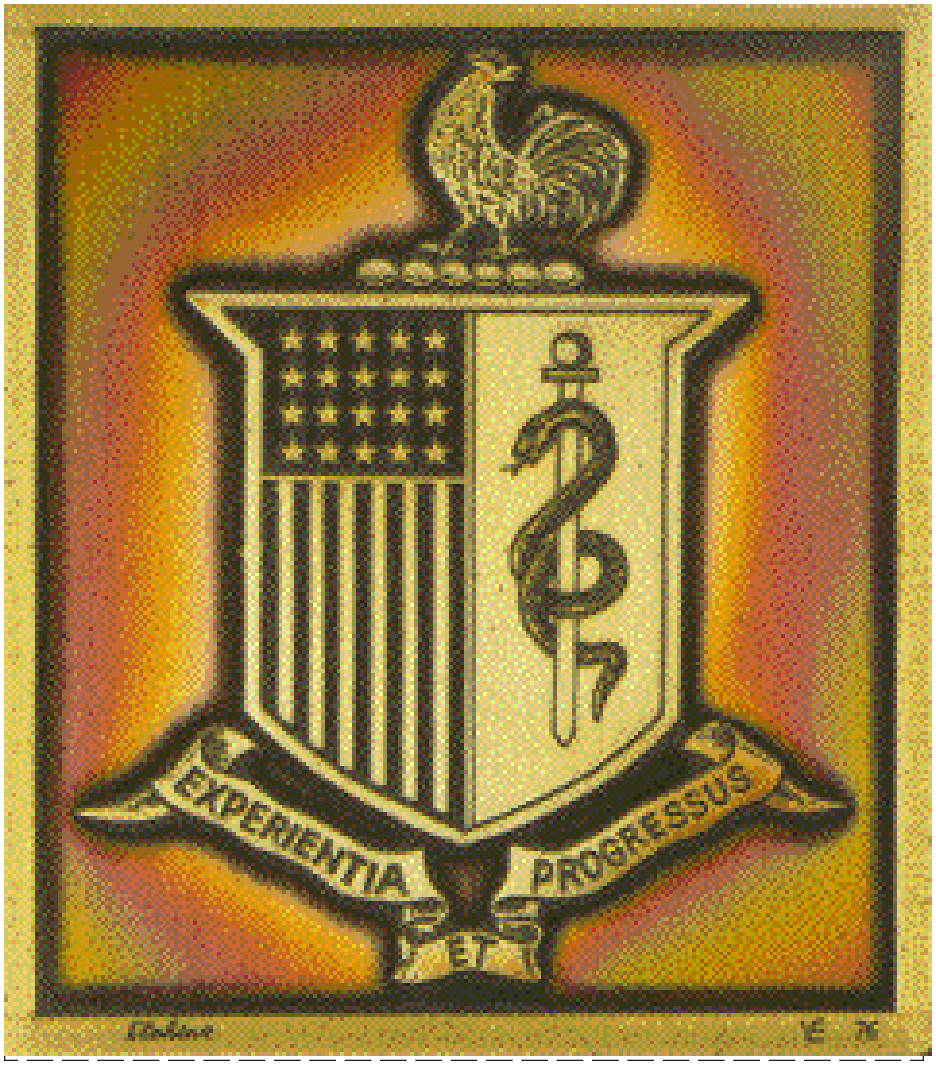
REHABILITATION OF THE INJURED COMBATANT
VOLUME 1



The Coat of Arms
1818
Medical Department of the Army

A 1976 etching by Vassil Ekimov of an
original color print that appeared in
The Military Surgeon, Vol XLI, No 2, 1917

The first line of medical defense in wartime is the combat medic. Although in ancient times medics carried the caduceus into battle to signify the neutral, humanitarian nature of their tasks, they have never been immune to the perils of war. They have made the highest sacrifices to save the lives of others, and their dedication to the wounded soldier is the foundation of military medical care.

Textbook of Military Medicine

Published by the

Office of The Surgeon General
Department of the Army, United States of America

Editor in Chief
Brigadier General Russ Zajtchuk, MC, U.S. Army
Director, Borden Institute
Commanding General
U.S. Army Medical Research and Materiel Command
Professor of Surgery
F. Edward Hébert School of Medicine
Uniformed Services University of the Health Sciences
Bethesda, Maryland

Managing Editor
Ronald F. Bellamy, M.D.
Colonel, MC, U.S. Army (Retired)
Borden Institute
Associate Professor of Military Medicine
Associate Professor of Surgery
F. Edward Hébert School of Medicine
Uniformed Services University of the Health Sciences
Bethesda, Maryland

Executive Officer
Lieutenant Colonel Elias G. Nimmer, MSC, U.S. Army
Deputy Chief of Staff, Comptroller
U.S. Army Medical Research and Materiel Command
Fort Detrick, Frederick, Maryland

The TMM Series

Part I. *Warfare, Weaponry, and the Casualty*

- Medical Consequences of Nuclear Warfare (1989)
- Conventional Warfare: Ballistic, Blast, and Burn Injuries (1991)
- Military Psychiatry: Preparing in Peace for War (1994)
- War Psychiatry (1995)
- Medical Aspects of Chemical and Biological Warfare (1997)
- Military Medical Ethics

Part II. *Principles of Medical Command and Support*

- Military Medicine: Expectation and Reality
- Military Medicine in Peace and War

Part III. *Disease and the Environment*

- Occupational Health: The Soldier and the Industrial Base (1993)
- Military Dermatology (1994)
- Military Preventive Medicine: Mobilization and Deployment
- Medical Aspects of Deployment to Harsh Environments

Part IV. *Surgical Combat Casualty Care*

- Anesthesia and Perioperative Care of the Combat Casualty (1995)
- Rehabilitation of the Injured Combatant. Volume 1 (1998)
- Military Surgery

Exercise Period for Wounded

Artist: Marion Greenwood ca. 1945

Art: Courtesy of US Center of Military History, Washington, DC.

REHABILITATION
OF THE
INJURED COMBATANT
VOLUME 1

Specialty Editors

TIMOTHY R. DILLINGHAM, M.D.
Associate Professor
Johns Hopkins University Hospital

PRAXEDES V. BELANDRES, M.D.
Colonel, Medical Corps, U.S. Army

Borden Institute
Walter Reed Army Medical Center
Washington, DC

Office of The Surgeon General
United States Army
Falls Church, Virginia

United States Army Medical Department Center and School
Fort Sam Houston, Texas

United States Army Medical Research and Materiel Command
Fort Detrick, Frederick, Maryland

Uniformed Services University of the Health Sciences
Bethesda, Maryland

1998

Contract Copyeditor: Ridge Hill Corporation
Joelton, Tennessee

Design and Layout: Lorraine B. Davis
Senior Editor
Borden Institute

This volume was prepared for military medical educational use. The focus of the information is to foster discussion that may form the basis of doctrine and policy. The volume does not constitute official policy of the United States Department of Defense.

Dosage Selection:

The authors and publisher have made every effort to ensure the accuracy of dosages cited herein. However, it is the responsibility of every practitioner to consult appropriate information sources to ascertain correct dosages for each clinical situation, especially for new or unfamiliar drugs and procedures. The authors, editors, publisher, and the Department of Defense cannot be held responsible for any errors found in this book.

Use of Trade or Brand Names:

Use of trade or brand names in this publication is for illustrative purposes only and does not imply endorsement by the Department of Defense.

Neutral Language:

Unless this publication states otherwise, masculine nouns and pronouns do not refer exclusively to men.

Published by the Office of The Surgeon General at TMM Publications
Borden Institute
Walter Reed Army Medical Center
Washington, DC 20307-5001

Library of Congress Cataloging–in–Publication Data

Rehabilitation of the injured combatant, Volume 1 / specialty editors, Timothy R. Dillingham, Praxedes V. Belandres.
p. cm. — (Textbook of military medicine. Part IV, Surgical combat casualty care)
Includes bibliographical references and index.
1. War wounds—Patients—Rehabilitation, 2. Soldiers—Rehabilitation. 3. Amputees—Rehabilitation. 4. Physically handicapped—Rehabilitation. I. Dillingham, Timothy R. II. Belandres, Praxedes V. III. Series.
[DNLM: 1. Wounds and Injuries—rehabilitation. 2. Wounds and Injuries—psychology. 3. Physical Therapy—methods. 4. Amputation—rehabilitation. 5. Military Medicine. UH 390 T355 pt.4 1995]
RD156.R44 1998
617.1'03—dc21
DNLM/DLC
for Library of Congress 90-17790
CIP

PRINTED IN THE UNITED STATES OF AMERICA

08, 07, 06, 05, 04, 03, 02, 01, 00, 99 5 4 3 2 1

Contents

Foreword

Highly skilled soldiers, sailors, airmen, and marines in today's military services possess knowledge of weapons systems that requires considerable training to acquire. These combatants are difficult to replace should they become severely injured during conflict or training. Providing the best medical care possible to America's military sons and daughters and returning these highly trained individuals to duty are, therefore, dual needs of paramount importance. Rehabilitation plays an essential role in the return to duty through both exercise, which prevents contractures, and beginning ambulation at the earliest possible time after injury, which prevents the deleterious effects of bedrest.

Lieutenant Colonel Howard A. Rusk, MD, the US Army Medical Corps officer in World War II who introduced active rehabilitation into Army Air Corps hospitals, and one of the founders of modern rehabilatative medicine, observed that "men did not get ready for full duty playing blackjack or listening to the radio."[1(p463)] It was apparent to physicians in World War I and World War II that physical rehabilitation was supremely important. In order to return to duty, aggressive therapies were necessary soon after injury. For those unable to return to duty immediately, early rehabilitative intervention prevented the effects of immobility and maximized the patients' functional potential.

Rehabilitation must be thought of as a continuum of care spanning the time from shortly after injury to full functional restoration. It is a common misconception that rehabilitative care should be relegated largely to Veterans Affairs hospitals. This argument has been made in the past, but we need only look at historical experience to realize that rehabilitation must begin soon after injury—while the patient is still being treated in military hospitals. During World War II, for example, the army established amputation centers where the highest quality rehabilitation could be provided. But in 1946 the Special Exhibit Committee for Rehabilitation stated:

Delay in inaugurating rehabilitation procedures is the most frequent cause of failure. If there is too much delay in instituting a program of rehabilitation, muscular atrophy, fixation of joints, and mental depression may progress to a point at which complete restoration becomes impossible.[2(p497)]

Far too often the hard-earned lessons of war are forgotten between conflicts. Physical medicine and rehabilitation developed as a specialty as a direct consequence of the great conflagrations of the two world wars. Although medical science has progressed at a phenomenal pace, more than 50 years have elapsed since the last book of rehabilitation specifically regarding war injuries, *Rehabilitation of the War Injured*, was published in 1943. It is fitting that the vastly improved diagnostic and therapeutic rehabilitation interventions be consolidated in the *Textbook of Military Medicine*, a series that will constitute an encyclopedia of combat casualty care. For this reason, this textbook, *Rehabilitation of the Injured Combatant*, will be a valuable reference for the physicians and allied providers who care for those who are injured while fighting for our nation.

Lieutenant General Ronald R. Blanck
The Surgeon General
U.S. Army

April 1998
Washington, DC

1. Rusk HA. The growth and development of rehabilitation medicine. *Arch Phys Med Rehabil*. 1969;Aug:463–466. Editorial.
2. Special Exhibit Committee on Physical Medicine of the American Medical Association. Exhibit on physical medicine: Physical therapy, occupational therapy and rehabilitation. *Arch Phys Med*. 1946;Aug:491–498.

Preface

> This nation has no more solemn obligation than healing the hurts of our wounded and restoring our disabled men to civil life and opportunity. The Government recognizes this and the fulfillment of the obligation is going forward fully and generously. ... It is merely the payment of a draft of honor which the United States of America accepted when it selected these men, and took them in their health and strength to fight the battles of the Nation. They have fought the good fight; they have kept the faith, and they have won. Now we keep faith with them, and every citizen is endorser on the general obligation.
>
> —Woodrow Wilson[1(pv)]

These words from the past ring as true today as they did in 1919. It is the responsibility not only of our nation but also of the medical corps of all the services to ensure the very best care possible for all combatants. This best possible care includes the responsibility to provide the highest-quality rehabilitative care when a soldier, sailor, airman, or marine has sustained a potentially disabling condition. For this reason, a textbook on rehabilitation is considered essential by the Borden Institute.

Rehabilitation traces its roots to the two world wars. The tremendous needs of injured combatants with amputations, severe hand injuries, spinal cord injuries, brain injuries, burns, and nerve injuries stimulated development of this field, which includes physiatry (physicians specializing in rehabilitation), physical therapy, and occupational therapy. As modern warfare has drastically improved its lethality, medicine has also improved its ability to save lives. But during their recuperative phase, almost all those with war wounds need at least strengthening to prevent complications of immobility, and range of motion exercises to prevent contractures.

Medical literature is replete with textbooks on the rehabilitative care of civilians. This textbook, however, focuses on the aspects of care that are specifically related to wounds sustained through combat and military training, for almost all of these require some component of rehabilitation to ensure full functional restoration. The textbook is published in two parts and organized into three sections. The first section introduces the field of rehabilitation, its history, and its functions in the modern military. The second section, the largest and most comprehensive, deals with injury-specific rehabilitation: of burn wounds, nerve injuries, spinal injuries, the special problems of amputees, and so forth. The authors of these chapters produced comprehensive treatises far beyond any preconceived expectations. They have captured the essence of modern rehabilitation and its application to the military. The chapter on preventing complications of immobility is an important contribution; all military physicians and healthcare providers must understand these important principles. The third section deals with exercise and training in ways to prevent injuries, yet maximize performance and strength. In addition, the army's medical boarding system has been outlined admirably and will guide the reader through this complicated system.

In the modern military services, which make substantial investments in training their personnel, vocational restoration encompasses returning to active duty. Depending on the needs of the military, the current national situation, and the special skills possessed by the injured combatants, rehabilitation to the point of return to duty can be an important source of force reconstitution during a conflict.

(Preface continues)

The efforts of the two specialty editors of this textbook, Timothy R. Dillingham, MD, and Praxedes V. Belandres, MD, Colonel, Medical Corps, US Army, have made this two-part volume a reality, and I thank them for their determination to provide nothing less than the best for those in their care. *Rehabilitation of the Injured Combatant* is a welcome addition to the *Textbook of Military Medicine* series. This volume symbolizes the armed forces' commitment to providing the finest rehabilitative care possible to those who "fought the good fight" in service to their nation and in so doing sustained grievous injuries.

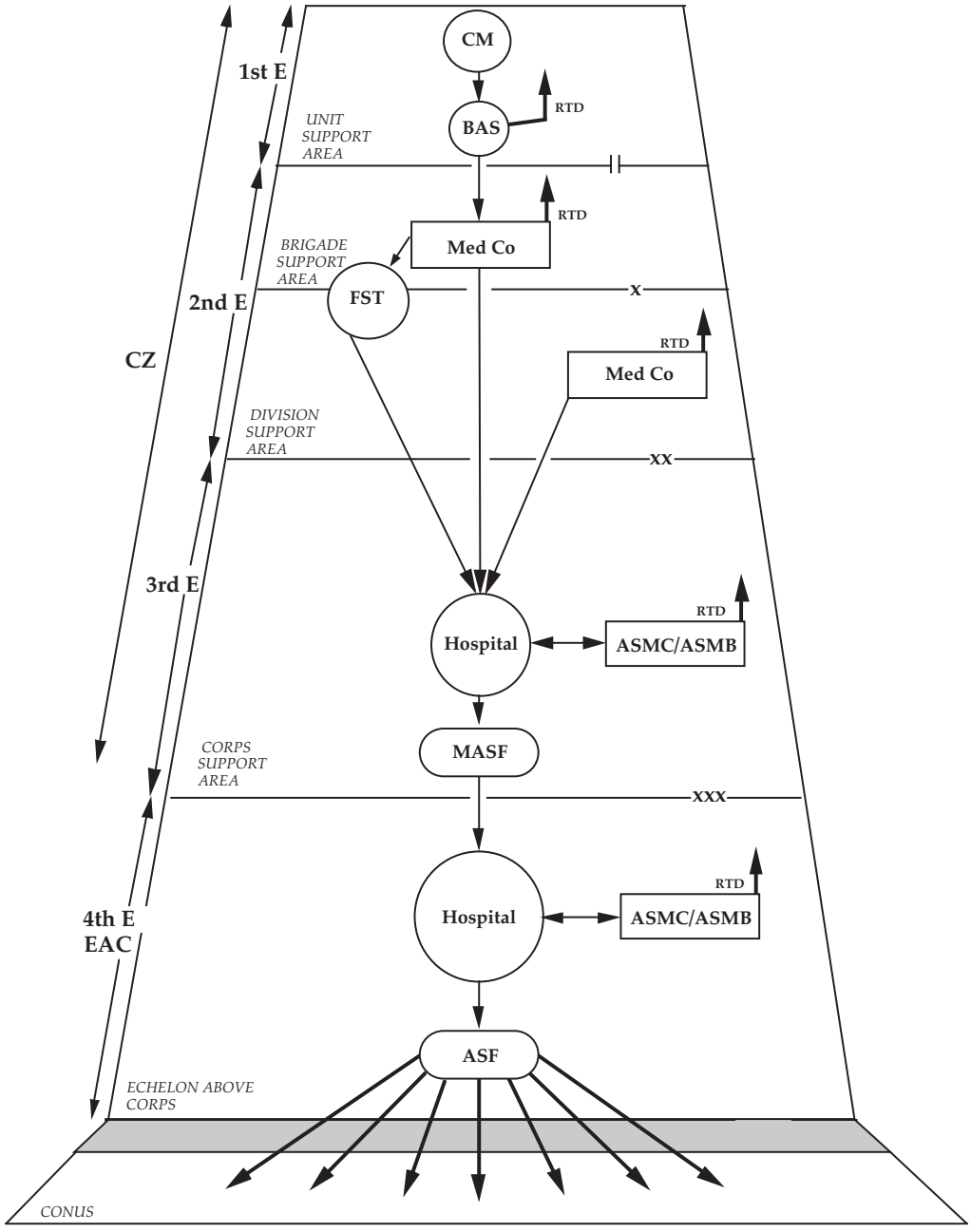
Brigadier General Russ Zajtchuk
Medical Corps, U.S. Army

April 1998
Washington, DC

1. Wilson W. Epigraph. In: Harris G. *The Redemption of the Disabled: A Study of Programmes of Rehabilitation for the Disabled of War and of Industry*. New York, NY: D. Appleton and Co; 1919: v.

The current medical system to support the U.S. Army at war is a continuum from the forward line of troops through the continental United States; it serves as a primary source of trained replacements during the early stages of a major conflict. The system is designed to optimize the return to duty of the maximum number of trained combat soldiers at the lowest possible echelon. Far-forward stabilization helps to maintain the physiology of injured soldiers who are unlikely to return to duty and allows for their rapid evacuation from the battlefield without needless sacrifice of life or function.

Medical Force 2000 (MF2K)
PATIENT FLOW IN A THEATER OF OPERATIONS



ASF:	Aeromedical Staging Facility, USAF	E:	Echelon
ASMB:	Area Support Medical Battalion	EAC:	Echelon Above Corps
ASMC:	Area Support Medical Company	FST:	Forward Surgical Team
BAS:	Battalion Aid Station	MASF:	Mobile Aeromedical Staging Facility, USAF
CM:	Combat Medic	Med Co:	Medical Company
CONUS:	Continental United States	RTD:	Return to Duty
CZ:	Combat Zone		

Chapter 1

PHYSIATRY, PHYSICAL MEDICINE, AND REHABILITATION: HISTORICAL DEVELOPMENT AND MILITARY ROLES

TIMOTHY R. DILLINGHAM, M.D., M.S.*; AND PRAXEDES V. BELANDRES, M.D.†

Formerly, Major, Medical Corps, US Army, Physical Medicine and Rehabilitation Service, Walter Reed Army Medical Center, Washington DC; currently, Associate Professor, Department of Physical Medicine and Rehabilitation, The Johns Hopkins University, Baltimore, Maryland 21239

†*Colonel, Medical Corps, US Army; formerly, Chief, Physical Medicine and Rehabilitation Service, Walter Reed Army Medical Center, Washington DC; currently, Director, Army Operational Medicine Research Directorate, and Director, Physical Medicine and Rehabilitation and Telemedicine Evaluation, US Army Medical Research and Materiel Command, Fort Detrick, Maryland 21702*

INTRODUCTION

Physical medicine and rehabilitation (PMR) has long been intimately associated with the military. In fact, the medical treatment required for war casualties during the conflagrations of World War I and World War II provided the primary stimulus for the extensive growth and development in the field of rehabilitation. The vast experience gained in rehabilitating the many wartime casualties educated military medical officers to the tremendous positive impact of rehabilitation on the care of all patients, not just soldiers. Indeed, the basic tenets of exercise, early range-of-motion exercise, and early mobilization and training, which underwent development and worked well during wartime, still hold true today. To a great extent, the medical specialty within PMR, Physiatry, owes its birth to the pioneering military medical officers who helped established it as a medical specialty in 1947.[1–3] Other rehabilitation professionals, physical therapists, and occupational therapists, also trace their beginnings to the military.

The purpose of this chapter is to summarize the historical development of physiatry and to define the role of PMR in the modern military. Historical support and recent experience will provide the basis for recommending that physiatrists and other rehabilitation professionals be utilized at large medical centers during conflict and peacetime, and at corps level theater hospitals during a major sustained conflict.

HISTORY OF PHYSIATRY

The field of physiatry dates back to the two major wars of the 20th century. Its development was in direct response to the considerable need for wounded soldiers to recuperate and return to duty. Initially, physiatry had close ties to the burgeoning application of physical modalities in the care of injured patients. The name physiatrist (pronounced fiz-ee-at'-rist) is derived from the Greek words *physis*, pertaining to physical phenomena, and *iatreia*, referring to healer or physician.[4] Thus, the physiatrist is a physician who employs physical agents.

During World War I, extensive utilization of "physical reconstruction services" was instituted to improve the functional restoration of injured soldiers. In 1917, Major Frank B. Granger, U.S. Army Medical Corps, was designated director of the physiotherapy service of the reconstruction division. Under his leadership, reconstruction units were set up in 35 general hospitals and 18 base hospitals throughout the United States.[1] Commanders were enthusiastic about the program because many injured soldiers could return to full duty status on discharge from the hospital.

Physicians who practiced physical therapy in the 1920s helped develop the field. Radiologists, strangely enough, were the first group to use physical measures to treat patients, and the first organization for physicians using physical agents was the American College of Radiology and Physiotherapy.[5] However, by 1938 it was obvious that physical therapy physicians had distinctly different interests from the radiologists, and at this time the name of the official journal, *Archives of Physical Therapy, X-ray and Radium*, was changed to *Archives of Physical Therapy*. In 1945, the journal became simply *Archives of Physical Medicine*.[5] Later, it became the *Archives of Physical Medicine and Rehabilitation*, the premiere journal in the field of rehabilitation. Those dedicated and enlightened physicians who implemented physical therapy as part of their treatment during these early years promoted, encouraged, developed, and established the field of rehabilitation medicine. The military history of physical reconstruction[6] illustrates well how occupational aides and physiotherapy aides worked together with physicians in rehabilitating wounded soldiers. Several books were published during and after World War I that described this early rehabilitation.[6–8] These texts described approaches employed in different countries to meet the overwhelming vocational needs of returning soldiers. The U.S. Army system was patterned after those of its allies—Great Britain, France, and Italy—who already had well developed systems for reconstruction. The German army had already perfected its physical reconstruction programs at this time, also.[6]

In the U.S. Army, certain military hospitals were designated as reconstruction centers. Colonel Frank Billings, Medical Corps officer, was chief of the reconstruction division during World War I. He described this program as "continued treatment of patients to the degree of complete physical and functional restoration as is consistent with the nature of their several disabilities."[6(p42)] Following World War I, the reconstruction organization de-

creased in size substantially. During the peacetime years between World War I and World War II, army physical therapy and occupational therapy departments continued to operate, albeit on a smaller scale, due to the vision and foresight of medical officers who advocated and supported these activities.[1] Many of these physical therapy physicians were from Europe and Scandinavia, where hydrotherapy or spa therapy enjoyed great respectability.[5] In 1938, a handful of physicians who practiced physical therapy in the United States founded the Society for Physical Therapy Physicians. These physicians included Dr. John S. Coulter, from Northwestern University Medical Center, Dr. Frank H. Krusen, of the Mayo Clinic, and Dr. Walter J. Zeiter, from the Cleveland Clinic. In 1939, the term "physiatrist" was proposed by Dr. Krusen who recognized the word's similarity to psychiatry and, therefore, suggested its current pronunciation.

The field of physiatry developed rapidly in response to social and medical cataclysms. Between 1939 and 1941, the demand for physiatrists in military hospitals far exceeded the availability. Many army physicians were sent to Rochester, Minnesota, for 3 months of training in physical therapy under Dr. Krusen at the Mayo Clinic.[5] These physiatrists were referred to as "90-day wonders." The focus of physical medicine was broadened from restoration of ambulation and strength alone to comprehensive rehabilitation of the individual—mentally, emotionally, vocationally, and socially.

Dr. Howard A. Rusk was a lieutenant colonel in the U.S. Army Medical Corps during World War II and he reintroduced active rehabilitation into U.S. Army Air Corps hospitals. Rusk noted that during convalescence, deconditioning and boredom were rampant.[9] He also noted that on discharge many soldiers could not return to duty and frequently required readmission to medical care. In his own words,

> I began to realize that military medicine was different from civilian medicine. From a military point of view, you were either a patient or a soldier. If you were a soldier, it meant you got full duty with whatever that involved, perhaps a 10-mile hike with full pack. . . . It seemed obvious to me that men did not get ready for full duty by playing blackjack or listening to the radio.[9(p463)]

Rusk introduced early ambulation and exercise following surgery. Aided by Bernard Baruch, he persuaded President Franklin D. Roosevelt to establish a rehabilitation program in the army air corps. Soon other branches of the military followed suit.[10] In 1943, seven convalescent hospitals were established.[9] "Here," Rusk stated, "*men* were treated, not diseases." During this time, scientific studies began to appear that indicated that early activity had far greater benefits than prolonged bed rest. In fact, in 1944 at the 94th annual session of the American Medical Association, there was a symposium entitled "The Abuse of Rest in the Treatment of Disease."[5] Soon, other military services adopted Rusk's concepts and after World War II, he referred to rehabilitation as "the third phase of medical care." This helped to clarify and to also defuse any possible misunderstanding or animosity about physiatry taking over some aspects of other specialties.

During World War II, several events propelled physical medicine forward. First, a noted philanthropist, Bernard Baruch, formed and financed the Baruch Committee. This committee, chaired by Dr. Ray Lyman Wilbur, was to develop ways to expand the field of physical medicine and maximize its contribution to the care of injured soldiers and sailors.[5] The committee was composed of subcommittees on education, teaching, research, public relations, rehabilitation, hydrology, occupational therapy, prevention, and body mechanics. The insightful work of the Baruch Committee members (which included Dr. Krusen; Lieutenant Colonel Benjamin A. Strickland, Jr., U.S. Army Medical Corps; Charles F. Behrens, U.S. Navy Medical Corps; and Dr. Rusk, to name a few) produced a blueprint for the organized growth, development, and promotion of physiatry. Five recommendations from this committee were: (1) the establishment of teaching and academic research centers at selected medical schools; (2) the establishment of residencies and fellowships in PMR; (3) promotion of teaching and research; (4) promotion of wartime and postwar physical rehabilitation; and (5) the development of an American Board of Physical Medicine under the auspices of the American Medical Association, Council on Medical Education, and the Advisory Board for Medical Specialties.

The Baruch Committee awarded funds to develop physiatry programs at selected universities. Perhaps one of the committee's greatest contributions was to award fellowships to selected physicians, enabling them to receive intensive training in PMR. These individuals included many of the early leaders in physiatry and provided a cadre of well trained academicians to direct residencies and PMR programs.[5]

Around 1945, a section on Physical Medicine and Rehabilitation was established in the American

Medical Association. By February 1947, the requirements for a specialty board were met and the American Board of Physical Medicine (ABPM) was recognized by the American Medical Association Advisory Council for Medical Specialties. Initially, the American Board of Medical Specialties saw no difference between the specialty of physical medicine and that of rehabilitation. Yet during World War II, these factions had been distinct, with the rehabilitation concept and its team approach to care including corrective therapists, occupational therapists, vocational education specialists, and recreation personnel organized under the team physiatrist. Because both factions were included in the ABPM, this continued the competition that had been spawned in the 1930s between physical medicine factions and those championing rehabilitation. In 1949, the ABPM, with guidance from the Advisory Board of Medical Specialties, became the American Board of Physical Medicine and Rehabilitation (ABPM&R),[5] which it remains to this day, and this quelled the discord between these factions.

The next impetus to the growth of physiatry was the polio epidemic of the late 1940s and 1950s.[11] The many patients afflicted with this disease required extensive rehabilitation, with joint, range-of-motion, and other exercises; gait training; activities of daily living (ADL) training; along with orthotic bracing. Concentration of interest in these areas due to tremendous patient needs stimulated PMR to cultivate research, education, and patient care activities, thus advancing the body of rehabilitation knowledge and experience.[11]

Through the 1950s, 1960s, 1970s, and 1980s, continued research, resident training, and greater recognition by other medical specialties firmly established physiatry as a vital, patient-care specialty. The concept of an interdisciplinary team establishing concurrent goals was pioneered by physiatrists and therapists and is now being emulated in other medical and surgical specialties. Legislation during this time (the Vocational Rehabilitation Act Amendments in 1954, the Hill-Burton Act Amendments including special programs for construction of rehabilitation facilities, and the enactment of Medicare and Medicaid in 1965) recognized the need for rehabilitation. The more recent Americans with Disabilities Act of 1990 (Public Law No. 101-336, July 26, 1990), clearly promotes the rehabilitation philosophy in protecting the rights of the disabled. Perhaps the strongest recognition of PMR as a scientific medical specialty was the establishment of the National Center for Medical Rehabilitation Research within the National Institutes of Health. The influential efforts of Dr. Justus F. Lehmann along with many others, led to creation of this landmark institution which solely supports PMR research.[10]

PHYSICAL MEDICINE AND REHABILITATION

Physiatrists are specialty-trained physicians educated through medical school, a year of internship, and three years of residency, then certified by specialty board certification in Physical Medicine and Rehabilitation. Whereas many other medical and surgical specialties have procedures or organ systems that, to a large extent, govern their interventions and define their specialty, physiatry crosses many disciplines. The physiatrist is concerned with the functional restoration of the patient, in addition to the care of medical and surgical needs. Both the army and the civilian sectors accept the role of the physiatrist as a consultant to the primary care team, such as an orthopedic service at a major military hospital, and as the primary physician on a rehabilitation unit.

Physical and cognitive limitations can occur as a result of disease and injuries. The physiatrist addresses the functional and vocational limitations resulting from a soldier's primary injuries. These limitations include muscle and joint contractures, deconditioning due to immobility, ambulation, and ADL impairments, and vocational (military service) impediments. The physiatrist possesses special knowledge of the pathophysiology of disease processes and the functional consequences that impact the patient. In addition, the physiatrist has a broad working knowledge of physical therapy, occupational therapy, and other interventions initiated by the rehabilitation team that can overcome patients' functional problems. Physicians who know the prognosis, the treatments required, and the current precautions regarding limitations of activities that allow healing of injuries, are in the best position to direct the team. For these reasons, a physiatrist, or other primary physician, coordinates the rehabilitation care team.

Rehabilitation services also encompass those professionals who utilize their special training in the prevention and treatment of disability. These include physiatrists, physical therapists, and occupational therapists. In the military, the development of physical therapy and occupational therapy is intimately related to that of physical medicine

and rehabilitation (physiatry). World War I saw the beginnings of physical therapy and occupational therapy. World War II witnessed dramatic expansion of these disciplines born from the necessity to care for vast numbers of war-injured soldiers. The roles of physical therapists and occupational therapists will be briefly discussed as they relate to the PMR team. However, the military specific functions of these professionals are described in other chapters in this textbook. Historical roles during conflict and evidence that strongly supports the vital role of PMR in the military will be presented.

The Rehabilitation Team

The rehabilitation team in the army is typically composed of physicians, physical therapists, occupational therapists, and therapy technicians. The team may also include speech therapists, psychologists, social workers, and nurses. This is referred to as "interdisciplinary team management." In contrast to conventional multidisciplinary care where each specialist forms his or her own diagnoses and set of goals, the interdisciplinary team establishes common goals that all members of the team adopt and implement with the injured soldier. This concept is crucial and provides the best possible rehabilitative care. The importance of close cooperation between physician and therapist was clearly underscored in World War II, when physical therapy was incorporated into the orthopedic sections in all hospitals.[12] Shands and Cleveland, of the OTSG in Washington, DC, stated that: "...the closer the cooperation between the chiefs of orthopedic surgery and physical therapy, the quicker and more satisfactory was their [the patients'] rehabilitation."[12(460)] This continues to be true today in the modern military and civilian sectors.

The rehabilitation team may greatly vary in composition depending on the combat situation, the military mission, and the needs of the military. For example, in an evacuation hospital in Vietnam, a team was composed of physical therapy aides and a physiatrist.[13] In contrast, at Walter Reed Army Medical Center, during the Persian Gulf War in 1991, the rehabilitation teams included nurses, psychologists, physical therapists, occupational therapists, prosthetists, orthotists, and social workers, with physiatrists and primary care physicians directing the rehabilitative efforts.[14]

The rehabilitation team in the British Royal Air Force during World War II consisted of a medical officer responsible for all aspects of care and rehabilitation of patients charged to him,[15] one physical training instructor, and one masseuse. They cared for about 50 patients. A sergeant assisted the team and a sports officer coordinated a variety of athletic activities (golf, tennis, swimming, and volleyball). These activities served to strengthen the recuperating wounded casualty and had "considerable remedial value."[15]

Rehabilitation: Not Just the Department of Veterans Affairs

It is a common misconception that rehabilitative care should be relegated mostly to the Department of Veterans Affairs (VA) Hospital system. This argument has been made in the past, but the historical record indicates that rehabilitative care must begin as early as possible in conjunction with military medical and surgical care. It is then continued throughout the injured soldier's recuperation after transfer to a VA hospital and until maximum functional restoration is achieved, including return to gainful employment or full military service.

At the beginning of the United States' participation in World War II, only the VA was responsible for rehabilitation of the casualty. It soon became apparent, particularly with respect to war-injured amputees, that the VA could not possibly handle the enormous workload.[16] Therefore, the army assumed responsibility for the rehabilitation of amputees and established amputation centers at large military hospitals in the United States.

The concept of early intervention with rehabilitation was recognized even during World War I. In the book *Problems of War and Reconstruction: The Redemption of the Disabled*, the authors stated that

> Again it has been amply demonstrated that the process of restoration, if it is to achieve the fullest measure of success, must be initiated early in the period of convalescence, and must be continuous and uninterrupted, sustaining the man with the inspiration of hope at every moment in his progress back from the front line first aid station, through the base and convalescent hospitals, on board the transport that brings him home from overseas, and during hospital convalescence after his return, until his restoration, physical, functional and vocational, is completed and he is fully established in a suitable employment.[17]

The thrust of PMR care in military hospitals is early intervention aimed at preventing complications of immobility that later hamper full rehabilitation. Rehabilitation, when instituted as soon

as injuries allow, prevents devastating joint contractures, deconditioning, and the psychological trauma of prolonged convalescence. Contractures develop after three weeks of immobilization, and significant deconditioning also occurs in this short time.[18] If soldiers were to wait until they reached a VA facility before getting rehabilitative care, their full functional potential most likely could not be reached. In many cases, if soldiers develop anklosed joints, deconditioning, decubitus ulcers, deep venous thromboses, or other complications of immobility related to not being mobilized and exercised early, they will continue to be impaired despite later rehabilitative efforts by the VA. Additionally the psychological trauma and adjustment to a new disability is lessened with such occupational interventions as hobbies and crafts, which focus a soldier's attention and fill the long hours spent recuperating. For these reasons, rehabilitative care must be available for the injured soldier within a short period of time following injury. The military is charged with providing vital rehabilitative care to injured soldiers, beginning at evacuation hospitals and in medical holding companies where range-of-motion exercise and strengthening exercises can be initiated, and continuing after evacuation to medical centers or prior to returning to duty.

The importance of early rehabilitation of the injured soldier was stressed during World War II in a report by the special exhibit committee on Physical Medicine (a subcommittee of the American Medical Association, under the chairmanship of Dr. Frank H. Krusen) where it was written

> Delay in inaugurating rehabilitation procedures is the most frequent cause of failure. If there is too much delay in instituting a program of rehabilitation, muscular atrophy, fixation of joints, and mental depression may progress to a point at which complete restoration becomes impossible.[4(p497)]

The role of military PMR with its various disciplines (physiatry, physical therapy, and occupational therapy) will vary depending on the current United States situation involving armed conflict; the needs of the military; the tactical situation; and the types, locations, and numbers of casualties being sustained. The remainder of this chapter will provide a historical framework on which to base recommendations for the various roles of physiatrists in the modern military.

PHYSICAL MEDICINE IN THE COMBAT THEATER

The U.S. Army medical corps' mission is "conserving the fighting force"; physiatrists, physical therapists, and occupational therapists greatly enhance and support that goal. The necessities of war demand that the rehabilitation teams in the combat theater be mobile and responsive to the needs of the hospital to which they are attached and, ultimately, to the field command. It must be remembered that the physiatrist is a trained physician and when necessary, due to the tactical needs of the unit, can provide advanced trauma life support, triage casualties, and care for medical problems (diarrhea, hepatitis, pneumonias, minor surgical injuries, and so forth) that frequently occur in army troops. In addition, physiatrists are recognized experts in the care of patients with musculoskeletal injuries. Because musculoskeletal problems frequently occur in active soldiers, a physiatrist can provide forward care for these specific problems. This allows the orthopedists to focus their energies on the many surgical needs of soldiers who sustained war injuries.

At an evacuation hospital during the Vietnam War, physiatrist Dr. Carl Hertzman provided unique insight into the abilities of a rehabilitation team composed of a physiatrist and two physical therapy technicians. The physiatrist and therapy aides supported the surgical subspecialists, primarily orthopedics, and demonstrated that "physical therapy in a combat theater is of considerable value."[13(p114)] Hertzman understood the enormous need for early rehabilitation of those casualties who did not have fractures or other injuries precluding return to duty. In Vietnam, many casualties sustained severe soft tissue injuries from fragment wounds that required extensive tissue debridement. In these casualties, contractures could develop rapidly, leading to marked disability and inability to return to duty. In addition, deconditioning and muscle weakness occurred to such an extent that "within days" severe impairment could result. With proper rehabilitation at an evacuation hospital, 80% of casualties with soft tissue extremity wounds were returned to duty without disability.

Interventions at the evacuation hospital, as described by Hertzman,[13] encompassed three major areas: (1) prevention and correction of disability for those soldiers returning to active duty; (2) early rehabilitation for those casualties who would be evacuated; and (3) proper diagnosis and rehabilitation for the many musculoskeletal complaints, cervical neck pain, lower back pain, and other ailments.

Early treatment while the casualty was at bed rest included fabrication of bivalved casts for the ankle and knee, thereby preventing contractures. Early ambulation was instituted whenever possible, minimizing contractures, weakness, and orthostatic hypotension. Of paramount importance was early initiation of range-of-motion exercise to joints, along with conditioning exercises. The soft tissue injuries to the thigh and calf were particularly prone to producing contractures. Through early range-of-motion exercise—first performed by the physiatrists or technicians, then later performed by the soldier after proper instruction—joint mobility was maintained. Muscles immobilized for a prolonged period can lose 7% of their strength per week and up to 50% after 1 month, but early rehabilitation with isometric and isotonic exercises can prevent this muscle wasting.[18] Hertzman[13] incorporated exercises to strengthen deconditioned and injured muscles.

Modern military medicine, using intervention strategies, saved many casualties during the Vietnam War, with a high percentage of soldiers returned to active duty. Curtis[19] reported that of the 75,000 patients seen at Da Nang Naval Hospital (a corps level hospital) between 1966 and 1970, 87% of the wounded who required hospitalization returned to duty. This implies that at the corps level hospitals in the theater of combat, rehabilitation specialists can improve the physical condition of injured soldiers expected to return to duty.

PHYSICAL MEDICINE AND REHABILITATION AT MEDICAL CENTERS

This section examines the rehabilitative efforts at medical centers in the continental United States during wartime. The literature concerning rehabilitation of the war injured soldier[1,2,4,14,20–23] provides unique insight into the accomplishments of PMR.

World War II

The birth of physical medicine occurred in World War I and greatly expanded in World War II. During World War II, army hospitals utilized PMR services with gratifying results both in large continental United States (CONUS) hospitals and in dispensaries in India and Burma.[1] Beginning with the advent of hostilities in December 1941, new officers who had been involved with physical medicine as civilians were assigned to large army hospitals. The Mayo Clinic and Northwestern University were asked to expand their training of officers in physical medicine.[1] A team of medical officers skilled in physical therapy was sent to smaller hospitals to train technicians in physical therapy techniques.

Care for soldiers with hand injuries was greatly improved during this time by establishment of "hand centers" at various military hospitals. Plastic, orthopedic, and neurosurgeons were given special instruction in salvage procedures and in reconstruction of injured hands.[20] The importance of early hand rehabilitation was stressed by the famous hand surgeon, Dr. Sterling Bunnell, who served as civilian consultant for hand surgery to the Secretary of War. In his words,

> Rehabilitation of an injured hand was always important and frequently difficult. In all patients in whom it was practicable, it was the general rule to institute early motion and mobilization by activity and steady traction.[20(p394)]

Bunnell also espoused the importance of occupational therapy and physical therapy, referring to these combined disciplines as "physical medicine." According to Bunnell,

> In the rehabilitation of the injured hand, occupational therapy played an extremely important role. The patient was assigned a job on the basis of his needs, not just to keep him working. The occupational therapist knew the results desired and devoted her efforts to restoration of the special function which had been lost.[20(p395)]

The Europeans also realized the value of physical medicine and rehabilitation during wartime. Dr. L. Guttmann, neurological surgeon in charge of the Spinal Injuries Center, Stoke Mandeville, Buckinghamshire, England, related the importance of physical therapy in the care of soldiers with paraplegia.[23] Interventions included proper positioning of paralyzed limbs, regular range-of-motion, dressing training, exercises for weak muscles, balance training, wheelchair ambulation, and walking with crutches and braces when feasible. In addition, avocational activities such as wheelchair polo were promoted, to improve the psychological well being of these soldiers.

At the end of the war, Lieutenant Colonel Benjamin Strickland, determined that the staffing goal for physical medicine physicians in the U.S. military should be 33 active duty physiatrists, assuming a permanent army of 850,000.[1] Strickland foresaw the need to train physical and occupational therapists along with enlisted technicians in the

application of rehabilitative treatments. Additionally, the special exhibit committee on Physical Medicine,[4] underscored the importance of an organized coordinated team of occupational therapists, physical therapists, and other rehabilitation specialists under the direction of a physical medicine specialist. This report stated

> Military hospitals have set new standards in physical medicine (coordinated physical therapy, occupatioÓ□Ÿl therapy, and rehabilitation) which must be emulated by civilian hospitals. [4(p494)]

The committee also highlighted the importance of vocational training and the participation of nurses familiar with the rehabilitation team's approach to care. While World War I established orthopedics as a specialty, it is generally felt that World War II did the same for physical medicine.[4]

Commander Thomas J. Canty[24] described amputee care during World War II at the United States Naval Hospital, Mare Island in Vallejo, California, the first Armed Service Amputation Center to be established. Over 2,500 amputees were rehabilitated. Early intervention played an important role, with physical therapists initiating bed exercises and occupational therapists providing arts, crafts, and hobbies as constructive activities during the often long recuperative period. Group support, through round table discussions, helped casualties address psychological issues involved with the new disability; this nurturing group of traumatic amputee soldiers supported each other through shared experiences and feelings. As the soldier improved, aggressive physical training was introduced along with provision of a prosthesis and gait training. Prevocational activities (such as driving, dancing, and various sports) were encouraged, thus facilitating the adjustment to a new disability. Canty[24] even reported the case of a pilot who lost a leg, was rehabilitated at Mare Island, then returned to flying duty.

