Symposium on the neurologic aspects of plastic surgery

Edited by

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Volume seventeen

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Proceedings of the Symposium of the Educational Foundation of the American Society of Plastic and Reconstructive Surgeons, Inc., held at Houston, Texas, January 27-30, 1977

with 385 illustrations

The C. V. Mosby Company

Saint Louis 1978

Volume seventeen

Printed in the United States of America

The C. V. Mosby Company
11830 Westline Industrial Drive, St. Louis, Missouri 63141

Library of Congress Cataloging in Publication Data

Symposium on the Neurologic Aspects of Plastic Surgery, Houston, Tex., 1977.
Symposium on the Neurologic Aspects of Plastic Surgery.

(Proceedings of the symposium of the Educational Foundation of the American Society of Plastic and Reconstructive Surgeons; v. 17)
Bibliography: p.
Includes index.
1. Neuromuscular diseases—Surgery—Congresses. 2. Nerves, Peripheral—Surgery—Congresses. 3. Surgery, Plastic—Congresses. I. Fredricks, Simon. II. Brody, Garry S. III. Series: American Society of Plastic and Reconstructive Surgeons. Educational Foundation. Proceedings of the symposium; v. 17. [DNLM: 1. Neurosurgery—Congresses. 2. Surgery, Plastic—Congresses. WL368 S992s 1977]
RD595.S95 1977 617'.483 78-7335
ISBN 0-8016-1679-4

TS/U/B 9 8 7 6 5 4 3 2 1

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The authors wish to dedicate this book
to their colleague

THEODORE F. WILKIE

whose efforts on behalf of cerebral palsy
children were unique.
His death at the height of his abilities
was precipitously premature.
His warmth and presence will be missed.

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Preface

We had a longing to perceive the possibilities still dim and in the distance.

The neuromusculo-receptor marriage is vital to a full-bodied and robust life. The nervous system serves as the coordinating communicator between the senses that reveal our environment and the muscular system, the motor-driven effector. Without one the other is nothing but wasted and lost potential.

To be afflicted by the conditions studied in this symposium is to endure and therefore to be miserable. They are conditions without the compassion of coma or an end point of death. These malfunctions are lifelong, agonizing, and frustrating. These patients truly suffer and deserve our attention.

To cure is elusive and for the foreseeable future perhaps impossible. But surely, to improve, to substitute for, or to mitigate is within our combined capabilities.

Problems of such complexity are best understood by a multidisciplinary approach. The interdigitation of all available knowledge by all concerned modalities was our intent and this volume the fruit of our combined deliberation.

Here we present in a portable, easily disseminated manner the current essence of understanding and therapeutics. The interface of such sincere and inventive workers was inspiring and appeared to produce a rededication of effort.

We wish to express our appreciation to the 125 participants of this symposium who made it possible.

We are humbled by the hopes and aspiration of the thousands whose lives we sincerely pray might be improved by the collection of offerings which is this text.

Simon Fredricks

Garry S. Brody

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Chapter 1

Metabolism of peripheral nerve injuries

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Michael Salcman
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Injury to the whole or any part of an organism causes marked changes in the existing metabolic status. The peripheral nervous system of man is no exception. Knowledge about the metabolic alterations associated with peripheral nerve lesions aids in determining the timing of an operation and the chances of a successful nerve repair.

The central and peripheral nervous systems are a single functioning unit whose purpose is to process information. Impulses travel down the spinal cord and synapse on the anterior horn cell to initiate action throughout the body or the periphery. Peripheral nerves are simply bundles on axons, the long extensions of neuronal cell bodies located within the spinal cord and brain stem. If the central system is diseased, then inadequate or incorrect information will be passed to and from the periphery. If the peripheral system is diseased, the same principle applies. In addition, if a segment of the system dies, for example, as in poliomyelitis, there are not only central nervous system changes but also degenerative changes in peripheral nerves and muscle.

We will approach the metabolic factors in the surgery of peripheral nerves in a dual fashion. The normal metabolic state will be described first. Then the changes in metabolism that are seen with injury or disease will be outlined. These normal and abnormal metabolic states will be reviewed from central to peripheral along the nerves, starting with the central cell or the dorsal ganglion cell and ending with the muscle cell or the sensory end organ.

