
HANDBOOK OF CLINICAL NEUROLOGY

Edited by

P.J. VINKEN and G.W. BRUYN

VOLUME 25

INJURIES OF THE SPINE AND SPINAL CORD
PART I

INJURIES OF THE SPINE AND SPINAL CORD

PART I

Edited by

P.J.VINKEN and G.W.BRUYN

in collaboration with

R. BRAAKMAN

(内部交流)

Associate Editor

HAROLD L. KLAUANS JR.



NORTH-HOLLAND PUBLISHING COMPANY • AMSTERDAM • OXFORD
AMERICAN ELSEVIER PUBLISHING CO., INC. • NEW YORK

© NORTH-HOLLAND PUBLISHING COMPANY – 1976

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, without the prior permission of the copyright holder

Library of Congress Catalog Card Nummer: 68-8297

ISBN for the complete set: 0 7204 7200 8

ISBN North-Holland for this volume: 0 7204 7225 3

ISBN American Elsevier: 0 444 10926 9

299 illustrations, 56 tables

PUBLISHED BY:

NORTH-HOLLAND PUBLISHING COMPANY • AMSTERDAM
NORTH-HOLLAND PUBLISHING COMPANY, LTD. • OXFORD

SOLE DISTRIBUTORS FOR THE U.S.A. AND CANADA:

AMERICAN ELSEVIER PUBLISHING COMPANY, INC.

52 VANDERBILT AVENUE

NEW YORK, N.Y. 10017

PRINTED IN THE NETHERLANDS

66522

INJURIES OF THE SPINE AND SPINAL CORD
PART I

HANDBOOK OF
CLINICAL NEUROLOGY

Edited by

P. J. VINKEN and G. W. BRUYN

Editorial Advisory Board

L. DEATAMS, B. P. BHARUCHA, PAUL CASTAIGNE
MACDONALD CRITCHLEY, RUSSELL N. DELONG
SHIGEO OKINAKA, S. REISUM, J. O. TRELLER, R. J. ZULCH

VOLUME 22



AMERICAN ELSEVIER PUBLISHING CO. INC. • NEW YORK
NORTH-HOLLAND PUBLISHING COMPANY • AMSTERDAM • OXFORD

INJURIES OF THE SPINE AND SPINAL CORD
PART I

HANDBOOK OF CLINICAL NEUROLOGY

Edited by

P.J.VINKEN and G.W.BRUYN

Editorial Advisory Board

R. D. ADAMS, E. P. BHARUCHA, PAUL CASTAIGNE
MACDONALD CRITCHLEY, RUSSELL N. DEJONG
SHIGEO OKINAKA, S. REFSUM, J. O. TRELLES, K. J. ZÜLCH

VOLUME 25



NORTH-HOLLAND PUBLISHING COMPANY • AMSTERDAM • OXFORD

AMERICAN ELSEVIER PUBLISHING CO., INC. • NEW YORK

Foreword to volumes 25 and 26

'Basically, management in neurology consists of prudently doing nothing'. This was the opinion of the neurology professor of one of the editors.

Many neurologists practising today would probably disagree with this statement. They would not be able to deny, however, that it has been pre-eminently applicable to the management of traumatic paraplegia for many thousands of years. The writer of the Edwin Smith papyrus considered tetraplegia as 'an ailment not to be treated'. Treatment of traumatic para- and tetraplegia was not even improved by the sadly all too rich experience of World War I. Although the literature of this period, particularly the British literature contained many excellent papers on spinal cord injuries, they dealt with neurophysiology and pathology, but were not at all concerned with therapy. In World War I, 90 % of patients suffering from this type of injury died within one year, and probably only 1 % survived for more than 20 years. The literature appearing between the two World Wars was scant and no real advance was made in the late care of spinal cord injuries. In 1924, the Medical Research Council summarised the situation at that time as: 'The paraplegic patient may live for a few years in a state of more or less ill-health'. Gowland's paper to the Harveian Society (1934) illustrates how these poor victims lived their short incarcerated lives in institutions. In 1937, Courville stated: 'It is a matter of preserving life by constant and meticulous care when life is of little value to the patient and costly to his relatives'.

After World War II, the influence of a few pioneers (in Europe, Guttman; in the U.S.A., Munro and Bors) caused a drastic reduction in mortality; hence the current survival rate is high. Guttman with his straightforward thinking and dynamism realised his concept of a paraplegic as a disabled but healthy person independent of mind and body and often with an independent future. In this new era, the spinal man (or woman) can usually be re-educated to perform a useful job and to take a useful place within the community.