The Vietnam War

Documented rehabilitation experience during the Vietnam War provides insight into a medical center's efforts to fully meet the needs of amputees resulting from the war. Fitzsimons General Hospital, for instance, treated over 500 soldiers with major amputations, many with multiple amputations.[25] The often complicated rehabilitation included substantial needs for physical therapy, occupational therapy, and prosthetic support; these, along with the psychological needs of the injured soldier, clearly required an interdisciplinary team approach to care.

Colonel Paul W. Brown, [25] Medical Corps, U.S. Army, Fitzsimons General Hospital, Denver, Colorado, reported that prosthetic fitting of amputees was often delayed due to residual limb problems. Because of this, rehabilitation efforts were started early—before transportation to a VA hospital and even before prosthesis fitting—and focused on the "physical and mental conditioning of the patient." Daily exercise routines were established to strengthen weakened muscles, and functional activities were vigorously addressed to ensure that the amputee could perform the basic ADLs: eating, dressing, bathing, toileting, and personal hygiene. Independence in these basic self-care skills provided the casualty with an important level of control over the environment, and contributed to improved psychological well being.

A unique aspect of care at the Fitzsimons General Hospital was the amputee skiing program. Over 100 amputees treated during 1968 and 1969 learned to ski using adaptive aids.[25] These casualties gained confidence and an enhanced sense that even with their disabilities they could find challenges and enjoyment through skiing and other recreational activities. Brown[25] described the incredible psychological trauma involved with amputation, and the Fitzsimons program stressed treatment of the whole individual with the goal of returning the soldier to an optimal level of function. The recreational activities had a positive impact on the mental well being of the soldier and were a vital part of the rehabilitation plan.

Amputees accounted for over half of the civilian casualties referred for rehabilitation in a Canadian medical rehabilitation project in Vietnam.[26] Eighty-five percent of these Vietnamese casualties had amputations involving the leg; the others had upper extremity and multiple amputations.

The Persian Gulf War

During the 1991 Persian Gulf War, a multicenter study assessed the injury characteristics and functional limitations in the casualty population referred to the army PMR services.[14] The results of this study provide insight into the many needs of the war casualty for early rehabilitation. Data were collected on 222 patients seen at participating medical centers: Walter Reed Army Medical Center in Washington DC; Fitzsimons Army Medical Center in Aurora, Colorado; Madigan Army Medical Center

in Tacoma, Washington; Second General Hospital in Landstuhl, Germany; and 97th General Hospital in Frankfurt, Germany.

Historically, musculoskeletal injuries have been significant problems for soldiers during wartime. Mullins et al[27] described the problems encountered with the "low back syndrome" in World War II, where lower back pain was described as one of the most common of all problems encountered. During World War I, General Pershing specifically stated that he wanted "no more men with flat feet, [and] weak backs." [27(p93)] These observations reveal that musculoskeletal complaints, particularly lower back pain, were exceptionally common.

The Persian Gulf War was no exception. At the five reporting centers, musculoskeletal injuries were the most frequently reported diagnoses, followed by nerve injuries, penetrating wounds, then fractures (Table 1-1). Casualties with amputations, burns, brain injuries, and spinal cord injuries were not as common, but these patients required extensive rehabilitative care with physical therapy, occupational therapy, psychological intervention, skilled nursing, and prosthetic and orthotic fabrication.

Fully 44% of all casualties cared for by PMR during the Persian Gulf War had at least one peripheral nerve injury (see Table 1-1). Nerve injuries were closely associated with penetrating wounds, amputations, and fractures (Table 1-2). Well over 60% of all amputees and soldiers with penetrating wounds and over 50% of soldiers with fractures sustained concomitant nerve injuries. This rate is much higher than previous war-related literature would suggest. Omer [28] found that 22% of the soldiers seen with arm injuries had peripheral nerve injuries as well. Nelson, Jolly, and Thomas[29] described brachial plexus injuries in nine cases that resulted from missile chest wounds. In a series of reports[30] describing vascular injuries of wounded soldiers, major nerve injuries were noted in 27.3% of casualties. A similar series by Jacob[31] describes 99 of 258 vascular trauma victims as having nerve contusions with 14 nerve divisions. The higher percentage of nerve injuries seen in Persian Gulf War casualties may reflect a referral bias, but nonetheless illustrates the need for nerve injury rehabilitation.

Electrodiagnostic evaluations are valuable in defining the extent of nerve injuries and were performed for 41% of the casualties referred to PMR services during the Persian Gulf War. According to the guidelines of the American Association of Electrodiagnostic Medicine, only physicians with special training in electrodiagnostic medicine are qualified to perform these consultations.[32] Physiatrists routinely perform electrodiagnostic studies for the army during peacetime, but the Persian Gulf War data documented for the first time the acute need for electrodiagnostic evaluations of injured soldiers during wartime,[14] and there will

TABLE 1-1

PRIMARY DIAGNOSES IN PERSIAN GULF WAR CASUALTIES REFERRED TO PHYSICAL MEDICINE AND REHABILITATION SERVICES

Primary Diagnosis	Number	Percentage
Musculoskeletal Injuries	126	57
Nerve Injuries	98	44
Penetrating Wounds	72	32
Fractures	62	28
Brain Injuries	17	8
Amputations	15	7
Burn Injuries	13	6
Spinal Cord Injuries	6	3

Adapted with permission from Dillingham TR, Spellman NT, Braverman SE, Zeigler DN, Belandres PV, Bryant PR, Salcedo VL, Schneider RL. Analysis of casualties referred to army physical medicine during wartime. *Am J Phys Med Rehabil.* 1993;72(4): 214–218.

TABLE 1-2

ASSOCIATED NERVE INJURIES

Primary Injury	With Coexistant Nerve Injuries (%)
Penetrating Wounds	66
Amputations	65
Fractures	58
Spinal Cord Injuries	43
Musculoskeletal Injuries	40
Burn Injuries	39

Adapted with permission from Dillingham TR, Spellman NT, Braverman SE, Zeigler DN, Belandres PV, Bryant PR, Salcedo VL, Schneider RL. Analysis of casualties referred to army physical medicine during wartime. *Am J Phys Med Rehabil.* 1993;72(4): 214–218.

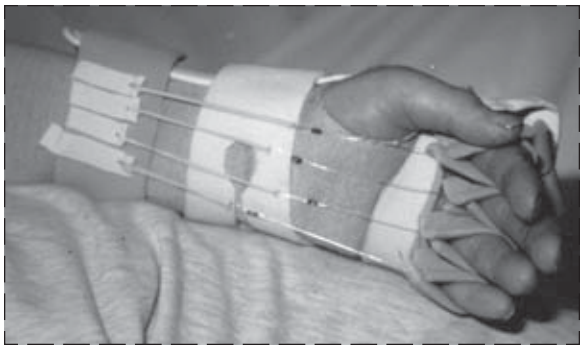


Fig. 1-1. An orthotic used during the Persian Gulf War to improve range-of-motion in the hand of a nerve injured soldier.

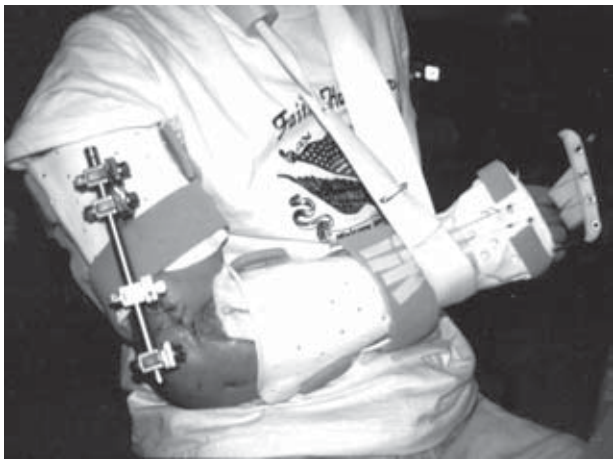


Fig. 1-2. An orthotic used for improving the function of a Persian Gulf War casualty with extensive nerve injuries, a humerus fracture, and a triceps tendon repair. This orthotic provided limited elbow flexion and static stretch to the long finger flexor tendons.

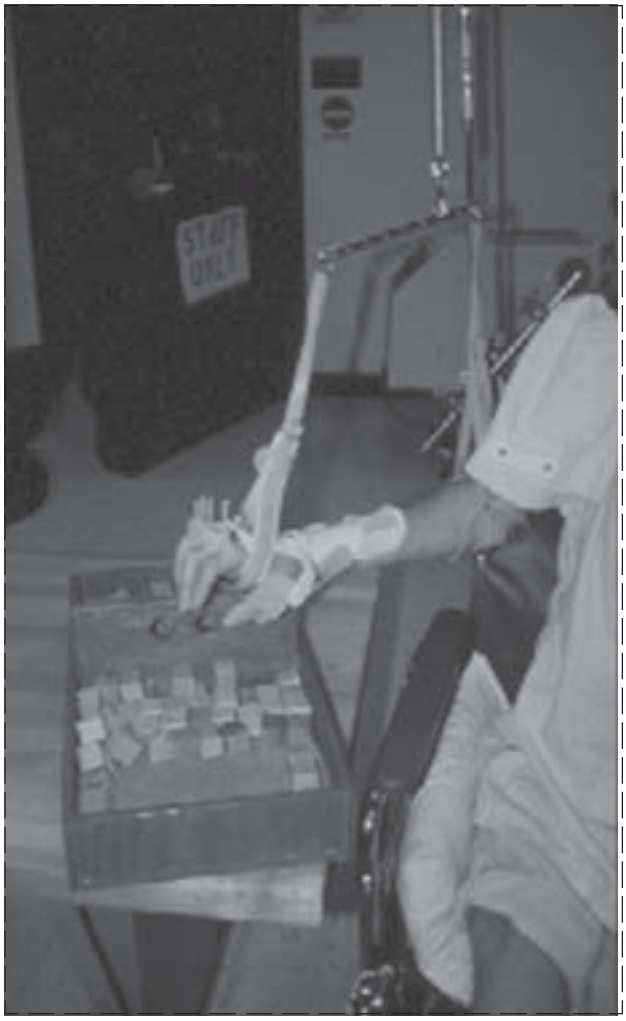


Fig. 1-3. An overhead sling assists weak shoulder muscles, allowing this Persian Gulf War casualty to exercise his right arm and hand despite a humeral fracture and peripheral nerve injuries.

likely be a continued need in future conflicts. The provision of electrodiagnostic consultations at corps level hospitals could enable accurate nerve injury diagnosis in the theater of war without evacuation. Portable commercial electrodiagnostic instruments are available and could be used for this purpose.

Nerve injuries in Persian Gulf War casualties required extensive rehabilitative intervention. Treatment of upper extremity nerve injuries accounted for a substantial part of occupational therapy involvement. Custom orthotics (splints) fabricated by occupational therapists, or therapy assistants were used to prevent deformity (Figures 1-1 and 1-2). Edema from lymphatic disruption and immobility was treated aggressively with elastic wrapping, pneumatic compression, and elevation. Strengthening of the involved muscles, in the case of incomplete injuries, was begun early (Figure 1-3). If the nerve injury was severe, with little chance of improvement, then functional orthotics were fabricated (Figure 1-4), and soldiers were given extensive training in their use and maintenance. Lower extremity nerve injuries were common with the peroneal nerve being most frequently injured. This injury often required orthotics to prevent foot drop (Figure 1-5) and improve ambulation, as well as strengthening, gait training, and range-of-motion exercises.

Amputations occurred in 7% of the casualties, but required considerable rehabilitation. A major goal was the fitting of temporary prostheses (Figures 1-6, 1-7, and 1-8) for both upper and lower extremity

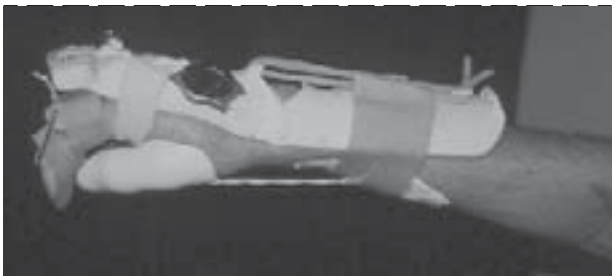


Fig. 1-4. Functional orthotic for a Persian Gulf War veteran with complete median and radial nerve injuries.

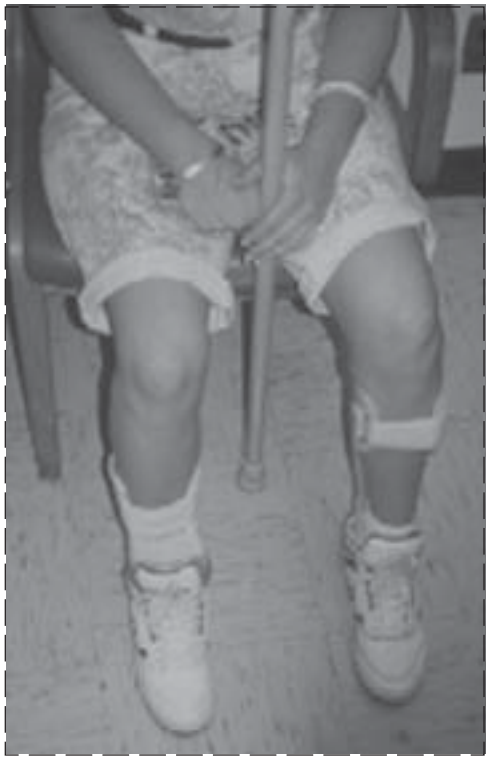


Fig. 1-5. A lower extremity ankle foot orthosis on the right leg, used by this Persian Gulf War casualty for toe pickup during ambulation due to a peroneal nerve injury.

amputees. Temporary prostheses enabled amputees to ambulate at the earliest possible time during recuperation (Figure 1-9). Early ambulation provides enormous psychological benefits to the amputee. In the case of traumatic amputations, early ambulation is possible within two weeks following definitive wound closure. Steinbach dealt with Israeli amputees over a 25-year period and felt it a major rehabilitation goal to fit temporary plaster prostheses for these amputees.[33] Steinbach felt that specialized rehabilitation centers delivered optimal amputee care, and that early vocational interven-

Fig. 1-6. A temporary plaster prosthesis fabricated for a below knee amputee, allowing early ambulation while the residual limb healed.

Fig. 1-7. A temporary adjustable thermoplastic prosthesis for an above knee amputee, allowing early ambulation.

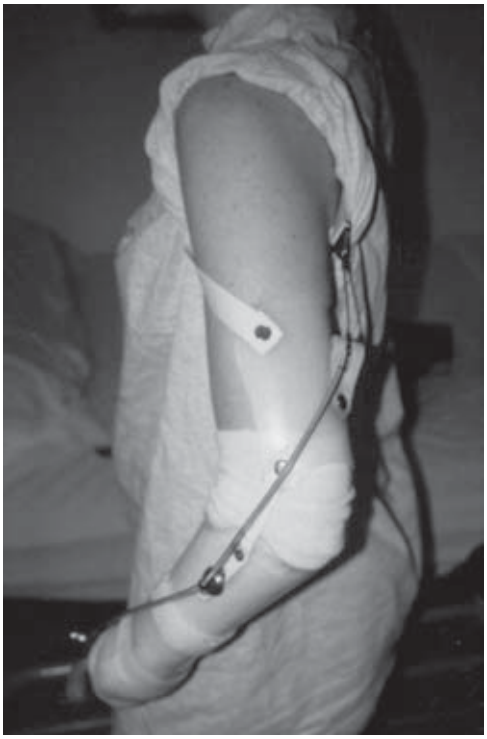


Fig. 1-8. A temporary prosthesis for a below elbow amputee, using a clear thermoplastic socket and fiberglass webbing to incorporate the terminal device (hook). This allowed early training and use of the prosthesis.

Fig. 1-9. A soldier with bilateral leg amputations ambulates in the parallel bars with a temporary prosthesis.

tion returned most amputees to productive work.[33] Nerve injuries were frequently found in Persian Gulf War amputees.[14] These injuries often involved the contralateral lower extremity, requiring orthotic bracing to overcome weakness, thus complicating functional restoration (Figure 1-10). The literature dealing with war amputees[1,16,33] supports the need for skilled prosthetists and orthotists at military medical centers.

The care of an amputee is maximized by a rehabilitation team's early intervention. Only 10% of amputees described by Steinbach[33] had phantom pains, whereas amputees from the Persian Gulf War frequently (80%) manifested phantom limb pain.[14] This difference in the prevalence of phantom pain may have been due to the retrospective nature of Steinbach's study, while the specific questioning of Persian Gulf War casualties probably accounts for the higher frequencies of reported phantom pain. Phantom pain and other residual limb pain syndromes can severely impede the soldier's functional rehabilitation and require a comprehensive approach to management involving early prosthetic fitting, medication trials, physical modalities, and occasional surgical interventions. The physiatrist possesses special knowledge regarding residual limb pain and can best coordinate this multifaceted treatment.

Although not as common, spinal injuries require a comprehensive rehabilitation approach, which addresses medical issues, functional limitations, and patient education. Adler echoed this need for a team approach to patient care.[34] In the United States military, traumatic spinal cord injured patients are stabilized and transferred from military hospitals directly to regional Department of Veterans Affairs Spinal Cord Injury centers.

Burn injuries were diagnosed in only 6% of Persian Gulf War casualties, but were occasionally extensive, requiring considerable rehabilitation (Figure 1-11). Shafir[35] and colleagues described the care of 119 burn casualties from the Lebanon War in 1982. The majority (57%) were sustained among tank crews. Psychological support was felt to be quite important to this casualty population. If the Persian Gulf War had involved greater numbers of intense tank battles, it is possible that more burn casualties might have resulted.

Fig. 1-10. A Persian Gulf War amputee ambulates using a crutch, a left above knee prosthesis, and a right ankle foot orthosis due to a coexistent right peroneal nerve injury.

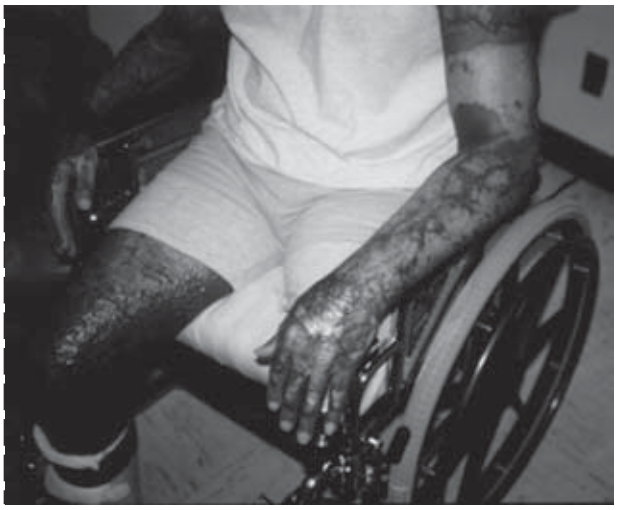


Fig. 1-11. A Persian Gulf War soldier who sustained extensive burns, a left above knee amputation, and nerve injuries in the right leg.

Of the brain injured casualties from the Persian Gulf War, 88% revealed cognitive impairment, requiring additional directed rehabilitation. Brain injured war casualties and their outcomes have been described in previous literature. Groswasser and Cohen[36] studied combat head injuries in Israel, comparing the rehabilitation outcomes of brain injured soldiers in the Lebanon War (1983) to those of their counterparts in the Yom Kippur War (1973). In terms of returning to work, the rehabilitation outcomes were better in the Lebanon War, possibly due to faster patient transfer to primary care hospitals, which yielded shorter periods of unconsciousness, and reduced secondary brain injury. Huusko, Nuutila, and Jarho[37] reported the excellent outcomes of open cerebellar injuries in Finnish War veterans. Katz, Galatzer, and Kravetz[38] described the successful use of sheltered workshops to improve the psychosocial and vocational outcomes in brain injured Israeli war veterans. These studies highlight the need for early rehabilitation of soldiers sustaining brain injuries during war to prevent complications, and initiate early rehabilitation that maximizes the functional outcomes of these soldiers.

The functional impairments noted most commonly among Persian Gulf War casualties involved ambulation.[14] Forty-eight percent of casualties had an ambulation impairment with 29% having limitations in one or more of the ADLs. Management of these problems included strengthening of weakened muscles, improving joint range-of-motion exercise, ADL training, prescription of orthotic devices, and gait training (Figures 1-9 and 1-10). Many of the soldiers required special wheelchair adaptations to accommodate fractures and amputations while allowing maximum mobility during recuperation. Walkers, crutches and canes were frequently required in the short term for early mobilization and for long term mobility in cases of severely injured soldiers. ADL limitations were noted frequently, but the duration and magnitude of the problems were not quantified. To overcome these functional limitations, occupational therapists trained the casualties to use assistive devices and employ compensatory strategies for accomplishing ADL.

The majority of referrals to physical medicine and rehabilitation services during the Persian Gulf War came from orthopedics (64%) followed by neurosurgery.[14] This is in keeping with findings from World War II which indicated that two thirds of the casualties in the European theater of operations sustained orthopedic injuries[27] and supports the close association between orthopedics and PMR services.

During a prolonged conflict, return of trained soldiers to duty may be of the utmost importance, particularly in the smaller and highly technical military envisioned for the future. In this scenario, the functional restoration of wounded soldiers by PMR could make a tremendous difference. Eldar and Ohry reported in their paper on establishment of rehabilitation systems for war that the British Royal Air Force found it "cost and time effective"[22(p106)] to rehabilitate injured air crewman instead of training new ones. This was also noted by Parry[39] in the case of certain highly skilled and valued upper extremity amputees who were retained on active duty in the British Royal Air Force. Israeli literature also reports rehabilitation efforts for selected burn casualties resulting in return to full active military duty.[21]

In summary, the experience during the Persian Gulf War in 1991 underscored Physical Medicine and Rehabilitation's valuable contributions to the optimal care and functional restoration of war injured soldiers.

OCCUPATIONAL THERAPY AND PHYSICAL THERAPY IN THE ARMY

Focus thus far has been primarily on the physiatrist's role in the military as the rehabilitation team leader. It is important to describe the expertise of other rehabilitation professionals who comprise the typical army rehabilitation teams. Other chapters in this textbook provide detailed descriptions of the training and qualifications of all potential members of the optimal comprehensive PMR team, discuss roles of physical therapists in the army, and clarify the special duties of an army PMR team, which are slightly different from those of their civilian counterparts. In general, these therapists treat functional problems of injured soldiers using strengthening exercises, range-of-motion exercises, orthotics, physical modalities, and gait and ADL training the same as in the civilian sector. When physiatrists are present at medical centers, however, physical therapy and occupational therapy departments are organized with physiatrists. When physiatrists are not available, orthopedic services typically incorporate the therapists.

In wartime and peacetime, there is often a shortage of orthopedists and physiatrists to evaluate and manage all of the musculoskeletal disorders in a concentrated troop population. For this reason, occupational therapists and physical therapists are authorized to function as physician extenders, providing primary evaluation and treatment of common musculoskeletal problems. Optimally, physicians skilled in musculoskeletal diagnosis and treatment are readily available for consultation. If the diagnosis remains unclear, or the treatment proves ineffective, early referral by a therapist to a physiatrist or orthopedist is important.

Occupational therapists also perform the vital function of combat stress control. In a war environment, psychological stress can become a major factor, which depletes the strength of an army. Occupational therapists in the theater of combat and at major medical centers provide support for these psychological casualties by providing structured therapies.

CONCLUSION

The evidence presented provides historical examples of physiatrists' roles during wartime. Based on this experience, the authors recommend utilization of physiatrists during wartime in the following roles.

Physiatrists should be placed at selected major medical centers in direct supervision of rehabilitation teams, providing early rehabilitative care to injured soldiers. PMR services here should be prepared to support surgical services, especially orthopedics, provide electrodiagnostic evaluations, and provide comprehensive rehabilitative services. Functional limitations, particularly ambulatory ones, require multidisciplinary rehabilitative care including trained prosthetists, occupational therapists, physical therapists, and orthotists. Designated continental United States medical centers capable of providing comprehensive rehabilitation, similar to the World War II model, can support these services and provide specialized care centers for soldiers requiring extensive rehabilitation. Such an organization functions equally well during wartime and peacetime.

Rehabilitation services at medical centers near troop concentrations also provide valuable peacetime musculoskeletal rehabilitation for soldiers injured during training, speeding their recovery, and optimizing return to duty.

Placement of physiatrists at large corps level hospitals in the combat theater during a prolonged conflict is another wartime role. Here physiatrists assume charge of the rehabilitation team (therapists

or technicians) closely supporting surgeons. In this capacity, physiatrists and therapists reconstitute forces by promoting rehabilitation of those casualties who can be effectively rehabilitated in the combat theater. This strategy also provides early rehabilitation for soldiers being evacuated. Electrodiagnostic evaluations, a major need in the war injured population, could be provided here by physiatrists using small, portable, commercially available electrodiagnostic instruments. This role is optimal during a protracted military involvement, in which case casualties at corps level theater hospitals can be fully rehabilitated during recovery, preventing deconditioning and contractures, and then returned to duty.

REFERENCES

1. Strickland BA. Physical Medicine in the Army. *Arch Phys Med Rehabil.* April 1947:229–236.
2. Garfield RM, Neugut AI. Analysis of warfare. *JAMA*. 1991;266(5):688–691.
3. Kottke FJ, Lehmann JF, Stillwell GK. Preface to the 4th ed. In: Kottke FJ, Lehmann JF, eds. *Krusen's Handbook of Physical Medicine and Rehabilitation*. 4th ed. Philadelphia: WB Saunders; 1990.
4. Special Exhibit Committee on Physical Medicine of the American Medical Association. Exhibit on physical medicine: Physical therapy and rehabilitation. *Arch Phys Med.* August 1946:491–498.
5. Kottke FJ, Knapp ME. The development of physiatry before 1950. *Arch Phys Med Rehabil*. 1988;69:4–14.
6. Crane AG. Physical reconstruction and vocational education. In: *The Medical Department of the United States Army in the World War, Volume XIII.* Washington, DC: GPO; 1927: 42.
7. Kelsey C. *Rehabilitation of the Wounded.* Philadelphia: The American Academy of Political and Social Science; 1918. Reprint. New York; 1980.
8. McMurtrie DC. *The disabled soldier.* New York: Macmillan; 1919.
9. Rusk HA. The growth and development of rehabilitation medicine. *Arch Phys Med Rehabil.* August 1969;463–466.
10. Verville RE. Fifty years of federal legislation and programs affecting PM&R. *Arch Phys Med Rehabil.* 1988;69:64–68.
11. Martin GM. Building on the framework: The academy in the 1950s. *Arch Phys Med Rehabil.* 1988;69:15–19.
12. Shands AR, Cleveland M. Physical therapy reconditioning and occupational therapy. In: Mullins WS, Cleveland M, Shands AR, McFetridge EM, eds. *Orthopedic Surgery in the Zone of the Interior: Surgery in World War II.* Washington, DC: Office of The Surgeon General, US Department of the Army; 1970: 460.
13. Hertzman CA. Special report: Rehabilitation of casualties in a combat theater. *Am J Phys Med Rehabil.* 1968;47(3):113–117.
14. Dillingham TR, Spellman NT, Braverman SE, et al. Analysis of casualties referred to army physical medicine during the Persian Gulf conflict. *Am J Phys Med Rehabil*. 1993;72(4):214–216.
15. Doherty WB, Runes DD. *Rehabilitation of the War Injured: A Symposium.* New York: Philosophical Library Inc; 1943.
16. Peterson LT. Administrative considerations in the amputation program. In: Mullins WS, Cleveland M, Shands AR, McFetridge EM, eds. *Orthopedic Surgery in the Zone of the Interior: Surgery in World War II.* Washington, DC: Office of The Surgeon General, US Department of the Army; 1970.
17. Harris G. *Problems of War and Reconstruction: The Redemption of the Disabled.* New York: Appleton; 1919: 24.
18. Halar EM, Bell KR. Rehabilitation's Relationship to Inactivity. In: Kottke FJ, Lehmann JF, eds. *Krusen's Handbook of Physical Medicine and Rehabilitation.* 4th ed. Philadelphia: WB Saunders; 1990.

19 Curtis DC. Military medicine from World War II to Vietnam. *JAMA.* 1990;264(17):2259–2262.

20. Bunnell S. *Hand Surgery.* Washington, DC: Office of The Surgeon General, US Department of the Army; 1955: 394–395.

21. Van Straten O, Mahler D. Pressure garments in the control of hypertrophic scarring and rehabilitation of the burn patient. *Isr J Med Sci.* 1984;20(4):320–322.

22. Eldar K, Ohry A. Preparation of a hospital rehabilitation system for war and other disasters. *Med War.* 1990;6(2):105–111.

23. Guttman L. Problems of physical therapy for persons with traumatic paraplegia. *Arch Phys Med.* December 1946:750–756.

24. Canty TJ. Amputee Rehabilitation. *Arch Phys Med.* 1951;32(April):219–221.

25. Brown PW. Rehabilitation of bilateral lower extremity amputees. *J Bone Joint Surg Am.* 1970;52-A(4):687–700.

26. Huston AF, Gringas G. The Canadian medical rehabilitation project in Vietnam. *Can Med Assoc J*. 1972;107:1213–1218.

27. Mullins WS, Cleveland M, Shands AR, McFetridge EM, eds. *Orthopedic Surgery in the Zone of the Interior: Surgery in World War II.* Washington, DC: Office of The Surgeon General, US Department of the Army; 1970: 9,93, 826.

28. Omer GE. Injuries to nerves of the upper extremity. *J Bone Joint Surg Br.* 1974;56-A(8):1615–1624.

29. Nelson KG, Jolly PC, Thomas PA. Brachial plexus injuries associated with missile wounds of the chest. *J Trauma.* 1968;8(2):268–275.

30. Levitsky S, James PM, Anderson RW, Hardaway RM. Vascular trauma in Vietnam battle casualties. *Ann Surg.* 1968;168(5):831–836.

31. Jacob JE. Vascular injuries in Vietnam: With special reference to compartment syndrome. *Int Surg*. 1972;57(4):289–293.

32. American Association of Electrodiagnostic Medicine. Guidelines in electrodiagnostic medicine. *Muscle Nerve.* 1972;15(2):229–253.

33. Steinbach T. Total Rehabilitation for amputees in special conditions. *Prosth Orthot Int.* 1977;1(2):125–126.

34. Adler E. Spine injuries during the Six–Day War. *Paraplegia.* 1969;7(1):28–33.

35. Shafir R, Ahronson Z, Hirshowitz B, et al. Various approaches to burn care in the Lebanon War, 1982. *Isr J Med Sci.* 1984;20(4):314–319.

36. Grosswasser Z, Cohen M. Rehabilitation outcome of combat head injuries: Comparison of October 1973 War and Lebanon War, 1982. *Isr J Med Sci.* 1985;21(12):957–961.

37. Huusko S, Nuutila A, Jarho L. Open cerebellar injuries in Finnish war veterans. *Scand J Rehabil Med.* 1974;6: 152–157.

38. Katz S, Galatzer A, Kravetz S. The physical, psycho-social and vocational effectiveness of a sheltered workshop for brain-damaged war veterans. *Scand J Rehabil Med.* 1978;10:51–57.

39. Parry CBW. The management of injuries to the brachial plexus. *Proceedings of the Royal Societies of Medicine.* 1974;67:488–490.

Chapter 2

PHYSICAL THERAPY IN A WARTIME ENVIRONMENT

DAVID G. GREATHOUSE, PH.D., P.T.[*]; JANE SWEENEY, PH.D., P.T., P.C.S.[†]; AND
ANN M. RITCHIE HARTWICK, PH.D., R.D.[‡]

[*]*Colonel (Ret), Medical Specialist Corps, US Army; formerly, Chief, Medical Specialist Corps, Headquarters, Office of The Surgeon General, Falls Church, Virginia; currently, Professor and Chairman Department of Physical Therapy, Belmont University, Nashville, Tennessee 37212-3757*
[†]*Formerly, Colonel, Medical Specialist Corps, US Army, Chief, Clinical Investigation and Research, Walter Reed Army Medical Center, Washington, DC; currently, Consultant in Physical Therapy, Tacoma, Washington 98335*
[‡]*Colonel (Ret), Medical Specialist Corps, US Army; formerly, Historian, US Army Center of Military History, Washington, DC 20374-5088*

INTRODUCTION

Military physical therapists (PTs) are essential members of the healthcare team and have proven their worth in rehabilitation and preventive care during peace and war. The mission of military PTs is to provide physical therapy evaluation and treatment to correct or prevent physical impairments resulting from injury, disease, or preexisting problems. This is done via utilization of physical modalities and therapeutic exercise to increase joint mobility, muscle strength, and cardiovascular endurance. Military PTs also serve as independent practitioners and nonphysician healthcare providers when administering primary care to patients with neuromusculoskeletal (NMS) conditions. Military PTs serve as technical advisors to commanders of troop units providing guidance in the areas of physical fitness, physical training, and injury prevention. During mass casualty situations, PT personnel assist in managing patients categorized as delayed or minor, and also augment the orthopedic section.

Historically, the requirement for extensive physical reconditioning of large numbers of soldiers committed by the United States in World War I was a major catalyst in promoting the formation of the profession of PT in the United States. Army PTs lead their profession and excel in clinical practice, education, and research. To observe the professional contributions that military PTs have provided in peace time and war time, the Diamond Jubilee (75th Year Anniversary: 1918–1993) of military PT education in the United States was celebrated at the 1993 Annual Conference of the American Physical Therapy Association.

The purpose of this chapter is to describe the past, present, and future professional roles of U.S. Army PTs. A perspective of the historical function of army PTs in World Wars I and II, the Korean War, and the Vietnam War is presented. During the Persian Gulf War, army PTs provided medical support services in Southwest Asia, Europe, and the continental United States (CONUS). An analysis of the involvement of army PTs in the Persian Gulf War is also provided.

The experiences of army PTs deployed in field training exercises and humanitarian missions to third world countries are also discussed. In addition, the evolving role of military PTs as nonphysician healthcare providers in the evaluation and treatment of patients with NMS conditions is described. As the U.S. Army Medical Department (AMEDD) progresses into the 21st century, army PTs will continue to perform their professional duties within the emerging structure of Medical Force 2000 (MF2K).

U.S. ARMY PHYSICAL THERAPY IN THE 20TH CENTURY

World War I

At the outbreak of World War I, the use of reconstructive therapy utilizing massage, electrotherapy, exercise, and hydrotherapy was not well established in the United States. Because the number of reconstruction aides needed by the Division of Special Hospitals and Physical Reconstruction was not obtainable from the nation's manpower pool in 1917, the Office of The Surgeon General (OTSG), U.S. Army, established emergency training programs for reconstruction aides in 1918[1] and the army recruited and trained "reconstruction aides" to fill this void in healthcare.

Reconstruction aide requirements included completion of at least a four-month course in theoretical and practical physiotherapy in two of the following modalities: hydrotherapy, mechanotherapy, massage, or electrotherapy. Also required were 240 certified hours of active clinical work, completion of a secondary school education, and a physical examination. The aides could be married and included men as well as women. Typically, the first reconstruction aides to serve in World War I were teachers of physical education in colleges and universities.[1]

Mary McMillan was an American PT trained in England under Sir Robert Jones, a noted orthopedic surgeon, and had attended war wounded at a base hospital in England. During World War I, McMillan structured the education programs for the reconstruction aides.[1] These programs were conducted in 7 of the 14 civilian institutions whose curricula met standards set by the OTSG. The courses taught at Reed College in Eugene, Oregon, contributed the largest number of graduates (over 200) for the U.S. Army Medical Department. To organize administrative details of the reconstruction program, establish the training curricula, and prepare reconstruction aides for overseas duty once they were trained, Marquerite Sanderson was appointed the first supervisor of reconstruction aides in the OTSG.[1]

Fig. 2-1. World War I—Army physical therapist instructing a below-knee amputee in range-of-motion and strengthening exercises. Photograph: Archives, U.S. Army Center of Military History, Washington, DC.

In February 1918, at the request of The Surgeon General, McMillan left Boston and joined Sanderson to assume duties as the head reconstruction aide at Walter Reed General Hospital in Washington, DC. In late 1919, she was appointed Superintendent of Reconstruction Aides in Physiotherapy, OTSG, and completed the first book to be published in the United States on physical therapy, *Massage and Therapeutic Exercise*.[1] McMillan is considered by many to be the founder of physical therapy in the United States and was elected the first president of the American Women's Physical Therapeutic Association in 1921.[2]

The stated mission of the U.S. Army Medical Department was to heal the injured soldier and to equip him with the training and skills necessary for becoming an effective member of society both industrially and socially.[1,2] In support of that mission, physiotherapy reconstruction aides of World War I served in large hospital complexes at home and abroad (Figure 2-1). Military hospitals in the United States were constructed, some entirely new structures, and some extensive additions to established hospitals. The reconstruction aides treated soldiers with battle injuries, including those caused by gases (chlorine, phosgene, mustard, and arsine) and shrapnel damage to the extremities.[1] The aides also managed patients with infectious diseases, especially influenza and pneumonia. Patients' hospital stays were often extended over several months until rehabilitation was completed.

In July 1920, 175 physiotherapy reconstruction aides were employed by the army, and by June 1921, the specialty was practiced in six U.S. Army hospitals.[1] In 1926, the term "reconstruction aide" was discontinued by directive of The Surgeon General, and the more definitive title "physiotherapy aide" was instituted. Following World War I, physical therapy was recognized in the United States as a profession, training programs were instituted, formal professional societies were formed, and civilian salaries for the specialty were increased.

World War II

Between 1934 and the onset of World War II, the number of physical therapists in the U.S. AMEDD remained consistent, absorbing graduates of the army training programs and allowing for attrition. PTs maintained civil service status during this time, and there was no longer a permanent staff position in the OTSG. Professional staff input concerning PT practice and personnel was provided to OTSG by Colonel Emma Vogel, who had served as an assistant instructor under McMillan. Colonel Vogel had assumed the chief's position at Walter Reed General Hospital in 1922, when McMillan returned to civilian practice.[1]

In September 1939, in response to the militarily enforced expansionist policies of Italy and Japan, and shortly after Germany occupied Austria and Czechoslavakia, President Franklin D. Roosevelt declared a national state of emergency. The President's declaration initiated a massive training effort by the military services to acquire and develop personnel necessary for overseas deployment. The anticipated army requirements for PTs far exceeded the staffing ceilings of the U.S. Army Medical Department,

and also exceeded the number of PTs recorded by the national professional organizations.[1,2]

Following Japan's attack on Pearl Harbor, the United States entered World War II. As in World War I, The Surgeon General , U.S. Army, organized hospitals to support the armed forces. Civil service PTs were recruited and trained by the army, and then deployed to military hospitals in the United States and overseas. U.S. Army physical therapists served in every Theater of Operations (TO) during World War II.[1]

In July 1943, the Reconditioning Division of the OTSG was established to direct an armywide reconditioning program under the leadership of Lieutenant Colonel Walter E. Barton, Medical Corps. In this division, Colonel Vogel was appointed Superintendent of the Physical Therapy Branch, OTSG.[2] As in the first World War, U.S. Army physical therapists played a major role in World War II in the evaluation and treatment of injured, wounded, and sick servicemen of our armed forces (Figure 2-2).[1]

On June 22, 1944, Public Law 78-350 was passed by Congress (House of Representatives, 78th Congress, 2nd Session). Known as the Bolton Bill in honor of Representative Frances P. Bolton, who introduced it, this milestone bill granted commissioned status for dietitians, physical therapists, and nurses in the U.S. Army, granting them the same allowances, rights, benefits, and privileges as other commissioned officers. The news of this landmark legislation and the protection under the war clause for women serving overseas was welcomed at home and abroad.[1,2]

PT equipment used during World War II had been evaluated and standardized in the late 1930s through efforts of the American Medical Association's Council on Physical Therapy.[1] In 1939, U.S. Army equipment purchase requests were processed in anticipation of wartime requirements. Even so, in the early months of the war in remote locations, PTs often rehabilitated the wounded with improvised equipment, demonstrating their ingenuity and a "can do" work ethic. Mess tables were used as plinths, and weights for resistance exercises were made from cans filled with dirt, rocks, and sand. Large basins and bathtubs became whirlpools, and water for these whirlpools was heated on field ranges. Bamboo, scrap lumber, and salvaged parts from vehicles or aircraft were used to construct and adapt inventive apparatus for treatment.[1]

By August 1945, 1,300 PTs were on active duty in the U.S. Army, and approximately 570 of them were assigned overseas in all theaters. Two of these American PTs, Brunetta A. Kuehlthau and Mary McMillan, were captured by the Japanese and interned in prison camps in the Philippines and China, respectively.[1] McMillan was repatriated in 1943 in China, and Kuehlthau was liberated by U.S. forces in February 1945.

The Korean War

In June 1950, North Korean forces accompanied by Soviet advisors and equipped with Soviet military hardware crossed the 38th parallel to invade South Korea. Within hours, President Truman di-

Fig. 2-2. World War II—Therapeutic Exercise Section, Physical Therapy Clinic, Walter Reed General Hospital, Washington, DC. Photograph: Archives, U.S. Army Center of Military History, Washington, D.C.

rected the United States Far East Command to use American combat forces in support of the Republic of Korea. The Korean War provided the first opportunity for the Women's Medical Specialist Corps (WMSC) to support the army medical department in a wartime situation.[1] The WMSC, which was established in 1947 by Congress, was composed of occupational therapists, physical therapists, and dietitians. In contrast to World War I and World War II, the WMSC facilitated the assignment and training of physical therapists, occupational therapists, and dietitians from both active and reserve components.

Led by Colonel Vogel, the WMSC increased and expanded training programs for physical therapy and began a recruitment program using corps officers in army area headquarters. The corps leadership appointed prominent members of the civilian specialties as consultants. By June 1953, the WMSC was composed of 607 women, 201 regular army and 406 reserve component officers.[1]

During the Korean War, U.S. Army PTs were deployed to hospitals in Korea, Japan, and Hawaii in support of the fighting forces. Additional U.S. Army PTs were mobilized and assigned to Continental United States hospitals. In support of the Republic of Korea, U.S. Army PTs instituted clinical training programs in physical therapy for Korean medical personnel and designed PT facilities for several Republic of Korea military hospitals.[1] To supplement the shortage of trained PT technicians in the Far East Command hospitals, PTs conducted "on the job" training for enlisted personnel within numerous army PT clinics during the early stages of the war. This training was formalized in 1952 into four 12-week courses that followed the recommended army program of instruction for PT technicians.[1]

The primary long-term rehabilitative effort for Korean War casualties occurred in general hospitals located in Japan, Hawaii, and the United States. After discharge from army hospitals, long-term rehabilitative efforts were continued for war injured patients by therapists assigned to Veteran's Administration hospitals. Reconditioning program development and innovative techniques were stimulated among army PTs by the rehabilitative requirements of patients who contracted poliomyelitis, tuberculosis, Japanese B encephalitis, or who were recuperating from the effects of cold weather injuries.[1]

Vietnam War

In 1955, through congressional action, the WMSC became the Army Medical Specialist Corps (AMSC) and male officers were added to the three specialty sections (Occupational Therapy, Physical Therapy, and Dietetics). AMSC officers played key roles in the U.S. Army Medical Department involvement in Vietnam, and in post-Vietnam military operations (Grenada, Panama, and the Persian Gulf War). In 1992, the Physician Assistant (PA) Section was created as the fourth section of the AMSC. Prior to 1992, PAs held the rank of warrant officer, and were assigned to the Medical Corps. The 1992 congressional action gave the AMEDD the ability to commission the army PAs and subsequently place them in the AMSC.

Between 1962 and 1973, in support of the military mission in South Vietnam, The Surgeon General of the Army deployed 24 hospitals, which were established as fixed medical installations with area support missions. These included surgical, evacuation, and field hospitals, and a 3,000 bed convalescent center. The first AMSC officer to arrive in Vietnam was Major Barbara Gray, a physical therapist, assigned to the 17th Field Hospital in Saigon. Her arrival in March of 1966 marked the first time an army PT or AMSC officer had been assigned to an active combat zone.[1]

Prior to her arrival in Saigon, Major Gray had developed a staff study indicating the need for physical therapy in the Vietnam scenario. As a result of this study, the OTSG deployed nine PTs and their enlisted physical therapy personnel to Vietnam in 1967. As Major Gray had hypothesized, the early intervention of PT in patient treatment programs improved the patients' medical prognoses, reduced healing times, and helped return soldiers to duty more rapidly. As a result of Major Gray's study, army PTs were assigned to the 3rd, 8th, and 17th Field Hospitals, the 12th, 24th, 29th, 36th, 67th, 71st, 93rd, and 95th Evacuation Hospitals, and the 6th Convalescent Center.[1]

The determining factor affecting assignment of PTs to Vietnam was direct requests for PT service by hospital commanders. Shortly after their arrival in Vietnam, U.S. Army PTs began treating Vietnamese military personnel and civilians in army hospitals. This interaction between U.S. and Vietnamese military personnel expanded to include PT instruction of patients and medical staffs regarding positioning and exercise in Army of the Republic of Vietnam (ARVN) hospitals. Between 1966 and 1973, 47 PTs served in Vietnam as members of the AMSC.