NERVE CELL BODY

Injury to a peripheral nerve or axon involves removal of a large portion of the nerve cell volume; however, the biochemical machinery required for repair remains largely intact because it is localized in the soma or cell body. Because of their unique geometry nerve cells must synthesize large amounts of structural material and transport these materials over relatively long distances. If the central cell body were the height of an average man, its axon would be 2 to 5 cm in diameter and would extend more than 3.2 km. The energy expenditure needed to synthesize structural materials, as well as factors associated with neurotransmission, and to transport these over long distances becomes very large and requires markedly enlarged and metabolically hyperactive cell bodies. The mature nerve cell is one of the most metabolically active cell types in our bodies.[67] With axonal injury or disease, even higher metabolic activity occurs, and profound changes take place in the structure as well as in the biochemical and physiologic properties of the cell body. Some of these changes may be viewed as particularly appropriate for the repair process. After axonal injury, chromatolysis, nuclear eccentricity, nucleolar enlargement, and cell swelling are the most conspicuous morphologic changes seen during the retrograde response.[47] For peripheral axons these changes have been associated with a reorganization and enhanced formation of cytoplasmic ribonucleic acid (RNA) to a more active state directed toward the reconstitution of lost axoplasm and recovery of peripheral connections.[6,55,69]

Increases in RNA synthesis, as expected, have been associated with enhanced cytoplasmic protein synthesis, demonstrated by the incorporation of radioactive amino acid precursors.[7,24,25,28,69] The changes in protein synthesis are complex and appear to involve a reordering of the various types of proteins synthesized by the injured neuron. Mate-

rials required for transmitter function are decreased, while materials required for regeneration of the axon are elevated. For example, in adrenergic neurons, decreases in dopamine-β-hydroxylase, dopa decarboxylase, monamine oxidase, and tyrosine hydroxylase activities occur after axonotomy.[15,45,60,61] Similar decreases in acetyl cholinesterase and choline acetyltransferase have been reported in axonotomized cholinergic neurons.[37,47] In contrast, the activity of glucose-6-phosphate dehydrogenase, a key enzyme by which glucose is converted to precursors required for the biosynthesis of nucleic acids and lipids, is significantly elevated in axonotomized neurons.[34,35,70]

A number of histochemical and biochemical studies have demonstrated that the activity of glucose-6-phosphate dehydrogenase is increased in axonotomized nerve cell bodies. Spinal anterior horn cells, facial nucleus, neurons, and sympathetic ganglion cells, all of which direct axons to peripheral structures, show such changes in the pentose phosphate pathway.[33,38,46,56,62] Increases in this activity are not observed in neuronal perikarya whose axons undergo wallerian degeneration.[50] In extended studies of metabolic changes in the rat superior cervical ganglion, strong evidence has been obtained that increases in glucose-6-phosphate dehydrogenase occurring shortly after axonotomy are associated with increased metabolism of glucose via the anabolic pentose pathway.[34,35] This increase occurs in the absence of any gross alteration in the major energy-yielding reactions such as ATP utilization and oxygen consumption.

Enhanced metabolism via the pentose pathway may have special significance in injured peripheral neurons because two functions of this pathway are to generate NADPH for reductive biosynthetic reactions and to form ribose phosphate required for nucleic acid and ribonucleotide biosynthesis. Studies carried out on nonneuronal tissue provide strong evidence that the formation of NADPH by the pentose pathway is intimately related to the biosynthesis of fatty acids.[4] A similar relationship may exist in regenerating neural tissuse because significant increases in lipid synthesis begin within the first week after axonal injury.[34,35,52]

The regenerative process in nerve cell bodies is complicated by the simultaneous occurrence of extensive hydrolytic activity during the period of enhanced biosynthetic activity. A marked increase in acid phosphatase and a corresponding increase in the internal complexity and number of lysosome-like dense bodies has been frequently noted in axonotomized nerve cells.[49,65] Matthews and Raisman[49] have suggested that the increased hydrolytic activity is related to a digestion of transmitter storage granules in nerve cells. It is not known whether these changes are a necessary prerequisite for axonal sprouting and regeneration.

The regenerative capacity of neurons varies with age. Axonal outgrowth from proximal stumps of transected nerves appears to be more rapid, and reconnection with denervated end organs occurs more rapidly in younger animals than in older animals.[31,53] Pertinent histologic and temporal characteristics of the retrograde response of various classes of neurons in young animals have been recently reviewed by Brodal.[10] Data concerning biochemical differences between young and mature axonotomized nerve cells is minimal, and it is not clear what mechanisms account for the greater regenerative capacity of neural tissue from immature animals. Nerve cells from very young mammals tend to degenerate when axonal lesions are introduced at or during the first few days after birth.[29] These differences reflect but one aspect of the major biologic problem of the nature and control of cellular differentiation, which is itself poorly understood. Nevertheless, these findings have important practical implications for the physician. For example, the neuron's reparative demands may be readily met in a teenager who has a sharp cut of a digital nerve, indicating immediate repair. On the other end of the spectrum, a person in his 50s with a proximal blast injury to the ulnar nerve does not have significant regenerative powers until 2 to 3 weeks after injury, and even then the neuron may be so busy in an effort to survive that its regenerative efforts are, in essence, ineffective.

