SPINAL CORD COMPRESSION

Spinal Cord Compression

MECHANISM OF PARALYSIS AND TREATMENT

By

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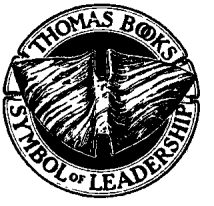

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To My Mother

. . ., *I would address one general admonition to all, that they consider what are the true ends of knowledge, and that they seek it not either for the pleasure of the mind, or for contention, or for superiority to others, or for profit, or fame, or power, or for any of these inferior things; but for the benefit and use of life; and that they perfect and govern it in charity.*"

FRANCIS BACON, in the Preface to
The Great Instauration, 1620.

PREFACE

SPINAL CORD COMPRESSION is a controversial field needing clarification. We therefore submitted some of its problems to experimental analysis in dogs. We then compared the laboratory evidence with reports on a large number of human cases of complete spinal paralysis due to trauma or to spinal cord tumors. The purpose of the experiments and comparison was twofold: (1) to collect a body of exact data on which it would be possible for the first time to base precise generalizations on prognosis in such cases, and (2) to provide the evidence necessary to determine whether to operate and, if so, when. We thought that a successful study would enable the physician to recommend proper therapy and to inform the patient or his family and friends of the outlook. Up to now it has been possible to make only grave and uncertain forecasts in cases of complete spinal compression paralysis.

To achieve the purposes of the investigation it was necessary to find answers to 5 critical questions: (1) How long can the spinal cord be acutely compressed before functional recovery becomes impossible? (2) What are the chances of recovery of spinal cord function in cases of paralysis, complete for any given time and caused by gradual compression occurring over any given time? (3) What is the sequence of loss of function with spinal cord compression and what is its clinical significance? (4) What is the cause of the paralysis? Is it mechanical deformation of cord tissue or is it anoxia? (5) In what circumstances should laminectomy be done?

To find answers to these questions we devised methods for producing acute and also gradually increasing spinal compression. These devices enabled us to study functional and histological results. These results were then compared with studies of spinal cord paralysis in man caused by injuries or neoplasms. These investigations, supplemented by special electrophysiological studies under the direction of Dr. Samuel Gelfan, threw light on the

basic cause of spinal compression paralysis. By correlating these studies we sought to provide the means for a surer interpretation of certain signs and a more reliable guide to therapy than have hitherto been available to physicians.

This monograph brings together in concise form details contained in a long series of publications by the author and various co-workers and adds new information on the subject.

ACKNOWLEDGMENTS

I WISH to express my appreciation to the men and women who have participated in the work upon which this monograph is founded: Dr. Robert Day, Dr. Samuel Gelfan, Dr. Clare C. Green, Dr. Ernst Herz, Dr. Arthur Jensen, Mrs. Elizabeth Kellerman, Mr. Harold Klinger, Dr. Harry McGuire, Miss Elizabeth Nesbitt, and Mr. Santo Vitale.

The Archives of Neurology and Psychiatry, The Journal of Neurophysiology, The Surgical Clinics of North America, and *The American Journal of Physiology* have kindly permitted me to quote and to use illustrations from articles appearing in them.

Special gratitude goes to Dr. Gelfan, since the electrophysiological data contained in Chapter 8 are based on studies made in our Laboratory of Neurophysiology under his direction. Many of the ideas in the chapter are his.

I. M. T.

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SPINAL CORD COMPRESSION

Section I

EXPERIMENTAL STUDIES—INTRODUCTION

It is hardly necessary to justify an experimental study in medicine aimed at providing more precise information under more controlled conditions than can be supplied by what Hughlings Jackson called "Nature's experiments." The purpose of accumulating such experimental, not to mention clinical, data is to enable one to foretell events in given circumstances. The ultimate end of this scientific approach is to understand the course of a disease and to attempt to control it.

Of course, the results of animal experiments are not always directly applicable to man because of anatomical, physiological, and biochemical differences among species. It remains true, nonetheless, that man has often been the beneficiary of critically evaluated experiments carried out on animals.

The need for treating acute spinal cord compression sometimes arises in the handling of patients suffering from vertebral fractures with or without dislocations, herniated intervertebral discs, neoplasms, or hemorrhage, either traumatic or spontaneous. Yet there is much controversy over the proper management of the victims of lesions producing acute paralysis. Because of the disagreements over the treatment of acute spinal cord compression and because of the considerable interest in the problem of compression of peripheral nerves and of the brain, it is surprising that before our own studies the problem of acute spinal compression had been so rarely subjected to experimental analysis.