The experience derived from observations on longer living paraplegics have initiated and stimulated clinical research, especially in Europe, thus shedding new light on spinal cord physiology. This gratifying development has given an enormous impetus to experimental research over the last ten years, particularly in the U.S.A.

Thirty years ago, a handbook editor would have found it difficult to think of more than a few chapters on injuries of the spine and cord. Today, even two volumes are insufficient to cover the field.

Volume 25 contains basic experimental, biomechanical, pathological and epidemiological aspects, as well as the various causes and types of acute injury to the different parts of the spine and cord.

In Volume 26, the non-acute aetiology of spine and cord lesions is covered, together with extramedullary haematomata, abscesses and some vascular lesions not dealt with in previous volumes. Clinical appraisal, management and prognosis of para- and tetraplegia with their influences on the cardiovascular, respiratory, gastrointestinal and urinary systems, are included. The effects of spinal injury on skin temperature regulation and sexual behaviour are also covered.

Physicians actively engaged in spinal units (paraplegists) who may feel that certain topics, such as psychological effects, vocational training, etc., have not been included, are kindly requested to bear in mind that this is a Handbook of Clinical Neurology and not of rehabilitation. The Editors had intended to include a chapter devoted to rehabilitation, but unfortunately the authors invited to undertake this assignment were unable to give to its completion that degree of priority which they and we would have wished. Faced with the problem of whether to extend the manuscript deadline or go ahead with publication, the latter course was adopted.

The Editors are grateful for the continuing enthusiasm of the editorial staff, Mrs. M. Posthuma and Mrs. B. Vollers-King, which has greatly contributed to the publication of these volumes within four years of their commencement.

P.J.V.

G.W.B.

R.B.

Acknowledgement

Several illustrations and diagrams in this volume have been obtained from other publications. Some of the original figures have been slightly modified. In all cases reference is made to the original publications in the figure caption. The full sources can be found in the reference lists at the end of each chapter. The permission for the reproduction of this material is gratefully acknowledged.

List of contributors

- John P. Allen
USAF School of Aerospace Medicine, Brooks Air Force Base, Texas 155
- George M. Bedbrook
*Departments of Orthopaedics and Paraplegia, Royal Perth Hospital, Perth,
 Western Australia* 27, 437
- Herbert S. Bell
Cleveland, Ohio 391
- R. Braakman
Department of Neurosurgery, Academic Hospital Dijkzigt, Rotterdam 145, 227
- David C. Burke
Spinal Injuries Unit, Austin Hospital, Heidelberg, Victoria 175
- H. V. Crock
Melbourne 481
- Thomas B. Ducker
Division of Neurosurgery, University of Maryland Hospital, Baltimore, Md. 9
- Robert Goodkin
*Division of Neurological Surgery, Barrow Neurological Institute, St.
 Joseph's Hospital and Medical Center, Phoenix, Ariz.* 1
- Peter Herman (Ben Zur)
Department of Neurology, Mount Sinai Hospital, New York 381
- V.-M. Huittinen
Kyyhkylä Rehabilitation Hospital, Mikkeli, Finland 467
- K. Jellinger
Neurological Institute, University of Vienna 43

- Byron A. Kakulas
*Department of Pathology, University of Western Australia and Department
of Neuropathology, Royal Perth Hospital, Perth, W. Austr.* 27
- R. Lipschitz
Department of Neurological Surgery, Baragwanath Hospital, Johannesburg 197
- L. S. Michaelis
London 141, 145
- L. Penning
Department of Neuroradiology, Academic Hospital, Groningen 227
- Robert Roaf
Department of Orthopaedic Surgery, University of Liverpool 123
- Sydney Sunderland
Department of Experimental Neurology, University of Melbourne 393
- Lionel Wolman†
Department of Neuropathology, Royal Infirmary, Sheffield 221
- David Yashon
*Division of Neurological Surgery, College of Medicine, Ohio State
University, Columbus, Ohio* 209

