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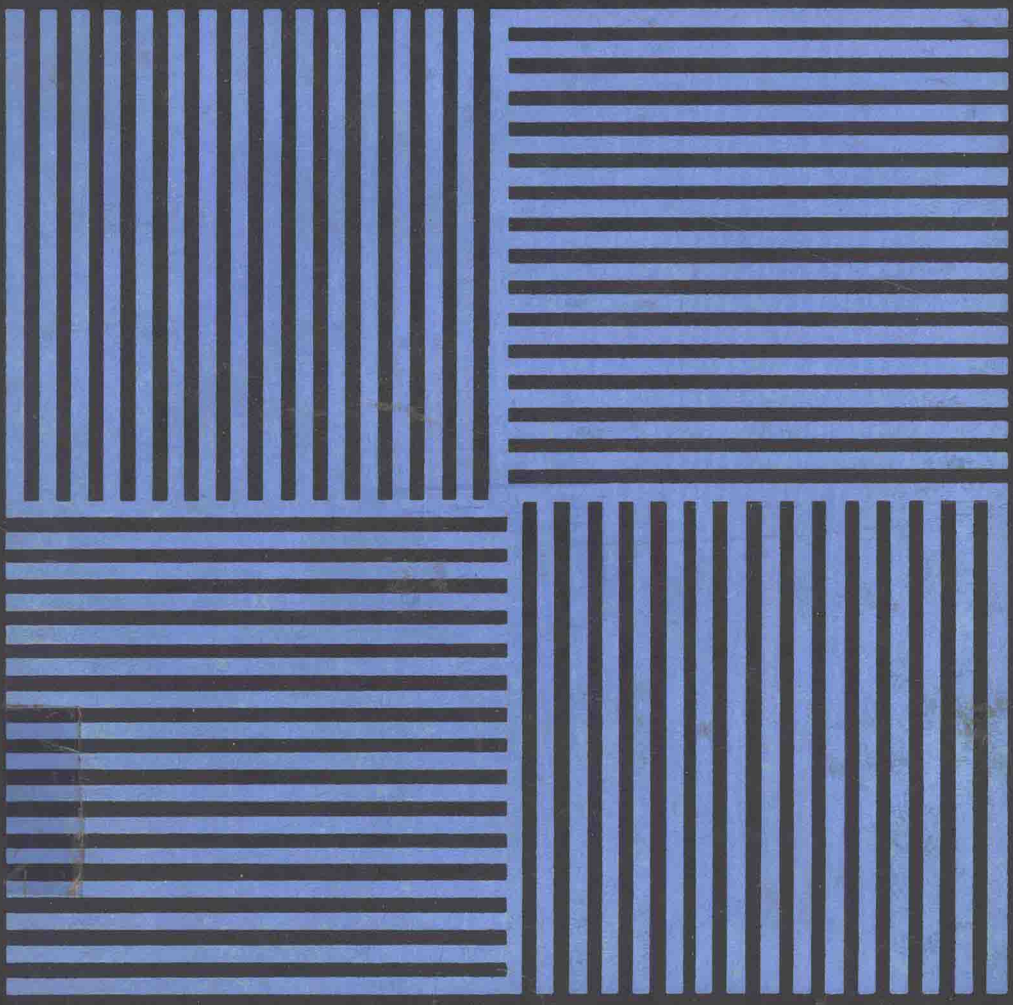

Medical Complications of Quadriplegia

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To the Memory of Samuel Repko, Jr.

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Foreword

Remarkable advances have occurred in the treatment of spinal cord injuries during the past 40 years. Based largely on experience gained in the fields of neurology, neurosurgery, and rehabilitation medicine, a considerable volume of data has accumulated. Both textbooks and the world medical literature have documented the transition from the early days—when fewer than 5% of men in the armed forces who incurred spinal cord injuries survived for more than a year after injury—to the present—when anticipated longevity of patients with spinal cord injuries is close to that of the general population.

Of the spectrum of spinal cord injuries, quadriplegia represents one of the most formidable challenges to even the most astute clinician. Rendering clinical services to quadriplegic patients requires the coordinated and cooperative efforts of a multidisciplinary medical group. The concept of organized team effort is the guiding principle of *Medical Management of Quadriplegia,* which is a thoughtful compilation of the viewpoints and approaches of physicians of various disciplines who are involved in the care of quadriplegic patients. The authors and contributors have created a pithy, important, information-filled work, which should serve to enhance and expand the resources of all colleagues, and especially those who encounter this catastrophic condition relatively infrequently in their specialties and practices.