The primary treatment goal of physical therapy in Vietnam was the rehabilitation of patients who were capable of being returned to duty. For patients requiring evacuation out of the theater, treatment

focused on basic rehabilitation procedures that would be continued at each evacuation stage. Because of the relatively short periods of patient hospitalization in Vietnam, physical therapy was typically limited to ward programs, although a small number of outpatients were also treated in selected hospitals. As facilities and equipment were improved, expanded treatment in physical therapy clinics was available (Figure 2-3).[3]

During the Vietnam War, orthopedic surgeons assigned to army hospitals in Vietnam found that their time was consumed with evaluating and treating patients requiring surgical management. This forced a large number of patients with nonsurgical musculoskeletal problems to wait until physician manpower became available to evaluate and treat them. Because of this, army PTs assigned to the combat zone (CZ) acquired a new and expanded role: nonphysician healthcare provider.[1] The newly designated function was the early evaluation and treatment of patients with NMS conditions without physician referral.[1,3] Army PT met that challenge and created programs and protocols where PTs evaluated and treated patients with nonsurgical orthopedic conditions. The program outcomes were decreased hospitalization, decreased patient waiting and treatment times, and facilitation of soldiers' rapid return to duty.[1]

Fig. 2-3. Vietnam War—Army physical therapist performing passive range-of-motion exercises for a patient with a femur fracture and associated nerve injuries of the lower limb. Photograph: Archives, U.S. Army Center of Military History, Washington, DC.

Physical Therapy in the 1970s: Evolution of the Profession

U.S. Army PTs have successfully practiced in a direct access environment as primary nonphysician healthcare providers for patients with NMS disorders since the early 1970s. The need for army PTs to assume this role and provide NMS evaluation and treatment for their patients without physician referral was a direct result of the evolving practice of army PT in a wartime environment (Vietnam) and the shortage of army physicians, especially orthopedic surgeons, following the Vietnam War.

In 1972, the AMEDD was plagued with a large number of patients with NMS complaints and a shortage of orthopedic surgeons to evaluate and treat these patients. This resulted in long delays in administration of primary healthcare to patients with NMS conditions. The traditional system of triage for these patients was initial evaluation and diagnosis by an orthopedic surgeon followed by a referral to PT for services. The modified system of triage for patients with NMS problems was evaluation, diagnosis, and treatment by PTs with appropriate referral to orthopedic surgery or other medical specialties as required.

Implementation of the NMS role for army PTs required formalizing extensive training and privileging protocols. The regulations that document the army PT nonphysician care provider role are Army Regulations 40-48 and 40-68.[4,5] Army PTs privileged to perform NMS evaluations and treatment may perform within the scope of physical therapy practice, refer patients to radiology for appropriate radiographic evaluation, restrict patients to quarters for 72 hours, place patients on temporary profiles up to 30 days, and refer patients to all medical specialty clinics. In some medical treatment facilities (MTFs) PTs may be privileged to order certain analgesic and nonsteroidal antiinflammatory medication.[4]

The advantages of having PTs perform NMS evaluation and treatment include (*a*) prompt evaluation and treatment for the patients with NMS complaints; (*b*) promotion of quality healthcare; (*c*) decrease in sick call visits; (*d*) more appropriate use of physicians; and (*e*) more appropriate use of PT education, training, and experience.[1] The use of PTs as nonphysician healthcare providers in the U.S. Army has been an overwhelming success. Presently, other branches of the uniformed services (Air Force, Navy, and Public Health Service) provide direct access to PT services for patients with NMS complaints.

The U.S. Army is fortunate to have an educational program to prepare PTs for their role in evaluating and treating patients with NMS dysfunction. The U.S. Army-Baylor University Graduate Program in Physical Therapy, located at Fort Sam Houston, Texas, is an entry-level master's degree program accredited by the Commission on Accreditation for Physical Therapy Education (CAPTE). The Army-Baylor PT program has a triservice function to prepare entry-level PTs for active duty service in the Army, Navy, and Air Force. The faculty of the Army-Baylor PT program is composed of representatives from these three services; however, the army remains the lead agent.

The Army-Baylor PT curriculum meets the accreditation requirements of CAPTE for an entry-level generalist PT, but the curriculum emphasizes the evaluation and treatment of patients with NMS problems. Following entry-level training, these army PTs are trained clinically, following specific credentialing protocols, and then credentialed to perform NMS evaluation and treatment. A two-week advanced NMS evaluation course was designed and implemented to enhance the continuing education of PTs performing NMS evaluations.

As the role of the PT in the U.S. Army changed in the 1970s, there was a concomitant change of the role of PTs in civilian practice. The evolution of PT practice proceeded toward a greater level of autonomy commensurate with increased levels of education and experience and an expanded research base.[1] The scope of PT practice changed from a limited role as technician to that of a professional member of the healthcare team.

With this evolution in the scope of civilian PT practice came an increased responsibility for PTs to provide optimal healthcare for their patients. At present, 28 states permit (by law) the evaluation and treatment of patients by physical therapists without physician referral. Forty-two states permit PTs to evaluate patients without physician referral but require physician approval before initiating PT treatment. The PT associations in an increasing number of states continue to pursue legislative changes to permit direct access to PT services.[6]

Persian Gulf War

In August 1990, the United Nations (UN) quickly responded to the Iraqi invasion of Kuwait by mobilizing United States and other UN coalition forces. As a part of this mobilization of American forces, U.S. Army physical therapists were deployed into the TO—Saudi Arabia, Kuwait, and Iraq—and communications zone—Europe—in support of the Persian Gulf War. In addition, army PTs were utilized in military hospitals in the CONUS in support of this operation. Active and reserve component PTs and PT enlisted specialists were deployed during this operation (Figure 2-4).

Fig. 2-4. Persian Gulf War—U.S. Army 47th Field Hospital, Bahrain. Photograph: Archives, U.S. Army Center of Military History, Washington, DC.

Fig. 2-5. Captain Don Hansen, U.S. Army physical therapist, performing cervical mobilization procedures on a patient with cervical joint dysfunction. Captain Hansen was assigned to the 47th Field Hospital, stationed in Bahrain. Photograph: Archives, U.S. Army Center of Military History, Washington, DC.

Six PT and 12 PT enlisted specialists were deployed to the TO during the Persian Gulf War. PT enlisted specialists (enlisted soldiers with the rank of Private First Class through Sergeant First Class) received 27 weeks of didactic and clinical PT training at the AMEDD Center and School at Fort Sam Houston, Texas. PT enlisted specialists assist the PT by performing modality and exercise treatments. PT personnel were assigned to the 47th Field Hospital (Bahrain), 50th General Hospital (Riyadh), 316th Station Hospital (Riyadh), and 300th Field Hospital (Saudi Arabia/Iraq border). Europe was designated the communication zone (COMMZ) for the Persian Gulf War. In addition to the active component PT personnel assigned to Europe/COMMZ (18 PTs and 32 enlisted specialists), an additional 11 PT officers and 21 enlisted specialists from the reserve component were deployed to Europe for augmentation of PT services.

At the onset of this operation, there were 186 PTs and 198 PT enlisted specialists on active duty and assigned to army hospitals in CONUS. An additional 40 PTs and 57 PT enlisted specialists from the reserve component were mobilized and deployed to army hospitals in the United States. One army PT and one PT enlisted specialist from the active component were deployed following the Persian Gulf War to serve six months with the 21st Evacuation Hospital in Saudi Arabia (Figure 2-5).

Navy and Air Force PTs were also deployed to the TO during the Persian Gulf War. Navy PTs were assigned to the navy hospital ships (*Comfort* and *Mercy*) and fleet marine hospitals. Air Force PTs were assigned to air transportable hospitals (ATHs) in the TO, and additional PTs were deployed to "warm base" hospitals in the COMMZ (United Kingdom and Germany).

The mission of army PTs in the Persian Gulf War was to provide physical therapy evaluation and treatment to correct or prevent physical impairments resulting from injury, disease, or preexisting conditions. PTs served as independent practitioners and nonphysician healthcare providers performing primary evaluation and treatment of patients with NMS complaints. Physical therapists also served as technical advisors to commanders, providing guidance in the areas of physical fitness, physical training, and injury prevention. U.S. Army PTs treated enemy prisoners of war and supplemented the staff in host nation hospitals, which required a shift from practice models to community health models with cultural integration.

Several significant issues pertaining to PT practice and personnel became evident during the Persian Gulf War. First, there was a lack of PT personnel to accomplish the mission at the hospitals deployed in the TO. This lack of PT personnel, especially in support of the orthopedic service, resulted in the needless evacuation of a large number of patients with NMS problems from the TO to the COMMZ (Europe) or CONUS. Second, an insufficient number of PT personnel in the TO was available to provide evaluation and treatment of work-related, training, and sports injuries. Insufficient staffing of PT personnel resulted in an increased evacuation rate of patients with NMS problems, and delayed soldiers' rapid return to duty following training or sports injuries (Figure 2-6).

During the period of August to December 1990, 986 orthopedic admissions were documented in the TO. As reported by the Patient Administration Division, OTSG, this included 270 patients (27%) with knee dysfunction, 202 patients (19%) with spinal dysfunction, 61 patients (6%) with foot/ankle dys-

Fig. 2-6. Persian Gulf War—Physical Therapy Clinic, 47th Field Hospital, Bahrain. Photograph: Archives, U.S. Army Center of Military History, Washington, DC.

function, and 35 patients (4%) with other musculoskeletal dysfunctions. Forty-two percent of the total number of patients evacuated to the COMMZ during the same time frame were patients with orthopedic conditions. These patients were evacuated to the COMMZ for further evaluation and treatment of their conditions.[7] After-action reports from army orthopedic and PT personnel hypothesized that increased PT personnel assets in the TO would have decreased the orthopedic hospital admissions in the TO and decreased the number of evacuations out of the TO during the buildup, preparation, and training phases of the operation.

In addition, the Army Patient Administration Division (OTSG), which was responsible for recording hospital admissions and dispositions, could not document the number of outpatient visits in the TO during the Persian Gulf War, including the buildup and training phases of the operation. Considering the number of orthopedic admissions during this time for back, knee, and ankle dysfunction, one can deduce that many more patients were evaluated and treated as outpatients for NMS conditions without the benefit of PT services. Early physical therapy evaluation and treatment of patients with NMS conditions have proven to be beneficial in rapidly returning soldiers to duty. Physical therapists are valuable in conserving the fighting force.

One significant "lesson learned" by the AMEDD for army PT services was a result of experiences in the Persian Gulf War. An insufficient number of PT personnel were deployed to the TO to optimally complement the assigned orthopedic physicians in accomplishing the mission of providing physical therapy evaluation and treatment of patients with work-related, training, and sports injuries.

Physical Therapy Study From The Persian Gulf War

To document the injury characteristics and recovery patterns in physical therapy referrals during the Persian Gulf War, a Physical Therapy Registry was developed by the Chief of Army Medical Specialist Corps Clinical Investigation and Research (AMSC-CIR). In previous mobilization environments, Army PTs did not implement systematic data collection as part of the clinical management of patients. Anecdotal reports and generalized historical reviews were the only sources of PT practice analyses from previous deployments.

Past attempts by PTs to quantify the information relevant to the recovery of individuals injured during deployment were limited to the data collected by army PT clinical researchers participating in the Vietnam Head Injury Study, a 10 to 14 year follow-up of veterans with penetrating head injuries incurred during the Vietnam War.[8,9] PT records during the Vietnam deployment were limited to treatment cards that were later destroyed, a common administrative procedure for army PT files retained for two years. At that time, PTs were not permitted to write in the progress note section of patients' medical records; therefore, retrospective record reviews for PT data were nonproductive. A clinical study of injury characteristics and a registry of injuries referred during a major combat-related, army PT practice have not been accomplished.[10]

During the period of October 1990 to May 1992, 1,433 patients were treated by army PTs in the Persian Gulf War field hospitals and evacuation sites. Each case was entered into the Persian Gulf War Army Physical Therapy Registry at three combat sites in the Persian Gulf region and three evacuation sites in West Germany.[10,11]

Design, management, and analyses of all data occurred at the AMSC-CIR office at Walter Reed Army Medical Center, Washington, DC. The qualitative data (patient count, work hours, military duties, adequacy of clinical and military training) from monthly surveys of practice were consolidated and forwarded by the Chief, AMSC-CIR, to the Physical Therapist Section, OTSG, for action.

The average PT patient profile from the combined caseload at combat and evacuation sites in the Persian Gulf War had the following characteristics: 29 years of age, caucasian, male, active duty army enlisted rank E1–E4, Army Physical Fitness Test score of 245/300 (82%), 94 weeks of experience in military service, and 13 weeks in the combat zone before injury. Only 42% (607) of the PT patients were treated in the Persian Gulf, compared to 58% (826) of the patients treated by PTs in the evacuation sites of Landstuhl, Frankfurt, and Nuremburg, Germany.[10,11]

Army physical therapy in the combat zone consisted of managing soldiers with injuries incurred primarily from training (69%) and off-duty (26%) activities. Patients treated by PTs in the COMMZ sustained injuries mainly during combat (75%) or training maneuvers (22%).[10,11]

The causes of injuries treated by physical therapists in CZ and COMMZ were primarily in two categories: (1) lifting or falling (37%); and (2) preexisting medical conditions (22%) such as degenerative joint disease, temporomandibular joint dysfunction, headaches, gout, or tumors. Less frequent mechanisms of injury were vehicle accidents (12%), weapons (7%), burns (6%), tools or machinery (4%), and unclassified (12%).[10,11]

The locations of injuries from this caseload were spine (38%), knee (27%), ankle or foot (12%), shoulder (7%), hip or thigh (6%), brain or cranium (6%), wrist or hand (3%), and elbow (1%). The average duration of PT treatment in the combat zone was 10.7 inpatient days and 1.8 outpatient days. If evacuated to Germany, PT patients were treated an average of 4.3 inpatient days before being transferred to stateside hospitals.[10,11]

Eighty-five percent of PT patients in the combat zone were returned to duty. On a case-by-case analysis, it was the professional opinion of PTs in the evacuation zone that 21% of their Persian Gulf caseload could have been returned to duty if treated in the combat zone by a PT. The shortage of PTs in the Persian Gulf contributed to excessive evacuation of troops with musculoskeletal injuries who could have remained in the combat setting for treatment. PTs in the field combat environment during the war played an important role in expedient assessment, treatment, and return to duty of acutely injured soldiers with only 15% of physical therapy patients requiring evacuation.[10,11]

U.S. Army Physical Therapists Deployed to Third World Countries

U.S. Army PTs and PT enlisted specialists have recently been deployed with AMEDD units and civilian humanitarian organizations to Third World countries. This humanitarian role for army PTs included assignments to El Salvador, Russia (burn care assistance), Turkey, Romania (pediatrics), and Croatia. In addition to the general practice of physical therapy, this type of assignment enables army PTs to serve as consultants and educators to the local medical community.

PHYSICAL THERAPY IN EVOLVING DOCTRINE

Recent events have dramatically changed the threat facing the United States. With those changes came the need to review and revise the army's warfighting concept. The army's current warfighting doctrine, "AirLand Battle," is primarily oriented toward conventional warfare within a European scenario. Today, that single entity threat no longer exists. Improvements in East–West relations have shifted the focus to regional threats of consequence to United States vital interests.[12]

The medical mission for the U.S. AMEDD of the future includes four aspects. First, AMEDD must maintain the health of the army. Second, it must conserve the army's fighting strength. Third, it must prepare for health support to the army in time of war, international conflict, or natural disaster. Fourth, it must provide healthcare for eligible beneficiaries in peacetime, concurrently with the above missions.[12]

To meet the requirements of the AMEDD mission for AirLand Battle doctrine, the Medical Force 2000 (MF2K) structure was developed to provide field medical support during the first decade of the 21st century. Medical Force 2000 places Army physical

therapists and physical therapy specialists in the Combat Support Hospital (NATO [North Atlantic Treaty Organization] Level III), Field Hospital (NATO Level III/IV), and General Hospital (NATO Level IV).[12]

Army physical therapists in MF2K are employed as healthcare providers in the evaluation and treatment of patients with NMS disorders along with their general PT duties. In addition, army PTs have been trained and should be used as consultants in injury prevention, physical training, and physical fitness. During mass casualty situations, PT personnel may assist in managing "delayed" or "minor" category patients, or augmenting the orthopedic section by evaluating and treating patients with nonsurgical NMS conditions.[13]

Army policy states that PTs plan, evaluate, supervise, and implement treatment regimens to correct, prevent, or retard physical dysfunction resulting from injury, disease, or preexisting biomechanical problems.[12] Physical therapists perform baseline and progress assessments of gait, structure, mobility (including strength and joint motion), neurological and circulatory status and function. Data from these assessments provide objective information for duty fitness determinations. Physicians reference PT data as indicators of the stability, improvement, or deterioration of a patient's condition.[13]

CONCLUSION

Army physical therapists are essential members of the healthcare team and have demonstrated their merit during wartime and peacetime. Historically, army PTs have contributed to the medical support of U.S. Forces in World War I, World War II, Korea, Vietnam, and, most recently, in Southwest Asia. In wartime deployments and field training exercises, army physical therapists have made substantial contributions to military healthcare in primary NMS assessment, rehabilitation, and injury prevention.

In peacetime, the scope of PT continues to evolve and expand into multiple practice settings and specialty areas. In the 21st century, army physical therapists will continue to lead the way in practice, education, and research, particularly in the advanced specialty area of orthopedic physical therapy.

REFERENCES

1. Hartwick AM. Army Medical Specialist Corps 45th Anniversary Commemorative Monograph. Washington, DC: Center of Military History, US Department of the Army; 1993.

2. Anderson RS, Lee HS, McDaniel ML. Army Medical Specialist Corps. Washington, DC: Office of The Surgeon General, US Department of the Army; 1968.

3. Neel S. *Medical Support of the US Army in Vietnam*. Washington, DC: US Department of the Army; 1973.

4. US Department of the Army. *Nonphysician Health Care Providers*. Washington, DC: DA; 1992. Army Regulation 40-48.

5. US Department of the Army. *Quality Assurance Administration*. Washington, DC: DA; 1992. Army Regulation 40-68.

6. Department of Practice, American Physical Therapy Association. Alexandria, VA.

7. Patient Administration Division. Washington, DC: Office of The Surgeon General, US Department of the Army.

8. Sweeney JK., Smutok MA. The Vietnam head injury study: A preliminary analysis of the functional and anatomical sequelae of penetrating head trauma. *Phys Ther*. 1983;63:2015–2018.

9. Smutok MA, Gradman J, Salazar A, Sweeney JK, Jones B, Dirocca P. Effects of unilateral brain damage on contralateral and ipsilateral upper extremity damage on contralateral and ipsilateral upper extremity function in hemiplegia. *Phys Ther*. 1989;69:195–203.

10. Sweeney JK, Sanders GT, Baehm D, Hansen D, Cook E, Greathouse D. Epidemiology of injuries requiring physical therapy during Operation Desert Shield/Storm. *Phys Ther*. 1992;72. Abstract RO86, S45.

11. Sweeney JK, Sanders GT, Baehm D, Greathouse D. Injury characteristics and recovery patterns of Persian Gulf War veterans receiving physical therapy during deployment. *Phys Ther*. 1993.

12. US Department of the Army. *US Army Health Services Long-Range Plan, 1993–2023*. Washington, DC: DA; 1992.

13. US Department of the Army. *DEPMEDS Policies/Guidelines & Treatment Briefs*. Washington, DC: Defense Medical Standardization Board; 1990 (revised 1992).

Chapter 3

REHABILITATION OF THE UPPER LIMB AMPUTEE

TIMOTHY R. DILLINGHAM, M.D., M.S.*

Formerly, Major, Medical Corps, US Army, Physical Medicine and Rehabilitation Service, Walter Reed Army Medical Center, Washington DC; currently, Associate Professor, Department of Physical Medicine and Rehabilitation, The Johns Hopkins University, Baltimore, Maryland 21239

INTRODUCTION

The devastating trauma suffered during armed conflicts results in substantial numbers of upper and lower extremity amputees. The care of amputees is a major problem facing any army during wartime. In the civilian setting, the primary cause of leg amputations is vascular disease,[1] which accounts for 90% of amputations. Other causes include trauma (7%), malignancy (2.5%), and congenital amputations (0.3%). Trauma is the leading cause of upper extremity (UE) amputations (75%), and typically involves males ages 15 to 45.[2]

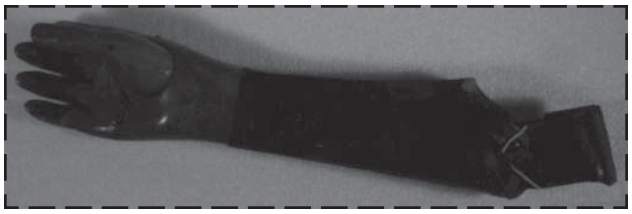
During the Civil War, 3 million troops were mobilized and 20,993 major amputations were documented in the Union Army.[3] Of these amputations, 8,518 were UE amputations and 12,475 were lower extremity amputations. Examples of 19th century post–Civil War era upper limb prostheses are shown in Figures 3-1 and 3-2.

The official statistics for World War II, covering the period between 1 January 1942 and 31 March 1946, indicate that in the Zone of the Interior (the continental United States), 14,912 amputees were treated, including 1,057 soldiers who had two amputations. This number does not reflect partial hand amputations not severe enough to impede continued military service.[3] Lower extremity amputations accounted for 10,620 of those treated, while 3,224 suffered UE amputations.

It is clear from these statistics that UE amputations are frequently seen in wartime and comprise a large percentage of the total number of amputees.

The importance of amputee care became obvious to planners during World War II when they realized that the Veterans Administration (currently, Veterans Affairs) hospital system could not handle the number of war injured amputees. Therefore, in 1943 the army was made responsible for both early care and definitive rehabilitation of amputees, ensuring that all amputees would receive maximum benefits prior to discharge from a military hospital.[4] Because of the enormous numbers of amputees, the U.S. military established five "amputation centers" at ports of debarkation. The major center was established at Walter Reed General Hospital (now known as Walter Reed Army Medical Center) in Washington, DC. The training of medical officers, therapists, and prosthetists was vigorously pursued to ensure that healthcare providers were up-to-date in the care of amputee soldiers.[4]

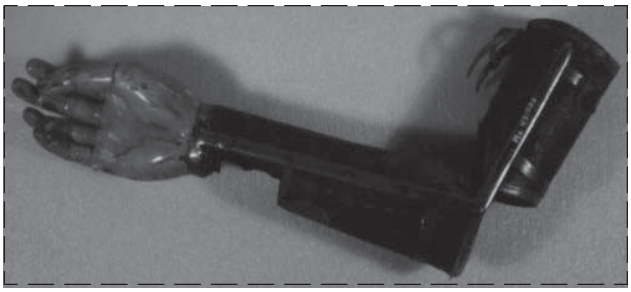
a



b

Fig. 3-1 (**a** and **b**). Below elbow prosthesis. Manufactured by Beaufort, in the United Kingdom, between 1875 and 1899. Photograph: Courtesy of Armed Forces Institute of Pathology Museum, Washington, DC.

a



b

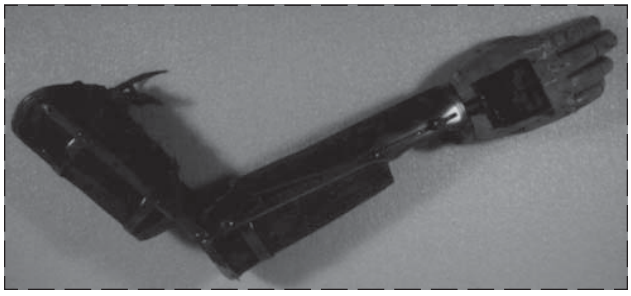


Fig. 3-2 (**a** and **b**)**.** Above elbow prosthesis. Manufactured by Doerflinger Artificial Limb Co., in the United States, between 1875 and 1899. Photograph: Courtesy of Armed Forces Institute of Pathology Museum, Washington, DC.

Even with substantial resources dedicated to amputees, additional development of prosthetic technology was required to best meet the soldiers' needs. To refine and develop prosthesis construction, civilian consultants were used. Because the army was responsible for definitive prosthetic fabrication, contracts were established with companies for the purchase of large quantities of prosthetic devices.[4] For instance, UE prosthetic terminal devices (TDs) were purchased from Dorrance, Hosimer, and Becker companies.

In April 1943, the Office of The Surgeon General directed that all amputees be transferred as soon as possible to designated amputation centers; however, by 1944, it was clear that the original five amputation centers (Bushnell General Hospital in Brigham City, Utah; Lawson General Hospital in Atlanta, Georgia; McCloskey General Hospital; Percy Jones General Hospital in Battle Creek, Michigan; and Walter Reed General Hospital in Washington, DC) were inadequate to meet the needs of amputees sustained in a long and protracted global war. This was particularly true during the intense fighting in Europe during the winter of 1944–1945. Because army hospitals were responsible for the amputee's full rehabilitation, as well as early care, longer military hospital stays were required. For these reasons, two other amputee centers were established: the Thomas M. England General Hospital in Atlantic City, New Jersey; and McGuire General Hospital in Richmond, Virginia. Each hospital had its own prosthetic shop with trained prosthetists. To educate "orthopedic mechanics," three-month training courses were established at the amputee centers. Here technicians were instructed in fabrication of prosthetic devices. Servicemen with amputations were sometimes trained as prosthetists and utilized in limb fabrication shops.[4] The U.S. Surgeon General was

> …insistent that extreme care be exercised to ensure that the fit of each prosthesis was entirely satisfactory and that each amputee be taught to use his prosthesis competently before his discharge.[4]

Hand injuries were tremendously common during World War II. Bunnell, a distinguished hand surgeon who served as civilian consultant for hand surgery to the Secretary of War, described the scope of hand injuries in World War II. Although the statistics for hand injuries during the war (based on Zone of the Interior hospital experience) were inaccurate, an estimated 22,000 major hand injuries occurred in World War II.[5] As Peterson[3] points out, the number of amputations involving the hand was probably much higher than 3,224. Many finger and partial (nondisabling) hand and toe amputations were not seen in the Zone of the Interior hospitals due to the fact that these soldiers were able to continue their military service and remained in the theater of war. Hand injured patients were sent to designated "hand centers," where specially trained surgeons and therapists managed their wounds. Surgeons at these hospitals became quite proficient in hand reconstruction, and occupational and physical therapists played extremely important roles. In fact, these therapists were classified under "physical medicine."[5] The importance of rehabilitation in the functional restoration of the hand following surgery cannot be overemphasized. Bunnell stated that "in all patients in whom it was practicable, it was the general rule to institute early motion and mobilization."[5]

The military has a rich tradition of caring for amputees injured as a result of armed conflict. Indeed, the U.S. Army pioneered the field of amputee rehabilitation out of necessity. Intense wars produce enormous numbers of traumatic amputations in distributions quite different from those seen in civilian medicine. For this reason, amputee care in the military must remain at the forefront of technology, maintaining its readiness to assume the full care of an amputee soldier. Organized multidisciplinary rehabilitation services, initially under the direction of the primary surgeon and then the military physiatrist, must be established at medical centers. The World War II system, where designated amputee centers were established, provides a model for optimal, present day military amputee care. Major military hospitals with modern prosthetic laboratories, where dedicated expert prosthetists, occupational therapists, and physical therapists are organized as a rehabilitation team, are best suited to meet the specific needs of individual amputee soldiers. Early temporary prostheses and definitive state-of-the-art prosthetic devices must be provided to the amputee for full rehabilitation to occur. Early weight bearing using temporary prostheses has been found to be very beneficial to amputees. In fact, in World War I, the Belgian Army Medical Corps demonstrated that early weight bearing improved circulation, hastened stump shrinkage, and prevented muscle atrophy and contractures.[6] The Belgians felt that early ambulation was "far more useful than any form of physical therapy."[6]

In the event of an intense conflict, even of short duration, substantial numbers of soldiers will sustain amputations. The military medical centers must

be able to accommodate these casualties. This chapter deals with the rehabilitative care of upper limb amputee soldiers, with continued emphasis on the importance of the amputee as the center of a coordinated interdisciplinary rehabilitation effort leading to fully functional restoration.

HAND AMPUTATIONS

Hand Amputations and Reconstruction

As discussed in the introduction, hand injuries are frequent war wounds. When a partial amputation of the hand is indicated, there are difficult choices regarding whether to reconstruct the hand or proceed directly with a prosthesis. Preserving all possible length is important for all amputations. This is particularly critical in the case of the hand.[5,7,8] There are many techniques for hand reconstruction following a partial amputation, but the scope of this text precludes a complete discussion of these. Interested readers are directed to a work by Bunnell titled *Management of the Nonfunctional Hand—Reconstruction Versus Prosthesis*.[9] This phenomenal work, according to Omer, presents principles which remain valid today.

The general principle regarding hand prostheses is that it is much better to have a painless hand with some grasp function and intact sensation than to have a prosthesis. The most important part of the hand is the opposable thumb. Preservation of sensate skin and all possible length of the thumb should be undertaken.[9] Reconstruction of the hand can provide greatly improved function after injury and should always be considered.

Phalangization of the metacarpals is a useful reconstructive technique in which the web space is deepened between digits, providing for a more mobile digit. This is often performed on the first web space and frequently coupled with rotation osteotomy of the first metacarpal, thus providing useful thumb opposition. An example of this phalangization of the first metacarpal is shown in Figures 3-3, 3-4, and 3-5. For this patient, the deepening of the web space provided improved opposition of the thumb.

Pollicization of a remaining finger can be used to reconstruct the thumb. For this procedure, a remaining finger with intact neurovascular structures and suitable length is moved with its nerve and blood supply to the site of the amputated thumb.[9] This reconstruction provides a sensate opposable digit to act as a thumb, enabling fine and gross grasp. Digit lengthening procedures involve creating a tube pedicle graft from the abdomen along with a bone graft.

The decision to reconstruct an injured hand requires the experience of a skilled hand surgeon who has knowledge of potential functional outcomes with both reconstruction and prosthetic training. In general, prostheses for hand amputations are inferior to the functional outcomes achieved with reconstructed hands.[9]

The optimal reconstructive techniques for thumb and partial hand amputations are delineated in Figure 3-6.[8] Reconstructive techniques for various levels of amputation, as described by Strickland,[8] are presented here. Adequate sensation at the opposing part of the thumb is very im-

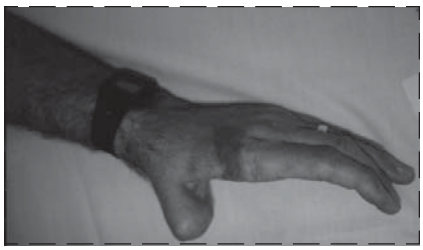


Fig. 3-3. Phalangization of the first metacarpal by deepening the first web space (dorsal view).

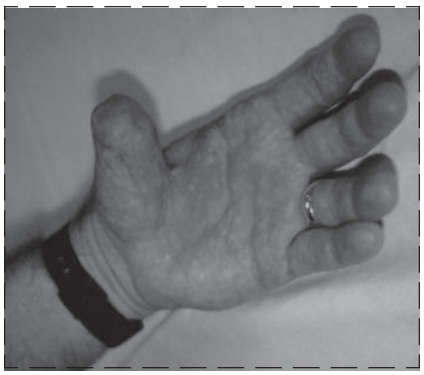


Fig. 3-4. Phalangization of the first metacarpal (palmar view). Additional skin grafting was required to cover the cleft.

Fig. 3-5. The patient shown in Figures 3-3 and 3-4 demonstrating improved grasp.

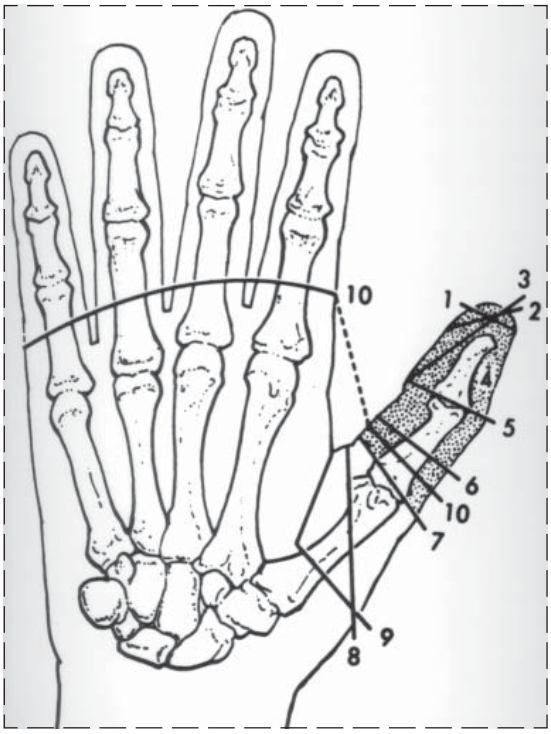


Fig. 3-6. The various thumb amputation levels. The optimal reconstructive procedure for each level is described in the text. Adapted with permission from Strickland JW. Restoration of thumb function after partial or total amputation. In: Hunter JM, Schneider LH, Mackin EJ, Callahan AD, eds. *Rehabilitation of the Hand*. 2nd ed. St. Louis: CV Mosby; 1984: 755–796.

portant. An amputation occurring at level 1 (see Figure 3-6), requires a full thickness skin graft. For loss of part of the volar pad (level 2), a volar advancement flap is used, whereby the innervated skin from the thumb is transferred distally, with its nerve and blood supply, to cover the defect. At level 3, where the entire volar pad of the thumb is removed, a cross finger flap using sensate skin from the index finger is transferred. Degloving injuries, represented by level 4, are reconstructive challenges and are best approached by using a tubed abdominal pedicle flap to cover the defect, followed by a neurovascular island pedicle flap from another finger to provide sensate skin.[8] Amputation at the interphalangeal (IP) joint results in a functional thumb (level 5). A more proximal amputation through the proximal phalanx of the thumb (level 6) requires deepening the first web space—a phalangization procedure.

Amputation at the metacarpophalangeal (MCP) joint (level 7) can be reconstructed in several different ways. The thumb can be reconstructed by grafting iliac bone to the remaining thumb giving added length, then using skin from the dorsal and lateral aspects of the first metacarpal to cover the bone graft. This procedure, referred to as the "cocked-hat flap," can extend the useful thumb length by 2.5 cm.[8] Another option for this level is pollicization of an adjacent or partially injured digit by transferring the digit with its neurovascular supply to the first metacarpal. Amputation through the distal one-third of the first metacarpal (level 8), can be managed by pollicization of an injured or normal digit, or a lengthening procedure with bone graft, tubed abdominal pedicle flap, and an island pedicle flap placed on the prehensile surface of the extended thumb. Amputation at the proximal two-thirds of the first metacarpal (level 9) requires complete thumb reconstruction by pollicization of the index finger or an injured finger. Toe transfer can also be considered in this case.[8] However, as Beasley and de Bese[10] point out, a toe transfer does not replace sensibility of the working thumb surfaces as would an island pedicle flap from a noninjured sensate part of the hand. Loss of all digits and the thumb (level 10) can be managed by phalangization of the thumb remnant by deepening the first web space, giving the thumb remnant better grasp and opposition.

For single-digit amputations, a distal interphalangeal (DIP) disarticulation is an acceptable procedure.[10] When an index or a middle finger is amputated close to the proximal interphalangeal (PIP) joint, the ability to oppose the thumb is compromised. Ray resection of the injured finger and rebalancing the hand can yield an excellent functional and cosmetic result. For loss of the fourth or fifth finger, or the fourth or fifth metacarpal bones, a ray resection can provide an acceptable cosmetic outcome (Figure 3-7). According to Beasley and de Bese,[10] finger amputations shorter than 18 mm distal to the web space will not accommodate finger prostheses, hence preservation of this minimal length is important.

In the decision to reconstruct a hand, one must weigh the benefits against risks of the procedure. Issues that must be considered are (*a*) whether the procedure will provide sensibility of the grasping surfaces, (*b*) if the treatment will be socially (cosmetically) acceptable, and (*c*) the consequence of the resulting scars.[10]

Hand Rehabilitation

The importance of hand rehabilitation, both concurrent with amputation and subsequent to recon-

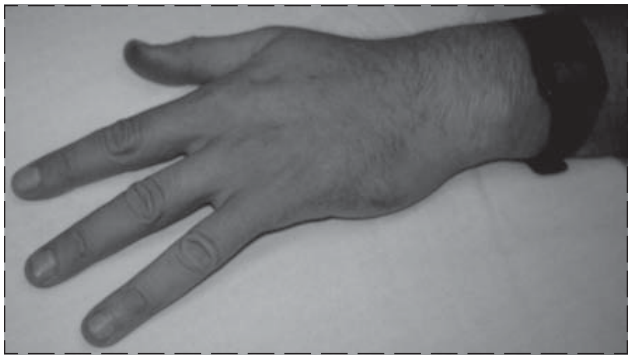


Fig. 3-7. A soldier who sustained a gunshot wound that injured the fifth metacarpal bone. Fifth ray resection resulted in a functional and cosmetically acceptable hand.

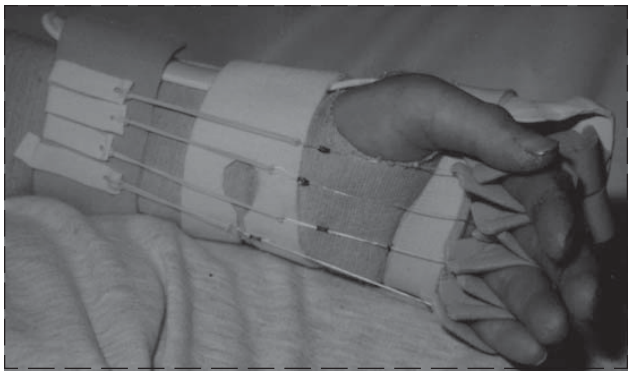


Fig. 3-8. A Persian Gulf War soldier who sustained fragment wounds of the arm, which caused severe nerve injuries, is shown with an orthosis utilizing elastic bands to provide prolonged stretch to contracted tendons and joints.

struction, cannot be overstated. In World War II, when the hand centers were established and Bunnell served as civilian consultant to The U.S. Army Surgeon General, Major General Norman T. Kirk, the rehabilitation of the hand was considered of paramount importance. Procedures for salvaging battle injured hands were taught to surgeons and rehabilitation teams at these centers:

> In all patients in whom it was practicable, it was the general rule to institute early motion and mobilization by activity and steady traction.[5]

Orthotics with traction devices, which applied steady pulling to mobilize joints, were used extensively. Figure 3-8 shows an example of such an orthosis used by a soldier wounded in the 1991 Persian Gulf War. This particular casualty sustained severe nerve injuries (not an amputation), but the same principles hold true for improving the range of motion with prolonged static stretch. Elastic traction is alternated in joint flexion and extension every few hours, providing prolonged range of motion in each direction.

Perhaps the most important means of ensuring optimal function following a reconstructive procedure is through rehabilitation that maintains or improves range of motion, increases strength of residual muscles, and incorporates the reconstructed hand into the casualty's daily activities. Occupational therapy plays a significant role in the rehabilitation process and works closely with surgical and rehabilitation teams. Education of the hand-injured soldier regarding injuries, prosthetic devices, and care of the residual limb, is crucial to maximal restoration. In addition, the amputee must be taught to use his noninjured limb and the prosthetic limb to achieve self-care and acquire vocational and leisure skills. Specially constructed adaptive devices may be required for these tasks[11]; for instance, Patricelli[12] describes a modified cutting board for a patient with a partially amputated hand. Early intervention, with temporary prostheses and rehabilitation training, greatly improves the ultimate acceptance and use of prosthetic devices.[13]

Hand Prosthetic Devices

The proper prosthetic device for a partial hand amputation must be prescribed based on a thorough knowledge of the patient. This includes obtaining detailed information about the soldier's daily activities, vocational interests and needs, avocational (recreational) desires, and expectations about the future with a prosthesis. A thorough physical examination, emphasizing the neurological assessment of strength and sensation, must be performed. Accurate assessment of the residual limb's range of motion and stability in all joints is also necessary. The soldier's cognition must enable the learning of necessary skills for prosthetic use. Vision is very important because a prosthesis provides little sensory feedback. There are many possible prosthetic devices that will effectively improve an amputee soldier's function. However, it is important to realize that function and satisfaction are the ultimate goals, and that frequently an amputee discards prosthetic devices, feeling they are no longer necessary and that they hinder optimal function. In general, partial hand amputations can be divided into several different categories with corresponding prostheses.

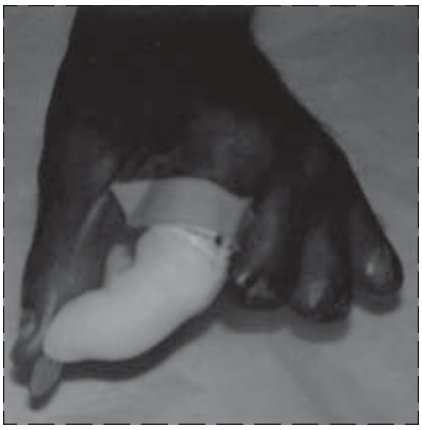


Fig. 3-9. A soldier who sustained frostbite injuries and who subsequently had transverse amputations of his second through fifth fingers is shown here using a finger prosthesis to grasp a coin.

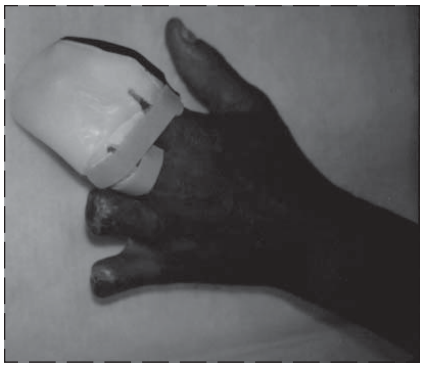


Fig. 3-10. The soldier shown in Figure 3-9 with finger prosthesis containing the index and middle fingers (dorsal view) showing the Velcro closure.

Fig. 3-11. Palmar surface of the prosthesis shown in Figure 3-10. This prehensile surface is coated with foam rubber, which increases grasping friction.

Transverse Amputations

Transverse amputations occur at any level and involve one or more digits that can be replaced with cosmetic prostheses or functional finger prostheses. The use of a finger extension prosthesis demonstrating fine pinch is shown in Figure 3-9. It is important to be mindful of prosthetic fabrication principles for all prostheses. Thermoplastic materials contoured to the skin and bony surfaces are very useful.[14] In addition, the prostheses must be lightweight, durable, and washable. Figures 3-10 and 3-11 show how a prosthesis is fitted onto two remaining fingers with sufficient length. Foam rubber, covering active surfaces, increases friction (see Figure 3-11). This concept, demonstrated in Figure 3-12, shows how larger objects can be manipulated and grasped.

Fig. 3-12. Finger prosthesis used to grasp a cup.

Radial Amputations

White and HilFrank[14] categorize amputations based on the amputated side of the hand: radial, ulnar, or central. These combinations of amputations are shown in Figure 3-13. They represent major functional deficits resulting from the amputations, and facilitate conceptualization of appropriate prostheses. Radial amputations involve the thumb and index fingers and compromise fine grasp.[14] Prosthetic devices are fabricated to replace the opposition role of the thumb. Prostheses can be used with or without prior thumb reconstruction, or can complement a reconstructive procedure. Figures 3-3 and 3-4 show a thumb amputation at the MCP joint managed by deepening the first web space. Complementing this reconstructive procedure, a thumb prosthesis was fabricated with orthoplast and a Velcro closure (Figure 3-14). The prosthesis is placed over the thumb (Figure 3-15), effectively lengthening the amputated digit. Grasping is aided by a rubber tip placed on the end of the prosthesis. Functionally, prostheses are often used only for certain activities.

Surgical reconstruction improves the fine grasp of this injured hand (see Figure 3-5). A special thumb prosthesis for the same soldier is shown in Figure 3-16. It fits over the thumb and presents a curved, rubber coated surface that allows fine three-jaw chuck grasp with either the second or third fingers (Figure 3-17). Fine grasping abilities of this amputee, with and without a prosthesis, are contrasted in Figures 3-17 and 3-18.