Allen[2,3] and, to be sure, considerably later, Freeman and Wright[20] attempted to determine how long a spinal cord may be compressed without precluding functional recovery. Their experiments, however, are not comparable to those herein reported,

and do not answer the questions posed in the Preface. Critical consideration of their technique and recommendations will be given in Section III.

Chapters 1 to 8, following, report on our own technique, findings and conclusions.

CHAPTER 1

TECHNIQUE TO PRODUCE ACUTE COMPRESSION

THE device used to produce acute compression of the spinal cord, shown in Figure 1, is hydraulic in principle. Its essential parts (Figs. 2 and 3) are a metallic compressor unit and a compression unit, consisting of two rubber balloons—the larger one semicircular, the smaller bulb-shaped—connected by polyethylene and

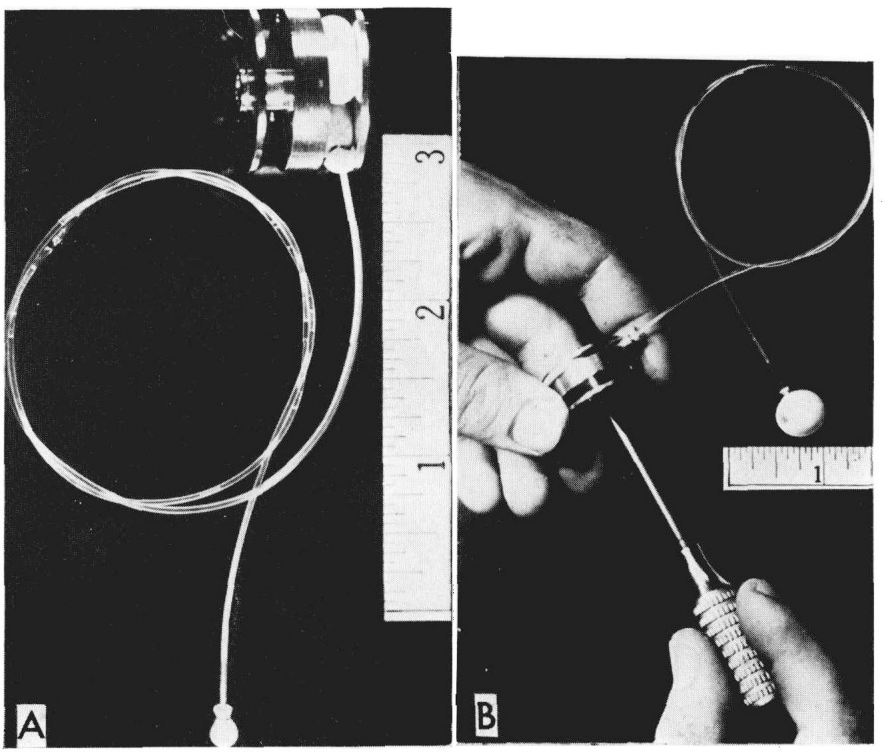

Fig. 1. A, Compressor and compression-units in operating position.
B, Compressing-screw being turned to compress semicircular balloon between bottom and middle flanges, thus inflating small balloon.