Contents

Foreword to volumes 25 and 26		v
List of contributors		vii
Chapter 1.	<i>Experimental injuries of the spine</i> – Robert Goodkin	1
Chapter 2.	<i>Experimental injury of the spinal cord</i> – Thomas B. Ducker	9
Chapter 3.	<i>Pathology of injuries of the vertebral column – with emphasis on the macroscopical aspects</i> – Byron A. Kakulas and George M. Bedbrook	27
Chapter 4.	<i>Neuropathology of cord injuries</i> – K. Jellinger	43
Chapter 5.	<i>Biomechanics of injuries of the spinal column</i> – Robert Roaf	123
Chapter 6.	<i>Epidemiology of spinal cord injury</i> – L. S. Michaelis	141
Chapter 7.	<i>Current terminology and classification of injuries of spine and spinal cord</i> – L. S. Michaelis and R. Braakman	145
Chapter 8.	<i>Spinal cord injury at birth</i> – John P. Allen	155
Chapter 9.	<i>Injuries of the spinal cord in children</i> – David C. Burke	175
Chapter 10.	<i>Stab wounds of the spinal cord</i> – R. Lipschitz	197
Chapter 11.	<i>Missile injuries of the spinal cord</i> – David Yashon	209
Chapter 12.	<i>Blast injuries of the spinal cord</i> – Lionel Wolman†	221
Chapter 13.	<i>Injuries of the cervical spine</i> – R. Braakman and L. Penning	227
Chapter 14.	<i>Ocular dysfunction resulting from spinal cord injuries</i> – Peter Herman (Ben Zur)	381

Chapter 15.	<i>Cruciate paralysis</i> – Herbert S. Bell	391
Chapter 16.	<i>Avulsion of nerve roots</i> – Sydney Sunderland	393
Chapter 17.	<i>Injuries of the thoracolumbar spine with neurological symptoms</i> – George M. Bedbrook	437
Chapter 18.	<i>Lumbosacral nerve injuries</i> – V.-M. Huittinen	467
Chapter 19.	<i>Traumatic disc injury</i> – H. V. Crock	481
Index		513

Experimental injuries of the spine

ROBERT GOODKIN

*Division of Neurological Surgery, Barrow Neurological Institute,
St. Joseph's Hospital and Medical Center, Phoenix, Ariz.*

Numerous experiments have been devised in an attempt to study and understand those factors and mechanisms which alter the integrity of the spine and spinal cord separately or together.

Schmaus (1890) introduced the first recognized experimental model to study spinal cord injury. It consisted of a board attached to the back of animals vertically suspended in air, subjecting them to repeated blows delivered to the board. There have been relatively few experiments recently dealing with trauma to the spine. Researchers' interests have tended to focus on the spinal cord injury either alone or in conjunction with the injury of the spine. The latter has been considered of secondary importance since it is the spinal cord lesion with which the patient must ultimately cope.

EXPERIMENTAL MODEL

The majority of experiments have dealt with human cadaver spines subjected to static loading compressive forces either as a single vertebral body, or two bodies with their common disc, or a series of vertebral bodies. Others have studied the influence of bending upon the spine, tensile strength and elasticity of the ligaments and discs subjected to increasing loads, acceleration injury to the spine of cadavers fixed with safety belts in

sleds or ejection seats, as well as acceleration injury in the intact experimental animal.

The author has elected to deal with those manuscripts which in his opinion address themselves primarily to experimental studies related to experimental injury of the spine. An attempt has been made not to conjecture, speculate, or interpret the material presented in the writings, but rather to preserve those authors' conclusions.

The experiments and their results

The disc. The properties of the intervertebral disc were studied initially by Petter (1933), who demonstrated the expansion of the intervertebral disc upon removal from the body and the pressure necessary to reduce that expansion. When the annulus itself was sectioned, further expansion occurred. He was apparently the first to suggest that the annulus exerted a limitation to the acceptance of exogenous fluid introduced into the center of the disc. The amount of fluid which could be forced into the center of the disc varied from 0.8 to 1.5 cm³ before bulging of the disc occurred.

Elasticity and hysteresis of the intervertebral disc were evaluated by Virgin (1951). He subjected a thin slice of bone, bordering each end of the disc, to compression loading and unloading

forces. The discs were reported to withhold great loads without disintegration and recovering after deformation by imbibition of tissue fluid and removal of the deforming force. He stated that the mechanical efficiency of the disc improved with use and during recovery the energy loss became less. Age influenced the elasticity of the disc. The greatest state of efficiency reached by the individual disc is during adult life when the nucleus pulposus is said to have disappeared as an entity.

