories: primary injury refers to physical tissue disruption caused by mechanical forces, and secondary injury refers to additional neural tissue damage resulting from the biologic response initiated by the physical tissue disruption. The extent of structural damage to neural tissue is indicated by other descriptive terms. Concussion refers to physiologic disruption without anatomic injury. Contusion refers to physical neural tissue disruption leading to hemorrhage and swelling (the most common type of spinal cord injury). Or laceration, which describes loss of structural continuity of the neural tissue (rare in blunt trauma). The clinical response to injury is typically described in temporal terms: acute refers to the first few hours after injury; subacute typically refers to several hours to days following injury, and chronic refers to intervals of weeks to months after the injury. The functional consequences of spinal cord injury are usually described by terms that refer to the severity and pattern of neurologic dysfunction. Complete spinal cord injury, incomplete injury, or transient spinal cord dysfunction describe different grades of severity of neurologic injury. Names for different types of spinal cord injury syndromes, such as anterior cord syndromes, central cord syndrome, and Brown-Séquard syndrome, refer to patterns of neurologic dysfunction observed during clinical evaluation.[295]

Mechanics of Neural Injury

Structural failure of the spinal column displaces bone and ligament structures into the neural space, the spinal canal, and neural frame. These displaced and disrupted structures apply forces on the neural tissue that result in either functional or anatomic disruption.[25] Most spinal cord injuries are crushing injuries resulting in acute neural tissue contusion from applied physical forces. Laceration or transection of the spinal cord is rare, even in markedly displaced fracture dislocations.[151]

Experimental models of spinal cord injury have identified several characteristics of the injury force that determine the extent of neural tissue damage. These include the rate of force application, the degree of neural tissue compression, and the duration of neural tissue compression.[150] The severity of neural tissue disruption is proportional to the energy absorbed from the injury mechanism.[10] For direct impact on neural tissue, contact velocity and maximum cord compression are better predictors of injury severity than either force or acceleration.

The viscoelastic properties of soft tissues provide the principal resistance to deformation in the early stages of impact during compression injures.[25,290] Spinal cord tolerance for compression decreases as the velocity of compression increases (Fig. 41-1).[150] Minimum compression of the cord at high-contact velocity may produce severe anatomic injury and limited functional recovery.[150] At about 50% cord compression, functional recovery is minimal regardless of the contact velocity.[150] Although this threshold effect denotes an upper limit of compression in the acute injury model, it is not apparent in extremely slow onset of cord compression seen in chronic degenerative conditions, such as cervical spondylotic myelopathy. Cord compression that develops over years of progressive arthritic changes can be quite severe and yet manifest minimal clinical symptoms.

The spinal cord can withstand considerable axial displacement without sustaining structural or neurologic deficit. Contrary to the relationship between nerve roots and neural foramina during physiologic movement, the spinal cord does not slide up and down in the spinal canal during spinal flexion and extension. Rather, the cord appears to deform like an accordion.[228] Physiologic movements can stretch the cord an average of 10%, and maximum change can be as much as 18% of the longitudinal length of the spinal cord.[228] Maximum stretching occurs between C2 and T1. Cord deformations may be more severe

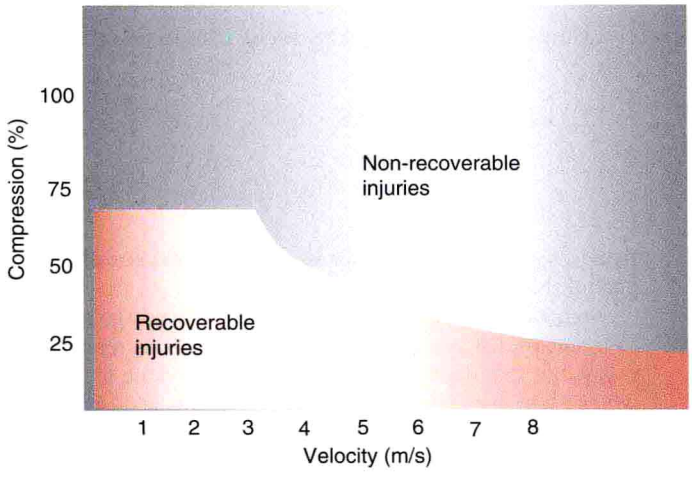

FIGURE 41-1 Effect of rate and severity of cord compression on potential for neurologic recovery. The threshold varies inversely with the magnitude of compression and the velocity of compression of the spinal cord.