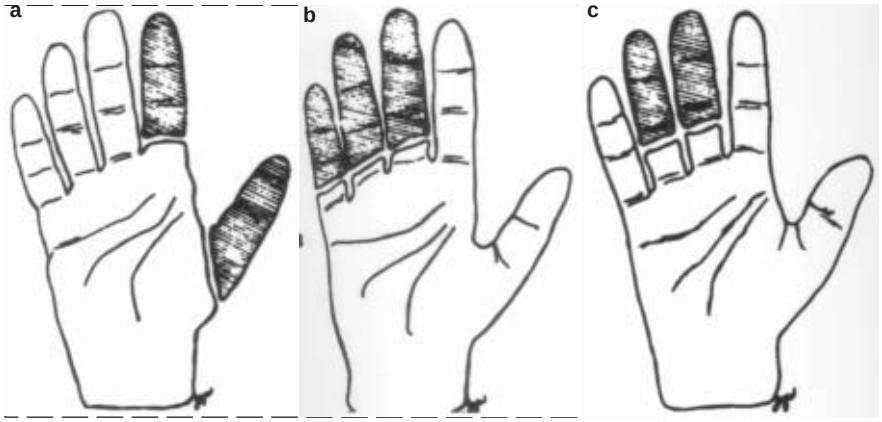


Fig. **3-13.** Classes of hand amputations. (**a**) radial amputations, (**b**) ulnar amputations, (**c**) central amputations. Adapted with permission from White JG, HilFrank BC. Prosthetic and adaptive devices for the partial hand amputee. In: Hunter JM, Schneider LH, Mackin EJ, Callahan AD, eds. *Rehabilitation of the Hand*. 2nd ed. St. Louis: CV Mosby; 1984: 755–796.

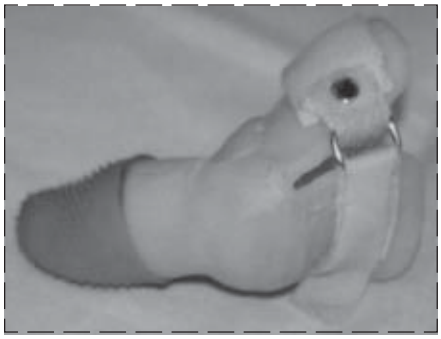


Fig. 3-14. Thumb prosthesis with rubber surface for opposition and a Velcro closure attaching it to the residual thumb.

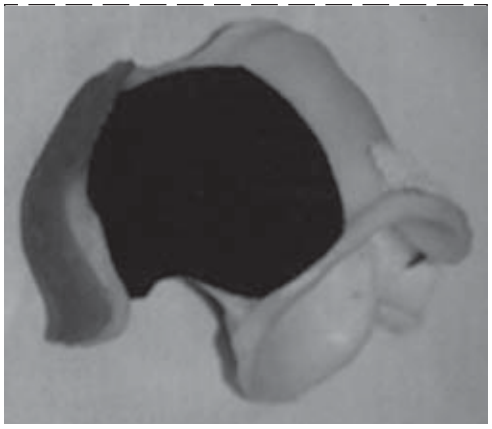


Fig. 3-16. Thumb prosthesis with curved surface for three-jaw chuck fine grasp.

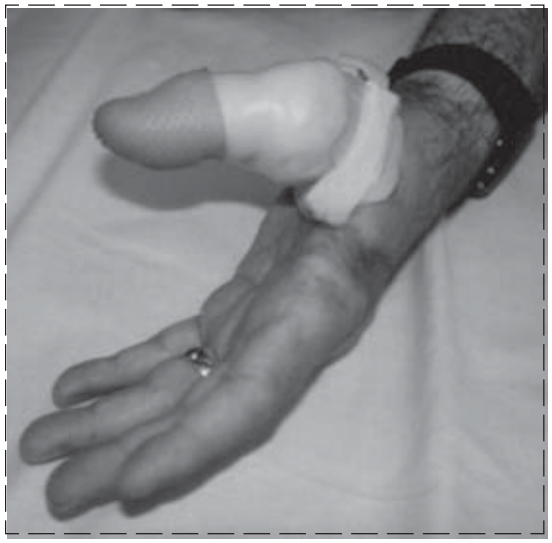


Fig. 3-15. Thumb prosthesis as worn on the residual thumb by the amputee.

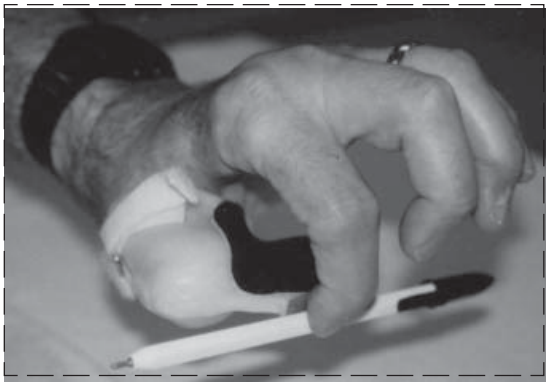


Fig. 3-17. Fine grasp with thumb prosthesis, which allows better opposition of the second and third fingers.

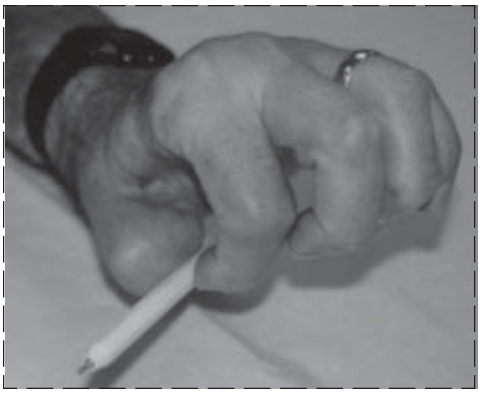


Fig. 3-18. Fine grasp without thumb prosthesis.

Amputation at the proximal metacarpal of the thumb requires a prosthesis or reconstructive surgery. A thumb prosthesis for the right hand of a soldier with an amputation at this level is shown in Figure 3-19. The prosthesis encircles the hand across the metacarpals and is firmly anchored with a Velcro closure (Figure 3-20). A functional, three-jaw chuck grasp is illustrated in Figure 3-21. Figure 3-22 shows fine opposition with a thumb prosthesis opposing the index finger. This active duty soldier, injured in an accidental grenade explosion, was an avid racquetball player. To pursue his avocational goal, the racket handle was encased in a polymer prosthesis (Figure 3-23). The amputee's hand was placed into the prosthesis, and a Velcro closure snugly anchored the prosthesis and the racket to the hand (Figures 3-24 and 3-25). Similar devices can be fabricated to hold cameras, golf clubs, ski poles, and so forth, and the prosthetist or occupational therapist must work closely with the amputee.

Fig. 3-19. Thumb prosthesis for an amputation at the level of the proximal first metacarpal.

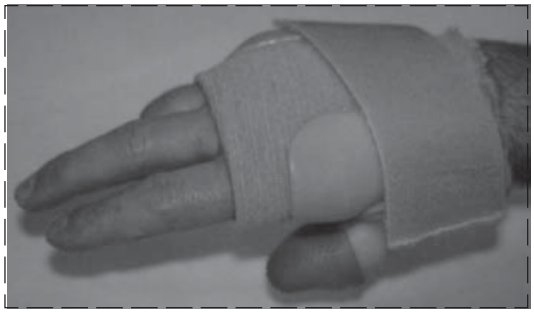


Fig. 3-20. Thumb prosthesis anchored to the residual limb.

Fig. 3-21. Three-jaw chuck grasp using a thumb prosthesis.

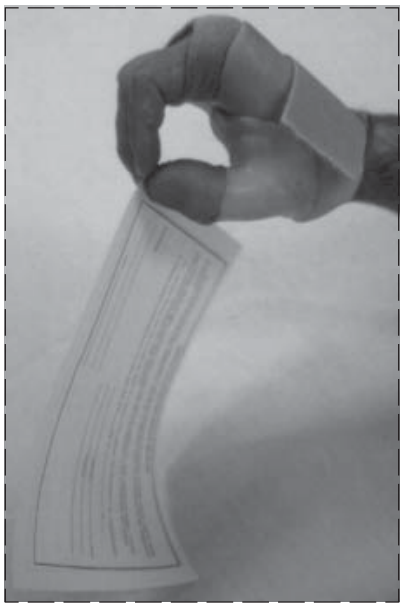


Fig. 3-22. Fine grasp using thumb prosthesis and second finger.

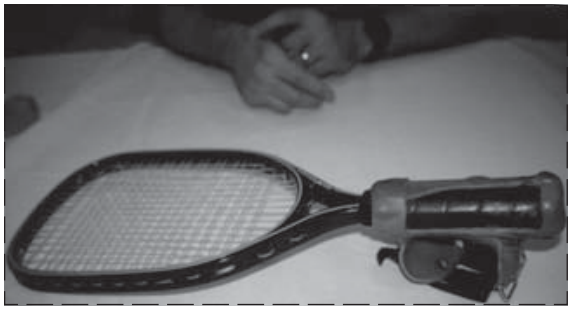


Fig. 3-23. Recreational prosthesis incorporating a racketball racket.

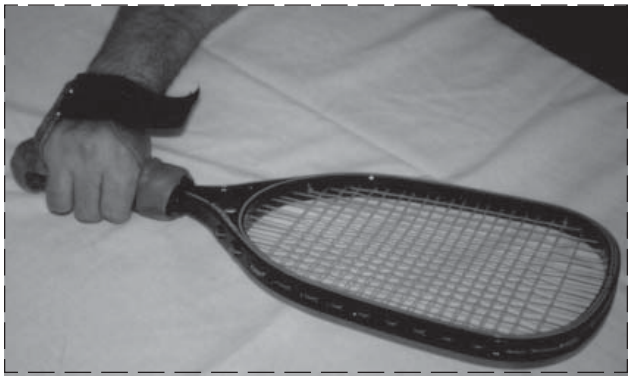


Fig. 3-24. Recreational prosthesis with Velcro closure enclosing the hand.

Fig. 3-25. Recreational prosthesis (palmar view).

Vocationally specific prostheses are also enormously valuable, particularly for soldiers pursuing careers requiring manual skills, such as a carpentry, machine work, auto mechanics, and so forth. Special prostheses enable an amputee to grasp tools and accomplish specific work tasks.

Ulnar Amputations

The fourth and fifth digits, when opposing the thumb or in a hook position, provide powerful grasp. Beasley[7] feels that the fifth finger's importance is greatly underestimated. Beasley and de Bese[10] state that a hand with only a fifth finger and a thumb function better than one with a thumb and an index finger. Full flexion of the fourth and fifth digits at the MCP and IP joints is crucial, and provides a powerful hook and cylindrical grasp.[7,14] Prosthetic substitutions for this function utilize the "scoop" concept.[14]

The scoop concept involves fabrication of a device, proportioned in size to the remaining digits, that will allow a large cylindrical grip and hook grasp. The thumb should be able to oppose this device comfortably. Figure 3-26 shows a soldier with transverse amputations of the middle and ulnar fingers at the MCP joints. A prosthesis for this soldier (Figures 3-27 and 3-28) fits over the distal end of the residual hand and extends proximally over the metacarpals on both palmar and dorsal surfaces, providing a firm attachment to the hand. In addition, a rigid loop passes between the thumb and index finger along with a Velcro strap encircling the wrist, firmly anchoring it to the hand, and enabling this soldier to carry books, briefcases, and other items. Again, a nonslip rubber palmar surface is

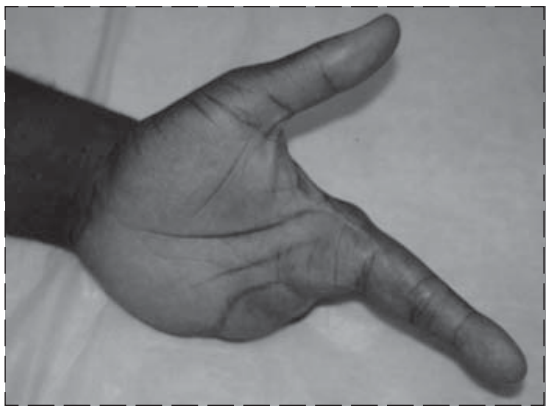


Fig. 3-26. Amputations of the middle, ring, and small fingers at the transmetacarpophalangeal level.

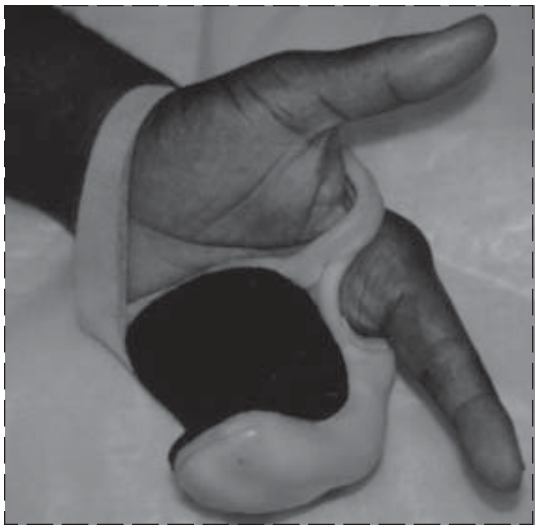


Fig. 3-27. "Scoop" type prosthesis (palmar view).

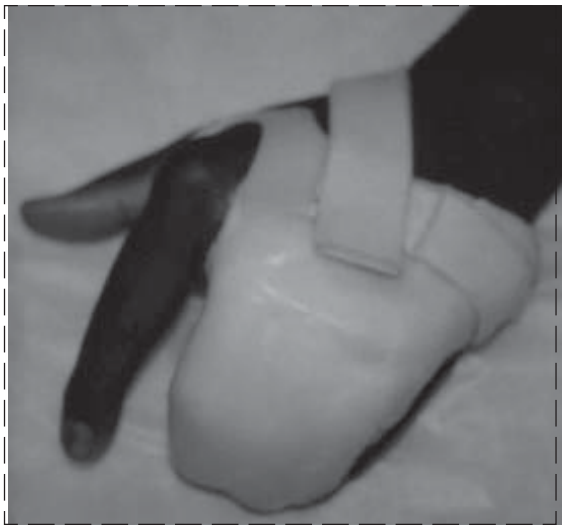


Fig. 3-28. Firm attachment of prosthesis to the residual hand by means of a Velcro closure (dorsal view).

incorporated into the prosthesis to facilitate grasp.

Central Amputations

Amputations of the middle or ring finger, if not surgically reconstructed, are easily managed with cosmetic prostheses.[7,14] Pillet[15,16] affirms the importance, to the amputee, of having a cosmetic substitute for the amputated part of the hand. For finger prostheses, there must be at least 15 to 18 mm of residual digit for the prostheses to be effectively secured. If the finger lacks this length, deepening the web space can improve suspension.[10,15] Indeed, many prosthetic users desire a functional prosthesis for work related activities and a more cosmetically acceptable prosthesis for social occasions.[10] To be of lasting benefit, cosmetic prostheses must be of high quality and must match the skin tone of the individual. Two shades of skin tone are recommended, one for winter and one for summer, so that skin tone can be matched to skin color changes due to tanning.[16] Also, fingernails and durability are important. Unilateral amputees, who have adjusted to the loss and express reasonable expectations, are optimal candidates for cosmetic prostheses. The bilateral amputee, however, usually places greater importance on the functional aspects of the prosthesis rather than on cosmetic issues.[15]

NOMENCLATURE AND FUNCTIONAL LEVELS

The following discussion addresses the nomenclature for all levels of UE amputations except the hand, which was discussed in preceding sections.

Broad categories of amputations include hand, below-elbow, and above-elbow amputations. Proximal upper limb amputations are called shoulder disarticulations and forequarter amputations (if they involve the pectoral girdle). Levels of amputation are shown in Figure 3-29.

As presented here, the term *residual limb* refers to the stump or remaining part of the amputated limb. To determine lengths of residual limbs, known anatomic points are used. Above elbow(AE) amputations are measured from the tip of the acromion to the bony end of the residual limb.[17] This length is compared to that of the noninjured side, from the acromion to the lateral epicondyle. The percentage of the amputated side relative to the intact side determines the percentages seen in Figure 3-29, which categorize the amputation levels based on functional implications.

Below-elbow(BE) measurements are made from the medial epicondyle to the end of the ulna or radius, whichever is longest in the residual limb. This measurement is divided by the length of the noninjured limb from the medial epicondyle to the ulnar styloid.[17] As in the case of AE amputation levels, functional implications are determined by the level of amputation. For the BE amputee, this primarily involves pronation and supination. BE levels determine forearm pronation and supination with a prosthetic device and affect the type of prosthesis prescribed.

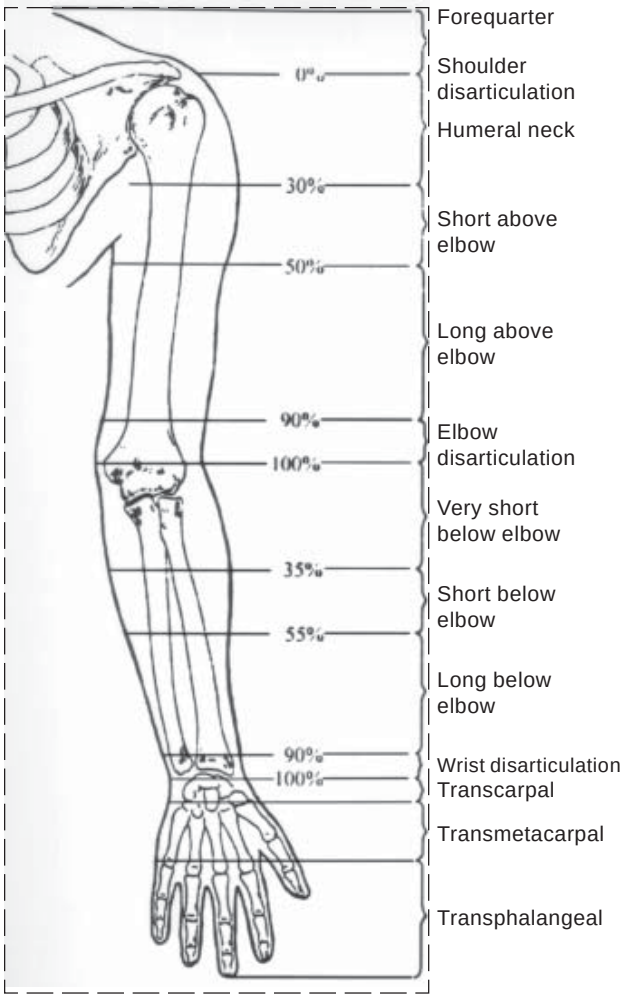


Fig. 3-29. The levels of amputation as defined by the length of the residual limb relative to the noninjured limb. Adapted with permission from Bender, LF. Upper extremity prosthetics. In: Kottke F, Lehmann JF, eds. *Krusen's Handbook of Physical Medicine and Rehabilitation.* 4th ed. Philadelphia: WB Saunders; 1990: 1011.

While wrist disarticulation amputations are rare,[18] Tooms [19]recommends that they are preferable to long BE amputations, because they preserve pronation and supination. Wrist disarticulations allow approximately 120° of pronation and supination compared with 180° in the normal case.[20] However, the actual amount of pronation and supination transmitted to the prosthesis is approximately 50% of that in the residual limb.[18] Modern wrist components easily accommodate this length of residual limb.

A long BE amputation is defined as 55% to 90% of the uninvolved extremity's length. This level preserves between 100° and 120° of pronation and supination.[20] However, pronation and supination decrease as amputation levels become increasingly more proximal. Long BE amputations provide residual limbs that are easily fitted with prosthetic devices. At this level, elbow flexion remains strong and easily transmitted to a prosthesis.

The short BE amputation, 35% to 55% of the corresponding noninjured side (see Figure 3-29), presents problems regarding pronation and supination. Here pronation and supination are absent, for all practical purposes.[20] Pronation and supination of the terminal device (TD) must be incorporated into the prosthesis by means of special wrist units.

Very short BE residual limbs (0%–35%) lack forearm pronation and supination. Additionally, elbow flexion range of motion and elbow flexion power are often reduced.[18] Suspension is often a problem. Elbow flexion range of motion can be increased with the step-up elbow joint, discussed later in this chapter.

The elbow disarticulation level poses some problems with prosthesis fitting, requiring an external elbow joint. Larger mediolateral dimensions of the humeral condyles pose difficulty in fitting a prosthetic socket, and a typical elbow unit would excessively extend the length of the residual limb. Shurr and Cook[18] feel the functional and cosmetic disadvantages of the elbow disarticulation make it suitable only for growing children, where preservation of the epiphysis for growth is important. However, Tooms[19] and McAuliffe[21] disagree and feel that elbow disarticulation is an excellent amputation level because it allows transmission of humeral rotation to the prosthesis. Modern prosthetic fabrication techniques can overcome the cosmetic and socket fit difficulties.[19,22]

The long AE level (50%–90% in Figure 3-29), is quite functional. A prosthesis can be fitted easily, glenohumeral actions are readily transmitted to the prosthesis, and ample muscles remain to control a myoelectric prosthesis (MP). In both the elbow disarticulation and long AE levels, the prosthetic socket terminates below the acromion and allows optimal shoulder movement.[20]

The short AE level (30%–50% shown in Figure 3-29) compromises transmission of glenohumeral motion to the prosthesis.[18] In this case, the prosthetic socket should extend over the acromion.[20]

Functionally, humeral neck and shoulder disarticulation levels can be classed together. These proximal levels lack the strong glenohumeral actions of flexion, extension, and abduction. Prosthetic sockets must extend over the shoulder and enclose part of the scapula and torso. It is important that all humeral lengths be preserved, even in the case

of humeral neck amputations, as this residual length aids prosthetic fitting and stabilization.[19] Prosthetic shoulders often include frictional rotation units positioned by the other extremity. Patients generally use these prostheses to stabilize objects, rather than to lift or manipulate them.

Forequarter amputations and shoulder disarticulations usually result from tumor resection. This disabling amputation diminishes biscapular abduction by one half.[18] Myoelectric prostheses (MPs) present a prosthetic alternative for this level; however, difficulty often exists in locating and training myoelectric control sites. The prosthetic arm is attached to a molded frame and fitted around the torso, which stabilizes and suspends the prosthesis.

PRINCIPLES OF AMPUTATION SURGERY

Detailed discussion of surgical procedures encompassing upper limb amputations is beyond the scope of this chapter. The general principles, however, are important to the rehabilitation specialist, and other physicians and healthcare providers who manage war injured amputees. In many ways, war surgery is not analogous to civilian surgery. Special missions of the military, the often austere medical environment, and changing combat situations that require mobility of medical services pose substantial challenges to amputee care. The war environment often limits the sophistication of surgical techniques. For these reasons, principles of military medicine evolved to maximize early care and safe evacuation of an amputee.

The primary indication for amputation is to preserve life and depends on three factors: (1) the extent of the injury, (2) the patient's condition, and (3) the expertise of the surgeon.[23] To enable subsequent reconstruction, all possible length, along with usable skin and soft tissues, should be preserved during an emergent amputation. Preservation of a joint greatly improves a patient's subsequent function, even when there is a short limb below the joint. Basic principles of prompt antibiotic treatment, early vascular repair, and early debridement, and immobilization should be followed.

The extensive contamination of war wounds and the need for evacuation from battlefield hospitals to medical centers has led to the use of the open

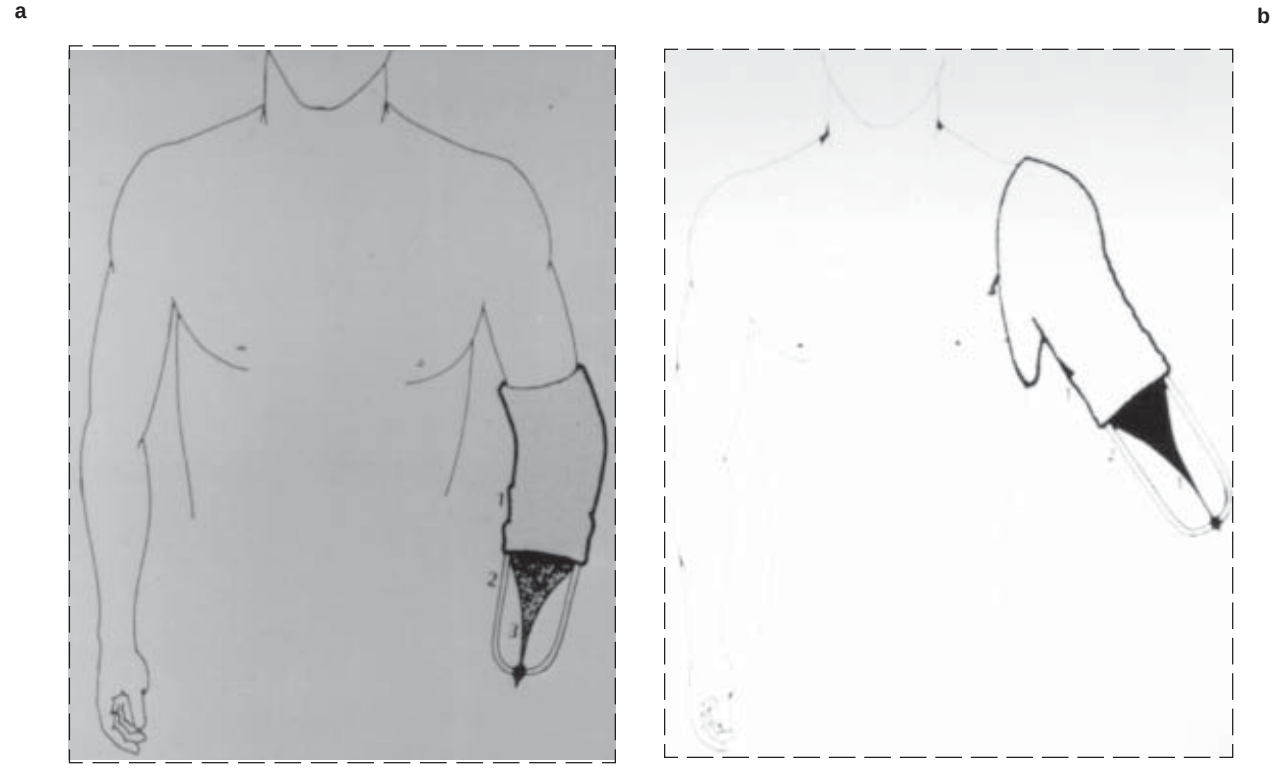


Fig. 3-30. Skin traction systems for (**a**) BE and (**b**) AE amputees. In both Figures, "1" is the cast placed over the residual limb, "2" is an attached frame, and "3" shows a stockinette placed under tension.

circular amputation as the most acceptable procedure in combat situations.[23] Open circular amputation involves severing the limb in layers: skin and subcutaneous tissues, muscle, then bone. Each layer is allowed to retract before incising the next layer, and muscle is retracted proximally before the bone is severed. This yields a stump with enough soft tissue to cover bone. During transport, skin traction is of paramount importance, and must be applied and maintained. The open tissues are dressed prior to transport, then a stockinette is placed over the residual limb and attached to the skin with tape or tincture of benzoin adhesive. Five or six pounds of traction are applied to the skin by means of a traction frame, often attached to a cast (Figure 3-30). All emergent amputations in the forward area are left open.[23] In many cases revision of a traumatic amputation is necessary.

For wrist disarticulations, the optimal surgical procedure utilizes a palmar flap from the hand to cover the distal stump, with resection of ulnar and radial styloids to minimize discomfort in the prosthesis.[19] Palmar and dorsal flaps in a 2:1 ratio provide good coverage.[24] The distal radioulnar joint, which preserves pronation and supination, should be saved.

Below elbow amputations use anterior and posterior flaps to cover the residual bone. Myodesis and myoplasty stabilize muscle ends and are important. For very short BE levels, the biceps tendon can be reattached to the ulna.[24]

Elbow disarticulation yields an optimal level.[21] The medial condyle, however, is sensitive to increased pressure, hence some surgical contouring of this prominence is useful.

For AE amputation levels, equal anterior and posterior flaps are used along with myoplasty of flexor and extensor muscles. If proximal amputations are necessary, it is important to retain the scapula whenever possible.[21] Sufficient skin and soft tissue to cover humeral disarticulations and forequarter amputations is quite important, and McAuliffe[21] recommends suturing remaining muscles to ensure padding and consistent muscle location for electromyographic (EMG) control should an MP be used. McAuliffe also recommends leaving the acromion and the coracoid process to increase leverage for body-powered prostheses. However, Baumgartner[25] suggests that for a shoulder disarticulation, removal of the coracoid process and articular part of the acromion eliminates excessively prominent bony projections, which may hinder prosthetic socket fit.

REHABILITATION

Early Rehabilitation

Rehabilitation of an upper limb amputee should begin as soon as possible following injury. Early intervention by a multidisciplinary team provides the best rehabilitative care, allowing the amputee to achieve the highest possible level of function. Following a traumatic amputation, primary treatment efforts at field hospitals include thorough debridement with preservation of the maximal limb length. Early rehabilitative interventions begun soon after injury can prevent complications of immobility, deconditioning, decubiti, and contractures. Preventing these problems facilitates rapid functional recovery. The concepts delineated in this section pertain to all upper limb amputees.

During armed conflict, there is often a significant time lapse between the time of the injury and the amputee's arrival at a medical center with rehabilitation services. Dillingham and colleagues,[26] demonstrated that most casualties arrived at medical centers in Europe and the United States about 19 days after evacuation from the Persian Gulf War theater. However, there was marked variability, with some casualties incurring much longer transport times.

During transportation, the primary concern with regard to the amputee is that adequate skin traction be constantly applied to prevent skin and subcutaneous tissues from retracting. By their very nature, battle wounds generate large amounts of debris that can contaminate wounds. Although most amputations should be allowed to close by secondary intention, in war situations it may be useful or necessary to perform secondary suture closure on some.[23] When the amputee is managed at a major medical center, surgeons decide when the wound can be closed.

In World War II, the open circular technique with skin traction was U.S. Army policy.[6] The three standard principles were (1) amputation at the lowest level permitting removal of devitalized tissues; (2) nonclosure of the wound; and (3) immediate skin traction, continuing until the residual limb healed. Certainly, during modern wartime these concepts should be followed.

As soon as possible, rehabilitation professionals should begin the rehabilitation process. Initially, this is often a consultative role. After surgery, trans-

fer to a rehabilitation service, with a team of professionals under the direction of a physiatrist, provides the best possible environment for the rehabilitation of an amputee.[27] The rehabilitation team optimally includes a physical therapist, a primary nurse, an occupational therapist, a prosthetist, a psychologist, a social worker or military administrative specialist, and a vocational counselor, in addition to a physiatrist. Although a vocational counselor frequently is unavailable at military medical centers, early consultation by a local Veterans Affairs Medical Center vocational counselor can improve vocational rehabilitation.[27] This large rehabilitation team is clearly possible only at designated military medical centers and not at corps level or evacuation hospitals.

The primary rehabilitation goals are prevention of contractures and decubiti, prevention of excessive muscle atrophy, and maintenance of skin traction on the residual limb. Contracture formation occurs very quickly due to multiple causes: edema, nerve injuries, fractures, and immobilization. During the Persian Gulf War, 10% of the lower limb casualties and 9% of the upper limb casualties who were referred to Army Physical Medicine Services suffered contractures, which complicated rehabilitation efforts.[26] To minimize these problems, all joints must be put through their full range-of-motion exercises whenever possible. Joints that are not moved regularly can form dense collagen in a disorderly fashion within four days, causing gross limitation of movement.[28] When joints are mobilized, loose connective tissue is continually formed.

Contractures are more easily prevented than treated. To prevent them, a joint should be put through its full range-of-motion exercises three times, twice a day.[28] If weakness prevents the patient from doing this, then a healthcare provider must perform this task. War injuries are often severe and life threatening. For these reasons, during acute care and evacuation, routine range-of-motion therapy may seem a secondary priority. It should be emphasized, however, that minimal intervention to prevent contractures will ultimately aid in the soldier's optimal functional restoration. Medics, nurses, therapists, and doctors can all provide joint range-of-motion therapy with very little training. Whenever extremities are taken out of their immobilization devices, if the attending physician gives approval, the joints of that extremity should be gently put through their full range of motion. For UE amputees, particular attention should be paid to preventing shoulder and elbow contractures. Maintenance of scapulothoracic motion, glenohumeral motion, elbow flexion and extension, and pronation and supination are very important.

With immobilization, muscle atrophy and deconditioning occur at an astonishing rate. At prolonged bed rest, a muscle will lose 10% to 15% of strength per week, and 50% in 3 to 5 weeks. Muscle contractions for a few seconds each day at 20% to 30% of maximal contraction will maintain strength.[29] Through early preventive measures, the amputee who maintains strength and mobility is better suited for rehabilitation.

A comprehensive rehabilitation program tailored to the individual soldier begins by obtaining a thorough database of knowledge regarding this person. The information is shared among the rehabilitation team members, and includes a detailed medical history and physical examination along with a comprehensive musculoskeletal evaluation for strength and mobility. Of particular concern to the UE amputee is adequate range of motion in all remaining joints of the residual limb; elbow flexion and extension; forearm pronation and supination; and shoulder flexion, extension, abduction, and adduction. Additionally, scapulothoracic motion and strength play critical roles in powering prostheses; hence, biscapular abduction, elevation, depression, and retraction must be evaluated. In the case of elbow disarticulations, humeral rotation is important. The residual limb must also be assessed for length, scars, and wound healing.

Current functional abilities of the casualty should be evaluated. During the Persian Gulf War, many UE amputees sustained serious wounds of other extremities, which compounded the functional problems brought on by the amputation.[26] Important evaluation elements include: hand dominance, phantom sensations or pain, education, military duties and other vocational interests, social support systems, current living situation, hobbies, ability to perform daily self-care activities, and recreational interests.[30]

Emotional aspects of a traumatic amputation pose considerable challenges for the patient and the care team. Psychological support in the structured, supportive, and educational environment provided by a cohesive rehabilitation team is vital and will help to ensure that emotional issues are adequately addressed.

Rehabilitation includes strengthening the residual limb muscles and the scapulothoracic muscles through active resistive training. Scapular abduction (or protraction) will generate tension in the control cable of a body-powered prosthesis. The

muscles that provide this function are the pectoralis major, pectoralis minor, and the serratus anterior. Elbow flexion by the biceps brachii and the brachialis provide lifting capability for the BE amputee. Because significant chest wall scar tissue may impede chest expansion, increased chest expansion should be pursued, particularly if scapulothoracic mobility and strength are compromised because this can improve control cable excursion. For an AE amputee, the elbow locking control cable is powered by combined action of shoulder (scapular) depression, extension, and abduction.[31] The muscles controlling scapular depression are latissimus dorsi, trapezius (lower fibers), and pectoralis major and minor. Major extensors of the glenohumeral joint are latissimus dorsi, teres major, and the posterior deltoid. Muscles abducting the shoulder are the deltoid and supraspinatus. Strengthening these important muscles should be aggressively pursued. Improved range of joint motion, through passive and active prolonged stretching, along with cardiovascular conditioning are important goals to pursue.

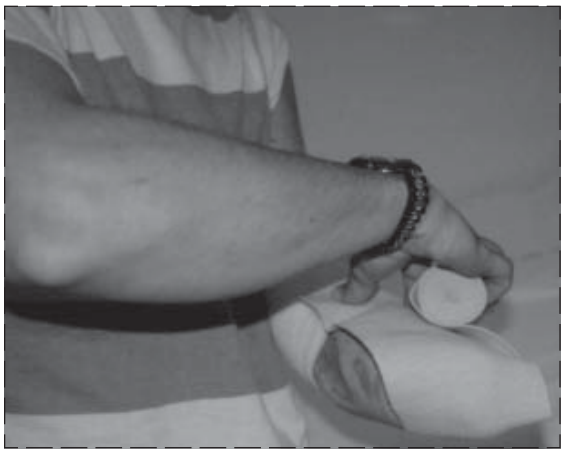
Postoperative Prosthetic Fitting

In the case of traumatic upper limb amputees, a temporary prosthesis should be fabricated when the residual limb will tolerate it. This allows the amputee early use of the residual limb with a functional prosthetic device. The term, immediate postoperative prosthesis (IPOP), refers to placement of an immediate rigid plaster or fiberglass dressing in sterile fashion over the wound; this is done in the operating room. IPOP placement minimizes pain, prevents edema formation, facilitates healing, and allows early prosthetic training.[13,17,32] War-injured amputees can be given this type of prosthesis while the open wound is healing by secondary intention, or immediately following definitive closure of the wound. This decision is, of course, made by the primary surgical physicians in concert with input from the rehabilitation team. Residual limbs that require daily monitoring for infection, skin graft success, and so forth, should not be fitted with an IPOP.

Construction of the IPOP is accomplished by covering the wounds with sterile dressings and stockinette followed by application of elastic plaster of Paris.[17] A rigid fiberglass cast material, applied over the plaster, gives added strength. Suspension straps are easily embedded into the layers of cast material. The prosthetist can add the cable housings and harnesses necessary to operate the prosthesis. The IPOP remains in place for a week or so, then is removed and replaced with a new rigid dressing. This process continues until the residual limb has matured and is ready for a more definitive prosthesis.[17] If an IPOP cannot be used due to surgical constraints (skin grafts, etc.), or the need to be able to view a residual limb, a temporary or intermediate prosthesis that can be easily removed, should be fabricated by the prosthetist at the earliest possible time.

Removable temporary prostheses are frequently fabricated from elastic plaster, forming a comfortable inner enclosure for the residual limb. Fiberglass cast material is placed over this to provide strength and a rigid frame onto which prosthetic components can be attached. If a temporary prosthesis cannot be used, then elastic wrapping (Figures 3-31 and 3-32) or elastic stockinettes should be used to mobilize and prevent edema formation in the residual

a



b

Fig. 3-31. (*Continues*)

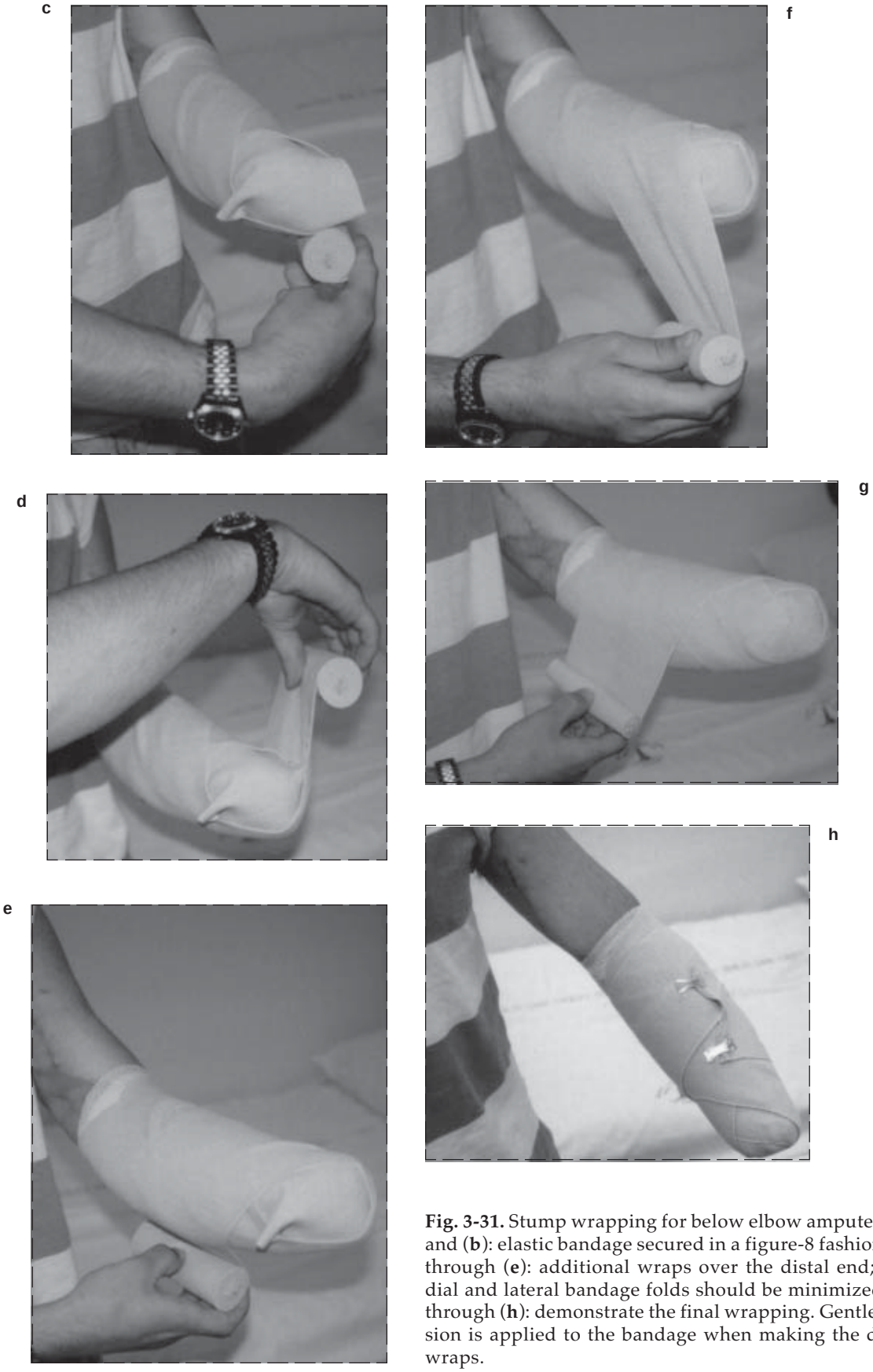


Fig. 3-31. Stump wrapping for below elbow amputee. (**a**) and (**b**): elastic bandage secured in a figure-8 fashion. (**c**) through (**e**): additional wraps over the distal end; medial and lateral bandage folds should be minimized. (**f**) through (**h**): demonstrate the final wrapping. Gentle tension is applied to the bandage when making the distal wraps.

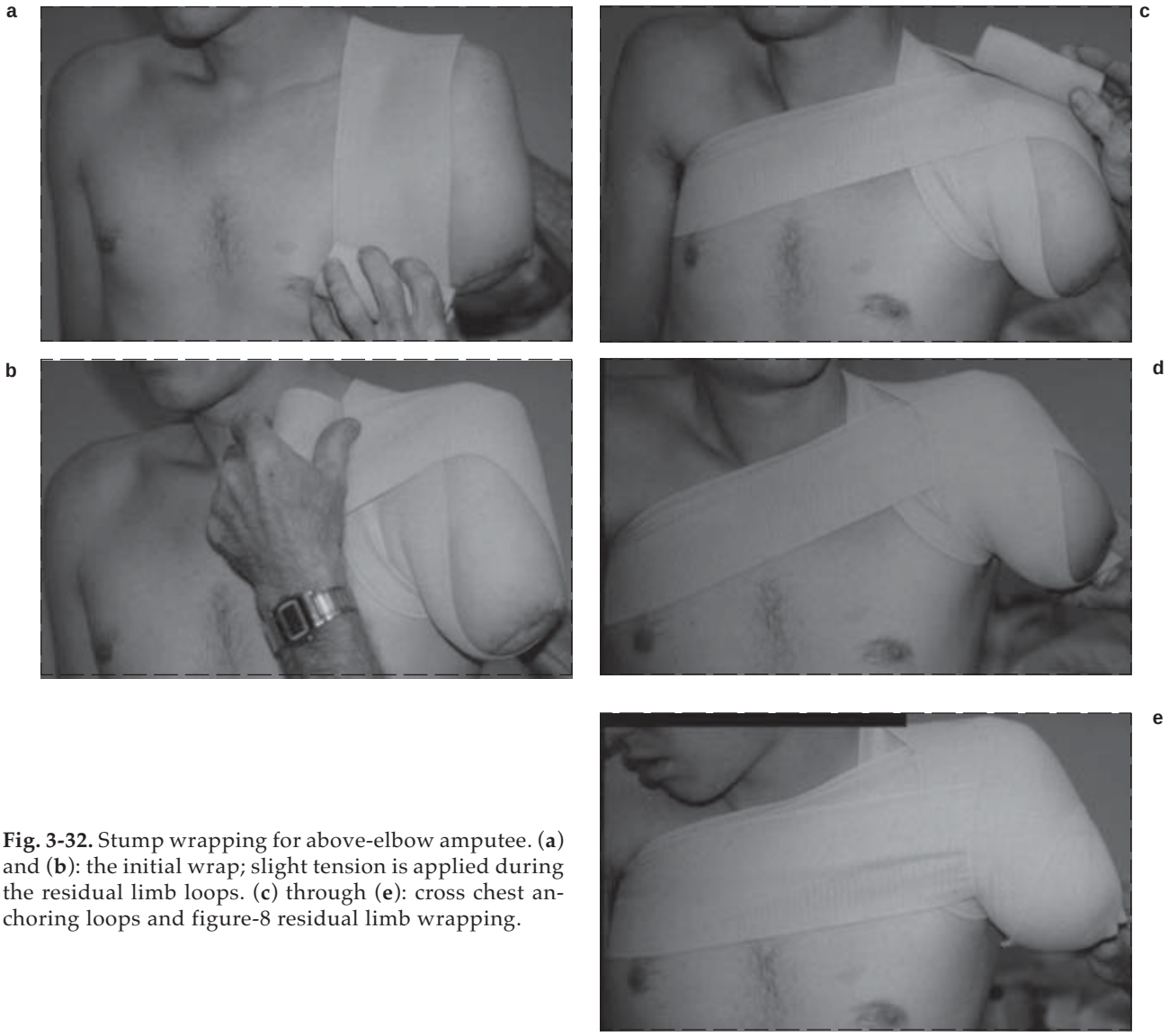


Fig. 3-32. Stump wrapping for above-elbow amputee. (**a**) and (**b**): the initial wrap; slight tension is applied during the residual limb loops. (**c**) through (**e**): cross chest anchoring loops and figure-8 residual limb wrapping.

limb. With elastic wrapping, it is important for the dressing to produce gentle distal compression of the limb. If proximal pressure is too high, the limb will be "choked," thus increasing distal edema.