Hirsch (1955) studied both static loading and dynamically applied stress to the disc. The intervertebral disc was found to react to a statically applied force with a slow change in form. A force of rapid action produced rapid alternating changes in the intervertebral disc, interpreted as indicating that the intervertebral disc acted as a shock absorber.

Nachemson and Evans (1968) reported on the tensile characteristics of the ligamentum flavum (intralaminar ligament) between the neural arches of L3 and L4 vertebrae of 10 post-mortem subjects. They demonstrated the almost perfectly elastic property of the ligament which is said to prevent it from protruding into the spinal canal in extension. The intralaminar ligament was reported to prestress the disc providing some intrinsic stability to the spine. As one would expect, the modulus of elasticity at rupture correlated well with age, being higher in the young than the aged.

The spine. Rechtman (1940) studied the use of pentetrazole (Metrozol) injections to induce convulsions in human patients which resulted in mid-dorsal spine compression fractures. The extent of the injury was attributed to the force of muscular contraction. It was observed and commented that the rupture of the nucleus pulposus with protrusion into the spinal canal was an unusual finding following fractures of the vertebral body. Speculations were made concerning the reasons for the site of injury at the middorsal level.

Perey (1957) performed static and dynamic loading experiments on the lumbar vertebra for the purpose of investigating changes occurring within the vertebral disc during motion and weight-bearing. He extended the experimentation

to test the resistance of the end plate and the vertebral body. Cadaver spines, including a portion of the sacrum, and in some instances the lower thoracic vertebrae, were subjected to a strong force during a short period of time or to the application of a static force. He reported finding both compression and end plate fractures. The end plate fractures varied from central to far peripheral with a portion of the vertebral body dislodged and a transverse fissure extending through the entire end plate. There was no statistical difference in the resistance of the central, lateral and vertebral portions of the normal end plates. Although radiographs did not always demonstrate the fractures, discography and tomography did disclose the fractures in those specimens studied.

Repeated axial compression and repeated transverse bending upon the intervertebral disc and bones of the lumbar spine were investigated by Hardy et al. (1958). Axial compression tests disclosed that severe compression fractures were associated with fractures of the posterior elements. The annulus did not rupture even with repeated compression. On subjection to very high loads the nucleus pulposus could rupture into the adjacent vertebral body. The authors concluded from their studies that with bending, the spine proved to be very weak and that the strength of the spine in bending appeared to come from the composite action of the bones, muscles, and ligaments. Despite tearing of the annulus, the nucleus pulposus ruptured only with gross avulsion.

A comparative study of embalmed versus non-embalmed lumbar spines and pelves of human cadavers, subjected to vertical and transverse loading stresses was made by Evans and Lissner (1959).

Roaf (1960) defined the basic spinal unit (Fig. 1) as that consisting of two intact vertebrae joined by an intervertebral disc, two posterior articulations and a number of ligaments. Subjecting such spinal units to forces of different magnitude and direction (compression, flexion, extension, lateral flexion, rotation and horizontal shear), he studied healthy cervical, thoracic and lumbar spines of fresh human cadavers. His writing discussed certain properties of the disc (the annulus and the nucleus pulposus). He claimed

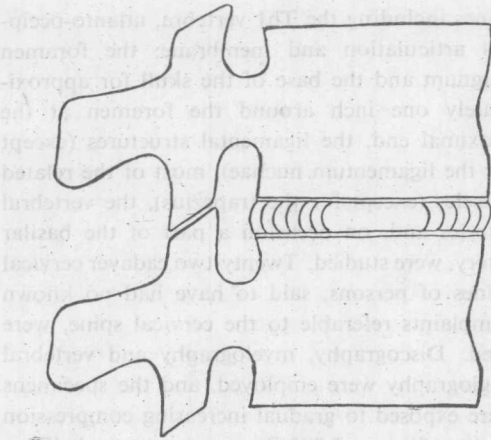


Fig. 1. A single spinal unit. (Roaf 1960.)