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Preface

The presentation of both common and uncommon problems of quadriplegia is distinctly out of the ordinary. In pediatrics there is a similar disparity between pathology and presentation. Whereas the pediatric patient is aware of pain but cannot communicate, the quadriplegic can communicate but cannot appreciate pain, distention of a viscus, heat, or any of the other sensations the ordinary patient experiences and upon which we depend so much in our evaluation of the clinical situation.

In a very real sense, the physician who cares for a quadriplegic must reorient his clinical thinking. He has to almost replace the history and instead emphasize the physical examination as well as the interpretation of the laboratory and other ancillary findings.

This book is designed to convey to all who take care of quadriplegics the crucial importance of meticulous, understanding, and sophisticated medical care.

One need not be a neurologist or neurosurgeon to have the necessary understanding of the distorted pathophysiology encountered in quadriplegics. We envisage that, with the increased survival of these patients, they will be cared for in much the same way as other patients, in their homes, doctors' offices, and community hospitals.

It is therefore that much more important that *all* physicians who care for general medical problems learn how to deal with the admittedly unusual but still ultimately predictable sequelae of spinal cord injury.

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Initial Clinical Evaluation and Management

Paul R. Cooper, M.D.

A HIGH DEGREE of suspicion is essential in order that injury to the cervical spine and spinal cord is not overlooked after trauma. As many as 10% of all patients with spinal injuries will develop new or progressive neurologic deficits during the initial stage of management—often from failure to recognize the presence or severity of bony injury.[10]

All patients who complain of neck pain after trauma—*no matter how trivial the injury appears*—should have a plain film examination. Similarly, patients who complain of weakness, hyperesthesias, dysesthesias, or paresthesias in the upper or lower extremities should be suspected of having a spinal cord injury. Patients whose level of consciousness is impaired as a result of head injury or shock must be suspected of having a cervical spine injury until proved otherwise.

A careful neurologic examination is essential to determine the presence of objective deficit and to serve as a baseline for evaluating subsequent improvement or deterioration.[8]

In the upper cervical spine (C1-C3) the spinal cord segments are opposite the same vertebral bodies. Below C4 the cervical spinal segment cord at any level is one-half to one level higher than the vertebral body of the same number.

Thus, the C7 spinal cord segment is opposite the C6 vertebral body. Nerve roots exit between adjacent vertebrae, and from C1–7 are numbered for the higher of the two vertebrae between which they exit. Thus, the C5 root exits between the C4 and C5 vertebrae. There are eight cervical nerve roots and cord segments and only seven cervical vertebrae; between the C7 and T1 bone segments, the nerve root that exits is the C8 root. Below this level the nerve root is numbered after the lower of the two vertebrae between which it exits.

Spinal cord injury at the T1 level will result in incomplete loss of function of the intrinsic hand muscles, including the interossei, lumbricals, and the abductor pollicis longus as well as all spinal cord innervated structures below this level. Injury at the C8 level will result in complete paralysis of the lumbricals and interossei muscles. At the C7 level the triceps muscle that controls extension of the forearms will be affected. Injuries at the C6 level will affect the biceps muscle that controls flexion of the forearm and is supplied by the C6 (and, to a lesser extent, the C5) nerve root. At the C5 level the deltoid muscle that controls abduction of the arms at the shoulders will be affected.

The diaphragm is supplied by spinal nerves C3–5. Thus, in patients who have spinal cord injuries at the C5 level and above, diaphragmatic movement will be compromised to varying degrees. Injury to the spinal cord above C3 will result in total respiratory muscle paralysis. In such patients, early intubation and mechanical ventilation are necessary to sustain life. Even in those patients with injury below C5, respiratory movement will be compromised because of paralysis of the intercostal muscles. Respiratory insufficiency may occur quite precipitously in patients with lesions below C5 as a result of fatiguing of the diaphragm, which must perform the entire work of respiration without the aid of the intercostal muscles. Frequent arterial blood gas studies will give the clinician early warning of a falling P_{O_2} and rising P_{CO_2} even when the patient is not in apparent respiratory distress.

Although there is some anatomic variation from patient to patient, the pattern of sensory loss is also helpful in delineating the level of injury. The undersurface of the proximal arm is

supplied by T1; the fourth and fifth fingers and ulnar aspect of the palm are supplied by C8; the midpalm and middle finger by C7; the first two fingers, radial side of the palm, and forearm by C6; the shoulder by C5; the area just below the clavicle by C4; the lower neck by C3; and the upper neck to the angle of the mandible by C2.

In addition to motor and sensory changes, patients with complete lesions experience spinal shock with absence of deep tendon reflexes and muscular hypotonia. Spasticity gradually appears after three to four weeks with increased (nonvoluntary) muscle tone and hyperactive deep tendon reflexes.