Military rehabilitation professionals can significantly impact on functional rehabilitation of upper limb amputees through early intervention and prosthetic training. The World War II experience proved this to be true. Malone and associates[13] showed that early prosthetic fitting improves the success of rehabilitation after an arm amputation.

Activities of Daily Living

The occupational therapist is the primary rehabilitation professional involved with prosthetic training in activities of daily living (ADL). When the amputated limb is the dominant limb, the amputee must be trained to use the contralateral upper limb as the new dominant limb.[33] The bilateral amputee poses tremendous rehabilitative challenges and is discussed in a separate section.

Each upper limb amputee must be independent in all basic ADLs before being discharged or sent to a Veterans Affairs Medical Center. A comprehensive list of activities that a unilateral amputee should accomplish with and without a prosthesis is given in Figure 3-33. Activities include eating, personal hygiene, bathing, dressing, and homemaking. Other activities, such as driving, are of great importance to the amputee and must also be addressed.

Name	Age	Sex	Occupation
Type of amputation	Type of terminal device		
Therapist	Date(s) of test		

RATING GUIDE

0. Impossible
1. Accomplished with much strain or many awkward motions
2. Somewhat labored or few awkward motions
3. Smooth, minimal amount of delays and awkward motions

PERSONAL NEEDS:	0	1	2	3
Put on shirt				
Fasten buttons: cuff and front				
Put on belt				
Put on glove				
Put on coat				
Lace and tie shoes				
Tie a tie				
File finger nails				
Polish finger nails				
Set hair				
Clean glasses				
Squeeze toothpaste				
Put on a bra and fasten				
Use a zipper				
Hook garters				
Take bill from wallet				
Light a match				
Open pack of cigarettes				
EATING PROCEDURES:				
Carry a tray				
Butter bread				
Cut meat				
DESK PROCEDURES:				
Use dial telephone				
Use phone and take notes				
Use pay phone				
Sharpen pencil				
Use ruler				
Use scissors				
Remove and replace ink cap				
Fill fountain pen				
Fold and seal letter				
Use card file				
Use paper clip				
Use stapler				
Wrap a package				
Type				
Write				
COMMENTS:				

GENERAL PROCEDURES:	0	1	2	3
Use key in lock				
Open and close window				
Play cards and shuffle				
Wind a clock				
Assemble wall plug				
HOUSEKEEPING PROCEDURES:				
Wash dishes				
Dry dishes				
Polish silverware				
Peel vegetable				
Cut vegetable				
Open a can				
Manipulate hot pots				
Sweeping				
Use dust pan				
Use vacuum cleaner				
Use wet mop				
Use dry mop				
Set up ironing board				
Iron				
Wash and wring out laundry				
Hang up and take down laundry				
Thread needle				
Sew on button				
USE OF TOOLS:				
Layout				
Saw				
Plane				
Sand				
Drive screws				
Hammer				
File				
Drill				
Power tools				
Gravel pit				
CAR PROCEDURES:				
Drive				
Change tire				
Use Jack				

Fig. 3-33. The rating guide for "Single Upper Extremity Amputation—Activities of Daily Living," which provides a comprehensive list of activities of daily living that a unilateral amputee should be able to accomplish. This list does not include any special recreational or vocational goals that the amputee may deem important. Adapted with permission from Atkins DJ. Adult upper limb prosthetic training. In: Atkins DJ, Meier RH, eds. *Comprehensive Management of the Upper-Limb Amputee.* New York: Springer-Verlag; 1989: 49.

Training in prosthetic use starts with education regarding basic prosthesis function. The amputee is first trained in opening and closing of the TD and, in the case of an AE amputee, in locking and unlocking the elbow. Residual limb care with proper hygiene and cleaning of the prosthetic socket is taught. Putting on and removing the prosthesis can be difficult and is practiced with the assistance of a therapist. All activities learned in therapy must be reinforced by nurses working with the patient. Prosthesis wear is advanced slowly, with initial periods of only 15 to 30 minutes, followed by careful skin evaluation for possible excessive pressure.[31] Basic activities, such as grasping and lifting, are taught. Realistically, the unilateral amputee will use his intact limb for most activities, with the prosthetic limb assuming a stabilizing and positioning role.[31] Functional modifications of clothing, with loops and assistive devices, are often used to help the amputee develop independence.[34] Knowledge of the amputee's vocational and avocational interests is important because training in these areas, along with fabrication of special adaptive devices, can dramatically improve an amputee's outlook for the future. The realization that previous recreational activities can still be pursued and that the ability to work is attainable will have a positive impact on the amputee's attitude.

Canty[35] reported on amputee care in World War II at Mare Island Naval Hospital in Vallejo, California. The rehabilitation program there included early stump conditioning by means of wrapping and exercises. Physical therapists initiated exercises early in the course of treatment, often while the casualty was still at bed rest. Occupational therapists provided the soldiers with a variety of art materials and hobby activities to use while recuperating. This gave them pursuits to fill nontreatment time and provided relaxation to further improve psychological adjustments to the new disability. Round table discussions provided valuable group support for the amputee. As the soldier improved, aggressive physical training was instituted. Prevocational activities, tool work, and driving were taught along with dancing and sports.

Brown[36] has described the rehabilitation at Fitzsimons Army Hospital in Aurora, Colorado, of amputees from the Vietnam War. This program stressed a holistic approach to rehabilitating the individual. In addition to functional activities, a vigorous avocational program was pursued. Using appropriate adaptive equipment, amputees were taught snow skiing, swimming, scuba diving, and water skiing. Other important skills such as driving were taught. These activities served to place the new amputee successfully in settings outside the hospital, furthering the optimal rehabilitation and psychological adaptation.

In the military, most amputees do not remain on active duty but are discharged to Veterans Affairs Medical Centers in their local area for continued care. It is important that consistent follow-up visits be established so that education and support continue. The rehabilitation process goes on for many years, and upper limb amputees require routine prosthetic repair and maintenance throughout their lives.

BELOW ELBOW PROSTHESES

The soldier with a BE amputation is best managed by a rehabilitation team utilizing the previously mentioned rehabilitation principles. Early prosthetic training, widely recognized as the optimal way to rehabilitate amputees,[13,17,32,37] along with comprehensive rehabilitation, promotes functional independence. BE amputees require prostheses with particular components and adaptations to meet their special needs. Components that comprise a permanent prosthesis for BE amputees can be broken down into a series of devices: a socket, prosthetic suspension, prosthetic control, a wrist unit, and a TD. The physician and care team, in concert with the patient, define an optimal prosthesis.

Prosthetic Sockets

The prosthetic socket is actually composed of two sockets. An inner socket conforms exactly to the residual limb, providing a firm purchase. The outer socket fits over the inner socket and matches the contour of the opposite arm. The extent to which the prosthetic socket extends proximally depends on the length of the BE amputation. For a wrist disarticulation or a long BE amputation where some pronation and supination remain, the prosthetic socket should only extend proximally to about 1.5 cm below the epicondyles of the humerus.[17] The socket should have adequate relief for the radius and ulna when the elbow is flexed.[18] Check sockets provide a means of ensuring adequate fit. These are made from a clear, low temperature thermoplastic, which is fit to the residual limb. An example is shown in Figure 3-34. The check socket is modified to accommodate any tender bony areas and to ensure adequate fit. Once fit has been optimized

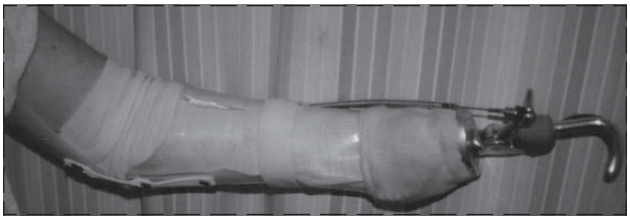


Fig. 3-34. A below-elbow amputee with clear thermoplastic check socket. The check socket can be modified, thus, optimizing fit and comfort. Note the attachment of a terminal device with fiberglass casting material.

with this socket, a positive mold, using plaster of Paris, is made from the socket. The final prosthesis is then fabricated using the positive mold as a template.[18]

For short and very short BE levels, the socket must extend more proximally. The special Muenster socket is a self-suspending socket that depends on pressure at the posterior olecranon over the triceps tendon and snug pressure around the biceps tendon to provide purchase on the limb. Suspension is not generated by attachment over the humeral epicondyles.[18] This socket is for light duty use and is well suited to myoelectric BE prostheses. If it is anticipated that the amputee must bring the prosthesis to the mouth or face, any socket can be put into some flexion.

For very short BE residual limbs, a split socket with step-up or variable-gear hinged elbow can be used. This consists of a small, mobile inner socket attached to variable-geared elbow hinges that move more than the forearm part of the prosthesis. With a 2:1 gear ratio, a very short residual limb can move less distance in flexion, while moving the external socket with the TD through twice as much excursion.[17,18] This is particularly helpful where the residual limb has limited range of motion in flexion, as in the case of contracture or heterotopic bone formation. It should be noted that the power (force) of lifting is decreased by a factor of 2, but this is the compromise required with a split socket and step-up hinges.

Terminal Devices and Wrist Units

A variety of TDs is available, which can provide specific and general functions desired by the user. Terminal devices are often easily interchanged. In most cases, they are used for prehension, but can also be specialized for hammering or other manual work. Considerable research has been invested on the improvement of both body-powered and myoelectric TDs.[11,38–44]

Common categories of TDs are the hook and the hand. Hooks are generally used in performing manual labor. Hands are thought to provide better cosmesis, particularly with myoelectric TDs (see the Myoelectric Prosthesis section in this chapter). Power to operate the TD is derived from other body muscles through a cable system (body powered) or by electric motors from a battery powered system (externally powered). Body-powered TDs typically produce voluntary opening, with rubber bands providing the closing force.[17,18,45]

In the United States, all hooks and hands have the same ½-in., 20-thread stud for attachment to wrist units. This allows ease of TD interchangeability.[17] Often amputees will have two TDs, one for functional activities and one for cosmesis.

Typical hook TDs are made from steel, for durability, or aluminum, for decreased weight.[18] Dorrance hooks are a common type of prescribed hook. These are made of either aluminum or steel and are numbered by size, with the largest number being the smallest size.[17,18] Hooks can be plastic coated, or neoprene-lined for better grip. The "thumb" of the hook is where the control cable attaches. A Dorrance hook is shown in Figure 3-35.

Recreational TDs provide the user with the ability to participate in a particular activity that would not otherwise have been possible. In a Canadian survey,[46] encompassing 2,176 amputees, the respondents reported that lack of information regarding

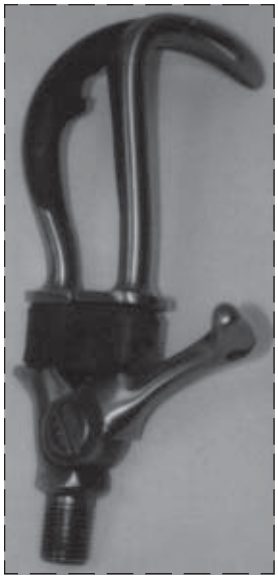


Fig. 3-35. A Dorrance Hook.

newer prosthetic components and lack of adaptive equipment for recreational activities were major concerns. Indeed, the ability to pursue avocational interests is valuable in terms of exercise and also in promoting psychological and social benefits.[11] Many TDs are made for special activities. A TD with a guitar pick attached can allow UE amputees to play the guitar. Another TD with a flexible cable attached between the prosthesis and a golf club, meets the U.S. Golf Association's regulations and enables the amputee to perform controlled powerful strokes.[11] This device can be changed from one golf club to another. Similarly, special attachments can be purchased or fabricated that allow amputees to grasp cross country and downhill ski poles.[11] The most common required characteristics of recreational prosthetic devices are durability, low weight, and strong suspension.

Vocationally specific TDs make life easier for the amputee performing special manual tasks. Driving often requires a cup or ring **(**Figure 3-36) attached to the steering wheel of a car, or a "Y" shaped TD with a nonslip rubber coating.[40]

A wrist unit is an important part of a prosthetic prescription. It connects the TD to the prosthesis and substitutes for the lost ability to pronate and supinate the forearm. This unit requires a detailed knowledge of the amputee's function and areas of vocational and avocational interest. Wrist units allow quick interchangeable use of various TDs. Some wrist units have a friction ring that limits TD rotation, and the TD is placed in the desired position by pushing it against an object or with the other hand.[17,45] The variable-friction wrist unit allows friction adjustability from low to high.[45] A thin friction wrist unit is available for amputees with wrist disarticulations.

Fig. 3-36. A steering wheel driving attachment for an amputee.

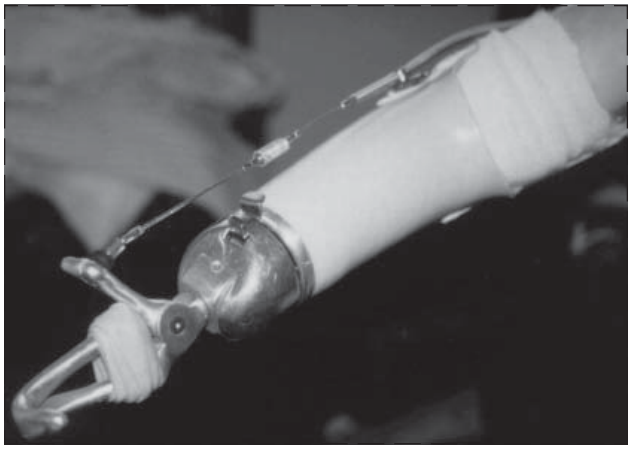


Fig. 3-37. A below-elbow prosthesis with a wrist flexion unit. The wrist unit is set without flexion.

Another type of wrist unit is the quick change unit, which has a mechanism that allows the amputee to set the TD in the desired position and then lock it.[17,45] This unit is preferred in cases where the person needs to perform heavy lifting or manipulate heavy objects.

Wrist-flexion units are special devices that allow the user to set the TD in some degree of flexion. This unit is useful for bilateral amputees who require the prosthetic extremity to perform dressing and personal hygiene activities with the TD close to the body (Figures 3-37 and 3-38).[17]

Suspension and Control

Suspension of the BE prosthesis and control of the TD are closely related and will be discussed simultaneously.

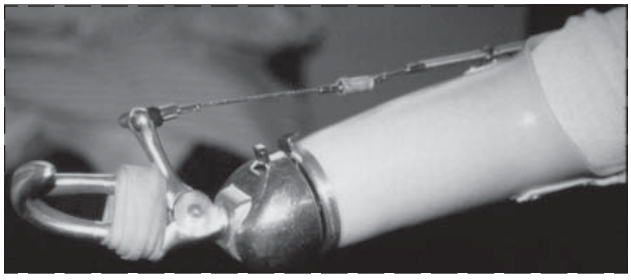


Fig. 3-38. A below-elbow prosthesis with a wrist flexion unit. The wrist flexion unit is set in a flexed position.

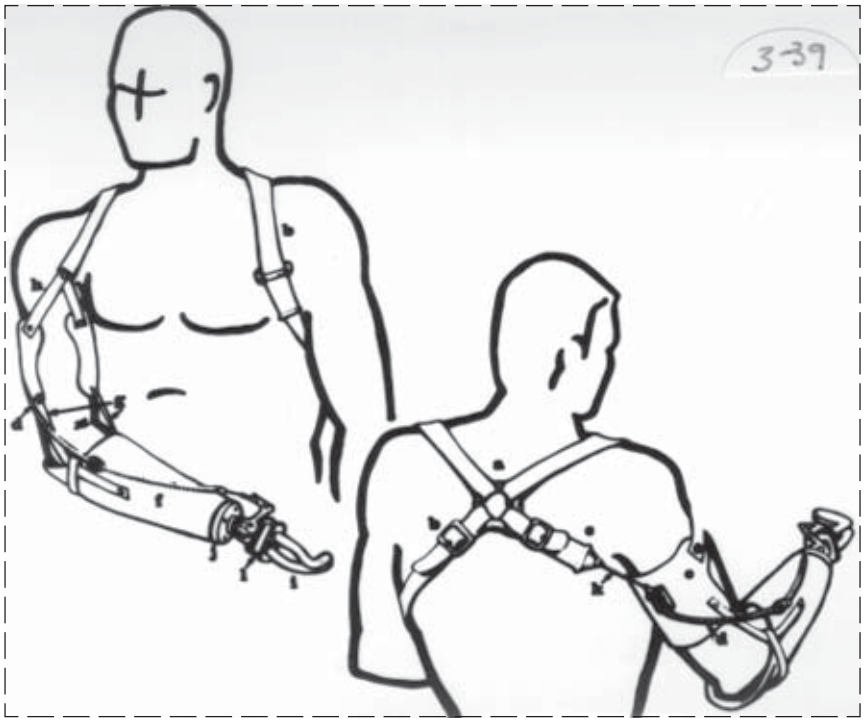


Fig. 3-39. The standard components of a BE prosthesis. The components are a: "O" ring of the figure-8 harness; b: axillary loop strap with adjustable buckle; c: Bowden cable control strap (adjustable); d: single-control Bowden cable outer cable housing; e: triceps pad; f: dual-walled socket; g: flexible elbow "hinges"; h: "Y" strap attaching figure-8 harness to the triceps pad; i: terminal device; j: wrist unit; k: inner braided steel cable that moves inside the cable housing; and l: elastic bands providing closure of the hook.

The BE amputee requires elbow joints (hinges), a triceps pad, and a figure-8 harness to suspend the prosthetic socket (Figures 3-39 and 3-40). The elbow hinges can be flexible straps (in the case of long BE amputees), or can be rigid metal hinges. The rigid hinges provide heavy-duty suspension for the aggressive user and for amputees with short and very short BE amputation levels. Rigid hinges do not allow pronation and supination, however.[17,18,45] Rigid hinges can have a single pivot, a polycentric pivot, or a step-up hinge used with a split socket.[17]

Figure 3-39 shows a figure-8 harness for a typical BE prosthesis. Posteriorly, an "O" ring is located below the C-7 spinous process and slightly to the sound side.[18] The anterior suspension strap is situated along the deltopectoral groove, and the control strap is attached posteriorly and inferiorly to the "O" ring. Shoulder abduction and scapular protraction through scapulothoracic motion provide force for the control cable.[18] The cable can also be combined with glenohumeral flexion.[31] The anterior suspension strap attaches to a "Y" strap and the triceps pad. The triceps pad redirects the suspensory force between the socket and the torso.[45] The cable operating the TD is called a Bowden control cable, and consists of a braided steel inner cable that moves inside a steel housing. The BE amputee control system is termed a single-control system, as the cable controls only one action: the opening of the TD. The cable housing is attached to the socket so that humeral flexion and scapular abduction produce tension in the inner cable, thus opening the TD. Elastic bands of varying tensions close it.

Fig. 3-40. Below elbow amputee with prosthesis.

ABOVE-ELBOW PROSTHESES

For the body-powered AE prosthesis, the same information applies regarding TDs and wrist units as in BE prostheses. Differences between AE compared to BE prostheses mainly involve the additional elbow mechanism, the humeral rotation device, and the suspension and control systems. Sockets are of dual-walled design. Suction sockets can be fitted to the AE amputee, providing better suspension, and allowing a figure-8 harness to be used alone for control.[22,47]

For the AE amputee with preserved humeral motion, a Utah Dynamic Socket (UDS) is available (Figure 3-41).[22] A conventional socket for a long AE amputation extends to the deltoid, but with abduction, it can "gap" at the proximal and lateral ends over the deltoid. It also rotates inwardly, with rotational stresses induced by lifting. The UDS has a lowered wall over the lateral deltoid and thus minimizes gapping. It contains added projections that extend over the chest anteriorly and posteriorly, providing rotational stability and minimizing suspensory needs.[22]

Elbow units for AE amputees can be flexed into the desired position and then locked with a locking control cable derived from the harness. Myoelectric elbows can also be used. Indeed, hybrid myoelectric and body-powered prostheses are quite functional. Passive control of humeral rotation is accomplished by means of positioning an elbow turntable (humeral rotation device) to the desired position.[45] Marquardt and Neff[48] describe a surgical procedure in which the residual humerus in long AE amputees is angulated to a 70° bend by means of an osteotomy. This angulated distal humerus more effectively transmits humeral rotation to the socket.

Although there are many different harness designs, their functions are identical: to suspend the prosthesis and operate active prosthetic components.[45] An example of a typical body-powered AE prosthetic system is shown in Figure 3-42. The TD and wrist units are similar to those of the BE prosthesis. The AE prosthesis requires the installation of an elbow joint and, generally, a turntable in the socket to allow placement of the prosthesis in the

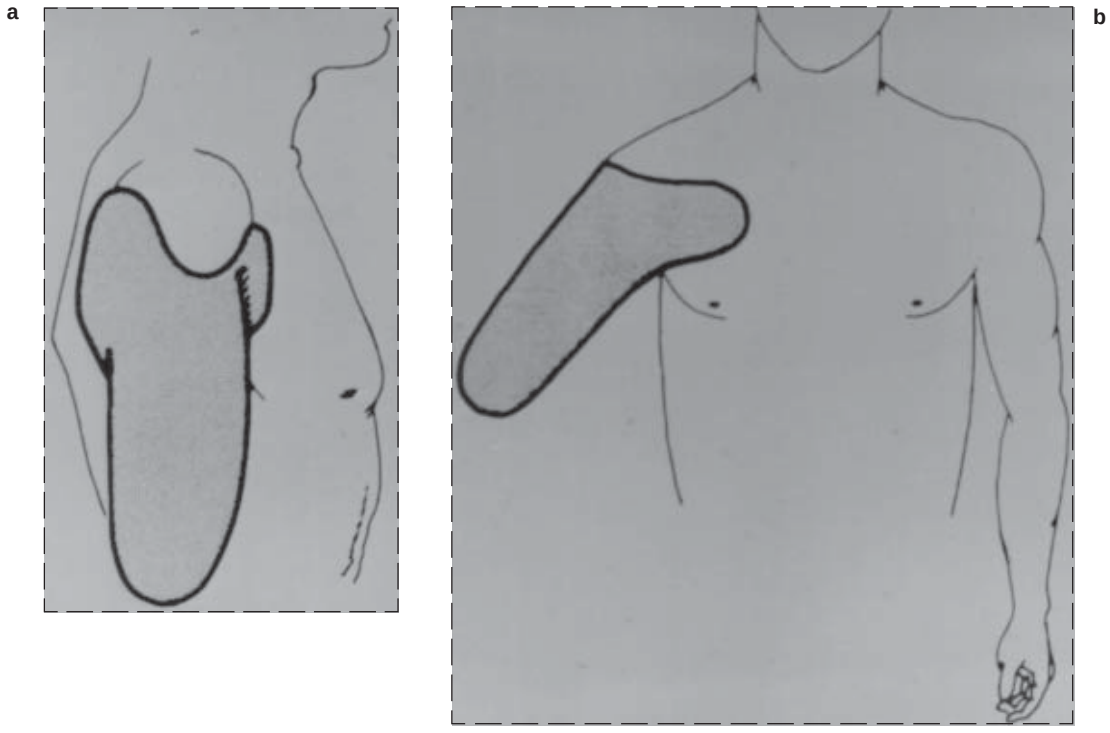


Fig. 3-41. The Utah Dynamic Socket, (**a**) lateral view and (**b**) frontal view, showing extension over pectoralis muscle. Reprinted with permission from Bowker JH, ed. *Atlas of Limb Prosthetics: Surgical and Prosthetic Principles*. St. Louis, MO: Mosby-Yearbook, Inc. 1992: 262.

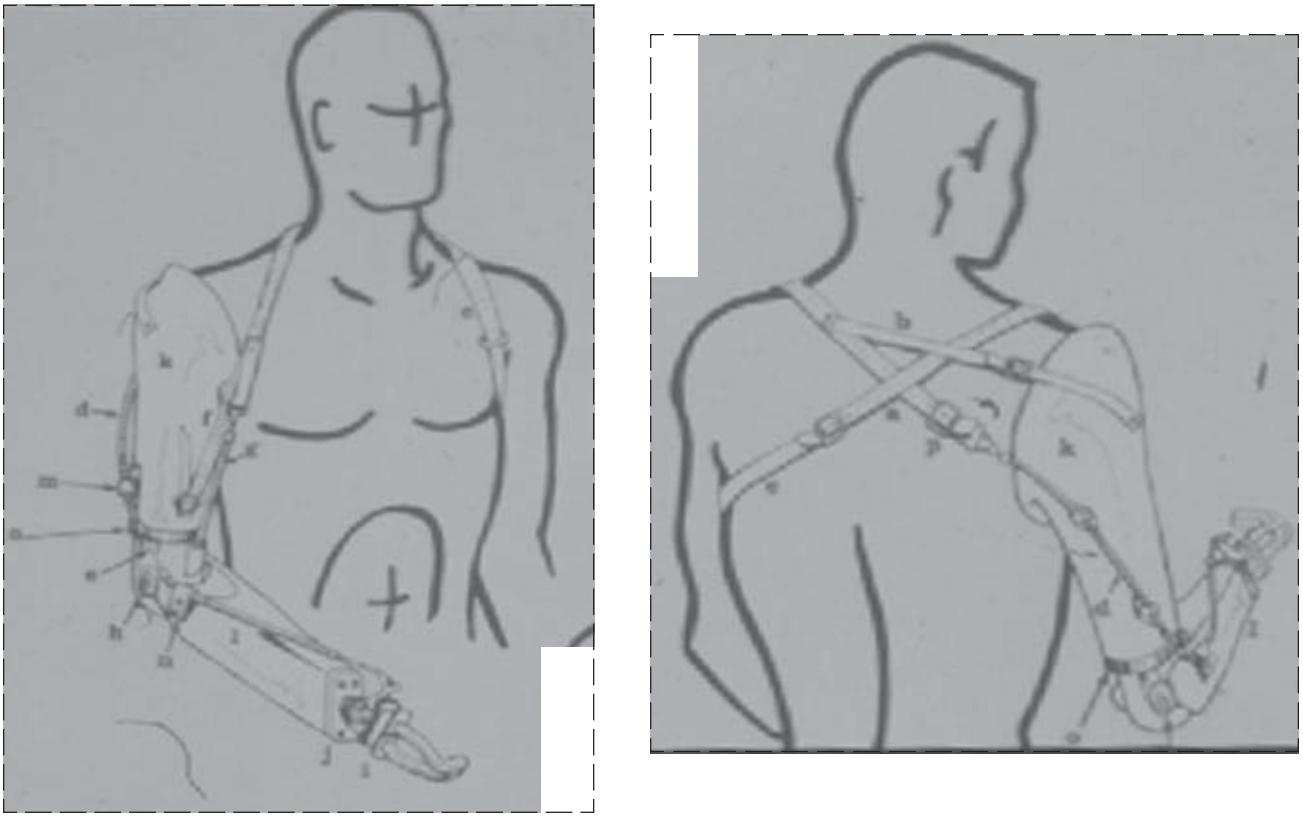


Fig. 3-42. The standard above elbow prosthesis. The components are a: modified figure-8 harness; b: lateral suspension strap for the harness (adjustable); c: axillary strap; d: dual control Bowden cable; e: bare area of Bowden cable; f: anterior elastic suspension strap; g: elbow locking control cable and adjustable strap; h: hinged elbow; i: terminal device; j: wrist unit; k: dual walled AE socket; l: forearm part of prosthesis; m: cable attachment to socket; n: cable attachment to forearm; o: friction joint for passive humeral rotation; and p: adjustable dual control cable strap.

desired position of humeral rotation. The control cable is called a dual-control cable because it operates both elbow flexion and TD opening. An elbow-locking cable (see Figure 3-42) locks the elbow when the desired position is achieved. The figure-8 adjustable harness forms the basis of the suspension system, but requires additional straps. The anterior adjustable suspension strap attaches to the prosthesis and has an elastic component. An adjustable lateral strap provides the primary suspensory force. The control cable is operated by means of an axillary loop of the harness around the contralateral limb, just as with the BE system. The adjustable elbow-locking cable originates near the anterior suspension strap. Elasticity of the anterior suspension strap allows sufficient excursion to operate the elbow lock.

Another type of suspension uses a saddle harness (Figure 3-43) and provides added suspension for someone who routinely performs heavy lifting. This padded saddle fits over the shoulder and provides a firm anchor for prosthesis suspension straps and control cables. Sometimes a cable housing, with a steel cable running through it and attaching anteriorly and posteriorly to the socket, suspends the prosthesis.[17] Figure 3-43 shows the saddle with two "V" straps that suspend the prosthesis and minimize internal and external socket rotation. An elastic suspensory strap and an elbow-locking cable passes anteriorly, in the deltopectoral groove. The elbow-locking cable is activated by shoulder extension, abduction, and depression.[31] Posteriorly, the dual-control cable is attached to the harness by another adjustable strap. Scapular abduction, shoulder abduction, and humeral flexion operate this cable. A strap passes around the chest and under the contralateral axilla, securing the saddle and prosthesis to the torso.

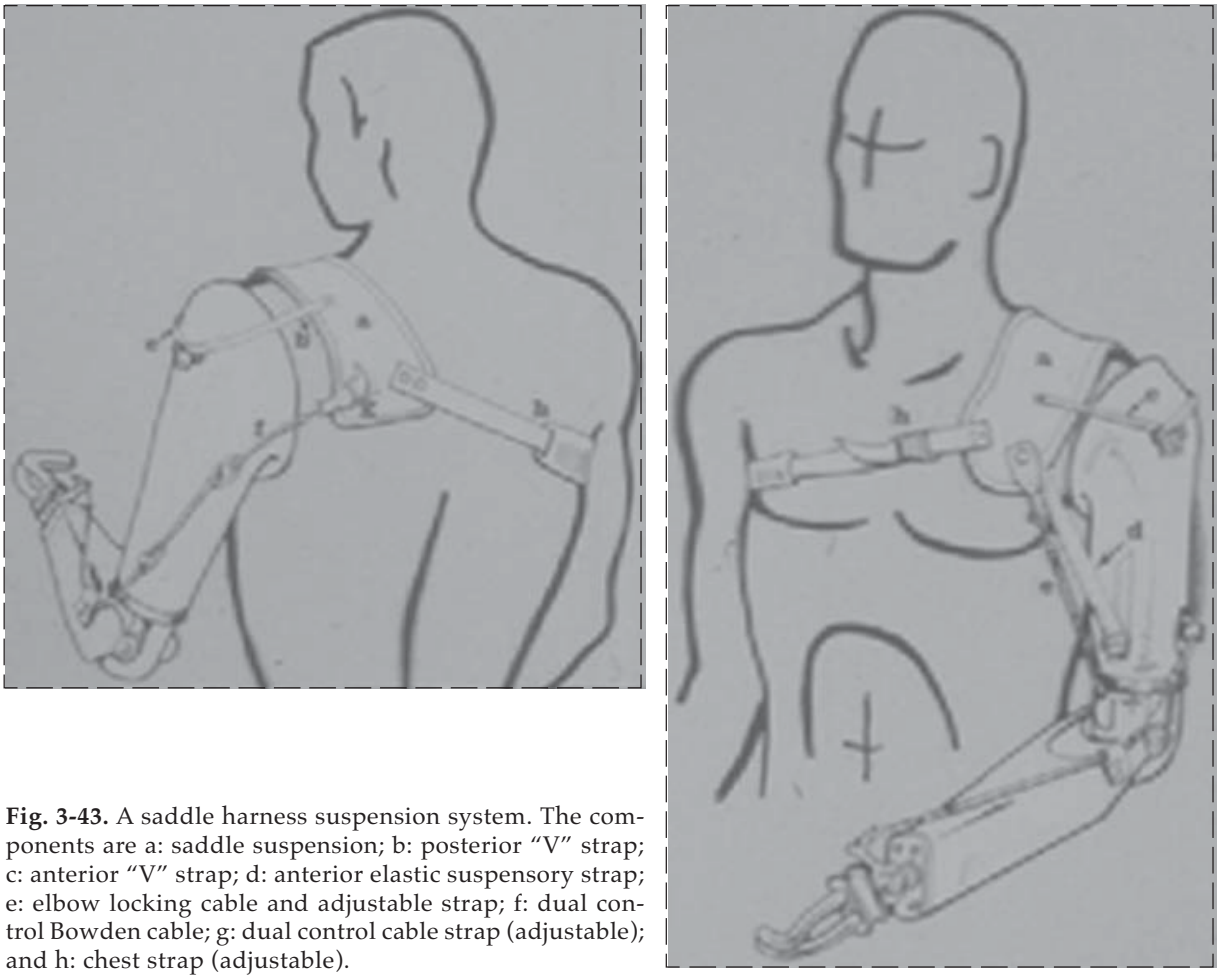


Fig. 3-43. A saddle harness suspension system. The components are a: saddle suspension; b: posterior "V" strap; c: anterior "V" strap; d: anterior elastic suspensory strap; e: elbow locking cable and adjustable strap; f: dual control Bowden cable; g: dual control cable strap (adjustable); and h: chest strap (adjustable).

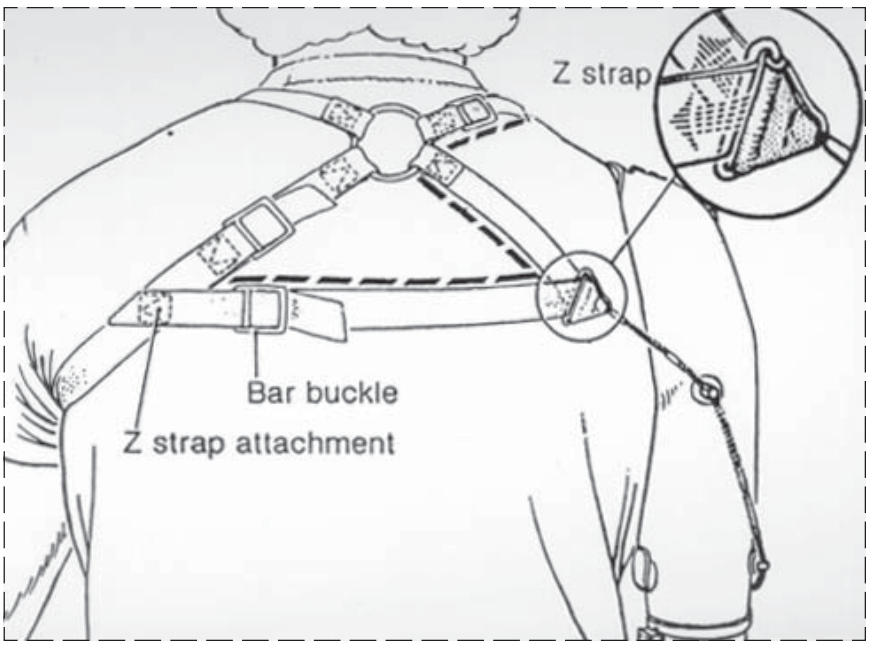


Fig. 3-44. A "Z" strap attachment allowing the control cable to glide back and forth as the prosthesis is used with the humerus in an abducted position. Reprinted with permission from Reyburn TV. The "Z" straps: Harnessing modifications for patients with upper-extremity amputations. *Arch Phys Med Rehabil*. 1991;72:250–252.

Many variations of the control and harness system exist. A modified figure-8 harness, with an anterior strap to operate the cable, can also be used.[45] A "Z" strap for the dual-control cable has been described.[49] This strap allows the TD to be used with the arm above raised 90°, and limits the problem of the "O" ring riding up the neck (Figure 3-44). In order to operate a dual-control cable, which flexes the elbow and also opens the TD, considerable cable excursion is required. Two inches of cable excursion are required to flex the elbow,[20] and approximately 2 in. of excursion can open the TD with the elbow in neutral.[18] Scapular abduction, along with humeral flexion and abduction, provide the force and excursion generated in the dual-control cable for elbow flexion and TD opening.

The humeral-neck amputee has difficulty both flexing the prosthetic elbow joint and opening the TD because all of the motion must come from biscapular abduction. The force and excursion generated by humeral flexion are approximately 63 lb and 2.1 in., respectively.[20] Biscapular abduction produces about 2 in. of excursion[18] with good force generation. According to Taylor,[20] arm (humeral) extension can generate 2.3 in. of displacement and 56 lb of force. This is much more than the 5⁄8- to 3⁄4-in. excursion and minimal force needed to operate the elbow lock. Chest expansion can also be used to operate an elbow lock.[18] Of course, the exact excursions and forces that each individual can generate are quite variable, depending on individual build, coexisting injuries, and the exact placement of control cables. However, the figures quoted above illustrate some of the difficulties in prosthetic control.

HUMERAL NECK, SHOULDER DISARTICULATION, AND FOREQUARTER PROSTHESES

At these amputation levels, the loss of humeral flexion (in the case of a shoulder disarticulation or humeral neck amputation) and unilateral loss of scapular motion (with a forequarter amputation) severely limits the amount of body-powered control. The prosthetic socket for a shoulder disarticulation and humeral neck amputation must extend over the shoulder to stabilize the prosthesis. A humeral neck amputation is shown in Figure 3-45. The prosthesis for this individual is illustrated in Figure 3-46. Forequarter amputations require an extensive prosthetic socket to stabilize the prosthesis; the socket is often attached to a frame that encompasses the torso. A prosthesis for an individual with a forequarter limb loss (a child with congenital limb loss) is shown in Figure 3-47. Here the prosthesis is firmly mounted to a body jacket, stabilizing it on the trunk. Loss of humeral extension to control the elbow-locking cable can be overcome by using chest expansion to operate the lock (see Figure 3-46). Another way to operate the elbow lock is a nudge control with a button attached to the prosthetic shell and depressed by the chin.[31] There are shoulder joints available that allow flexion, extension, abduction, and adduction.[45] However, the joint must be passively placed in the desired position by the other hand.

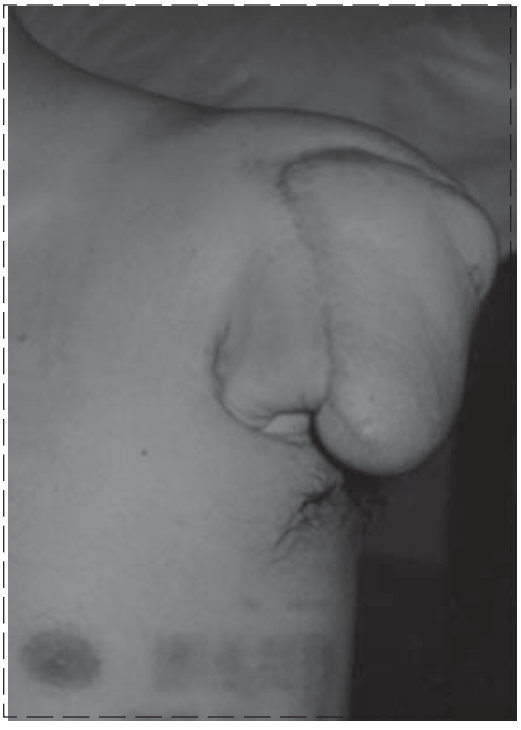
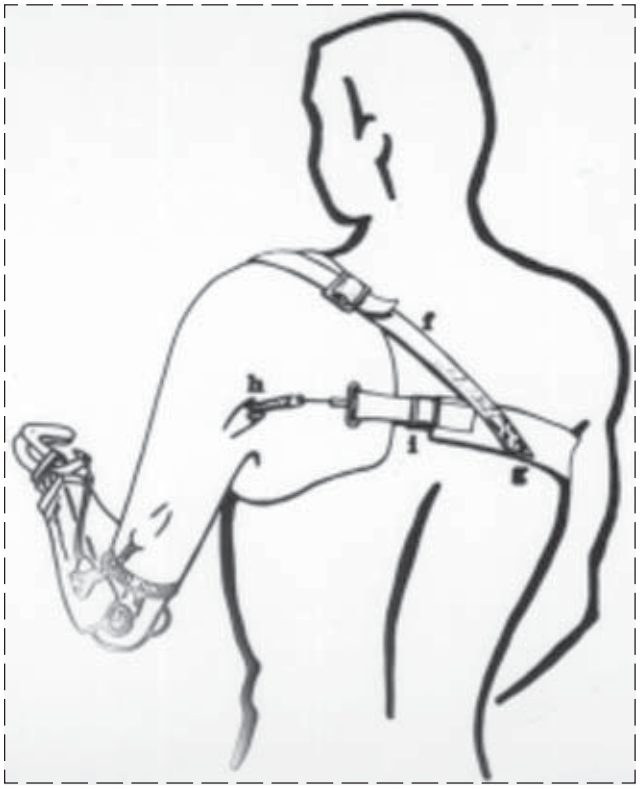


Fig. 3-45. Humeral neck amputation.

An important principle to remember is to save any residual humeral length. This allows better prosthetic fit, improves stability of the socket, and minimizes its movement. If myoelectric controls are required, the socket must consistently provide an intimate fit with respect to those control muscles.

Hybrid prostheses, with some body-powered actions and some myoelectric actions, can be used.[45] An example of this is an AE amputee could operate elbow flexion with a body-powered cable, and control the hand myoelectrically. The hybrid prosthesis can be particularly useful in the situation where body power cannot provide sufficient force and excursion in the dual-control cable to operate both actions.



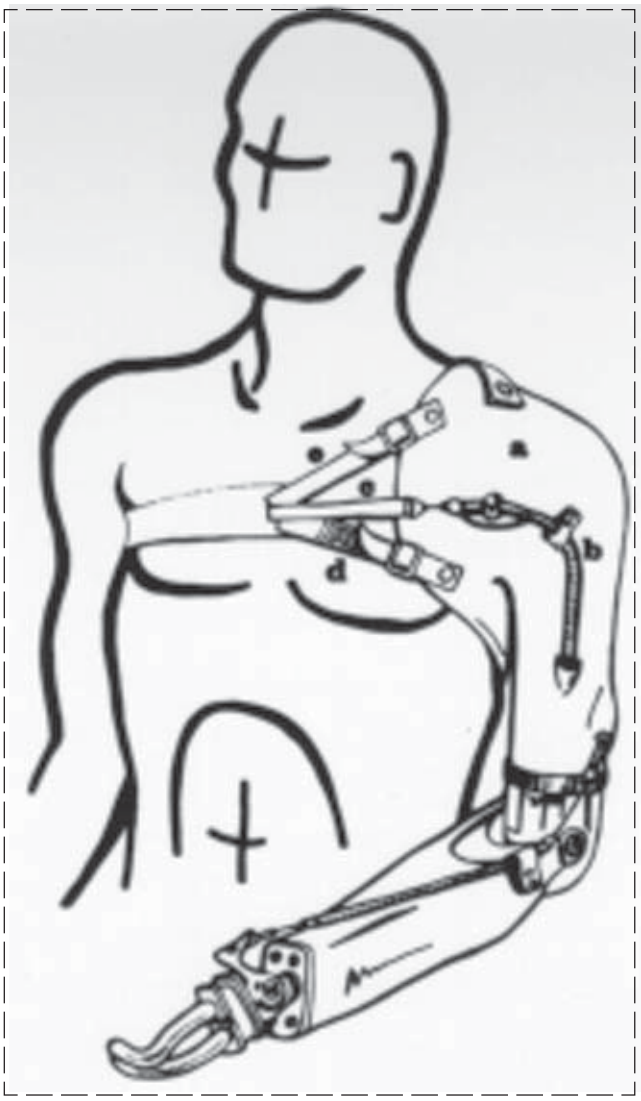


Fig. 3-46. Illustration of humeral neck amputation prosthesis using chest expansion to operate the elbow lock. Note the socket extending over the shoulder. The components are a: dual walled socket; b: elbow locking cable and housing; c: chest expansion elbow locking control strap (adjustable); d: elastic suspensory strap (adjustable); e: anterior suspensory strap (adjustable); f: posterior suspensory strap (adjustable); g: chest strap (adjustable); h: dual control cable and housing; and i: dual control cable strap (adjustable).