that the nucleus pulposus is incompressible, being a liquid; and, according to Roaf, the nucleus pulposus does not alter in shape or position on compression or flexion and has no active role in the production of a disc prolapse. When the nucleus pulposus has lost its turgor, a disc prolapse will take place, consisting primarily of the annulus. The annulus protrudes on the concave and not the convex side. In the normal state it bulges little. The vertebral body mainly absorbs compressive forces and always bulges before the normal disc gives way. The break occurs at the vertebral end plate resulting in a vertical fracture. Hyperextension (hyperdeflexion) and hyperflexion did not produce rupture of the normal spinal ligaments until after the bone had sustained a compression fracture. Ligaments ruptured and dislocations readily occurred with horizontal shear, particularly with rotation forces. Rotational forces were found in general to produce dislocations whereas compression forces produce fractures. The association of a body fracture with spinal dislocation was said to depend on the degree of associated compression. The fluidity of the nucleus pulposus influenced the type of injury sustained. If the fluid nucleus pulposus was mechanically removed and the spinal unit subjected to compression, there resulted an abnormal mobility between the vertebral bodies. The annulus bulged without bulging of the vertebral end plate; no oozing of blood from the vertebral body occurred, but a typical annular disc prolapse took

place. In older subjects in which the nucleus pulposus is no longer fluid, compression of the specimens resulted in either a tearing of the annulus, general collapse of the vertebra due to buckling in of the sides, or a marginal plateau fracture.

Evans et al. (1962) investigated controlled vertical acceleration upon the vertebrae of intact human cadavers and the strains produced.

Compression fractures of the lumbar spine utilizing electrotensometric methods were investigated by Kriukov and Gorobetz (1971). They confirmed the clinical observation that deformation prevails on the frontal part of the vertebral body while the posterior portion remains relatively uninjured.

Plaue (1972) showed that the weight-bearing capacity of the compressed fracture of the vertebral body retained 60–70% of its original capacity. A vertebral body compressed to about one-half of its original height was said to have the same capacity for weight-bearing as a normal vertebral body.

The cervical spine. Although cervical spinal cord injuries are frequently associated with cervical spine fractures and/or dislocations, there is a significant group in which a spinal cord injury exists without concomitant radiologically detectable injury of the spine. Taylor and Blackwood (1948) introduced the concept that hyperextension (deflexion) injuries were responsible for this type of cord damage. Experimentally, Taylor (1951) placed radio-opaque oil in the cervical spinal canal and performed radiograph studies in various positions, disclosing in the full hyperextended (deflexed) cervical spine indentation of the ligamentum flavum cord. This was interpreted as suggesting that the injury to the spinal cord resulted from inward bulging of the ligamentum flavum impinging on the spinal cord.

Beatson (1963), using fresh cadaver cervical spines of young adults, studied the radiographic appearance of the cervical spine after manipulation injury. He demonstrated the importance of oblique radiographs to determine the side of a unilateral facet dislocation. Unilateral dislocation and locking of an interfacetal joint occurred only if the corresponding intraspinal ligament and

joint capsule on the dislocated side were completely ruptured. To cause a bilateral interfacetal joint dislocation, it was necessary to rupture the capsules of both interfacetal joints, the intra-spinous ligaments, the annulus and the posterior longitudinal ligament. Experimentally, it was difficult to reduce a unilateral interfacetal dislocation by a straight pull in the longitudinal axis of the spine. With a slight amount of lateral flexion away from the dislocated side, reduction happened easily. Bilateral interfacetal joint dislocations were considered to be grossly unstable.

Acceleration injuries utilizing cadavers fixed in sleds were studied by Hinz et al. (1969). They found that those specimens with pre-existing cervical disease suffered a greater degree of injury than those without. Pressure gauges were placed in the seat belts and speed photography was employed in their studies.

One of the most comprehensive studies of the experimental mechanisms of injury to the cervical spine and adjacent structures was presented by Selecki and Williams (1970). Cadaver cervical

spines including the Th1 vertebra, atlanto-occipital articulation and membrane, the foramen magnum and the base of the skull for approximately one inch around the foramen at the proximal end, the ligamental structures (except for the ligamentum nuchae), most of the related muscles (except for the trapezius), the vertebral arteries and, on occasion a part of the basilar artery, were studied. Twenty-two cadaver cervical spines of persons, said to have had no known complaints referable to the cervical spine, were used. Discography, myelography and vertebral angiography were employed, and the specimens were exposed to gradual increasing compression forces of up to 5,000 lbs. per square inch (Figs. 2 and 3), positioned in various planes (Fig. 4). Selecki and Williams' findings as summarized by them, are divided as follows:

(a) *Injuries in hyperextension.* Injuries favored the lower cervical region and the following fractures attending pathological and/or radiologic examination of the specimen:

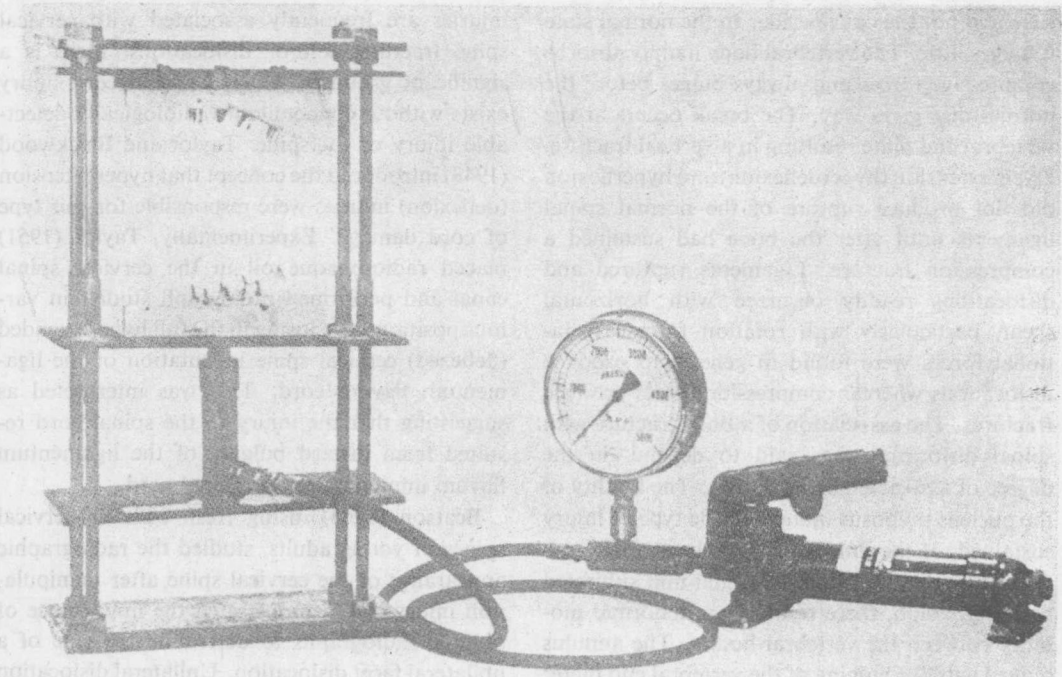


Fig. 2. Apparatus used to exert pressure on specimens of cadaver cervical spines. (Selecki and Williams 1970.)

- disruption of the anterior longitudinal ligament;
- equatorial rupture of the intervertebral disc(s);
- compression of the cervical cord between posteriorly extruded disc material and plications in the ligamentum flavum;
- compression of the radicular arteries;
- avulsion fractures at the anterior vertebral angles;
- fracture of the spinous process;
- fracture of the lamina; and
- fracture/dislocations of the upper cervical spine (appearing only when force was applied directly to the vertex of the specimen).

(b) *Injuries in hyperflexion.* The spinal model contained the following injuries at pathological examination:

- rupture of the posterior longitudinal ligament;
- rupture of the interspinous ligaments;
- avulsion of the ligamentum flavum;
- tearing of the anterior longitudinal ligament at a high level (commonly at C1, C2 and C3);
- rupture of the posterior halves of the intervertebral discs C2/3–C7/Th1, usually equatorial;
- posterior protrusion of the discs;

- separation of the zygo-apophyseal joints;
- anterior wedge fractures of the vertebral bodies;
- anterior chip fractures of the vertebral bodies;
- fracture/dislocation of the atlas on the axis;
- widening of the disc space in the lower cervical spine;
- bilateral zygo-apophyseal fractures;
- fractures of laminae and spinous processes;
- anterior dislocation C2 on C3;
- compression-transection of the spinal cord; and
- stretching and laceration of the vertebral arteries.

(c) *Injuries in rotation, lateral flexion and in combined or multiple directions:*

(1) Extension with rotation:

- equatorial rupture of the C3–4 disc (two specimens);
- equatorial rupture of the C2–3, C3–4, C4–5 discs (one specimen);
- transection of the cord at C1;
- compression of the cord at C3–4 and C6–7;
- tearing and separation of the posterior ligament complex;
- rupture of the interspinous ligaments;
- rupture of the atlanto-occipital membrane;



Fig. 3. Specimen of cadaver cervical spine under pressure. (Selecki and Williams 1970.)