Injury to sympathetic pathways in the ventrolateral white matter of the spinal cord commonly accompanies complete lesions. The clinical manifestations include hypotension to a moderate degree (70 to 90 mm Hg systolic) and bradycardia. Bradycardia is not seen in hypovolemic shock and serves to distinguish this entity from hypotension caused by trauma to sympathetic pathways in the spinal cord.

Ileus and fecal retention are also common in the acute phase. Urinary retention with a large hypotonic bladder is invariably seen with complete lesions and should be anticipated and treated early with catheterization of the bladder.

When there is incomplete motor or sensory loss, the remaining neurologic function sometimes fits into one of several syndromes. Schneider et al.[12] in 1954 described the central cervical spinal cord syndrome, which consists of relatively greater weakness in the arms as compared to the legs. Bladder involvement is inconsistent, and sensory changes are variable. The injury generally occurs following severe hyperextension in patients with narrow spinal canals and osteophytic ridges. The predominance of upper extremity motor deficit is due to the central location of these fibers in the cervical spinal cord. A varying degree of recovery is possible, but many patients are left with permanent deficit.

The syndrome of hemisection of the spinal cord was described by Brown-Sequard in 1850 and is manifest by motor deficit on the side of the lesion, ipsilateral loss of joint position sense, and contralateral loss of pain and temperature sensation beginning one or two segments below the level of the lesion.

At the level of trauma there will also be lower motor neuron deficits from injury to the anterior horn cells.

The syndrome of acute anterior spinal cord injury was described by Schneider in 1955.[11] It occurs as a result of spinal cord compression by herniated cervical discs or fracture-subluxations. Clinical features include total motor paralysis below the level of the lesion, decreased to absent pin sensation below the lesion, and the preservation of joint position sense.

Usually, none of these syndromes prevails and motor loss of varying degrees is present below the level of the injury accompanied by spotty sensory loss without apparent anatomic logic.

RADIOGRAPHIC EVALUATION

All patients who are suspected of having a cervical spine fracture or subluxation because of clinical signs or symptoms, should be treated as if a fracture or subluxation actually exists. Such patients should have their necks immobilized with a collar or have a piece of tape passed over their forehead to hold their heads firm to their bed or stretcher. If possible, the patient should not be moved and a portable x-ray should be taken in the lateral projection on the emergency room stretcher. The C7-T1 junction is often obscured by the shoulders in young husky males and can be visualized by pulling the patient's arms caudally or, if that is not helpful, by performing a swimmer's view.

The diagnostic evaluation of patients with spine injury has been summarized previously in an article by Maravilla et al.[9] In those patients whose films demonstrate fractures or subluxations but who do not have neurologic deficit, appropriate immobilization is established according to principles and techniques that are detailed in subsequent sections. In the past, full definition of the nature of the fracture has been obtained using polytomography. Recent experience with the CT scanner shows that this modality, when skillfully used, can identify fractures as accurately as polytomography. In general, myelography is not performed on patients without neurologic deficit.

In patients with neurologic deficit, the sequence and goals of the diagnostic evaluation are different. Immobilization is instituted in the same fashion as in those patients without deficit and plain films are obtained to define the level and nature of bony injury. In patients with complete loss of motor and sensory function below the level of the lesion, neurologic recovery of any significant degree is unlikely and further radiologic evaluation is limited to defining the pathologic anatomy of the fracture and/or subluxation at a convenient time using polytomograms or the CT scanner.

On the other hand, patients who have neurologic deficit with preservation of some motor or sensory function should have the earliest and most aggressive diagnostic evaluation to determine the presence of spinal cord compression. Plain films are first taken and the site of injury is defined. The patient is kept immobilized in the supine position, the subarachnoid space is punctured laterally at the C1–2 level, and 5 to 6 ml of metrizamide, a water-soluble contrast agent, is introduced. A myelogram is performed to determine the presence of spinal cord compression. If a high-resolution CT scanner is available, transaxial cuts are performed to further define the extent and nature (blood, disc, bone) of spinal cord compression and the anatomy of the bony injury.

The patient with incomplete neurologic deficit and apparently normal plain films presents a particularly difficult problem. The possible diagnoses are several. A small spinal canal with osteophytes that intrude on the canal may contuse the spinal cord in the absence of a fracture or subluxation, an intervertebral disc herniation may be present that cannot be seen on plain films, or a subluxation with spinal cord injury may have occurred at the time of trauma with reduction taking place prior to the time that cervical spine films are obtained. It is essential that all patients with incomplete neurologic deficit and normal cervical spine films have a metrizamide myelogram followed by CT scanning. If the myelogram is normal, plain x-rays in the lateral projection may be taken in extension and flexion to rule out the possibility of an occult subluxation.