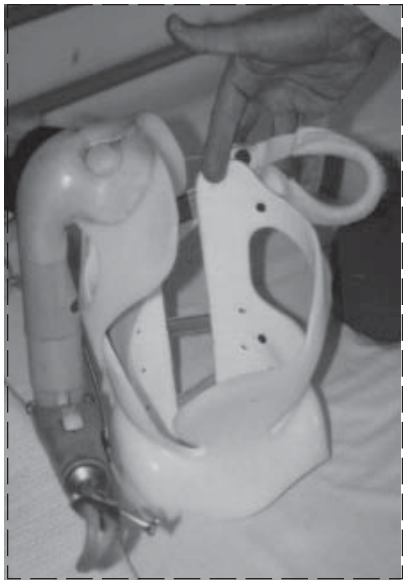


Fig. 3-47. Prosthesis for a forequarter amputee.

RESIDUAL LIMB PROBLEMS

Skin and residual limb problems in the upper limb amputee are similar to those of the lower limb amputee; however, because residual upper limbs are not load bearing like lower limbs, less abnormal forces are applied. Abnormal stresses are caused by lifting, in which case the socket presses on the distal residual limb. While lifting, the proximal socket in an AE amputee can exert high forces on the shoulder. If pain in the BE or AE amputee at the distal anterior end increases with resisted elbow or humeral flexion, the cause is probably excessive pressure or boney osteophytes.[50] Treatment is to relieve the pressure of the inner prosthetic socket over the painful area by placing a distal foam pad in this area. Other effective relief measures include increasing the surface area of the socket to distribute forces over a larger stump area. Wrist disarticulations may have styloid pain, which also can be treated with distal padding or surgical styloid reduction.[50]

Residual limb choking can occur if the proximal socket is too small and lacks total distal contact. The residual limb end may become bulbous and edematous. Distal end contact with the socket should be evaluated. This evaluation can be accomplished by inserting a clay ball into the socket and determining if it is deformed when the prosthesis is worn, or by making a lipstick mark over the residual limb and seeing if the lipstick rubs onto the inner socket. If total contact is lacking, the socket can be refabricated or a distal foam pad can be inserted inside the socket. If sensitive skin due to skin grafts or surgical scars is present, the socket can be lined with a soft inner liner to protect the limb from shear and compressive forces.

Phantom sensations after the amputation involve a common feeling that the limb is still present. Phantom pain is the abnormal increase in these sensations with painful, disturbing qualities.[50] Reasons for the pain are not clear and no single treatment strategy is optimal.[50,51] Schnell and Bunch[50] recommend a systematic approach to the painful limb, including a thorough history and examination to differentiate limb infection, postsurgical pain, or other referred pain. For the upper limb amputee it is important to remember that ischemic cardiac pain and chest wall lesions can be referred to the arm or shoulder; liver or gallbladder problems may cause diaphragm irritation. Cervical radiculopathy can cause a painful residual limb. If phantom pain is present, the frequently recommended treatment is early prosthetic fitting and use. Desensitization therapy may be of benefit. Transcutaneous electrical nerve stimulation (TENS) is often helpful, as are a variety of medications, including tricyclic antidepressants, anticonvulsants, or beta blockers.[51]

Neuromas are naturally occurring phenomena resulting from sectioned nerve ends that attempt to regenerate. If a tender area on the residual limb causes a sharp shooting pain with light tapping or palpation, then a neuroma may be present. This can be managed with local steroid and anesthetic injections. Surgical resection may be necessary if there is no resolution and the pain is functionally limiting.[50,51]

Tendinitis and bursitis may develop due to overuse of, or abnormal chronic stress caused by, the socket. These conditions can be treated with nonsteroidal antiinflammatory medications, modification of activities to include relative rest and stretching, socket modification to decrease abnormal stresses, local steroid injection, or a combination of these measures.

Skin problems, such as fungal infections, can be reduced by daily washing of the residual limb and socket with soap and water followed by thorough drying.[50] Occasionally, contact dermatitis occurs due to a local allergic reaction to a particular material, such as foam, tape, leather, and so forth. Treatment involves identification of the agent and its removal from the prosthesis.

BILATERAL UPPER LIMB AMPUTEES

The loss of both upper limbs is a tremendous psychological trauma for the wounded soldier. In addition to the immense difficulty in accepting the loss of normal arm function, the amputee must overcome special functional difficulties that are not faced by the unilateral arm amputee, who can use the remaining limb for most activities.

The bilateral proximal upper limb amputee is totally dependent for all self-care activities until optimal rehabilitation occurs. Wounded soldiers with additional injuries such as blindness, deafness, or brain injury, pose substantial rehabilitation challenges.

The amputee may become independent with prostheses, but proper prescription and training in the use of assistive devices is often more important for independence.[52] The bilateral BE amputee has more chance of regaining independence than the bilateral AE amputee. If the bilateral AE amputee

cannot oppose the residual limbs, this person may never gain independence. For some bilateral AE amputees, one residual limb is longer and more functional than the other, hence this limb takes over most of the prosthetic activities. Baumgartner[52] and Hermansson[34] each recommend that the bilateral UE amputee use an MP on one side and a body-powered prosthesis on the other. Bilateral upper limb amputees show preference, however, for body-powered prostheses, citing proprioceptive feedback, fewer repairs, and increased fine motor dexterity as reasons for the preference.[31] Bilateral amputees may be totally dependent on their prostheses for achieving function in their daily activities.

Figure 3-48 is a comprehensive list of activities the rehabilitation team can use as a guide to ensure that the amputee can meet all functional needs. The list includes many activities that may

Fig. 3-48. (*Continues*)

Fig. 3-48. The rating guide for "Bilateral Upper Extremity Amputation—Activities of Daily Living," which provides a comprehensive list of bilateral upper extremity amputee activities. Adapted with permission from Atkins DJ. Adult upper limb prosthetic training. In: Atkins DJ, Meier RH, eds. *Comprehensive Management of the Upper-Limb Amputee.* New York: Springer-Verlag; 1989: 52, 53.

not necessarily be the goals of the patient; however, this list serves as a guide to comprehensive rehabilitation training, environmental modifications, and adaptive equipment needs. Other activities, vocational and avocational, may also be pursued if they are goals of the individual amputee.

Dressing poses major difficulties for the bilateral upper limb amputee. Congenital amputees, who grow up as bilateral amputees, learn to use their feet with astonishing dexterity and are able to become quite independent in this manner.[52] Of critical importance to success in this area is modifica-

tion of clothing. These modifications include loose shirts, elastic waistbands, well placed loops, and Velcro fasteners that replace buttons and belts. A dressing hook or "tree" can position items of clothing such that the amputee can maneuver into them. The mouth and teeth are important and are frequently used to assist the amputee with grasping cuffs and collars.

Devices to position washcloths, soap, and facial care articles are very helpful.[31,52] Other helpful devices are foot-operated sinks, push button telephones that can be dialed using the nose, and automobile adaptations that enable independent driving.[52]

Toileting, understandably, is an area of self-care that is of major concern for the bilateral amputee, and there are many strategies for overcoming the inherent problems. Toilets with a water jet and air blower can be used.[52] However, this limits the amputee to using only that particular modified toilet. A fixed, wall-mounted device that consists of a stick projecting from the wall near the toilet can be used.[53] This device is approximately the height of the toilet seat and can swivel to a position over the toilet. Toilet paper is wrapped around the stick by the foot, and the patient wipes by squatting on the stick. The used toilet paper is then eased off of the stick into the toilet. Some amputees can use their prosthetic devices to wipe themselves. Wrist flexion units are particularly important to incorporate into these prostheses. Friedman[54] describes a number of devices used by the bilateral upper limb amputee to perform toileting. These include long sticks with end pieces to hold toilet paper, and variations on the wall-mounted toilet paper holder. Some of these devices can be easily transported, making the individual independent when using other toilets. Clothing adaptations; Velcro zippers; and loose, easily removed pants are helpful. There are even toilet seat–mounted devices to aid females in placement of vaginal tampons. Feminine hygiene can be managed by sanitary napkin attachment to undergarments. Particular techniques for independent perineal wiping involve placing the toilet paper on the toilet seat with feet, then rocking the buttocks over the toilet seat. Alternatively, the amputee can place toilet paper on the heel of one foot and squat over the heel.

One particular surgical procedure described and used extensively in India and Third World countries, is the Krukenberg procedure.[55,56] This procedure is used for BE residual limbs. It effectively divides the radius and ulna with their respective groups of forearm flexor and extensor muscles and creates a "claw-like forearm." The ulnar and radial halves of the "claw" can be opened and closed voluntarily, to effectively grasp objects (Figure 3-49). The major advantage to the procedure is that the opposing surfaces, which grasp objects, retain tactile sensation. The procedure is especially helpful for amputees who also have impaired vision.[55,56] These patients cannot adequately use conventional prostheses (CPs) due to lack of visual input. The disadvantage of the Krukenberg procedure is what some consider the unsightly appearance of Krukenberg limbs.[56] For Third World countries with limited availability of trained prosthetists, the Krukenberg procedure is an alternative to conventional prostheses.[55]

Fig. 3-49. Illustration of a bilateral below elbow amputee with Krukenburg limbs. Adapted with permission from Mathur BP, Narang IC, PipLani CL, Majid MA. Rehabilitation of the bilateral below-elbow amputee by the Krukenberg procedure. *Prosthet Orthop Int*.1981:5:135-140.

MYOELECTRIC PROSTHESES

Myoelectric prostheses represent significant technological developments offering alternatives for selected upper limb amputees beyond conventional prostheses. The first practical MP was demonstrated in Hanover, Germany in 1948, by Reinhold Reiter of Munich.[57] It was not until 1960 that another practical device was presented; this was in Moscow, USSR, at the First Conference of the International Federation of Automatic Control. Development of improved MPs continued throughout the 1970s in the United States, Canada, England, Denmark, Sweden, and Japan. Commercial systems became available during this time. For the BE amputee, the prescription and provision of these devices is now common in some European countries, even more so than the CPs. By 1985, between 10,000 and 20,000 MPs had been fitted to upper limb amputees worldwide.[58] Significant developments and refinements have occurred in the microprocessor control of these prostheses. Additionally, the continued advancement of power source (battery) and drive motor technology enhances the functional usefulness of the MP.

As with any prosthetic device, the advantages and disadvantages of prosthetic fitting must be carefully balanced in order to optimize the ultimate functioning of an individual. Myoelectric prostheses are only a part of comprehensive amputee rehabilitation management. The patient must continue to remain the center of informed decision making with regards to the fitting of the appropriate prosthetic device. The rehabilitation team must comprehensively evaluate the amputee. The amputee should be educated in all aspects of self-care and prosthetic needs, including being made fully aware of the special requirements of an MP, and being helped to develop a realistic perception of MP capabilities. Many patients may choose to abandon their prostheses (MP and CP) altogether in favor of unencumbered independence with the remaining upper extremity. The experience reflected in the literature suggests that for the BE amputee the MP presents a satisfactory and often appealing alternative to the CP, particularly when cosmesis is an issue.[57–63] For progressively higher levels of amputation, the functional improvements and performances are less satisfactory.[57] Myoelectric prostheses can improve the function of selected UE amputees and should be available for soldiers.

Bioengineering and Myoelectric Control

Control of myoelectric prostheses involves deriving myoelectric signals from voluntary control muscles. The signal results from the contraction of a chosen muscle on the residual limb and is recorded by surface electrodes implanted in the prosthetic socket. Electrodes must maintain contact with those particular muscles from which the control signals are derived, and the signal varies with the force of contraction. (The biophysics of myoelectric control and many of the technical considerations regarding signal extraction are available from other authors.[64–66]

The recorded myoelectric signal is first amplified, then processed into a control signal governing the electric motors that operate the prosthesis. The magnitude of the processed signal is roughly proportional to the isometric force exerted by the muscle,[67] and the microprocessor makes decisions based on the strength of the myoelectric signal.

The myoelectric signal can control a prosthetic movement or force through either digital or proportional control.[68] In proportional control, the magnitude of the myoelectric signal determines the speed or force of the prosthetic action. For example, if a particular muscle controls grip force, then the larger the myoelectric signal, the greater the grip force would be. Digital control systems determine the force or speed of prosthetic action by the duration of muscle contraction up to a preset limit. Hence, a prolonged myoelectric signal in a control muscle would cause grip force to increase. Sears and Shaperman[68] compared these two control types in a survey of MP users. They found that digital control users who subsequently switched to proportional control reported improved responsiveness with proportional control. Proportional control, however, resulted in shortened battery life. Amputees who had used digital control prior to the proportional control, preferred the latter. It was also noted that patient education is of great importance in the level of effectiveness achieved with these devices.

Two-state control involves the use of one muscle to control one action. When the amplitude of the electrical signal reaches a preset level, a particular action occurs. This is shown in Figure 3-50. As myoelectric activity increases to a certain point, an action occurs—in this case, closing the TD. For ex-

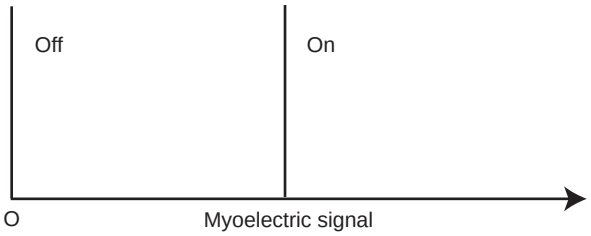


Fig. 3-50. A two-state control system. Adapted with permission from Scott RN. Biomedical engineering in upper extremity prosthetics. In: Atkins DJ, Meier RH, eds. *Comprehensive Management of the Upper-Limb Amputee*. New York: Springer-Verlag; 1989.

ample, this control system would utilize the biceps to open a TD, and the triceps to close the device. Proportional control can be combined with this system, varying the rate or force of opening or closing in response to the amount of myoelectrical signal.

An obvious disadvantage to the two-state control system is that the need for multiple muscles to control prosthetic movement makes the complex control of the TD and other actions, such as wrist flexion and extension, difficult to incorporate into an MP system. Three-state control overcomes some of these limitations. This is diagrammatically illustrated in Figure 3-51, and involves using three levels of muscle contraction (three states), each state controlling one action. In Figure 3-51, A through D represent preset cutoff points that define the states of control. Point A represents maximum background noise level. This level should be less than level B, where a particular action begins. From zero to B is state I and the device driver remains off. With B greater than A, the background noise will not accidentally operate the prosthesis. At point B, a defined action occurs and, in this case, the device closes. As the myoelectric activity increases, level C is reached, at which time the device opens. The action continues until maximum voluntary contraction occurs, level D. Level C must be substantially less than D to allow prolonged control of the prosthesis without muscle fatigue.

More sophisticated MP control methods utilizing myoelectric signals have been reported. Scott[57,65] determined that using individual motor units to control an action is possible, but requires intense concentration and, consequently, is not widely used. The technical details of myoelectric signal extraction for multidegree freedom prostheses is discussed by other authors.[66,69]

Herberts and colleagues[70] described a pattern recognition system in which a phantom limb was "moved" by the amputee, and EMG patterns were analyzed. The amputee visualized the phantom limb as if it were moving. During wrist flexion, wrist extension, pronation, supination, and finger flexion and extension, myoelectric patterns, from six electrodes attached to the residual limb, were analyzed. Four subjects were evaluated and clear patterns were identified, yet these subjects did not have the opportunity to use this complicated prosthesis at home.[70]

In another study, Almstrom and colleagues[44] reported on five subjects who used Swedish multifunctional prosthetic hands. Maintenance of this complicated device was a major problem. Fatigue in residual limb muscles from controlling the hand was also noted as a drawback. The amputees reported that compensatory shoulder and arm movements were decreased due to the many motions the hands provided. The authors concluded that compactness and reliability are necessary for prosthetic acceptance. It was pointed out that this type of system could also eventually incorporate proportional control. Myoelectric pattern recognition as a method of controlling myoelectric prostheses requires considerable technological progress before it can be widely used. Bergman and coworkers[43] demonstrated that amputees may choose conventional myoelectric hands over the more complex ones.

A new system for MP control is the Servo Pro system (marketed by Motion Control, Inc., a division of IOMED, Salt Lake City, Utah). For difficult-to-fit amputees who lack appropriate muscle sites for myoelectric control, this system uses a force sensor, which is placed in the harness, to control the MP from graded tension produced by the ampu-

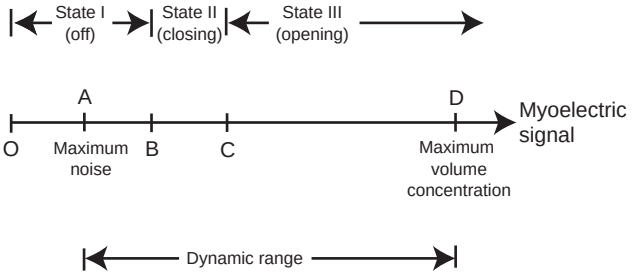


Fig. 3-51. A three-state control system. Adapted with permission from Scott RN. Biomedical engineering in upper extremity prosthetics. In: Atkins DJ, Meier RH, eds. *Comprehensive Management of the Upper-Limb Amputee*. New York: Springer-Verlag; 1989.

Fig. 3-52. Myoelectric electrodes embedded in a prosthetic socket.

tee. Proportional control is maintained. This system presents a myoelectric alternative for patients without adequate muscle control, such as those with brachial plexus injuries, shoulder disarticulations, or forequarter amputations.

Sensory feedback with an MP occurs from vibration of the prosthesis and from the noise of the motor. Various attempts have been made to improve the sensory feedback.[71] Pressure sensors in the fingertips of the prosthesis can register pinch force and process the information. Chappell and Kyberd[41] describe such a prosthetic hand, which governs grip through sensors incorporated into the fingertips. The residual limb was then stimulated by electrical impulses of increasing frequency, which corresponded to the increasing levels of force being exerted. Even though electrical stimulation of the residual limb can interfere with the myoelectric control signals,[64] this interference can be minimized by stimulating the remaining nerves of the residual limb.[71] Korner[71] showed that this form of sensory feedback is feasible and could provide the amputee with information about the MP. Implantable electrodes can be used; however, they frequently fail.[72]

Prosthetic Components

The myoelectric device incorporates special systems into a properly fitted prosthesis. The prosthetic socket contains embedded electrodes that contact control muscles on the residual limb (Figure 3-52). The myoelectric signal is amplified and then processed by a microprocessor responsible for interpreting the EMG signal. Rechargeable nickel cadmium batteries provide energy for drive motors that operate elbow, wrist, and TDs.[73] A commercially available MP is shown in Figures 3-53 and 3-54.

Fig. 3-53. The Utah myoelectric prosthesis. Photograph: Courtesy of Motion Control, Inc., a division of IOMED, Salt Lake City, Utah.

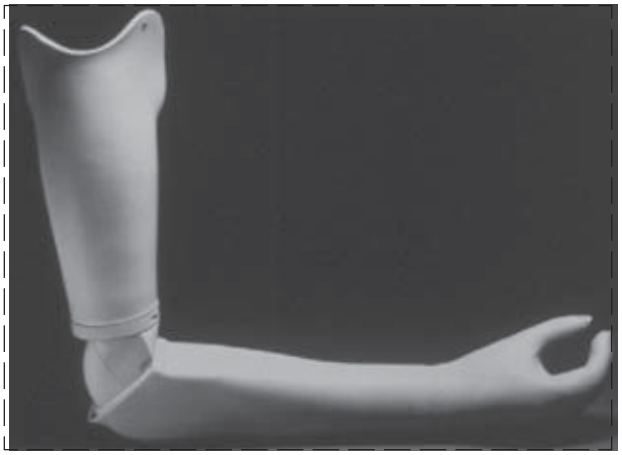


Fig. 3-54. The Utah myoelectric prosthesis with cosmetic cover. Photograph: Courtesy of Motion Control, Inc., a division of IOMED, Salt Lake City, Utah.

Two examples illustrate MP prescriptions.

Example 1

A 20-year-old female soldier sustaining a long traumatic BE amputation and desiring comfort and cosmesis in her prosthesis, decides she wants an MP. A prosthetic prescription could include some of the following variations in components:

1. BE myoelectric prosthesis with total contact, double walled, hard socket (Muenster socket) for suspension. The Muenster socket is similar to that used with a CP, and provides suspension.
2. Wrist flexor and wrist extensor controls for the myoelectric hand. The supinator muscle can also perform this activity in the case of a short BE amputation.[73]
3. Proportional two-state control. This is generally determined by the type of MP available from manufacturers.
4. A powered wrist rotator. This device demands an additional muscle control site. One in four Utah arm users have a powered wrist unit.[73] Another option is a friction wrist unit placed into the desired position with the opposite hand.

Example 2

A 34-year-old male soldier with an AE amputation decides on an MP. A prescription might include these variations:

1. AE myoelectric prosthesis.
2. A total contact, dual walled, hard socket with the inner wall containing myoelectric electrodes.
3. Figure-8 suspension.
4. Biceps and triceps control of the myoelectric elbow and hand. The Utah arm locks the elbow when the elbow position is maintained for a brief, preset period of time. Unlocking occurs with a brief cocontraction of both control muscles. Control transfers to the TD after the elbow is locked.[73] Proportional control can be used.
5. Conventional body-powered elbow flexion and myoelectrically controlled TD. This hybrid prosthesis combines the advantages of an MP and a CP.

Evaluation of Myoelectric Prosthetic Candidates

The comprehensive evaluation of an amputee who chooses an MP is similar to that of an upper limb amputee who uses a CP. There are, however, additional training issues specific to MP users.

Each amputee should be evaluated by the rehabilitation team as early as possible. Comprehensive assessments of the amputee's thoughts regarding the amputation should be discussed. A detailed history concerning the patient's vocational and avocational interests along with a complete medical evaluation should be obtained. The amputee's wishes, in terms of ultimate function and appearance of a prosthesis, should be thoroughly addressed.

A physical examination, as with any amputee, includes a complete evaluation of all organ systems, with close attention paid to the musculoskeletal and neurological systems. A candidate for an MP must have available muscle sites on the residual limb that can be trained to provide independent voluntary contractions. These muscles will control the prosthesis. An intact motor control system to the residual limb includes: (*a*) upper motor neuron input from the brain to the spinal cord; (*b*) lower motor neuron continuity to the control muscles; and (*c*) intact, nonatrophied muscles capable of generating and sustaining a useful myoelectric signal.

The decision to use an MP instead of, or in addition to, a CP is complex. Selection of the optimal prosthesis incorporates patient wishes, status of the other organ systems, adequate residual control muscles to control a prosthetic limb, functional goals of the patient (vocational and avocational) and issues of cosmesis. The patient and the rehabilitation team together make these decisions, remembering that independence and maximal function of the individual patient is the ultimate goal.[74] Substantial experience with BE amputees supports routine use of myoelectric BE prostheses for appropriate candidates.

Rehabilitation of the Myoelectric Prosthesis User

If the decision is made to provide and train an amputee in the use of an MP, additional education

must be employed beyond the comprehensive rehabilitation described for all amputees. This involves the selection of specific myoelectric control muscle sites to control the prosthesis. The type of prosthesis to be used, its control system (two-state or three-state), and any other special requirements such as cocontraction of muscle groups, should be known in advance. A myoelectric testing device identifies suitable muscle sites; it measures the surface EMG signals. Typically, BE amputees use wrist flexors and wrist extensors to operate the TD. Above-elbow amputees typically use biceps and triceps to control the prosthesis.[73] Proximal muscles of the scapula can be used. A biofeedback system is useful for training control muscles.[74] As the amputee contracts a muscle, feedback is provided to the patient regarding the contraction. According to Spiegel,[74] fine control of myoelectric activity by the amputee, is the most important part of the rehabilitation process. If a patient can consistently control muscle signals, successful MP training often occurs. The minimum myoelectric signal amplitude required is approximately 15 μV.[73]

As in the case of a body-powered prosthesis, early fitting optimizes rehabilitation. When the residual limb tissues will accommodate a temporary socket, surface electrodes embedded into it can be used to train the amputee and also to determine if the MP will be accepted and incorporated into the patient's activities.

Training initially involves learning to perform the simple activities of opening and closing the TD or flexing and extending the elbow. As progress is made, other ADLs are addressed. To practice fine control, the amputee can learn to pick up styrofoam without crushing it.[74] The unilateral amputee should also be independent without a prosthetic device, and every MP user should be trained in body-powered prosthesis use for special activities and in case of MP malfunction.[27,59]

All aspects of prosthetic maintenance must be learned by the amputee, and the MP user must treat the device with care. General precautions are to avoid carrying loads greater than 50 lb, lifting more than 2 lb, and not using the arm for hammering or with vibrating machinery.[73] In addition, the MP should be kept clean and dry.

Trends in Myoelectric Prosthetic Use

MPs have been extensively prescribed in Europe whereas there has been limited use in North America.[58] The literature provides insight into issues centering around acceptance and function of amputees using MPs.

Stein and Walley[63] studied the functionality of 20 MP users who had previously used CPs, and compared them to 16 current CP users. They found that MP users completed tasks much more slowly than CP users. However, the MP users preferred myoelectric to CPs in 60% of the cases.

Weaver and colleagues[62] measured arm function and subjective assessments in unilateral congenital amputees before and after being fitted with an MP. Pinch force was increased. A 65.6% increase in the Bimanual Functional Assessment was documented. Eight of 10 adolescents fitted with an MP preferred it to the CP, citing better cosmesis as a major advantage. Of note was the fact that cosmetic gloves covering the Otto Bock hands required frequent replacement.

Northmore-Ball and associates,[61] in a retrospective study of injured workers in Ontario, Canada, found a low rejection rate for MPs. People with desk jobs used their MPs at work more frequently than those amputees performing manual labor. The most common reason for not using the MP was fear of damaging it.

Although not applicable to wounded soldiers, Sorbye[60] documented that children with BE amputations could be successfully trained with an MP even when fitted as young as age 2.5 years. Increased maintenance requirements were again noted to be a drawback.

It appears that MPs can provide better cosmetic outcome and increased hand function. The technical drawbacks of frequent glove tears and the need for increased maintenance when compared with CPs, can possibly be eliminated as technology progresses. In certain war-injured amputees, an MP could provide optimal function. As with all amputees, the rehabilitation principles must be followed. The multidisciplinary team must be involved in the functional restoration of war injured amputees from the earliest time after injury to the time when the casualty has achieved optimal independence.

PROSTHESIS CHOICE

Prosthesis choice for AE and BE amputees is a complex decision made by the patient with advice and guidance from the rehabilitation team. The first decision involves which type of prosthesis to provide. The general categories are body powered or CPs and MPs. Sears[75] provides a useful framework for deciding which type of prosthesis to choose.

Categories of basic needs (see Table 3-1) are prioritized by each prosthetic candidate. The importance of each need depends on individual career goals, functional goals, cultural background, and daily activities. These categories are further subdivided providing additional information as to what a patient may prefer. In each category, the optimal control choice and TD (shape) choice is shown with an X.[75] For example, the Greifer Otto Bock hook is a myoelectric hook that is durable and used for rigorous work.

An excellent conceptualization for determining the optimal prosthetic prescription is shown in Figure 3-55. This figure presents two axes, the vertical being a control axis and the horizontal the TD axis. This vector approach helps define the most functional prosthesis and TD. The primary areas in relative importance are comfort, cosmesis, function, reliability, and cost.[75] The vector sum of these needs indicates the quadrant that represents the optimal prescription for that particular individual. For example, a young female soldier with a traumatic BE amputation may prefer comfort and cosmesis with less concern for function or cost (Case 1). The net vector would place her desires in the quadrant representing an MP with a hand as the TD.

Active duty amputees rarely return to active military duty following injury, so consideration as to the optimal prescription would likely include future vocational plans along with current considerations as to functional activities. Suppose an amputee enjoys carpentry work, and wants a simple, reliable, and durable prosthesis as he spends free time camping and pursuing other outdoor activi-

TABLE 3-1

EXPANDED BASIC NEEDS TABLE USED FOR EVALUATING PROSTHETIC ISSUES

	Control Choice		Shape Choice	
Basic Needs	**Body Power**	**Myoelectric**	**Hook**	**Hand**
Function				
Fine tip prehension	—	—	x	—
Cylindrical Grip (large diameter)	—	—	—	x
Cylindrical Grip (small diameter)	—	—	x	—
Flat prehension	—	—	x	—
Hook and pull	—	—	x	—
Pushing/holding down	—	—	—	x
Handling long-handled tools (handle must slide)	—	—	x	—
Ruggedness	x	—	x	—
High grip force	—	x	—	—
Delicate grip force	—	x	—	—
Visibility	—	—	x	—
Cosmesis	—	—	—	x
Comfort				
Low weight	x	—	x	—
Harness comfort	—	x	—	—
Low effort	—	x	—	—
Reliability and convenience	x	—	x	—
Low cost	x	—	—	—

Adapted with permission from Sears HH. Approaches to prescription of body-powered and myoelectric prostheses. *Phys Med Rehabil Clin North Am.* 1991;2(2):364.

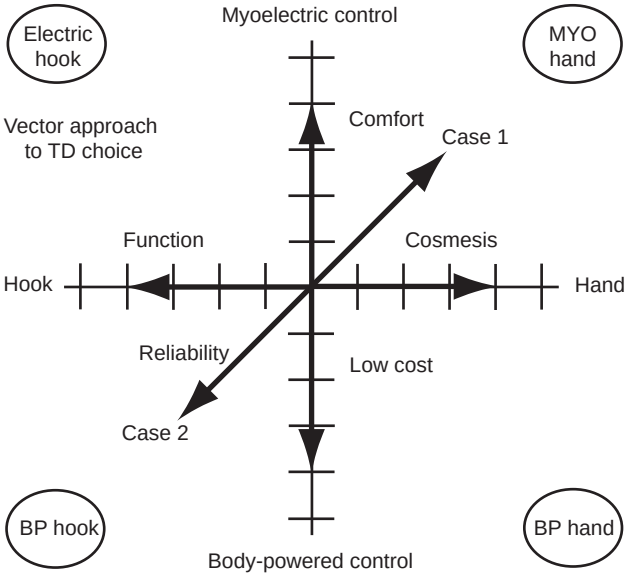


Fig. 3-55. The vector approach to terminal device prescription. Adapted with permission from Sears HH. Approaches to prescription of body-powered and myoelectric prostheses. *Phys Med Rehabil Clin North Am.* 1991;2(2):365.

ties. Cosmesis and cost are not issues to him. He knows other amputees who use hook devices so he is familiar with their appearance. In this case, reliability and function produce a net vector indicating a body-powered hook (Case 2).

This view of prescription provides an estimation of what is optimal. Sears[75] recognizes that there are many other factors that impact on the prosthetic decision, such as motivation, body image, and expectations. Early fitting and training with a temporary prosthesis allows a trial period in which the amputee can judge whether an MP or CP is suitable. All myoelectric users should be proficient with a CP because a CP will likely be used while the MP is being serviced, and because certain activities require a more rugged and durable prosthesis.

When deciding between an MP and a body-powered device, the strengths and weaknesses of each device must be considered. Spiegel[74] lists the relative advantages and disadvantages of MPs. It is important to remember that as technological improvements in myoelectric components, fitting, and production occur, many of the disadvantages may no longer apply.

Cosmesis is a major advantage of the myoelectrics. Weaver and colleagues[62] fitted 10 adolescents with myoelectric BE prostheses with Otto Bock hands and skin tone cosmetic gloves. Eight of the 10 preferred the MP, citing better cosmesis as one of the reasons.

The ability to use an MP in all planes of arm motion and overhead has been cited as one advantage.[74] Other advantages are higher pinch strength with an MP, and a graded grip strength or speed due to proportional control. Stump socks are not used with MPs, because they interfere with electrode contact. In BE prostheses, often no auxiliary suspension is necessary.

The disadvantages of MPs include high cost, frequent breakdown with high maintenance costs, technical complexity, and greater weight when compared to CPs. Reduced durability is also a major issue. Northmore-Ball and coworkers[61] in a retrospective survey of injured workers with MPs, found the major reason for not wearing MPs all the time was fear of damaging them. Sorbye[60] also noted that maintenance of MPs in children was a problem. According to Sears and associates,[73] only one week of training is necessary to train prosthetists in fitting and prescribing myoelectric devices. However, it is only the larger medical centers, which have trained, experienced rehabilitation teams, that routinely prescribe MPs and rehabilitate the receiving amputees.

VOCATIONAL OUTCOMES OF AMPUTEES

The complete rehabilitation of an amputee requires the achievement of all functional goals and the assumption by the amputee of an expected societal role involving productive work. This is particularly important for the war-injured soldier who will have many productive years remaining after injury.

Statistics regarding amputees who return to active military duty are not readily available, but a review of data from a U.S. Army Physical Evaluation Board over a 1-year period (1988–1989), revealed that only 2.3% of amputees return to active duty. Historically, during times of prolonged major conflict, amputees were utilized to perform many noncombat tasks. In World War II, amputees were sometimes trained in prosthesis fabrication and utilized in military hospitals.[4] The British Royal Air Force retained amputees on active duty, finding it more costly and time consuming to train new aircraft mechanics than to retain the amputees.[76,77] During the Civil War, amputees and other disabled soldiers frequently guarded bridges, maintained

prisoner of war camps, and performed other necessary duties. These soldiers belonged to the "Corps of Invalids," later renamed the Veterans Reserve Corps because of the negative connotation of the former name.[78]

In a follow-up study of amputees from the Vietnam War, Curley and colleagues[79] found that when comparing the social and vocational outcomes of amputees to those of noninjured Vietnam veterans, the amputees fared less well. The amputees showed twice the unemployment rate, earned less money, held more blue collar jobs, and obtained fewer college degrees than their noninjured counterparts. These results underscore the need for emphasis on vocational rehabilitation. Steinbach described the Israeli experience in rehabilitation of war-injured amputees and pointed out the importance of vocational counseling "as soon as possible after the injury."[80] Steinbach reported that 96% of Israeli amputees at discharge had vocational plans with 28% returning to their previous jobs. Unfortunately, the percentage who returned to active military duty was not stated. Ryan and coworkers[81] conducted a follow-up study of World War II amputees treated by U.S. Navy physicians. These authors followed 200 amputees and found that 78% were working or pursuing higher education. They again pointed to the need for addressing vocational issues while the injured soldier convalesced at a military hospital, stressing the inclusion of driving.

The war injured amputee poses many vocational challenges to the rehabilitation team, but the field of vocational rehabilitation has dramatically expanded in the last few decades.[82] Vocational rehabilitation includes an accurate evaluation of functional limitations of the disabled individual, particularly in work simulation tasks, and reintegration into a vocation in which the person can succeed.

The civilian experience relates similar findings and also highlights interventional strategies for improving vocational outcome. The literature[83,84] suggests that amputees do have higher unemployment than their able bodied counterparts. Only a small percentage of amputees return to their previous jobs.[83] Shepherd and Caine[85] noted in their series on traumatic UE amputees, that at follow-up only 36% had returned to their previous work, with 32% requiring retraining before doing other work, and 32% not working at all. The reasons for reduced vocational outcomes were addressed by Sheikh[86] in a study of limb injuries and amputations. Surprisingly, this author found that the exact type of limb injury (fracture, amputation, or soft tissue injury) had little if any effect on vocational outcome. However, variables such as motivation, low level of disability, short duration of unemployment, a vocational retraining program, and low unemployment in the general population strongly influenced return to work. Some of these variables are potentially modifiable through appropriate rehabilitation. Millstein and associates[87] in their review of 1,010 Canadian amputees, found that 89% of upper- and lower-limb amputees returned to work, but most (75%) changed jobs. For upper-limb amputees the return to work rate was 93%. The authors found that younger ages, comfortable and routine prosthetic use, and provision of vocational services were associated with return to work. Phantom pain and residual limb pain, along with multiple amputations, negatively impacted on return to gainful employment. Most of these Canadian amputees were injured as a result of work-related accidents.

Helm and associates[88] in their series on lower extremity amputees, found that prosthetic fit and pain were important variables affecting amputee function. Other authors[89,90] strongly support early vocational intervention. Brown[90] notes that simulated work tasks coordinated with occupational therapy and other rehabilitation professionals, can develop skills used in pursuing alternate careers. Returning to work on a part-time basis can also be advantageous.[90]

CONCLUSION

From the preceding discussion it is clear that many amputees require a change in career after their amputations, but that the majority can successfully find and maintain gainful employment. Intervention by rehabilitation professionals is very important. A comfortable and functional prosthesis, achieved through trained physiatrists and prosthetists working to optimize construction, alignment, and fit, is enormously important. Vocational counseling early in the amputee's postoperative period, with subsequent retraining, improves vocational outcomes. Earlier return to work favors a better vocational outcome and should be pursued whenever possible. Phantom limb pain and other painful residual limb problems should be aggressively addressed.

Return to active military duty following amputation is a complex issue involving the soldier's motivation; command support; special, highly developed skills that the soldier may possess; and the policy and needs of the armed forces at that particular time. The vocational restoration of injured soldiers is an important goal. Indeed, if the nation becomes engaged in a prolonged conflict, the rehabilitated soldier could potentially be a vital asset to the war effort, particularly in the small, and more technically complex army that is envisioned for the future.

There are amputees who remain on active duty, some in combat units; however, they are the exceptions. These individuals find that they require durable prosthesis that can accommodate active vocations and that they also require routine prosthetic replacements.

Upper limb amputees pose special rehabilitative challenges to the military. Designated amputation centers where skilled surgeons and rehabilitation professionals work in a coordinated fashion can best meet the needs of amputee soldiers.

ACKNOWLEDGMENTS

The author thanks Bill Discher, Armed Forces Institute of Pathology, Walter Reed Army Medical Center, Washington, DC, for his superb illustrations of prosthetic devices; and Harold H. Sears, PhD, Vice President and General Manager, Motion Control, Inc., a division of IOMED, 1290 West 2320 South, Suite A, Salt Lake City, Utah 84119, for the myoelectric slides and technical advice regarding myoelectric prostheses.

REFERENCES

1. Stern PH. The epidemiology of amputations. *Phys Med Rehabil Clin North Am*. 1991;2(2):253–261.

2. Leonard DA, Meier RH. Prosthetics in rehabilitation medicine. In: DeLisa JA, ed. *Rehabilitation Medicine Principles and Practice*. Philadelphia: JB Lippincott; 1988.

3. Peterson LT. Amputations: Historical note. In: Mullins WS, Cleveland M, Shands AR, McFetridge EM, eds. *Surgery in World War II*: *Orthopedic Surgery in the Zone of the Interior*. Washington, DC: Office of The Surgeon General, US Department of the Army; 1970.

4. Peterson LT. Administrative considerations in the amputation program. In: Mullins WS, Cleveland M, Shands AR, McFetridge EM, eds. *Surgery in World War II: Orthopedic Surgery in the Zone of the Interior*. Washington, DC: Office of The Surgeon General, US Department of the Army; 1970.

5. Bunnell S. Conclusions on the care of injured hands in World War II. Derived from the experiences of the civilian consultant for hand surgery to the Secretary of War. In: Bunnell S, ed. *Surgery in World War II: Hand Surgery*. Washington, DC: Office of The Surgeon General, US Department of the Army; 1955.

6. Peterson LT. General clinical considerations of amputation. In: Mullins WS, Cleveland M, Shands AR, McFetridge EM, eds. *Surgery in World War II: Orthopedic Surgery in the Zone of Interior*. Washington, DC: Office of The Surgeon General, US Department of the Army; 1970.

7. Beasley RW. Surgery of hand and finger amputations. *Orthop Clin North Am*. 1981;12(4):763–803.

8. Strickland JW. Restoration of thumb function after partial or total amputation. In: Hunter JM, Schneider LH, Mackin EJ, Callahan AD, eds. *Rehabilitation of the Hand*. 2nd ed. St. Louis: CV Mosby; 1984.

9. Bunnell S. Management of the nonfunctional hand—Reconstruction vs. prosthesis. In: Hunter JM, Schneider LH, Mackin EJ, Callahan AD, eds. *Rehabilitation of the Hand*. 2nd ed. St. Louis: CV Mosby; 1984.

10. Beasley RW, de Bese GM. Upper limb amputations and prostheses. *Orthop Clin North Am.* 1986:17(3):395–405.

11. Michael JW, Gailey RS, Bowker JH. New developments in recreational prostheses and adaptive devices for the amputee. *Clin Orthop.* 1990:256:64–75.

12. Patricelli J. Adapted knife for partial hand amputated patients. *Am J Occup Ther.* 1982;36(3):193–194.

13. Malone JH, Childers SJ, Underwood J, Leal JH. Immediate post-surgical management of upper extremity amputation: Conventional, electric and myoelectric prosthesis. *Orthot Prosthet*. 1981:35(2):1–9.

14. White JG, HilFrank BC. Prosthetic and adaptive devices for the partial hand amputee. In: Hunter JM, Schneider LH, Mackin EJ, Callahan AD, eds. *Rehabilitation of the Hand*. 2nd ed. St. Louis: CV Mosby; 1984.

15. Pillet J. The aesthetic hand prosthesis. *Orthop Clin North Am.* 1981;12(4):961–969.

16. Pillet J. Aesthetic hand prostheses: Its psychological and functional potential. In: Hunter JM, Schneider LH, Mackin EJ, Callahan AD, eds. *Rehabilitation of the Hand*. 2nd ed. St. Louis: CV Mosby; 1984.

17. Bender, LF. Upper-extremity prosthetics. In: Kottke F, Lehmann JF, eds. *Krusen's Handbook of Physical Medicine and Rehabilitation*. 4th ed. Philadelphia: WB Saunders; 1990.

18. Shurr DG, Cook TM. Upper-extremity prosthetics. In: Shurr DG, Cook TM, eds. *Prosthetics and Orthotics*. Norwalk, Conn: Appleton and Lange; 1990.

19. Tooms RE. Amputation surgery in the upper extremity. *Orthop Clin North Am.* 1972;3(2):383–395.

20. Taylor CL. The biomechanics of control in upper-extremity prostheses. *Orthot Prosthet*. 1981;35(1):7–28.

21. McAuliffe JA. Elbow disarticulation and transhumeral amputation/shoulder disarticulation and forequarter amputations. In: Bowker JH, Michael JW, eds. *Atlas of Limb Prosthetics: Surgical, Prosthetic, and Rehabilitation Principles*. 2nd ed. St. Louis: Mosby-Year Book; 1992: 22.

22. Andrew JT. Prosthetic Principles. In: Bowker JH, Michael JW, eds. *Atlas of Limb Prosthetics: Surgical, Prosthetic, and Rehabilitation Principles*. 2nd ed. St. Louis: Mosby-Year Book; 1992.

23. Bowen TE, Bellamy RF. *Emergency War Surgery*. 2nd ed. Washington, DC: US Government Printing Office; 1988.

24. Ouellette EA. Wrist disarticulation and transradial amputation. In: Bowker JH, Michael JW, eds. *Atlas of Limb Prosthetics: Surgical, Prosthetic, and Rehabilitation Principles*. 2nd ed. St. Louis: Mosby-Year Book; 1992.

25. Baumgartner RF. The surgery of arm and forearm amputations. *Orthop Clin North Am.* 1981;12(4):805–817.

26. Dillingham TR, Spellman NT, Braverman SE, et al. *Analysis of Casualties Referred to Army Physical Medicine Services During Wartime. Am J Phys Med Rehabil.* 1993;72(4):214–218.

27. Van Lunteren A, Van Lunteren-Gerritsen GH, Stassen HG, Zuithoff MJ. A field evaluation of arm prostheses for unilateral amputees. *Prosthet Orthot Int*. 1983;7:141–151.

28. Kottke FJ. Therapeutic exercise to maintain mobility. In: Kottke FJ, Lehmann JF, eds. *Krusen's Handbook of Physical Medicine and Rehabilitation*. 4th ed. Philadelphia: WB Saunders; 1990.

