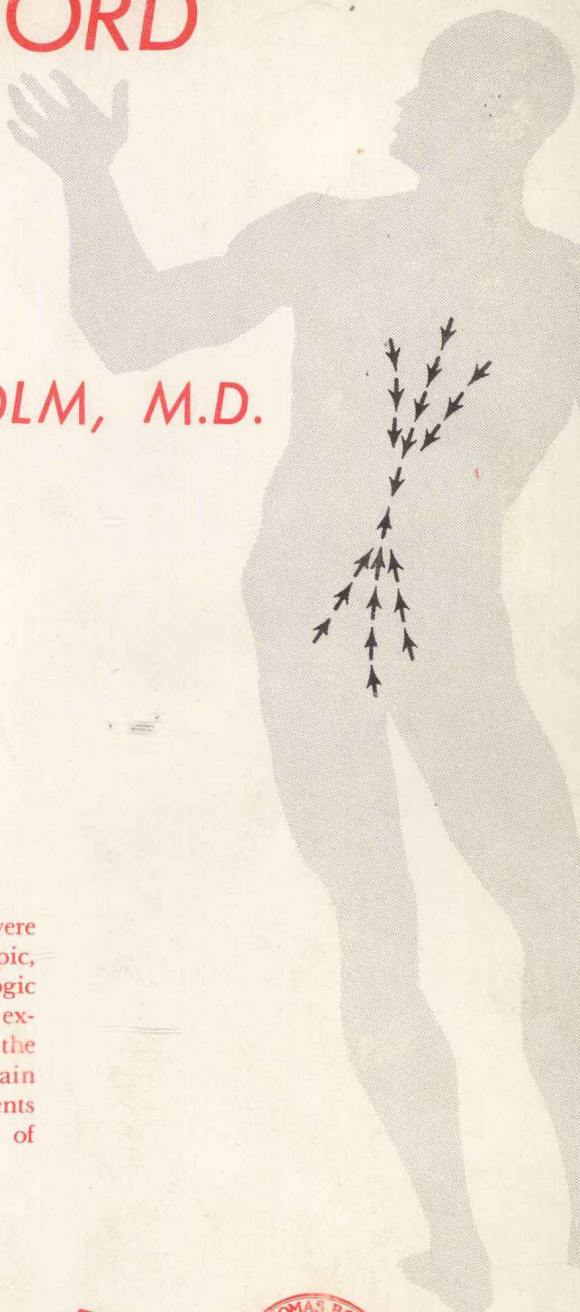
THE PATHOPHYSIOLOGY OF SPINAL CORD TRAUMA



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Focusing on the response of neural tissue to severe injury, this monograph documents the microscopic, histochemical, biochemical and pathophysiologic events which follow extreme trauma. The author explains metabolic changes which involve not only the locally injured area but the entire spinal cord, brain stem and suprasegmental structures. He also presents in-depth reviews of the basic etiologic concepts of posttraumatic neural tissue loss.

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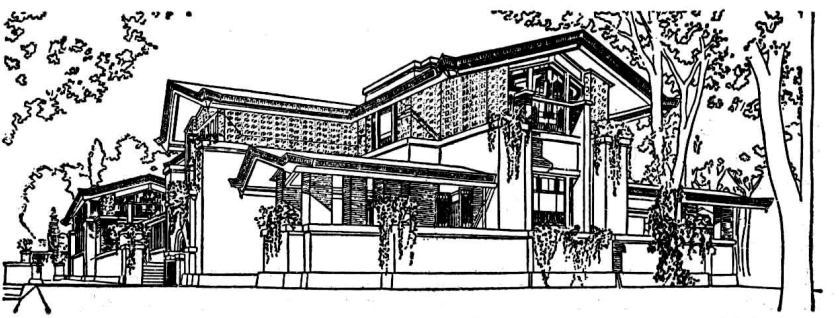

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AMERICAN LECTURE SERIES®

A Monograph in
The BANNERSTONE DIVISION *of*
AMERICAN LECTURES IN NEUROSURGERY

Edited by
ROBERT H. WILKINS, M.D.
Professor and Chairman
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This monograph is dedicated to all the spinal injured persons I have known and for whom I have cared. One cannot help but admire their courage, determination and continued belief that something better might be done.

CONTRIBUTORS

Although the author has written this monograph, this is not meant to imply that all work contained is his alone. Important contributions of individual workers in this laboratory are indicated in each chapter heading where they are listed as co-authors.

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PREFACE

The author has made no attempt to review in totality all available spinal cord injury knowledge or clinical treatments. Emphasis is rather placed upon the pathophysiologic and biochemical responses of neural tissues to severe trauma. In this theme the common clinical aspects of blunt or penetrating spinal injuries will be mentioned only in passing. The major thesis will relate to microscopic, histochemical, and biochemical events which have been documented to follow severe trauma. Metabolic changes which involve not only the locally injured area but the entire spinal cord, brain stem, and suprasegmental structures will also be discussed. Present concepts of the basic etiology of posttraumatic neural tissue loss are elaborated in detail and thus become a compilation of seven years of production from this laboratory by several researchers. Some measure of success has been achieved in reversing the anticipated cellular postinjury response as well as preserving cord tissue and chronic spinal function in rigidly controlled injury test systems. I sincerely hope these observations will be but the dawn of a long productive day leading to more effective therapy for spinal injured man.

This work has been possible through the combined assistance of the National Institute of Nervous Diseases and Stroke, the Hines Chapter of the National Paraylzed Veterans of America, the Susan Adler Foundation, and the National Paraplegia Foundation. Because of their great interest, the Veterans Administration has provided a wide forum for these works. The author has been intellectually enriched and the project enhanced by many students who have earnestly labored in this laboratory.