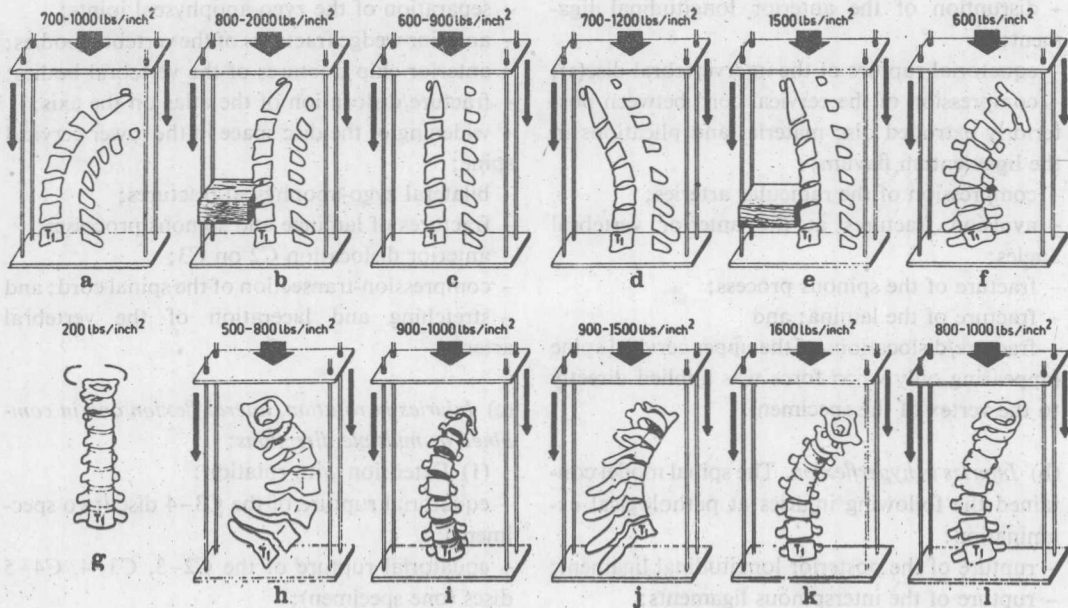


Fig. 4. Schematic representation* of pressure exertion on cadaver cervical spine specimens in various planes. (Selecki and Williams 1970.)

- fracture of the odontoid process;
- anterior dislocation of C1-2-3 and C4;
- separation of the zygo-apophyseal joints;
- compression fracture of the body of C2;
- fracture of the laminae; and
- dislocation of the atlanto-occipital joints.

(2) Flexion with rotation:

- fracture of the spinous processes;
- fracture of the odontoid process;
- posterior fracture dislocation of C2 on C3;
- compression fractures C3, C4, C5;
- fracture of the pedicles;
- rupture of the mid-cervical discs;
- tearing of the anterior spinal ligament; and
- transection of the cord.

Selecki and Williams (1970) suggested that, in contradiction to Roaf's (1960) results, their studies provided ample evidence in middle-aged and elderly specimens with either degenerative or healthy discs, that rupture of the anterior longitudinal ligament in hyperextension occurred as a rule before any damage to the bone structure was observed. They criticized Roaf's basic unit in particular where hyperextension and rotation studies under compression were concerned.

Selecki and Williams stated that hyperextension injuries occurred in the lower cervical spine whereas rotation damage occurred in the upper spine, and Roaf's basic unit was not applicable for comparison. The types of lesion relative to the compression forces in pounds per square inch were identified. The clinical application of the experimental findings were commented on throughout Selecki and Williams' (1970) presentation. Based on their understanding of the pathogenesis of the lesions, they reached certain conclusions concerning treatment. The acute injury of every fracture/dislocation, regardless of the mechanism, is to be treated with skeletal traction, but in extension injuries without fracture or dislocation skeletal traction is contraindicated. They further concluded from their experiments that X-ray evidence of enlargement of the lower disc space with associated neurological findings is evidence of a disc rupture occurring in extension.

An in vivo dynamic experimental model for the study of cervical spine injury and concomitant cervical spinal cord injury was described by Gosch et al. (1972). Macaca mulatta monkeys were subjected to acceleration injury, forces of different direction and magnitude being delivered to the