29. Halar EM, Bell KR. Rehabilitation's relationship to inactivity. In: Kottke FJ, Lehmann JF, eds. *Krusen's Handbook of Physical Medicine and Rehabilitation*. 4th ed. Philadelphia: WB Saunders; 1990.

30. Meier RH. Evaluation of and planning for acquired upper-limb amputee rehabilitation. In: Atkins DJ, Meier RH, eds. *Comprehensive Management of the Upper-Limb Amputee*. New York: Springer-Verlag; 1989.

31. Atkins DJ. Adult upper limb prosthetic training. In: Atkins DJ, Meier RH, eds. *Comprehensive Management of the Upper-Limb Amputee*. New York: Springer-Verlag; 1989.

32. Dalsey R, Gomez W, Seitz WH, Dick HM, Hutnick G, Akdeniz R. Myoelectric prosthetic replacement in the upper-extremity amputee. *Orthop Rev*. 1989;18(6):697–702.

33. Atkins DJ. Postoperative and pre-prosthetic therapy programs. In: Atkins DJ, Meier RH, eds. *Comprehensive Management of the Upper-Limb Amputee*. New York: Springer-Verlag; 1989.

34. Hermansson LM. Structured training of children fitted with myoelectric prostheses. *Prosthet Orthot Int*. 1991;15:88–92.

35. Canty TJ. Amputee rehabilitation. *Arch Phys Med Rehabil*. April 1951;32:219–221.

36. Brown PW. Rehabilitation of bilateral lower extremity amputees. *J Bone Joint Surg Am.* 1970;52(4):687–700.

37. Bailey RB. An upper extremity prosthetic training arm. *Am J Occup Ther*. 1970;24(5):357–359.

38. Meeks D, LeBlanc M. Preliminary assessment of three new designs of prosthetic prehensors for upper limb amputees. *Prosthet Orthot Int*. 1988;12:41–45.

39. Kruit J, Cool JC. Body-powered hand prosthesis with low operating power for children. *J Med Eng Technol*. 1989;13(1/2):129–133.

40. Sensky TE. A simple and versatile driving appliance for upper-limb amputees. *Prosthet Orthot Int*. 1980;4:47–49.

41. Chappell PH, Kyberd PJ. Prehensile control of a hand prosthesis by a micro-controller. *J Bioeng*. 1991;13:363–369.

42. Lewis EA, Shevedos CR, Sowell TT, Houston VL. Clinical application study of externally powered upper-limb prosthetics systems: The UA elbow, the UA hand, and the UA/NU myoelectric hand systems. *Bull Prosthet Res*. 1975;10-24:51–136.

43. Bergman K, Ornholmer L, Zackvisson K, Thyberg M. Functional benefit of an adaptive myoelectric prosthetic hand compared to a conventional myoelectric hand. *Prosthet Orthot Int.* 1992;16:32–37.

44. Almstrom C, Herberts P, Korner L. Experience with Swedish multifunctional prosthetic hands controlled by pattern recognition of multiple myoelectric signals. *Int Orthop*. 1981;5:15–21.

45. Muilenburg AL, LeBlanc MA. Body-powered upper limb components. In: Atkins DJ, Meier RH, eds. *Comprehensive Management of the Upper-Limb Amputee*. New York: Springer-Verlag; 1989.

46. Chadderton HC. Consumer concerns in prosthetics. *Prosthet Orthot Int*. 1983;7:15–16.

47. Levack H. Suction socket prosthesis for A.E. amputees. *Can J Occup Ther*. 1971;38(3):121–124.

48. Marquandt E, Neff G. The angulation osteotomy of above-elbow stumps. *Clin Orthop*. 1974;104:232–238.

49. Reyburn TV. The "Z" straps: Harnessing modifications for patients with upper-extremity amputations. *Arch Phys Med Rehabil*. 1991;72:250–252.

50. Schnell MD, Bunch WH. Management of pain in the amputee. In: Bowker JH, Michael JW, eds. *Atlas of Limb Prosthetics: Surgical, Prosthetic, and Rehabilitation Principles.* 2nd ed. St. Louis: Mosby-Year Book; 1992.

51. Vaida G, Friedmann LW. Postamputation phantoms: A review. *Phys Med Rehabil Clin North Am.* 1991;2(2):325–353.

52. Baumgartner RF. Management of bilateral upper limb amputees. *Orthop Clin North Am*. 1981;12(4):971–977.

53. Weiss-Lambrou R, de Sart M, Moreau R, Duquette J. Brief on new toilet independence for the severe bilateral upper-limb amputee. *Am J Occup Ther.* 1985;39(6):397–399.

54. Friedman L. Toileting self-care methods for bilateral high level upper limb amputees. *Prosthet Orthot Int.* 1980;4:29–36.

55. Visuthikosol V, Wonglousarakum S, Navykarn T, Druavit A. The Krukenberg procedure in the bilateral amputee after electrical burn. *Ann Plast Surg*. 1991;27(1):56–60.

56. Mathur BP, Narang IC, PipLani CL, Majid MA. Rehabilitation of the bilateral below-elbow amputee by the Krukenberg procedure. *Prosthet Orthot Int*. 1981;5:135–140.

57. Scott RN. Biomedical engineering in upper extremity prosthetics. In: Atkins DJ, Meier RH, eds. *Comprehensive Management of the Upper-Limb Amputee*. New York: Springer-Verlag; 1989.

58. Kritter AE. Current concepts review myoelectric prostheses. *J Bone Joint Surg Am.* 1985;67(4):654–657.

59. Datta D, Kingston J, Ronah DJ. Myoelectric prostheses for below-elbow amputees: The Trent experience. *Int Disabil Stud.* 1989;11(4):167–170.

60. Sorbye R. Myoelectric prosthetic fitting in young children. *Clin Orthop*. 1980;148:34–40.

61. Northmore-Ball MD, Heger H, Hunter GA. The below-elbow myoelectric prosthesis: A comparison of the Otto Bock myoelectric prosthesis with the hook and functional hand. *J Bone Joint Surg Br.* 1980;62(3):363–367.

62. Weaver SA, Lange LR, Vogts VM. Comparison of myoelectric and conventional prostheses for adolescent amputees. *Am J Occup Ther*. 1988;42(2):87–91.

63. Stein RB, Walley M. Functional comparison of upper extremity amputees using myoelectric and conventional prostheses. *Arch Phys Med Rehabil*. 1983;64:243–248.

64. Scott RN. Feedback in myoelectric prostheses. *Clin Orthop*. 1990;256:58–63.

65. Scott RN. Myoelectric control systems research at the Bio-engineering Institute, University of New Brunswick. *Med Prog Technol*. 1990;16(1–2):5–10.

66. Kelly MF, Parker PA, Scott RN. The application of neural networks to myoelectric signal analysis: A preliminary study. *IEEE Trans Biomed Eng.* 1990;37(3):221–230.

67. Winter DA. *Biomechanics of Human Movement*. New York: Wiley and Sons; 1979.

68. Sears HH, Shaperman J. Proportional myoelectric hand control: An evaluation. *Am J Phys Med Rehabil.* 1991;70(1):20–27.

69. Herberts P, Kaiser E, Magnusson R, Peterson I. Power spectra of myoelectric signals in muscles of arm amputees and healthy normal controls. *Acta Orthop Scand*1973;44(2):161–193.

70. Herberts P, Almstrom C, Caine K. Clinical application study of multi-functional prosthetic hands. *J Bone Joint Surg Br.* 1978;60(4):552–560.

71. Korner L. Afferent electrical nerve stimulation for sensory feedback in hand prostheses. *Acta Orthop Scand*. 1979;Suppl:178.

72. McNeal DR, Waters R, Reswick J. Experience with implanted electrodes. *Neurosurgery*. 1977;1(2):228–229.

73. Sears HH, Andrew JT, Jacobsen SC. Experience with the Utah arm, hand, and terminal device. In: Atkins DJ, Meier RH, eds. *Comprehensive Management of the Upper-Limb Amputee*. New York: Springer-Verlag; 1989.

74. Spiegel SR. Adult myoelectric upper-limb prosthetic training. In: Atkins DJ, Meier RH, eds. *Comprehensive Management of the Upper-Limb Amputee*. New York: Springer-Verlag; 1989.

75. Sears HH. Approaches to prescription of body-powered and myoelectric prostheses. *Phys Med Rehabil Clin North Am*. 1991;2(2):361–371.

76. Wynn Parry CB. The management of injuries to the brachial plexus. *Proc R Soc Med*. 1974;67:488–490.

77. Eldar R, Ohry A. Preparation of a hospital rehabilitation system for war and other disasters. *Med War*. 1990;6(7):105–111.

78. Adams GW. *Doctors in Blue: The Medical History of the Union Army in the Civil War*. Dayton, Ohio: Morning Side House; 1985.

79. Curley MD, Walsh MJ, Triplett RG. Some adjustment indices of oral-maxillofacial war casualties, limb amputees, and non-injured veterans. *Mil Med*. 1982;147:572–574.

80. Steinbach T. Total rehabilitation for amputees in special conditions. *Prosthet Orthot Int*. 1977;1(2):125–126.

81. Ryan TC, Jones DT, White WL, Orr TE, Walters W. Management and rehabilitation of naval battle casualty amputees. *Ann Surg.* 1946;124(6):1047–1056.

82. Davis DJ, Paasuke L. Vocational evaluation and rehabilitation. In: Delisa JA, ed. *Rehabilitation Medicine Principals and Practice*. Philadelphia: JB Lippincott; 1988.

83. Khoury SA. Evaluation of amputees. *Int J Rehabil Res.* 1983;6(4):449–454.

84. Kejlaa GH. The social and economic outcome after upper limb amputation. *Prosthet Orthot Int*. 1992;16:25–31.

85. Shepherd WG, Caine D. Vocational end results following rehabilitation of upper-extremity amputees. *Med J Aust*. 1968;2(4):167–169.

86. Sheikh K. Return to Work following limb injuries. *J Soc Occup Med.* 1985;35:114–117.

87. Millstein S, Bain D, Huster GA. A review of employment patterns of industrial amputees: Factors influencing rehabilitation. *Prosthet Orthot Int*. 1985;9:69–78.

88. Helm P, Engel T, Holm A, Kristiansen VB, Rosendahl S. Function after lower limb amputation. *Acta Orthop Scand*. 1986;57:154–157.

89. Schafter R. Occupational therapy for lower extremity problems. *Am J Occup Ther.* 1973;27(3):132–137.

90. Brown LKW. Traumatic amputation: Mechanism of injury, treatment and rehabilitation. *AAOHN J*. 1990;38(10):483–486.

Chapter 4

REHABILITATION OF THE LOWER LIMB AMPUTEE

CHRISTOPHER HUSTON, M.D.[*]; TIMOTHY R. DILLINGHAM, M.D.[†]; AND ALBERTO ESQUENAZI, M.D.[‡]

[*]*Formerly, Major, Medical Corps, US Army, Physical Medicine and Rehabilitation Service,Walter Reed Army Medical Center, Washington, DC; currently, private practice; The Orthopedic Clinic, 2700 North 3rd Street, Suite 300, Phoenix, AZ 85004*

[†]*Formerly, Major, Medical Corps, US Army, Physical Medicine and Rehabilitation Service, Walter Reed Army Medical Center, Washington, DC; currently, Associate Professor, Department of Physical Medicine and Rehabilitation, The Johns Hopkins University, Baltimore, Maryland 21239*

[‡]*Director, Gait and Motor Analysis Laboratory and Regional Amputee Center, MossRehab, 1200 West Tabor Road, Philadelphia, Pennsylvania 19140; and Associate Professor, Department of Physical Medicine and Rehabilitation, Temple University, 3401 North Broad Street, Philadelphia, Pennsylvania 19141*

INTRODUCTION

The care of war injured amputees is a major problem facing any army during wartime. Historically, amputations are a very common consequence of modern warfare. In the civilian setting, the primary cause of leg amputations is vascular disease,[1] accounting for 93% of amputations. Other causes include trauma, malignancy, and congenital amputations. The devastating trauma suffered during armed conflicts results in substantial numbers of traumatic upper and lower extremity (LE) amputees.

During the Civil War, 3 million troops were mobilized and 20,993 major amputations were documented in the Union Army. Of these amputations, there were 8,518 upper extremity and 12,475 LE amputations.[2]

The official statistics for World War II, covering the period between 1 January 1942 and 31 March 1946, indicate that 14,912 amputees were treated in the Zone of the Interior (the continental United States). These figures do not reflect those casualties who died overseas, or the nondisabling toe amputations that did not preclude continued military service. There were 10,620 amputations of the lower limb, of which 870 were bilateral amputations. The majority of the leg amputations were at the below-knee level.[2]

During World War II, because of the enormous numbers of amputees, the United States military established five "amputation centers" at ports of debarkation. The major center was at Walter Reed General Hospital (currently Walter Reed Army Medical Center), in Washington, DC. Training of medical officers, therapists, and prosthetists was vigorously pursued to ensure that healthcare providers were up-to-date in procedures used in caring for amputee soldiers. Even with substantial resources dedicated to amputees, additional development of prosthetic technology was required to best meet soldiers' needs. To refine and develop prosthesis construction, civilian consultants were used. Because the army was responsible for definitive prosthetic fabrication in World War II, contracts were established with companies for purchasing large quantities of prosthetic devices.[3]

In April 1943, the U.S. Army Surgeon General directed that all amputees be transferred "as soon as possible" to designated amputation centers. The original five army amputation centers (Bushnell General Hospital in Brigham City, Utah; Lawson General Hospital in Atlanta, Georgia; McCloskey General Hospital in Temple, Texas; Percy Jones General Hospital in Battle Creek, Michigan; and Walter Reed General Hospital in Washington, DC) were not adequate to meet the treatment needs of the many amputees sustained in such a long and protracted global war. By 1944, it was clear that the original amputation centers could not handle the enormous workload. This was particularly true after the intense fighting in Europe during the winter of 1944–1945. Because U.S. Army hospitals were responsible for the amputee's full rehabilitation, long military hospital stays were required; therefore, two additional army amputee centers were established: Thomas M. England General Hospital in Atlantic City, New Jersey; and McGuire General Hospital in Richmond, Virginia. Each hospital had its own prosthetic shop with trained prosthetists. In order to educate these "orthopedic mechanics" (prosthetists), 3-month training courses were established. Amputee servicemen, themselves, were sometimes trained as prosthetists and utilized in the limb fabrication shops.[3]

The Army Surgeon General was insistent that extreme care be exercised to ensure that the fittings of prostheses were entirely satisfactory and that each amputee be taught to use his prosthesis competently before his discharge.[3]

That considerable numbers of amputees required care at a continental military hospital was illustrated during the Vietnam War, when between May 1966 and May 1969,[4] Fitzsimons General Hospital in Colorado, treated over 500 casualties with major amputations. Of these 500 patients, 342 sustained loss of an LE, with 44 of those 342 losing parts of both lower limbs.

Even a brief conflict can produce a substantial number of amputees requiring rehabilitation. The relatively short Persian Gulf War in 1991 resulted in a single medical center (Walter Reed Army Medical Center, Washington, DC) receiving 14 amputees for rehabilitation.[5] These casualties frequently sustained comorbid conditions, such as peripheral nerve injuries, which complicated functional restoration.

Intense wars produce tremendous numbers of traumatic amputations in distributions much different from those seen in the civilian world. For this reason, amputee care in the military must remain at the forefront of technology, maintaining its readiness to assume the full care of an amputee soldier. This requires the establishment of designated ma-

jor medical centers with organized multidisciplinary rehabilitation services that would initially be directed by the primary surgeon, then by a military physiatrist. The World War II rehabilitation system, with designated amputee centers, provides a model for optimal present day military amputee care. Rehabilitation services at major military hospitals with modern prosthetic laboratories, expert prosthetists, physiatrists, occupational therapists, physical therapists, psychologists, and social workers, are best suited to meet the specific needs of the individual amputee soldier. Early temporary prostheses and definitive state-of-the-art prosthetic devices must be available to the amputee for full rehabilitation to occur. Early weight bearing, using temporary prostheses, was found to be quite beneficial to amputees. In fact, during World War I, the Belgian Army Medical Corps demonstrated that early weight bearing improved circulation, hastened stump shrinkage, and prevented muscle atrophy and contractures.[6] Waiting until the casualty transfers to a Veterans Affairs (VA) hospital to institute rehabilitation and prosthetic training yields suboptimal care; if rehabilitation has not already begun by the time casualties reach a VA hospital, contractures will develop and deconditioning will occur. These conditions make full functional restoration difficult.

In summary, military medical centers must be able to accommodate many amputees, initiate rehabilitation, and provide temporary prostheses and state-of-the-art permanent prostheses. To maximize the functional potential of the soldier, the amputee must be the center of a well-coordinated, interdisciplinary rehabilitation effort. This chapter addresses such an approach.

MANAGEMENT OF NEW AMPUTEES

Management of the soldier with a traumatically injured extremity initially begins with resuscitation measures. Life threatening injuries need to be evaluated and the soldier must be stabilized. Once the soldier is stabilized, attention may be directed toward the traumatized extremity. Thorough debridement of devitalized tissues should be performed by surgeons in the operating room. Necrotic muscle left in the wound is a good medium for bacterial growth and subsequent infection. Furthermore, necrotic muscle may result in severe myoglobinuria and subsequent renal failure.[7] Initial evaluation by an experienced orthopedic and vascular surgeon is preferable. Further testing may be needed, such as radiographic and vascular studies. The surgical team can then decide to proceed with limb salvage or amputation.

Limb Salvage vs Amputation

With advances in both vascular and orthopedic reconstructive surgery, limb salvage has become an option for limbs that previously would have been amputated. Today, the surgical team must decide whether to attempt limb salvage or perform amputation. The main goal of treatment is to provide the optimum functional result—physically, cosmetically, and psychologically. Limb salvage may not provide the best functional outcome. Saving an insensate, paralyzed limb may be considered a failure when compared to an optimal residual limb with a prosthetic device. Additionally, limb salvage may be emotionally taxing and require longer hospital stays than a primary amputation.[8] Furthermore, individuals may have limitations of mobility and function despite limb salvage. However, a prosthetic limb is not a substitute for a sensate limb with residual motor function.[9] Hence, the goal should be limb salvage, providing that the salvaged limb is more functional than a prosthetic replacement.

The decision as to when to amputate or when to attempt limb salvage remains controversial. General concepts and scales (presented below) developed to aid in this decision remain subjective and controversial. Additionally, the military environment and tactical needs of the unit pose further problems related to surgical options and must be considered in the surgical decision. For the most part, combat wounded amputees present with completely amputated limbs, which the surgeon then debrides and provides hemostasis (Figures 4-1 and 4-2).

In examining the traumatized limb, the limb may be divided into skin, bone, muscle, nerve, and vascular components. If three of the above five components are destroyed beyond repair, then amputation should be considered. Enough viable skin is needed to provide eventual wound coverage. Myocutaneous flaps, free skin grafting, and porcine heterografts may be options when insufficient skin is present. After thorough debridement, evaluation of the amount of musculature remaining needs to be determined in reference to functional use of the limb. Tendon repairs may be performed, but they require immobilization and skin coverage. Fractures will need to be evaluated in terms of status of blood supply to the fracture, joint injury, limb shortening,

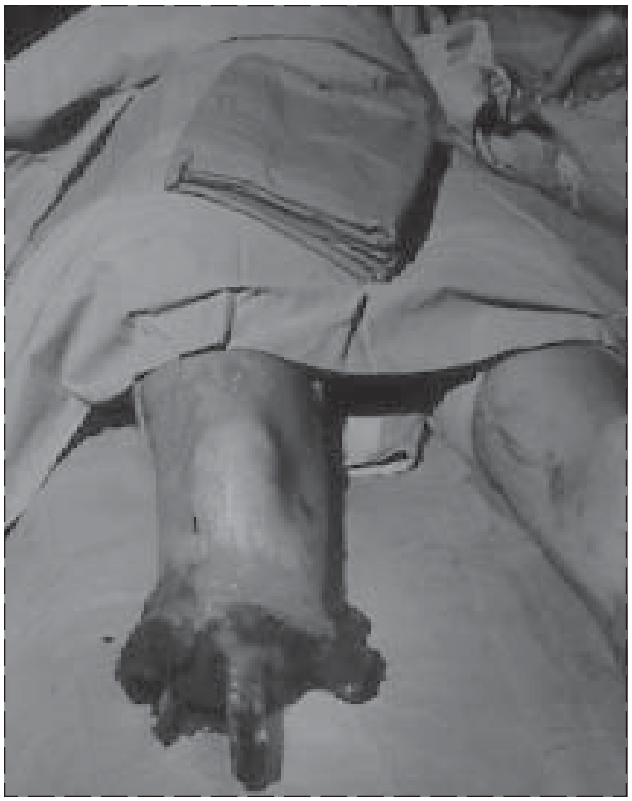


Fig. 4-1. This injury was caused by a Claymore mine. Note the extensive soft tissue damage and the resulting exposure of the tibia and fibula. A high below-knee amputation was performed. However, given the paucity of soft tissue, a knee disarticulation or even a low above-knee amputation would have been options.

and ability to achieve fixation.[7] External fixation of bone should be performed early, before vascular repair, to prevent further soft tissue or vascular damage.[7,8]

Nerve status must be evaluated to establish the degree of nerve injury—neurapraxia, axonotmesis, or neurotmesis. A poorer prognosis exists for nerve recovery in the case of neurotmesis, with better outcomes for axonotmesis. Results from microfascicular nerve repair has been disappointing. Also, nerve recovery in an LE is poor compared to that in an upper extremity. Both venous and arterial damage need to be assessed. A variety of vascular reconstructive procedures may be performed, depending on the injured structures and potential graft sources. Usually, amputation for vascular damage alone is not indicated except when warm ischemia lasts more than 6 hours.[7] Hence, flow should be reestablished before 6 hours has elapsed. Mechanism of injury, interval from injury to treatment, and degree of wound contamination are important factors in the decision process; however, when applied to the individual patient, these general concepts do not necessarily take into account all factors in the decision process. Furthermore, the concepts are dependent on the subjective interpretations and experience of the surgeon. For these reasons, scales to assist the surgeon have been developed.

The Mangled Extremity Syndrome Index (MESI)[10] was developed to aid decision making for limb salvage vs amputation. If 3 of 4 organ or tissue systems are significantly injured, the scale classifies an extremity as mangled. The systems are skin, nerve, vascular, and bone. The scale takes into account severity of general injuries, shock, age, preexisting disease, skin, nerve, vascular supply, bone, and time postinjury greater than 6 hours. Retrospectively, 7 of 17 casualties with an MESI less than 20 had limb salvage success. The other 10 with MESIs greater than 20 eventually had to have amputation. Average hospital stays for patients with salvaged limbs vs those with primary amputation were 75 and 36 days, respectively.[10]

Lange and associates[11] evaluated 23 cases retrospectively with Type IIIC tibial fractures (a Type IIIC tibial fracture is an open fracture with concomitant arterial injury) and concluded *absolute* indications for amputation were anatomic complete disruption of the posterior tibial nerve and crush injuries with

Fig. 4-2. This is a forefoot amputation of the type that typically results from the detonation of a buried antipersonnel mine. There was insufficient viable plantar skin to allow reconstruction as a Syme's amputation at the ankle joint. A below-knee amputation was performed.

warm ischemia for longer than than 6 hours. *Relative* indications for amputation were associated serious polytrauma, severe ipsilateral foot trauma, and an anticipated protracted course of treatment to obtain soft tissue coverage and tibial reconstitution. The presence of 2 to 3 relative indicators was considered to be justification for amputation as opposed to salvage.[11] In addition to the small sample size (23) and retrospective design, the study was criticized for the indicators being subjective and requiring substantial experience on the part of the surgeons.[8]

Another scoring system for Type IIIC tibial fractures has been developed based on a modification of the MESI.[12] This score is based on the mechanism of injury to the musculoskeletal system, degree of shock, degree of ischemia, ischemia greater than 6 hours, and patient age. Those with scores greater than seven had to undergo amputation.

Despite the scales and general concepts, there is currently no definitive method for objective determination of whether a patient should undergo limb salvage or primary amputation.[8] If feasible, a multispecialty surgical team with experience in both limb salvage and amputations should be consulted. Additionally, the patient should be included in the decision process, and a visit by an amputee may be helpful. A physiatrist is a valuable member in the decision process because of the physiatrist's expertise in function, especially with amputations complicated by other musculoskeletal or neurologic injuries elsewhere in the body.

Amputation

The trauma and treatment before amputation usually determines the level of amputation or, in the case of an already amputated limb, reamputation (revision).[13] In selecting the level, attention must be given to providing the best functional outcome. Improved function is usually associated with levels that require less energy consumption. Additionally, preserving joints is beneficial secondary to their mechanical advantages and preservation of proprioception. More proximal amputations are associated with increased energy consumption and sacrifice of the benefits from retained joints. Hence, preservation of limb length is functionally beneficial.

Functionally, the better amputation levels in order of preference are transmetatarsal, Syme's, below-knee, knee disarticulation, above-knee, hip disarticulation, and hemipelvectomy.[14] Advantages of the knee disarticulation are improved weight bearing distally and improved prosthetic control. In the past, prosthetic components for a knee disarticulation were a problem. However, newer components, especially in knee designs, have solved the prior disadvantage. The Lisfranc's (tarsometatarsal) and Chopart's (tarsotarsal) amputations may be the exception to improved outcome with preserving length. In many cases, these amputations have the disadvantage of developing equinus deformities, thus prohibiting optimal prosthetic fitting[14]; performing these amputation levels yields suboptimal results.

In the battlefield environment, there may be delays in seeing a wounded soldier, and the wounds may be grossly contaminated. The combination of delayed surgical treatment and grossly contaminated wounds has rendered the open circular amputation the method of choice.[15] Briefly, this technique consists of circular incision of layers, allowing each layer to retract before proceeding to the next layer. The procedure is then followed by continuous skin traction. Skin traction is maintained during transportation[6] and is very important in avoiding soft tissue retraction of the open amputation. If soft tissue retraction or skin retraction is allowed to occur, significant bone loss with revision is frequently necessary. This can lead to loss of function for the amputee and should be avoided. In the past, a common error was the early discontinuance of skin traction, which led to a sacrifice of limb length.[16] Skin traction should continue until the limb is ready for revision or has healed from secondary intention.

Some open amputations and unsuccessful limb salvage procedures require revision or definitive amputation, respectively. The definitive amputation procedure is performed on an elective basis. The procedure is directed toward healing, providing an optimum residual limb for prosthetic ambulation, and is viewed as a reconstructive procedure. The various procedures are described by the specific levels of amputation; however, general concepts should be followed.

During secondary closure, the skin should be closed without tension. The scar should be placed to avoid scar adhesion to bone and trauma from pressure points in the prosthesis. Bone should be beveled distally to prevent a sharp end from causing discomfort or soft tissue breakdown with prosthetic wear. Myodesis or myoplasty should be performed to provide better muscle balance and control of the residual limb[17]; this also provides soft tissue coverage distally by preventing muscle retraction. Nerves should be sharply divided under

tension, thereby facilitating retraction into healthy soft tissue. This prevents later irritation of the nerve endings by the prosthesis.[13]

Rehabilitation

Rehabilitation should begin as soon after the emergent amputation as possible. The main goal of rehabilitation is to prevent any complications of immobility while awaiting the definitive amputation procedure. Other goals include patient education, conditioning, functional training, and psychologic support.

The bedbound patient is at risk of developing myriad complications, such as deep vein thrombosis, pulmonary embolus, atelectasis, pneumonia, orthostatic hypotension, decubitus ulcers, loss of strength, osteopenia, and contractures.[18] These complications can often be prevented by early mobilization. The specific prevention strategies for many of these problems will be addressed in Chapter 12, Prevention of Medical Complications of Immobility Through Early Rehabilitation, in the second of this two-book series on rehabilitation. Problems specific to the amputee are the development of contractures and deconditioning. Contractures can seriously affect final outcome for the amputee. Proper bed positioning, with scheduled turnings, is necessary to prevent contractures.[18] Range-of-motion exercises should be performed four times daily.[14] To prevent deconditioning, the patient must follow a daily exercise program. A physical therapist may be helpful in exercise training, and early mobilization will help in strengthening, improving endurance, and preventing contractures.

Functional training should begin early, with a physical therapist in attendance. The patient is taught to independently transfer from bed to wheelchair and to use crutches. Once independent in these functions, the patient progresses to toilet transfers. Mobility training begins in the wheelchair or with crutches, depending on the functional level of the patient. Early mobilization, besides preventing complications of immobility, provides valuable preprosthetic training. Furthermore, independence in ambulation helps the patient's morale. Not being able to move from one room to another or to use the bathroom without assistance can be disheartening.

Independence should be further encouraged by training to enhance skills in activities of daily living (ADL), where dressing, eating, toileting, and showering are addressed. An occupational therapist will assess and teach these skills.

At the start of rehabilitation the patient is given information regarding the surgical, rehabilitation, and prosthetic training processes. Education becomes more specific as the patient progresses. Often, a meeting with a successful amputee is helpful to the new amputee.

The new amputee undergoes a grieving process and will need to adjust to an altered body image. Concerns of self identity, social acceptance, employment, and sexual function should be addressed. Patient education helps in the psychologic adjustment of the patient. Formal psychologic counseling is usually not required unless the patient's emotional status interferes with the rehabilitation process.[19] Also, early rehabilitation helps patient adjustment.

Postoperative rehabilitation begins after the definitive amputation procedure. The goals in this phase are wound healing, early limb maturation, prevention of complications of immobility, functional independence, and acquisition of a prosthesis. Postoperatively, wound healing and early limb maturation may be accomplished through the use of a nonremovable rigid dressing. The rigid dressing protects the wound, prevents the development of edema, and assists healing. The rigid dressing also provides for early maturation through edema control and shaping of the limb. The dressing is changed weekly. Usually, after the second week, the sutures are removed, and the rigid dressing is changed to a removable rigid dressing with a pylon and prosthetic foot (see Below-Knee Amputation section).

Gradual increase in weight bearing is begun, which allows early limb maturation and quicker fitting of a prosthesis. This is followed with gait training by a physical therapist. Experienced personnel educate the amputee in how to properly fit the removable rigid dressing with residual-limb socks and how to check the residual limb for signs of excessive weight bearing; proper limb hygiene is also taught.

Mobility and ADL training are continued to achieve the maximal functional result for the patient. The functional training should be specifically directed toward vocational and avocational goals, and the amputee should be referred to a vocational counselor for guidance regarding future vocational plans.

Once the residual limb volumes have become relatively stable, the limb is fitted with a temporary prosthesis. The amputee is evaluated in the prosthesis and then educated in proper prosthetic care and fitting. The independent amputee with a properly fitting prosthesis is discharged with close

follow-up. Thereafter, the amputee is followed on a regular basis indefinitely. During the first year after discharge, because of continued shrinkage of the residual limb, most new amputees require refitting of their prosthesis.

The use of immediate postoperative prosthetic fitting and a rehabilitation team concept has been shown to result in shorter hospital stays, more efficient rehabilitation, and a greater likelihood of ambulation.[20] A retrospective study[21] of 182 diabetic amputees revealed quicker healing and earlier prosthetic fitting when rigid dressings were used. Another study[22] demonstrated that only 4 of 238 lower limb amputees referred from a tertiary care hospital to a rehabilitation center had good enough residual limbs to allow preparatory prosthetic fitting. Many of the limbs were nonhealed, bulbous, edematous, infected, or dehised. Many amputees also had hip or knee (or both) flexion contractures greater than 15°.[22] These studies support the need for early comprehensive rehabilitation to prevent complications and to achieve a higher functional outcome. The physiatrist should be consulted early to plan and manage the rehabilitation program.

PARTIAL FOOT AND SYME'S AMPUTATIONS

The goal of surgical treatment is to remove as little tissue as possible while treating the limb injury. Partial foot amputations should be performed with a clear understanding of the potential functional outcome for the patient. Partial foot amputations are particularly problematic because of the rather poor quality of the arterial vascular system. As a general rule, the greater the anatomic loss of the foot, the more involved will be the rehabilitation and the prosthetic restoration.

The selection of the surgical level of amputation is probably one of the most important decisions that will be made. The viability of the soft tissues, as determined by skin bleeding at the time of surgery, will usually determine the most distal possible level for amputation. Sophisticated predictive techniques to optimize the level of amputation are in use, but their reliability is limited.[23–26] After surgery, the patient will have to use the residual limb as a weight bearing structure. Ideally, full body weight of the patient will be carried on a newly created man–machine interface (the socket/residual limb). Bony prominences, adherent skin scars, traction, shear, and perspiration will complicate this function. For these reasons, the residual limb must be surgically constructed to optimize the transfer of loads, maintain muscle balance, and assume the stresses inherent in its new function. The more bone and muscle lost as a result of amputation, the greater the loss of the normal locomotor mechanisms and, therefore, the greater the energy cost of ambulation and the greater the degree of impairment and need for prosthetic restoration. Syme's amputation (ankle disarticulation), when correctly performed, will provide an excellent weight bearing surface that permits short distance ambulation without the use of a prosthesis (helpful for the inevitable middle of the night bathroom trip).

The skin is the crucial interface between the residual limb and the modified footwear or prosthesis. For this reason utmost care in the management of the skin is essential to (*a*) provide a pain free extremity that can tolerate weight, (*b*) have enough sensation to provide protective feedback, and (*c*) have a durable soft tissue cover. Distal metatarsal and toe amputations should be considered only when full skin thickness coverage can be provided. Midfoot amputations, such as that described by Lisfranc in 1815 (tarsometatarsal) and Chopart in 1792 (tarsotarsal), both cited in Pinzuer etal,[27]should be contemplated only under special circumstances and when primary skin coverage can be obtained. Insensate skin graft coverage is an inadequate interface surface that will tend to become adherent and will have frequent breakdowns; additionally, the inevitable foot deformities caused by muscle imbalance may further increase disability.

Surgical Technique

Toe. Toe amputations can be performed either with side to side, or plantar to dorsal flaps and must utilize the best available soft tissue. The bone should be shortened to a level that allows adequate soft tissue closure without tension. In great toe amputations, if the entire proximal phalanx is removed, often the sesamoids will retract and expose the keel shaped, plantar surface of the first metatarsal to weight bearing. This may lead to high local pressures, callous formation, or ulceration. The sesamoid bones should be removed.

Isolated second toe amputations should be avoided, as a severe hallux valgus deformity commonly results. This deformity may be prevented by second ray amputation or first metatarsal phalangeal fusion. In metatarsal phalangeal joint toe amputations, transfer of the extensor tendon to the capsule may help elevate the metatarsal head and promote a more even distribution of weight.

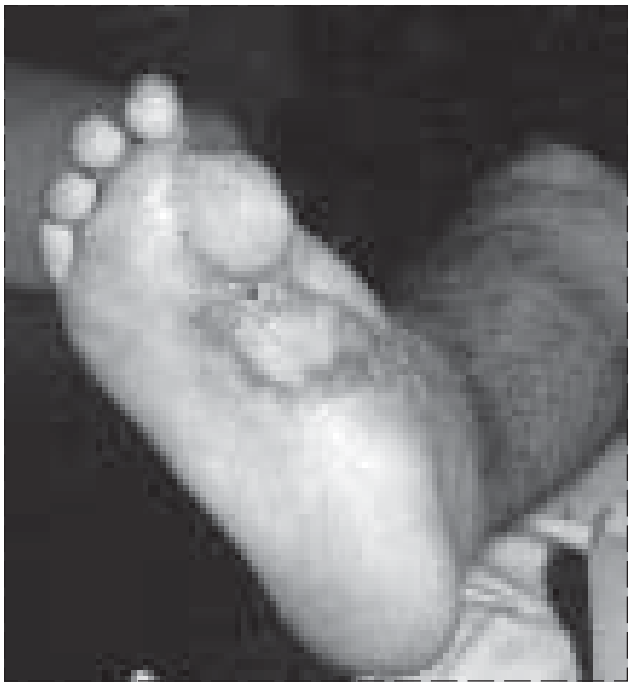


Fig. 4-3. First ray resection requiring flap coverage.

Ray. A ray amputation removes the toe and all (or some of) the corresponding metatarsal. Isolated ray amputations are useful; however, multiple ray amputations can narrow the foot excessively. This results in reduction of the weight-bearing area with potential for callous formation and ulceration. Surgically, it is often difficult to achieve primary closure of ray amputations, as more skin is usually required than is readily available (Figure 4-3). Instead of closing these wounds under tension, it is advisable to leave the wound open and allow secondary healing, although this may result in a less than optimal residual limb and interfere with the rehabilitation program; a reevaluation of the level of amputation may be required.

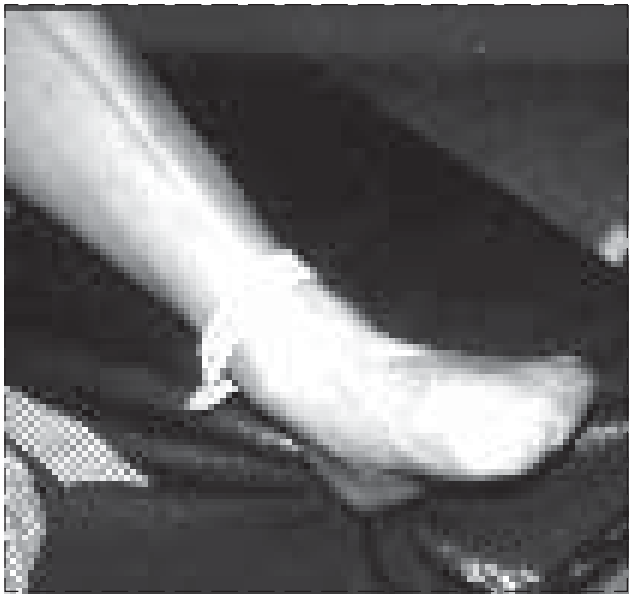


Fig. 4-4. Transmetatarsal amputation.

The fifth ray amputation has been the most useful of all the ray amputations. Plantar and lateral ulcers around the fifth metatarsal head often lead to exposed bone and osteomyelitis. A fifth ray amputation allows the entire ulcer to be excised and the wound to have primary closure.

Midfoot. The transmetatarsal (Figure 4-4) and Lisfranc (tarsometatarsal) amputations produce highly acceptable functional and cosmetic residual limbs. Surgically, a healthy, durable soft tissue envelope is more important than a specific anatomic amputation level, so bone should be shortened to allow soft tissue closure without tension, rather than to a specific described surgical level. A long plantar flap is preferable, but equal dorsal and plantar flaps work well, especially for metatarsal head ulcers. The major disadvantage of transmetatarsal amputation is the high risk of nonhealing.

Preoperatively, careful evaluation should be made of the muscle balance around the foot, with specific attention to heel cord tightness, anterior tibialis, posterior tibialis, and peroneal muscle strength. Midfoot amputations significantly shorten the lever arm of the foot, so Achilles tendon lengthening may have to be done. Tibial or peroneal muscle insertions should be reattached if they are released during bone resection. Postoperative casting prevents deformities, controls edema, and speeds rehabilitation.

Hindfoot. A Chopart (tarsotarsal) amputation (Figure 4-5) removes the forefoot and midfoot while preserving the talus and calcaneus. Tendon transfers for rebalancing are required to prevent equinus and varus deformities. Achilles tendon lengthening, transfer of the anterior tibialis or extensor digitorum tendons to the talus, and postoperative casting are all usually necessary. The Boyd hindfoot amputation consists of a talectomy and calcaneal-tibial arthrodesis. The Pirogoff hindfoot amputation is a talectomy with calcaneal-tibial arthrodesis after vertical transection of the calcaneus through the midbody, and a forward rotation of the posterior process of the calcaneus under the tibia. These latter two amputations are performed mostly on children to preserve length and growth centers, pre-

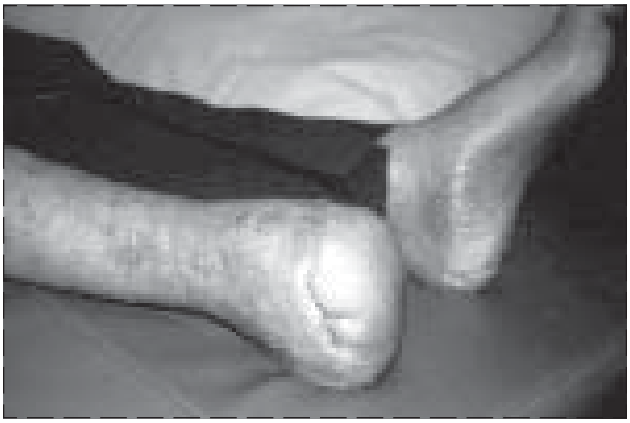


Fig. 4-5. Chopart amputation.

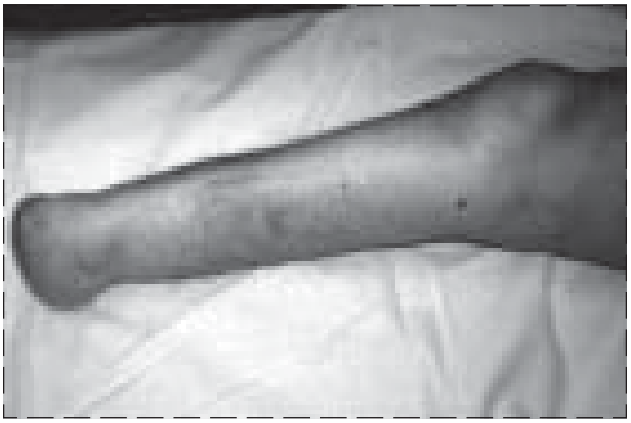


Fig. 4-6. Symes amputation.

vent heel pad migration and improve socket suspension.[28] The hindfoot prosthesis requires more secure stabilization than a midfoot prosthesis to keep the heel from pistoning during gait.

Partial Calcanectomy. Partial calcanectomy, excision of the posterior process of the calcaneus, should be considered a proximal amputation of the foot. In patients with large heel ulceration or calcaneal osteomyelitis, this can be a very functional alternative to a below-knee amputation.[29]

Syme's Amputation. The Syme's level of amputation was described in 1843[30] as a disarticulation of the ankle, affording ease of execution, less risk to life, a comfortable residual limb, and resulting in some distinct advantages regarding mobility and prosthetic fitting. The surgical technique, as described by Harris[31] in 1961, requires that the calcaneus and talus be removed with careful dissection of bone to preserve the heel skin and fat pad to cover the distal tibia (Figure 4-6). The malleoli must be removed and contoured. A late complication of the Syme's amputation is the posterior and medial migration of the fat pad. Options to stabilize the fat pad include (*a*) tenodesis of the Achilles tendon to the posterior margin of the tibia through drill holes; (*b*) transfer of the anterior tibialis and extensor digitorum tendons to the anterior aspect of the fat pad; or (*c*) removal of the cartilage and subchondral bone to allow scarring of the fat pad to bone, with or without pin fixation. Careful postoperative casting can also help keep the fat pad centered under the tibia while it heals.[31–33]

The Syme's amputation is an end-bearing level. Retention of the smooth, broad surface of the distal tibia and the heel pad allows direct transfer of weight from the end of the residual limb to the prosthesis with improved proprioception. Because of the ability to end bear, the amputee can ambulate without a prosthesis in emergency situations, or for bathroom activities. The larger circumference of the distal leg at the level of the malleoli and their flares allows the use of a socket that is totally self suspending, and eliminates the need for any form of proximal auxiliary suspension. These same two advantages are also the two main drawbacks for prosthetic fitting. The long length of the residual limb places some minor limitations on the options for prosthetic ankle/foot systems that can be used. The bulbous nature of the distal residual limb that permits excellent self suspension of the prosthetic socket can also result in a prosthesis that is cosmetically unacceptable. A leg length discrepancy can be a problem with and without the prosthesis.

Acute Postamputation Rehabilitation

Pain control, maintenance of range of motion and strength, and promotion of wound healing are the goals of this stage, which begins with the surgical closure of the wound and culminates with healing after the sutures are removed. Pain control and residual limb maturation should be aggressively pursued. For edema control, use of an immediate postoperative rigid dressing (IPORD), or a soft elastic bandage and subsequent pneumatic compression are indicated. Another method of wound protection and early shaping and shrinking is the use of the removable rigid dressing, as proposed by Burgess and associates.[34] This dressing is easily changed to accommodate stump shrinkage until the residual limb stabilizes in size and is ready for the first prosthetic casting. In addition, this dressing protects the residual limb while ambulation training with gait aids and other essential mobility skills are practiced

by the patient. For the Syme's and partial foot amputee, the IPORD is more difficult to apply and it makes it more difficult to maintain skin integrity because of the patient's tendency to attempt weight bearing. IPORD techniques offer the advantages of early rehabilitation and control of edema and pain, and are preferred if the patient has no history of chronic arterial compromise, and if the expertise to apply it is available. Soft compressive dressings alone are used in many centers.[21] The dressing should be extended proximal to the midtibia to improve its suspension. Proper postoperative positioning and rehabilitation are essential to prevent ankle and knee contractures.