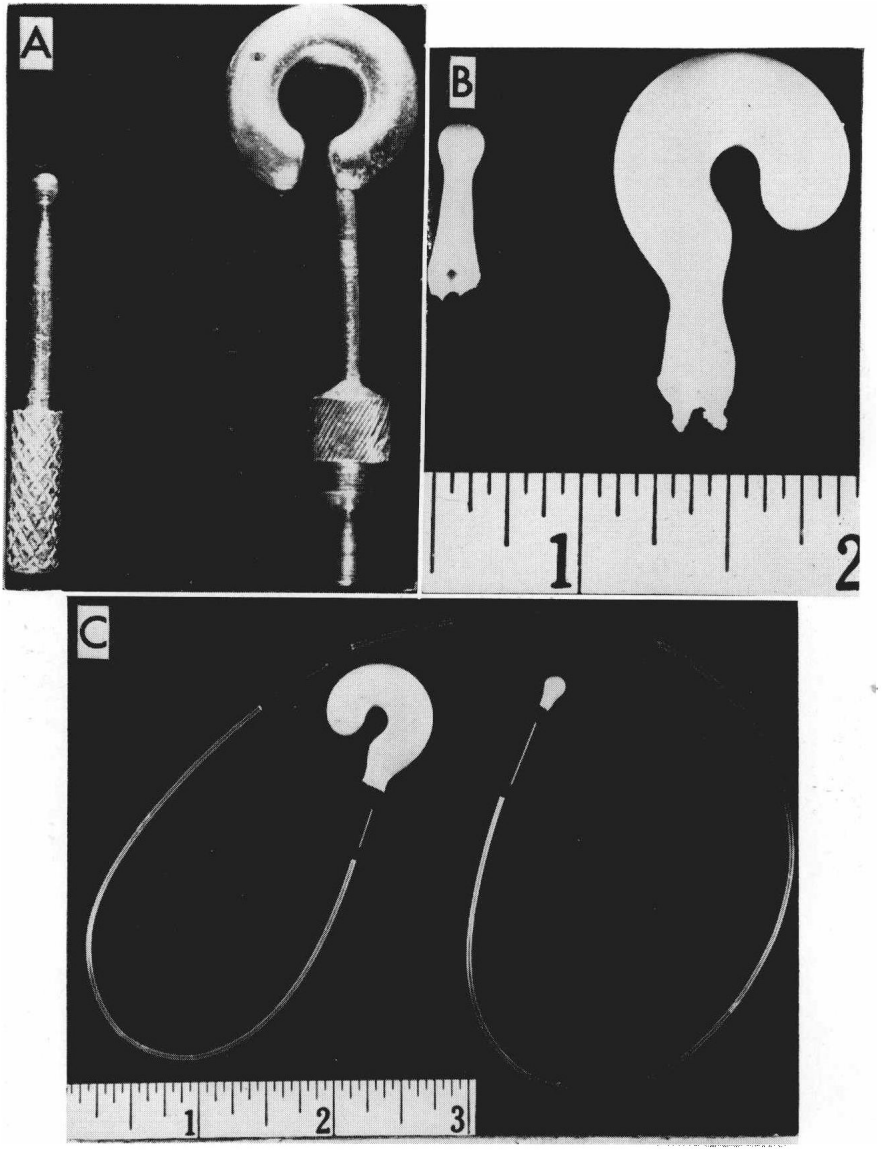

Fig. 2. Parts of the compression-unit with metal forms which are dipped into latex to make the balloons.

metallic tubes. The compressor unit has five parts, machined from stainless steel. When the compressing screw is tightened (Figs. 1 and 4), the bottom flange moves up toward the middle flange, compressing the semicircular balloon and forcing fluid through the connecting tubes to the small balloon, which is thereby inflated.

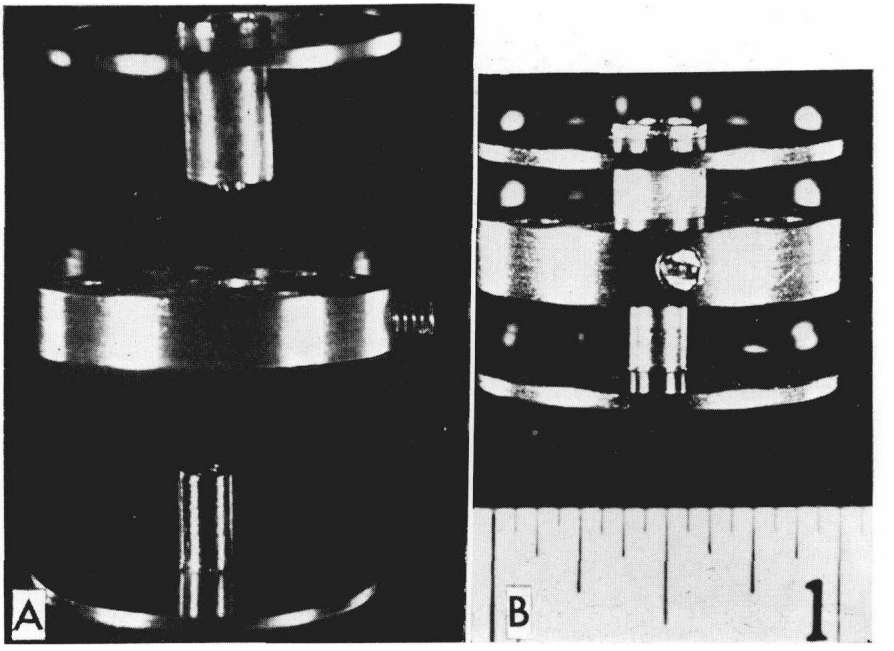

Fig. 3. A, Parts of the compressor unit machined from stainless steel. B, Assembled compressor-unit showing the set-screw that fixes the middle flange to the female collar of the top flange.

Insertion of Device in Animal

A 1-segment laminectomy is made several segments caudal to the site chosen for compression. The dura is exposed laterally to the ventral wall of the vertebral canal.

The top flange of the compressor unit is then fixed to the skin by forcing its female collar through a stab wound made several centimeters to one side of the laminectomy incision. The middle flange is then slipped under the skin and over this projecting