Miss Benedette R. D'Amore has greatly contributed to the research since the laboratory was established in 1968. Miss Francine E. D'Amore deserves much praise for her organization and preparations of innumerable manuscripts, grants, and this

monograph. I am indebted to my entire staff, who have admirably met many crises and deadlines.

Most especially, my wife deserves credit for her patience, understanding, and help during the preparation of this manuscript.

J. L. O.

INTRODUCTION

The spinal cord has long been identified as an extremely trauma-sensitive organ. Seemingly moderate traumatic injury or tumor pressure is clinically associated with total and permanent paralysis. For this reason neurosurgeons have long employed their most gentle manipulative techniques when approaching or operating upon the cord for fear of producing intraoperative injury. Throughout this monograph we have sought to scientifically identify the truth of these propositions, remove them from the anecdotal sphere, and to suggest means of providing trauma resistance and ways of treating the severely traumatized spinal cord.

Is the cord indeed more sensitive and reactive to traumatic forces than the brain or other body tissues? The undeniable answer to that question is yes. We and others have experimentally graded the spinal cord resistance to trauma. It is now clear that 400-500gm/cm force to larger mammals provokes lasting sensory motor paralysis. This injuring force can be crudely duplicated by dropping a rather heavy fountain pen a distance of about 1 foot. There is no pain or discomfort if this force is applied to one's hand. The tactile sensation from 500gm/cm is roughly approximated by sharply tapping the finger pad upon a blunt surface. By comparison, the permanent injury cord force (5×10^2gm/cm) is nearly one thousand times less than that required to provoke thirty minute apnea in a similar experimental head injury model (1.5×10^5gm/cm).[1] Thus the cord is quantitatively identified as unique in terms of injury sensitivity.

This is not to say that the cord tissues are themselves excessively fragile. It is rare for even major fracture dislocations to lacerate or tear the cord. We have found it necessary to deliver twice the paralyzing force (1000gm/cm) before the pia or underlying white matter is physically disrupted. Albin has calculated the instantaneous paralyzing forces generated from

weight dropping to approximately 22 lbs./in.[2] Circumferential pressures of 300mm/Hg delivered by encircling cuff are reportedly tolerated for one hour with some functional restitution, although this does not obtain after three-hour compression.[3] This observation is somewhat surprising in the context of the injury sensitivity developed here and requires further documentation.

Even at this early juncture we have seemingly conflicting statements, for on one hand the cord has certain physical toughness, but on the other is exquisitely sensitive to wounding forces. These statements are not mutually exclusive. In fact they are compatible in terms of our present knowledge. The cord is quite resistant to physical disruption, yet even nondisruptive injuries cause total malfunction. Two types of spinal paralysis can be advanced to explain these seemingly divergent facts.

1. *Physiologic.* Spinal transmission ceases immediately after a severe wound. Histopathological and ultrastructural studies conducted in the first thirty minutes reveal, for the most part, structurally intact white fibers at the wounded site. The conduction failure in this setting must relate to micromolecular membrane alterations. An outpouring of potassium has been found in the ambient spinal fluid by Eidelberger[4] and may be a responsible factor in such membrane alterations and attendant neuronal conduction loss.

2. *Structural.* With the passage of time an inexorable autodestruction of spinal tissues follows major trauma. This pathophysiological response resides entirely within the spinal cord. First the grey and later the white matter undergoes progressive dissolution. This process predictably follows severe wounding even when the vertebral lamina are removed prior to wounding and the injured cord exposed only to atmospheric pressure after the blow. The autodestructive time course is described below in detail and is essentially complete within twenty-four hours. There are no remaining fibers at or about the wound at this time.

It is to the further understanding of the belated autodestruction that we have long labored. Theoretically, if the cord structural components could be treatment saved, then the initial malconduction is probably a repairable and therefore reversible process.

Clinical science has ineffectively struggled with the problem to the present time. It is our opinion that past failures obtain because therapy has not been properly conceived and directed toward the offending lesion which lies deep within the spinal cord parenchyma. Routine spinal injury management has largely related to mechanistic idea and theory. Treatments are directed toward improving the local spinal cord environment. In this vein we actively pursue reduction of fracture dislocations and remove foreign intraspinal masses such as bone fragment, extruded discal materials, or hematomas. Unfortunately, these efforts, as extrapolated from the experimental data, will not be anticipated as associated with successful future spinal cord function. This statement is modified for mild or moderate injury where some distal spinal function can still be appreciated. Under these conditions correction of cord distortion may be very rewarding. A severely injured cord as defined by immediate and total paralysis undergoes autophagic dissolution when laying free in the air. The removal of bone in this setting has no relationship to the causal lesion and will be ineffective as a treatment modality for that lesion.

For these reasons a very strong school of conservatism has developed. This conviction is based upon extensive long-term experience with these patients. Many clinicians are of the opinion that surgery offers nothing for the spinal injured victim. Perhaps in the present setting this is so. We have all, however, seen recovery of an important spinal segment or more after well-planned and executed surgery. In terms of our present thesis, and as is so often the case, probably neither the conservative nor surgical approach is totally correct.

Because of our rather radical departure from accepted concepts, much of the material presented here has become controversial. Its truth will probably not be wholly confirmed or disproved for many years. New concepts are difficult for many and impossible for a few. We have attempted to maintain scientific unbias, although it is difficult at times when supporting evidence can be garnered at every level in favor of our theories. We have interpreted the controversy based on available scientific data and merit. Even if the current hypothesis is proven untenable, it has

succeeded to the extent that a new focus has been made and the community stimulated to labor so that the precise autodestructive mechanism may be eventually elaborated.

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CONTENTS

Chapter

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