SUPPORTIVE GLIAL CELLS

The glial cells form the supportive structure of the central nervous system and are thought to help regulate metabolic events external to the neuronal membrane. They invest the neurons in such a way that sugars, amino acids, and other solutes freely circulating in the extracellular space are made available for the nerve cell's metabolism.[68] Neuroglia also have comparatively high resting membrane potentials and a high internal potassium level, which may serve to distribute potassium ions to aid or create surface potentials in active axons.[11,54]

Glial cells in close association with axonotomized neuronal perikarya undergo alterations that influence metabolic changes accompanying neuronal regeneration. A proliferation of microglia close to perikarya occurs shortly after axonotomy; in some

cases this change is associated with displacement of synaptic boutons from the perikarya and dendrites of nerve cells. Activation of metabolism in glia surrounding injured neurons is suggested by hypertrophy of astrocytes in the vicinity of axonotomized nerve cell bodies.[48] Literature dealing with glial responses during antegrade and retrograde neuronal responses has been recently reviewed.[10] Relevant to defining those events that take place in glial elements and nerve cells, Watson[71] has shown that in contrast to neuronal responses the onset of metabolic changes in astrocytes surrounding nerve cells in the hypoglossal nucleus is independent of the level at which the hypoglossal nerve is injured.

PROXIMAL NERVE TRUNK

Regeneration of a damaged axon requires the restoration of large quantities of axonal lipids and proteins, which are synthesized in the nerve cell body and delivered to the growing axon by transport systems. There is more than one distinct transport process, and materials are transported at different rates. The slow component may be mediated by peristalsis in the nerve trunk plasms, and the fast component involves active participation of microtubules. Inconsistent results have been obtained in studies of the effect of axonotomy on fast axonal transport.[14,26,28,57] These inconsistencies may be explained by the recent findings that axonotomy produces different responses in different nerves of the same animal.[26] Griffin and associates[30] studied fast axoplasmic flow in regenerating rat sciatic nerves. They demonstrated that the rate of fast flow was not altered in the proximal nerve trunk and that the rapidly transported proteins were carried past the level of axonotomy into regenerating nerve sprouts. Materials moved down the sprouts at the normal rate of about 400 mm per day. Although rates of axoplasmic flow may not be altered in the regenerating axon, evidence has been obtained that the amount of protein transported is increased.[30] Functional adaptation of axoplasmic flow in regenerating axons may also involve a reordering of the types of proteins transported. As discussed above, the production of proteins associated with transmitter function seems impaired while formation of proteins associated with the repair process is enhanced in nerve cell bodies that have sustained axonal injury.

PROXIMAL NERVE STUMP

Within 1 hour of cutting a peripheral nerve, there is marked proximal swelling, as much as 1 cm, from the point of disruption. The amount of swelling is greater than previously realized, for the cross-sectional area of the nerve increases three times.[21] The swelling consists of both intracellular and extracellular edema, mostly a gellike amorphous substance that contains a large quantity of acid mucopolysaccharides.[13,59] It persists for a week or more and subsides slowly thereafter.

Within 2 to 3 days after transection of a nerve there are signs of demarcation of the nerve stump or healing over of the open end of the neuron. After a week there is vigorous sprouting of the axons. In traumatic blast wounds the axonal sprouting may be 1, 2, or even 3 cm proximal to the point of the actual severed end. In a clean operative sectioning this occurs for only a few millimeters retrograde. Between 1 and 3 weeks, axon buds begin to advance across the anastomosis of a primary neurorrhaphy.[43,44] The onset of this budding and regeneration occurs concomitantly with the anabolic hypertrophic phase of the cell body in the spinal cord or dorsal ganglion.

Thus all evidence indicates that there is a delay from a few days to a few weeks before the axon buds and crosses a repair site. Thus, operations for the repair of an injured nerve need to be planned when the regenerative efforts of the nerve are at their initial maximum performance.

Axon elongation and the transduction of chemical energy to sustain this process are central issues in the ongoing nerve regeneration. The process of axon elongation appears to originate at growth cones situated at the tip of axons.[74] Growth cones are characterized by a profuse elaboration of microspikes that explore the environment and establish direction for growth.[74] The direction as well as the amount of branching displayed by a growing axon is influenced by adhesion between the cell surface and the substratum upon which it grows.[64] Thus, initiation as well as maintenance of axon elongation may involve changes in the cell surface. Extension of a growing fiber seems to take place at the tip of the fiber.[9]

It is unlikely that sustained axonal growth could occur without the enhanced metabolic activity generated in the cell body during the retrograde reaction. This is supported by the finding that the rate of axonal outgrowth is faster after the second of two successive axonal injuries.[51] Presumably the necessary metabolic adaptation for optimal axonal regeneration has taken place prior to the second injury.[27]

Little information exists concerning the biologic signal that initiates nerve sprouting. Considerable