Pain Management. Pain control can be best achieved initially with a patient controlled analgesia (PCA) system, followed by the use of scheduled oral analgesia. A skin desensitization program that includes gentle tapping, massage, soft tissue mobilization, and skin lubrication is recommended for the patient who uses a removable, soft, or elastic dressing.

Postoperative Care. Postoperative edema is common following amputation; if soft dressings are used, they should be combined with stump wrapping to control this, especially if the patient is a prosthetic candidate. A major complication from stump wrapping is too tight an application of the elastic wrap at the proximal end, which actually causes congestion and worsens edema. A figure-8 wrapping technique is best, and it should be reapplied every 4 to 6 hours. The preferred treatment approach is the use of an IPORD to control postoperative edema, protect the limb from trauma, decrease postoperative pain, desensitize the limb, and allow early mobilization and rehabilitation. Immediate postoperative weight bearing can be initiated safely in selected patients, usually young traumatic amputees where the amputation was performed above the zone of injury. An IPORD and an immediate postoperative prosthesis (IPOP) need to be applied carefully, but their application is easily learned and well within the scope of interested physicians.[34]

When the patient is medically stable, early mobilization is initiated, along with general endurance and strengthening exercises (with emphasis on the knee flexor and extensor muscles and the avoidance of joint contractures) and improvement in sitting and standing balance. It is important physically to emphasize the strength and function of remaining limbs, with specificity of training as the preferred type of training. Strengthening of upper limb musculature is essential for wheelchair propulsion, transfers, and walker and crutch ambulation. Whenever possible, patients should be placed in a cardiovascular conditioning program before the amputation.

At this time also, emotional counseling should be implemented for the patient and family, with special attention to the significant other and children. The counseling should include a psychosocial evaluation of the patient and family to assess and manage the existence of depression, anxiety, or both. Patient participation in the decision making process during this phase is important to encourage independence.

Phantom Limb. Phantom limb sensation is the feeling that all or a part of the amputated limb is still present. This sensation is perceived by nearly all acquired amputees, but is not always bothersome.[35] Phantom sensation usually diminishes over time, and the sensation that the phantom foot or hand has moved proximal toward the stump (telescoping), commonly occurs.

As many as 70% of amputees perceive phantom pain in the first few months after amputation. However, such pain will usually disappear or decrease sufficiently so as not to interfere with prosthetic fitting and day to day activities.[36] A smaller percentage will experience pain long term, while others will have recurrence later in life. When the phantom pain persists for more than 6 months, it usually becomes chronic and is extremely difficult to treat. Perceived pain intensity is closely related to anxiety level, depression, prosthetic fitting problems, and other personal factors.[37]

Joint Contractures. Joint contractures usually occur between amputation and prosthetic fitting. In the Syme's and partial foot amputation, the deforming forces are to knee flexion and ankle plantarflexion and inversion. Tibialis anterior and extensor hallucis reattachment during surgery can prevent the deforming forces. After surgery, patients should avoid (*a*) propping the leg up on a pillow, (*b*) prolonged sitting, and (*c*) should be started on active and passive motion exercise early. Sitting with the knee fully extended prevents knee flexion contractures. Strengthening of the knee extensors should be encouraged. Efforts should be directed at prevention of joint contractures, with aggressive rehabilitation beginning soon after surgery.

Gait Training. Gait training is integral in the rehabilitation process. This program should be a coordinated effort between the physical, occupational, and recreational therapists and the prosthetist, with frequent physiatric input. Each team member will use different techniques to teach and review all of

the important topics that need to be learned by the amputee.

Initial gait training should address technique and velocity on flat surfaces. Then training for mobility on uneven surfaces and elevations is introduced by all the therapists. A review and practice of the use of the prosthesis in transfers, driving, sports, and other activities should always be included.

Prosthetic Fitting And Training

Prosthetic prescription options for the amputee have changed dramatically over the past decade. Selection of the most appropriate prosthetic devices for functional restoration of the lower limb amputee is an extremely challenging task in view of the variety and complexity of new prosthetic feet, socket fabrication techniques, suspension systems, and available materials. Ideally, this task should be accomplished by an expert team of professionals in close communication with the patient. Members of the team should include a surgeon, a physiatrist knowledgeable in amputee rehabilitation and prosthetics, a certified prosthetist, an occupational therapist, a physical therapist, a recreational therapist, a psychologist, a social worker, and the patient and his family.

The patient should learn prosthetic management, including the basic principles behind the function of each of the components in the prosthesis, its maintenance and care. The patient should practice how to put on and take off the prosthesis, the techniques to adjust it, and how to determine the appropriate sock thickness to wear. Skin care and inspection techniques are also reviewed.

Prosthetic Restoration and Socket Characteristics. Following a lower limb amputation, the residual limb, instead of the foot, must bear the weight of the body when standing on the prosthesis. The socket provides the surface for contact and transfer of body weight from the residual limb to the prosthesis. It is this interface between residual limb and socket that is probably the most critical factor in determining the successful fit and function of a lower limb prosthesis. Considerable forces of varying types and magnitudes (eg, weight bearing, shear, traction, hemodynamics) occur at this interface. To provide adequate comfort and avoid breakdown, the socket must be designed to assure these forces do not exceed tolerances of the residual limb tissues. The amount of force the residual limb can tolerate varies with the amputation level.[38] Disarticulation (eg, Syme's or through-knee amputations) with broad intact distal joint or bone surfaces are able to tolerate high forces concentrated on a small surface area. The end-bearing capability of disarticulation amputations can minimize many of the socket interface problems seen with other levels of amputation.

Toe and Ray Amputation. Loss of one or more toes with preservation of the metatarsals will have minimal impact on level walking. But these amputation levels will have an impact on more demanding activities such as running or jumping. Within the shoe, a toe filler can be used to prevent collapse of the toe box, prevent irritation of the residual foot skin, support the foot, and improve the shoe's appearance. A soft shoe with a carbon graphite insert may be used instead of an orthopedic shoe. The loss of two or more rays will result in a narrower forefoot. Loss of the first or fifth toe will shift the normal weight bearing pattern to the adjacent metatarsal head. A modified shoe insert to support the metatarsals, redistribute weight bearing to the remaining metatarsal heads, and provide a filler for the absent rays and toes may be necessary.

Transmetatarsal Amputation. With the transmetatarsal amputation, the anatomic toe lever of the foot is shortened, which reduces the ability to control dorsiflexion, push off normally, and elevate the body's center of gravity. The traditional prosthetic restoration includes a transmetatarsal silicone custom shoe insert (Figure 4-7) with an arch support attached to a toe filler. This can be used in conjunction with an extended steel or carbon graphite

Fig. 4-7. Springlite toe fillers for partial foot amputations. Photograph: Courtesy of Springlite, 97-E Chinook Lane, Steilacoom, WA 98388.

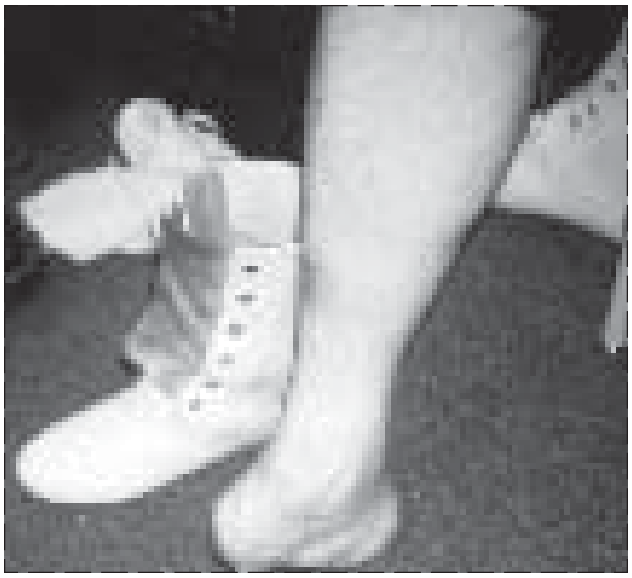


Fig. 4-8. Self-suspending partial foot prosthesis socket made of flexible resin and silicone.

shank in the shoe. The shank should be extended distally to the point of the former metatarsal heads to restore normal toe lever length and improve push off. The addition of a rocker sole to the shoe can also help to restore a more normal gait pattern. With the development of very thin, lightweight carbon fiber shanks or inserts, such as those made by the Springlite Company (Steilacoom, Washington), it is possible to incorporate the shank directly into the insert itself. An ankle/foot orthosis (AFO) can also be attached to this partial foot prosthesis.

Life-like prosthetic replacements are available for transmetatarsal amputations and are designed to provide comfortable residual limb and prosthesis interface, redistribution of weight bearing, acceptable cosmesis and shoe fit, and to maintain the biomechanics of walking. They are made of silicone, which should be shaped and intrinsically colored to match the remaining foot. The major drawback of this form of prosthesis is the high cost and limited durability. In very active individuals, the silicone prosthesis may not provide sufficient resistance to ankle dorsiflexion late stance, which would result in a drop-off gait. This problem can be corrected by reinforcing the shoe with a metal or carbon graphite shank, or attaching it to an AFO.

With amputation through the more proximal portion of the metatarsals, it may be more appropriate to consider a partial foot prosthesis similar to those used for the more proximal partial foot amputations, such as for the tarsometatarsal and midtarsal amputations.

Tarsometatarsal and Midtarsal Amputations. Tarsometatarsal and midtarsal amputations present the special problem of the partial foot being short in length, which makes it difficult for the individual to keep on a low-quarter shoe. In the past, prosthetic options have utilized either a high-top, boot, or AFO type of design to extend the prosthetic replacement well above the ankle in an attempt to provide adequate suspension and restore the biomechanics of walking. Several partial foot prostheses, including self-suspending sockets made of flexible resins or silicones with laminated forefoot and shank, have resulted in highly cosmetic and functional prosthetic options for these previously difficult to manage amputation levels (Figure 4-8).[39,40] These latter designs often make it possible for the amputee to wear low-quarter shoes instead of boots. Yet even with the best of these prostheses, it is often necessary to add a rocker sole or rigid sole plate to the shoe to simulate the action of normal push off (Figure 4-9).

Syme's Amputation. In fitting a prosthesis for Syme's amputations, the distal half of the socket must have either an opening or a circumference slightly larger than that of the distal residual limb so the residual limb can fit into the socket. The pos-

Fig. 4-9. Springlite Chopart prosthesis. Photograph: Courtesy of Springlite, 97-E Chinook Lane, Steilacoom, WA 98388.

terior (Figure 4-10) or medial (Figure 4-11) opening Syme's prostheses permit widening or removal of part of the distal portion of the socket to allow passage of the bulbous distal residual limb into the socket. Once in the prosthesis, the wall or door is replaced and held in place with a strap that provides excellent suspension of the prosthesis. Because of the tibial flare, the Syme's sockets are usually self suspending, and their key functions are that they provide a comfortable residual limb or prosthesis interface; an efficient energy transfer to the prosthesis; a secure suspension of the prosthesis; and acceptable cosmesis. Although these socket designs provide an outer socket contour that more closely resembles the anatomic leg, they still result in a limb that is larger in circumference than the nonamputated limb. The difficulty with these two designs is a potential anterior failure (fracture) of the socket due to the concentrated stresses, especially at push off. Reinforcing this area of the socket with carbon fibers is a good solution for this problem, but may increase the overall weight of the device.[41]

The closed socket design is inherently stronger than those with anterior or medial windows. Thus a thinner lamination is possible, which results in a lighter prosthesis. To permit entry of the residual limb into the socket, the distal diameter must be sufficient to allow passage of the bulbous residual limb. This means that the outer shape of the distal one third to one half of the prosthetic socket will appear cylindrical and much larger than the nonamputated leg. The normal anatomic contours will not be present, resulting in poor cosmesis. With this type of design either a removable expandable liner or a fixed silastic expandable air-filled inner liner may be used. In the former, the friction between the removable liner and the socket lamination provides the suspension; in the latter, the compression of the expandable inner wall of the socket, as well as increased pressure of the enclosed air chamber, provides for suspension of the prosthesis. This closed chamber prosthetic system will not permit transpiration and the air chamber will work as an insulator, further trapping heat, and thereby making this design less desirable in warm climates.

There are several choices for weight bearing in the Syme's amputation. A distal end pad can be used to optimize weight bearing comfort. If the amputee is unable to tolerate distal end bearing, it is possible to add a patellar tendon-bearing proximal brim trim line to the prosthesis and partially unload the distal end. It is also possible to manufacture a prosthesis that will share the load between

Fig. 4-10. Posterior windowed Symes socket attached to a Flex foot.

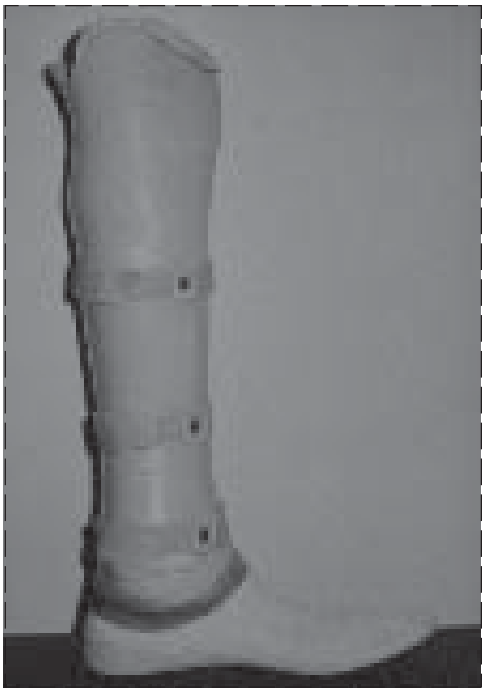


Fig. 4-11. Medial opening Symes socket.

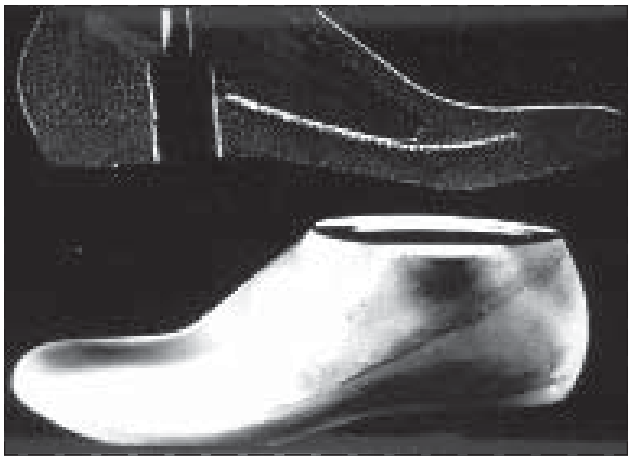


Fig. 4-12. Solid-ankle cushion heel (SACH) foot.

proximal and distal ends, as tolerated by the patient. If necessary, any of the common transtibial prosthetic suspension systems can be added, such as a sleeve or supracondylar cuff.

Modifications to Syme's sockets include a carbon graphite reinforced hybrid of the posterior opening with a flexible inner socket, without the use of an outer door to close the opening. This is usually done to provide relief for tender bony areas. The open socket designs permit the prosthetist greater access to the distal socket should any modification or adjustment of the socket be necessary for changes in fit.

Traditionally, a modified solid-ankle cushion-heel (SACH) foot, which has a lower profile, has been used in the Syme's prosthesis (Figure 4-12). Several of the low profile dynamic response feet are available (ie, Seattle light, Carbon Copy II, and SAFE) for the Syme's amputees.[42,43] The Flex Syme's (see Figure 4-10) and the low profile Springlite prosthesis (Figure 4-13) incorporate carbon graphite construction and longer lever arms to improve their dynamic response.

Long-Term Follow-up

The patient who has successfully completed a rehabilitation program should be seen for follow-up by at least one team member a minimum of every 3 months for the first 18 months. These visits may need to be more frequent and will include other members of the team if the patient is having difficulties with prosthetic fitting, the residual limb, specific activities, or psychosocial adjustment. After this critical 18-month period, the patient should be seen at least every 6 months to assure adequate prosthetic fit and function, for prosthetic maintenance, and for assessment of overall patient condition. It may be necessary to replace a Syme's prosthesis or parts of it every 2 to 3 years. For the partial foot, more frequent replacements may be necessary. Table 4-1 illustrates the increased metabolic demand of walking for foot amputees and highlights the need for optimal prosthetic fit and comfort to achieve the best possible functional result.[44–46]

If a pain-free and scar-free residual limb was created, if optimal prosthetic restoration and training were provided, if no other significant comorbidity exists, patients with partial foot and Syme's amputations can be expected to return to a high functional level. The rehabilitation team should be able and ready to assist the patient throughout the rehabilitation program.

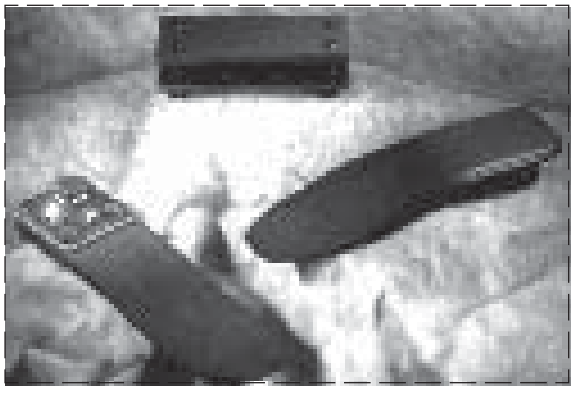


Fig. 4-13. Springlite Symes prosthetic foot. Photograph: Courtesy of Springlite, 97-E Chinook Lane, Steilacoom, WA 98388.

TABLE 4-1

AVERAGE ENERGY CONSUMPTION INCREASE AT DIFFERENT LEVELS OF LOWER LIMB AMPUTATION

Level of Lower Limb Amputation	Metabolic Energy Increase (%)
Transmetatarsal	10–20
Symes	0–30
Transtibial	40–50
Transfemoral	90–100
Bilateral transtibial	60–100
Hip disarticulation	> 100

BELOW-KNEE AMPUTATIONS

Historically, the below-knee amputation (BKA) has been quite common among war-injured soldiers. In World War I, from 1917 to 1918, there were 525 BKAs and 1,282 above-knee amputations (AKAs); 19.5% and 47.7% of all amputations, respectively.[3] During World War II, from 1 January 1943 to 1 May 1944, five medical centers reported 627 BKAs and 550 AKAs, 35.7% and 31.3% of all amputations, respectively.[3] In the continental United States, from 1 January 1942 to 31 March 1946, 11,631 soldiers were treated for at least one LE amputation, with the majority sustaining BKA. These numbers demonstrate the importance of knowing how to manage BKAs that result during times of conflict.

Mortality rates are lower in BKAs compared to AKAs. In World War I, mortality rates were reported to be 18% BKAs and 40% AKAs, respectively.[47] Mortality rates in AKAs of three- to four-times that of BKAs have been reported.[7] This higher mortality is likely due to femoral artery injuries and a greater potential for sepsis due to larger muscle mass. The importance of salvaging the knee joint should not be underestimated, as this procedure offers the amputee numerous advantages compared to AKA. One advantage is the reduced energy the amputee expends with ambulation. For below-knee amputees, energy expenditure has been estimated to increase by 9% to 40% compared to nonamputees, and increases by 25% to 50%, or greater, for above-knee amputees[44,45] (see section on Through- and Above-Knee Amputations). Another advantage of BKA compared to AKA is walking speed. In comparing comfortable walking speeds, Waters and associates[45] found the following gait velocity differences: normal was 82 m/min; traumatic BKA was 71 m/min; traumatic AKA was 52 m/min; dysvascular BKA was 45 m/min; dysvascular AKA was 36 m/min; and dysvascular Syme's amputation was 54 m/min.

The length of the residual limb in below-knee amputees also affects energy expenditure. Gonzalez et al[46] evaluated nine below-knee amputees who were divided into short and long lengths defined as 6% and 8% of body height. They found the increase in energy expenditure compared to nonamputees was 10% and 40% for long and short residual limbs, respectively.[44] Hence, not only is saving the knee joint important, but salvaging length is also important.

In evaluating functional results, Purry and Hannon[48] studied 25 traumatic below-knee amputees and found the following: 84% wore a prostheses for more than 13 hours per day; 32% left their prosthesis off for more than 4 days in the previous year; 72% could walk one mile; 84% drove cars; 96% worked; 64% did not require any sick leave; 72% participated in sports; 84% considered themselves not very or not disabled at all; and 68% felt their lives were similar to the lives of nondisabled people.[48] In regards to employment, one study[49] on LE traumatic amputees found an overall return to work rate of 87%. However, jobs were usually changed to less physically demanding work. Positive factors for return to work were use of a prosthesis, age less than 45 years, and availability of vocational services. In comparing below-knee and above-knee amputees, the use of the prosthesis has been reported to be approximately 74% and 27%, respectively.[13] Of note, is that these estimates included mainly dysvascular amputees.

Another reported advantage of a BKA compared to an AKA is spared sensory input from the knee joint. In the dark, the above-knee amputee is unable to sense the location of the prosthetic foot; but the below-knee amputee knows the prosthetic foot is in line with the remaining tibia.[14]

Classification

BKAs can be classified by the length of the residual limb. A long BKA is from above the Syme's level to the junction of the lower and middle thirds of the tibia.[14] A standard or medium length BKA is from the junction of the lower and middle thirds to the junction of the middle and upper thirds of the tibia. A short BKA is from the middle and upper third junction to slightly below the tibial tubercle. The ultrashort BKA is just below the tibial tubercle. The tibial tubercle is critical in BKAs because it is the site of insertion of the patellar tendon, which is needed for knee extension.

Surgery

Amputation for trauma is either emergent or elective.[15] Indications for emergent amputation include a nonviable extremity secondary to massive injury, infection, or gangrene that endangers life; hemorrhage in the presence of severe infection; and trauma with serious associated polytrauma.[11,15] The level selected for amputation is usually predetermined by the trauma and treatment prior to amputation.[13] However, it is paramount to preserve the

knee and as much below-knee length as possible.[15] Indications for a BKA may include (*a*) crush injury with warm ischemia greater than six hours; (*b*) severe ipsilateral foot trauma; (*c*) traumatic injury to the leg with complete, anatomic severance of the posterior tibial nerve; and (*d*) severe soft tissue and bone destruction not amenable to reconstruction or more distal amputation.[11,13,47]

Because war injuries are usually grossly contaminated,[50] an open amputation is usually performed. The open, circular technique, as described by Peterson,[6] followed with immediate skin traction, has been the standard amputation for the war injured soldier.[6,15,16] Basically, this technique consists of a series of circular incisions performed in layers, with each layer being allowed to retract before the next layer is cut. The first layer is cut down to deep fascia at the lowest viable level of the extremity. A short flap is allowed to conserve skin for later closure.[51] Muscle is cut in layers, allowing retraction before proceeding to the next layer. After the last layer retracts, the bone is cut. Nerves are transected under tension and large vessels are double ligated. The end of the stump is covered with a fine mesh gauze soaked in betadine and a fluff gauze. A stockinette is applied and secured with benzoin tincture.[15] The residual limb is then wrapped with an elastic bandage with gentle compression, decreasing compression proximally. Finally, skin traction of 5 to 6 lb of force is applied immediately using weights and pulleys or a contained traction unit. A self-contained unit is made from rubber tubing extending from a wire ladder splint to the stockinette (Figure 4-14). Traction is applied continuously, except for dressing changes.[15]

Failure to use skin traction often results in unacceptable soft tissue retraction, an error commonly seen early in World War II.[6,51,52] Thompson and Alldredge[16] state that the most common mistake in open amputations is failure to use, or the premature discontinuation of, skin traction. Soft tissue retraction results in bone projection beyond the level of skin, and surgical closure results in sacrifice of considerable bone length.

Another error in skin traction management is casting the transtibial amputee in knee flexion. This is done to prevent rotation of the cast, but results in knee flexion contractures. Better results are obtained by casting the knee in extension, thereby avoiding flexion contracture.[51]

Skin traction is applied until healing occurs by secondary intention or until the limb is ready for revision with closure. When good granulation tissue is present, cultures will be unremarkable, radiographs will show no signs of infection, and normal skin can be closed without tension or bone shortening; the limb is ready for revision.[16] Revision consists of removal of granulation tissue, scar, and just enough bone for subcutaneous and skin closure without tension. The tibia is beveled, fibula cut just proximal to tibia, and skin undermined to secure smooth approximation of skin margins.[16,51] If tension on the suture line becomes evident, skin traction is applied. The advantages of the open, circular technique include the following:

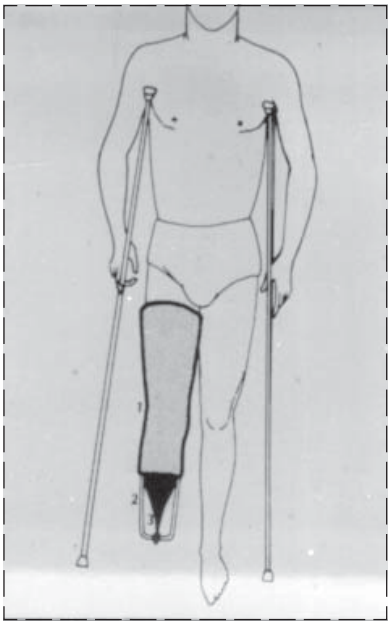


Fig. 4-14. Self contained skin traction unit. Tension in the stockinette provides skin traction and should be adjusted to provide approximately 5 to 6 lb of force. (1: cast material, 2: frame, and 3: stockinette with traction).

- The procedure can be performed quickly under adverse conditions.
- Safe evacuation from a field hospital can occur after 48 hours or even earlier.
- The technique provides optimal removal of all devitalized tissue that may otherwise serve as a source of infection.
- It provides wide drainage.
- Soft tissue or bone infections are rare.
- Limb length is preserved.
- Blood supply is good.
- Muscle and deep fascia adhere to the bone.
- Only a simple revision is needed.[6,16]

The disadvantages include the following:

- The technique requires a protracted period of treatment.
- There is a need for secondary reconstructive operations.
- Frequently there is excessive bone at the end of the residual limb that requires surgical excision.[51]

Other techniques are frequently used for limb revision or subsequent closure. The open, flap technique consists of conserving all viable tissue as a myofasciocutaneous flap.[13] Orientation and length of the flaps are dictated by the trauma. The open flap technique was used in World War I when there were surgical resources available.[6,51] However, in times of heavy conflict, difficulties with the large number of casualties and the need to evacuate to other hospitals led to problems. In these delayed circumstances, the open flap technique could be complicated by serious infection if the wounds were closed too soon.

In World War II, use of long flaps was avoided because of the prevalence of infection when early wound closure was employed. However, the use of short flaps was not without problems.[51] Whatever approach was used, the goals were prevention of infection, preservation of maximum length, and simplified revision without loss of residual limb length.[13,47] The main disadvantage was infection.[2,6,51]

Internal and external fixation has been used to preserve length in the traumatic amputee. Segmental fractures can be stabilized to proximal segments using fixation.[13] However, Thompson and Alldredge,[16] reporting on their experience in World War II, state that in the presence of a compound fracture, pin fixation should not be used proximal to the site of injury because of risk of subsequent amputation at the proximal pin site, resulting in loss of length.

Elective transtibial amputations may be performed after unsuccessful salvage of the traumatically injured leg. The posterior flap method uses a posterior myofasciocutaneous flap created from the posterior compartment muscles, which is brought anteriorly to cover the amputated limb in a semicircular fashion, avoiding dog ear formation.[13] In the operation, nerves are cut sharply under tension and allowed to retract into muscle. The tibia is stripped of periosteum to the level of transection, cut, beveled, smoothed, and contoured to avoid bone spur formation or sharp edges. The fibula is transected obliquely to form a posterolateral facet at the same level or slightly shorter than the tibia, which creates the desired cylindrical shaped limb. The posterior compartment muscles are tapered distally. The gastrocnemius can be excised and the soleus trimmed to decrease bulk, thereby providing the optimal distal tibial padding. Myodesis is created by fixing the posterior compartment myofascia and anterior investing fascia to the tibia. An alternative to myodesis is myoplasty, which consists of attaching the posterior to the anterior myofascia. Either myodesis or myoplasty should be performed to avoid undesirable muscle retraction. Myodesis is contraindicated in severe dysvascular cases.[13] The main advantage of the posterior flap method is improved healing in the dysvascular patient because the major source of collateral blood supply is through the posterior compartment muscles.[47,53]

Another method consists of equal anterior and posterior flaps. In this method, a midlateral incision is made to create equal anterior and posterior flaps. Myoplasty of the anterior and posterior flaps is created over the distal tibia.[13] This procedure is indicated to salvage bone length.

The sagittal flap method uses equal medial and lateral flaps with side-to-side myoplasty over the tibia.[54] This method has been used in dysvascular cases. The procedure should not be used in the presence of a rigid knee flexion contracture, debility that would preclude a second operative procedure, failure of the skin to bleed at that level, or infection near the operative site.[55] A reported advantage of the sagittal flap method in dysvascular cases is improved healing.[13,54] The postulated reasons for the improved healing are that the wide based, very short flaps used in this procedure improve viability compared to long flaps; and the use of sagittal flaps reduces usage of poorly vascularized anterior skin.[13,54] Improved bone coverage with side-to-side myoplasty is also reported.[54]

In trauma with damage to posterior or anterior skin, the intact skin with sagittal flap usage may allow bone coverage without sacrificing length. The skew flap method combines posterior and sagittal flaps to create posterolateral and anteriomedial flaps. This procedure has been used in dysvascular patients with major vessel occlusion. The skew flaps contain the collateral circulation that accompanies the sural and saphenous nerves, providing blood supply to the flaps.[13]

The ERTL procedure, osteomyoplasty, was designed for revision of transtibial amputations in the war wounded.[13] The procedure uses osteoperiosteal bone flaps created from the tibia distal to the level

of bone transection. The osteoperiosteal bone flaps are sutured to the fibula and to each other, and create an osteoperiosteal bridge from the tibia to the fibula for weight bearing. The main disadvantage cited is the sacrifice of bone length to make the bridge.

The Singer procedure uses the heel pad and sole as a flap to give an end weight bearing residual limb. This procedure has limited use and is indicated when there is unreconstructable tibial diaphyseal bone loss, but there is an intact posterior tibial nerve and an artery to the foot.[13]

Skin flaps and split-thickness skin grafts (STSGs) have been used in special circumstances to preserve length. Intact skin is rotated over the anterodistal tibia, where prosthetic stresses are highest.[13] Uncovered areas posteriorly receive STSGs. The procedure is indicated in degloving injuries and in the presence of sufficient bone and muscle, but insufficient skin coverage.[13,47] The skin graft should not be taken from the amputated side because healthy skin is optimal for prosthetic stress tolerance.[16] Reported complications with STSG usage include the need for subsequent revision with excision of the grafted skin, and bone resection in 50% of BKAs.[47] However, modern prosthetic sockets may allow suboptimal skin to tolerate the pressures of weight bearing.

Late revision of healed, very short residual limbs can include the Ilizarov technique of stump lengthening. A few case reports have been published in British literature.[56,57] Latimer and associates[57] report three cases of below-knee amputees who underwent residual limb lengthening ranging from 1.2 to 4.9 cm with a mean of 3.5 cm. Eldridge and colleagues[57] report one case with a lengthening of 4.5 cm using the Ilizarov technique. Complications in these cases include thinning of the skin distally with subsequent breakdown, which may necessitate further bone debridement, pain with lengthening, loss of knee range of motion, and loss of distraction length with too early weight bearing.[56,57]

There is a variety of BKA procedures that can be performed. As mentioned before, the open, circular technique followed by immediate skin traction has been the standard procedure in the war casualty requiring amputation. Other techniques have evolved in the civilian treatment of traumatic amputation. The Emergency War Surgery handbook[15] states there is no ideal or standardized level of amputation in the combat theater. The final decision on the type of amputation is up to the surgeon, and is influenced by the surgeon's experience and skill and the tactical situation. The physiatrist can assist the surgeon by offering expertise in functional outcomes for various amputation levels, especially when associated with other injuries or disabilities, and by coordinating and organizing rehabilitation interventions.

Postoperative Dressing

Once the definitive amputation procedure has been performed and there is wound closure, a decision regarding which type of postoperative dressing needs to be made. A variety of postoperative dressings exist. The predominant choices are soft dressing, IPOP, and rigid dressing.

Soft Dressing. The soft dressing consists of a conventional dressing of sterile and fluff gauze.[21] A soft dressing allows easy wound inspection and dressing changes. Soft dressings may be used with or without an elastic wrap. Without an elastic wrap, the soft dressing lacks edema control, risks knee flexion contracture, lacks protection to the residual limb from bed trauma, and slows limb maturation.[58] A soft dressing with an elastic wrap improves edema control, but has the disadvantages of sometimes choking the residual limb from a tourniquet effect, and skin breakdown from excess pressure.[58,59]

Immediate Postoperative Prosthesis

The IPOP utilizes a plaster dressing with a pylon and SACH foot applied in the operating room at the completion of surgery.[60] At the completion of the amputation a sterile, nonadherent dressing is placed over the suture line. Three to four layers of fluff gauze are placed over this dressing and the distal residual limb. An Orlon-Lycra-Spandex sock is placed over the residual limb and suspended above the knee. Preformed polyurethane pads, with adherent backing, are used to provide relief to pressure-intolerant areas of the residual limb (Figure 4-15). A longitudinal

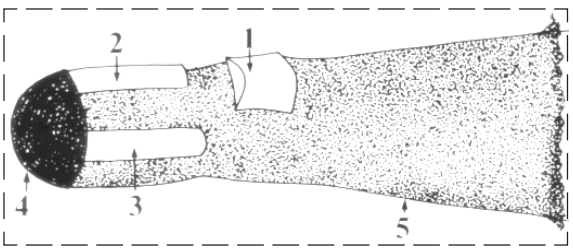


Fig. 4-15. IPOP construction. Pad placement provides relief or padding to pressure intolerant areas: (1) Patellar pad, (2) lateral tibial crest, (3) medial tibia, (4) distal tibia, and (5) stockinette over residual limb.

pad is placed medial to the tibial crest and extends past the distal tibia. Additionally, the pad has a proximal posterior extension along the concave aspect of the medial tibial condylar flare. The lateral pad is placed approximately 0.25 in. distal and lateral to the tibial crest. Again the pad extends distal to the tibia. The medial and lateral pads provide a relief channel for the tibial crest. In addition to the medial and lateral padding the anterior tibia can also be padded if there is friable skin in this area. The distance between the pads is approximately 0.5 in. A prepatellar pad is placed over the entire patella. A reticulated polyurethane pad, 4 to 5 in. in diameter is placed over the distal limb in a hemispheric shape to overlap the distal aspect of the medial and lateral pads. The preformed pads are trimmed and beveled to fit the patient. The knee is kept in 5° to 15° of flexion for the rest of the procedure.

Two layers of elastic plaster of Paris are applied to give tissue compression and a smooth, total contact fit. The wrap starts distally and in a direction that will take tension off the suture line (Figure 4-16). The elastic plaster is brought 3 to 4 in. proximal to midthigh, with tension decreasing proximally. A tourniquet effect must be avoided to prevent constriction and distal limb ischemia. The elastic plaster is then reinforced with either conventional plaster or fiberglass. Fiberglass results in a lighter dressing. A 1-in. cotton strap with a safety buckle is affixed to the dressing and provides attachment for waist belt suspension. This webbing is folded in a loop, with the safety buckle attached, and incorporated into the fiberglass wrapping (Figure 4-17). The strap should be centered anteriorly. Taking care to avoid the bony prominences of the condyles, anterior and medial-lateral (ML) compression is applied to the cast just proximal to the femoral condyles (Figure 4-18). This compression

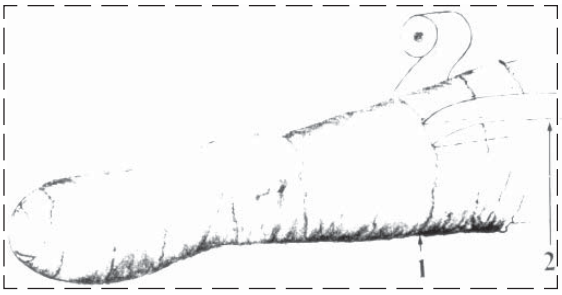


Fig. 4-17. IPOP construction. Incorporation of cotton strap (2) for suspension into the cast. Plaster rolls (1) anchor the suspension strap.

Fig. 4-16. IPOP construction. Elastic plaster compression dressing placed over stockinette and padding. The wrap is placed such that tension is removed from the incision. Additionally, the wrap should provide mildly increased pressure distally with gradually decreasing compression proximally. The decreasing compression gradient and avoidance of circular wraps are important in preventing a tourniquet affect. The wrap should extend to the midthigh level.

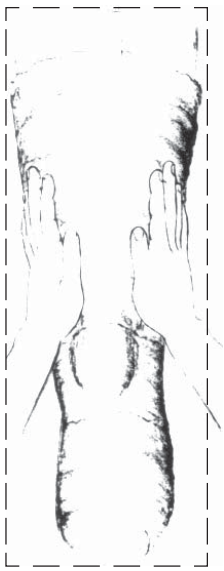


Fig. 4-18. IPOP construction. Gentle compression is applied just proximal to the femoral condyles. The slight indentations provide some suspension and resistance to rotation. Care must be taken to avoid excessive compression causing a tourniquet affect.

Fig. 4-19. IPOP construction. Attachment of the pylon plate (1), and pylon (2). The cage is molded to fit the dressing. Plaster or fiberglass is then used to attach the cage. All gaps under the plate should be filled with casting material.

provides some suspension for the dressing. Tension on the sock is now released. The sock is trimmed, and the proximal free edge is folded back and incorporated with fiberglass into the cast. The completed dressing is now attached to the waist belt.

Next the pylon and SACH foot are attached to the dressing. The body and leg must be positioned for proper alignment of the pylon and prosthetic foot. The socket attachment plate is now fitted to the cast. The plate for pylon attachment is positioned at a 90° angle to the operating table (Figure 4-19). The center of the plate is inset 0.5 in. from the line drawn from the middle of the knee. The cage is molded to the exterior of the cast. Strips of fiberglass casting material are folded onto and placed between the distal cast and pylon plate. The cage is attached to the cast with the fiberglass casting material. The pylon and SACH foot in neutral position are attached, and the pylon is cut to the proper length. The pylon and foot are then removed and utilized later in therapy when controlled weight bearing can be ensured. Finally, a circular cut is made in the cast over the prepatellar region (Figure 4-20). The cut should be made smaller than the pad to allow the pad to be removed and also provide relief over the patella.

On postoperative day 1, the patient may do touchdown weight bearing at the bedside. After the first few days, the patient may be advanced to 10% weight bearing in the parallel bars. During the second week, weight bearing is increased to 50%. After two weeks (at the time of the second cast change), the patient may be advanced to full weight bearing with gait training in the parallel bars.[61] Full weight bearing with IPOP is controversial as some proponents are not in favor of full weight bearing.[60,61] For traumatic amputees IPOP is indicated, but for dysvascular amputees, this early weight bearing may result in skin breakdown.

The cast is changed at 1-week intervals until the wound has healed and the sutures are removed. This usually occurs two weeks postoperation in the uncomplicated traumatic amputation. At each IPOP change, the wound is inspected and, if no complications exist, another cast is applied immediately. While the limb is not in a rigid dressing, it should be elevated. If there is a delay in applying the next rigid dressing, the limb should be wrapped in an elastic bandage and kept elevated. In the presence of increased or unexplained pain, discomfort,[60] fever, or excess drainage, the cast should be removed and the wound appropriately investigated.[62] The surgeon will then decide whether to reapply the dressing. Once the wound is healed and sutures are removed, a temporary prosthesis or a removable rigid dressing with a pylon/foot assembly can be applied.

The advantages of the IPOP include edema prevention,[17] edema reduction,[58] trauma protection, reduced pain, decreased phantom pain,[58] psychological benefit, early weight bearing, and prevention of knee flexion contracture.[62] Disadvantages include the inaccessibility of the wound, the need for a prosthetist, and poor wound healing if weight bearing is excessive (caused by a lack of close monitoring during therapy).[21,58,63]

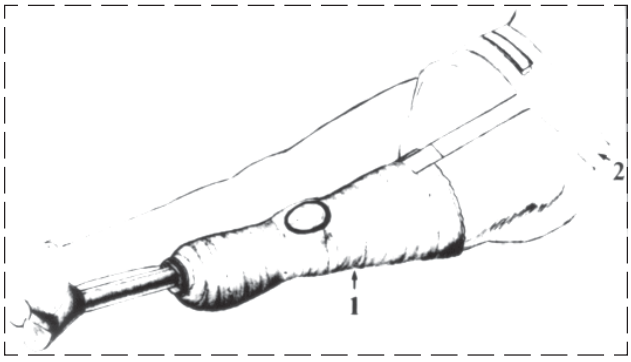


Fig. 4-20. IPOP with pylon and SACH foot (1), and waistbelt suspension (2). Note the patellar cutout that was made over the patellar pad.

Long Rigid Dressing. The application of an IPORD is similar to that of an IPOP except that a pylon and foot are not added. This IPORD is applied using the same techniques as when applying an IPOP, except the knee is cast in full extension.[62] The dressing is changed weekly until sutures are removed and the wound has healed. The patient is then fitted with a removable rigid dressing with pylon and foot, or with a temporary prosthesis. The reported advantages are the same as those for the IPOP, except there is no early weight bearing and no psychological advantage of awakening to see a foot. However, there is no wound breakdown secondary to excessive weight bearing,[21] and a prosthetist is not required.

Short Removable Rigid Dressing. A short, removable rigid dressing may be used immediately postoperatively or after the first or second IPORD change. The dressing is constructed of elastic plaster reinforced with conventional plaster or fiberglass. The dressing is carried anteriorly to midpatella, and trim lines are cut posteriorly in the popliteal space to accommodate the hamstring tendons and allow comfortable knee flexion. Instead of pads to create pressure relief, cotton cast-padding is used to make spacers over the tibial tubercle, tibial crest, and fibular head. Additionally, before casting, padding is added proximally over the residual limb. Once casted, the spacers and padding are removed. This allows the dressing to be removable even for a bulbous limb.[64]

This dressing may be suspended by a supracondylar cuff or waist belt attached directly to the dressing (Figure 4-21). The patient is instructed in proper donning of the rigid dressing, the suspension, and the varying thicknesses of stump socks to achieve a proper fit.

A short, removable rigid dressing with a pylon and foot is considered a preparatory or temporary prosthesis for the below-knee amputee. It is "temporary" because it requires changing as the residual limb decreases in size, but it is quite beneficial for early ambulation.

At 10 to 14 days postoperation, a gradual increase in weight bearing is started.[64] Weight bearing in the rigid dressing can be with or without a pylon and foot (see Figure 4-21). Weight bearing without a pylon is accomplished by standing with the dressing resting on a stool. A gradual weight bearing program is begun while standing. Before each increase in weight bearing, the limb is checked for duration and amount of skin erythema. Additionally, sock thicknesses are increased to give a continually snug fit, resulting in gradual decrease in stump edema.

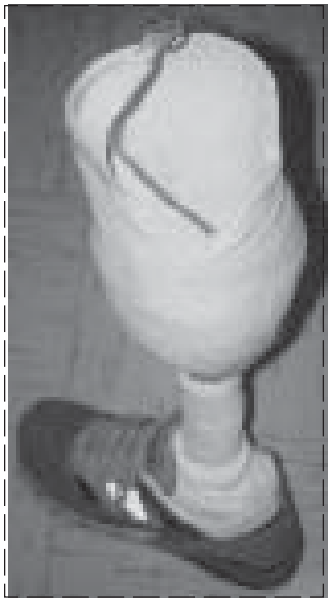


Fig. 4-21. A short removable rigid dressing (temporary prosthesis) with attachment strap for waistbelt suspension. Pylon and foot are attached.

The advantages of a short, rigid dressing compared to an IPORD are wound accessibility for easy wound examination during increases in weight bearing, and easy addition of socks, which facilitates quicker shrinkage.[64] Disadvantages of a short, rigid dressing include the possibility for development of knee flexion contracture, and for skin breakdown from excessive weight bearing before wound healing is complete.

Experience with Postoperative Dressings. The use of rigid dressings or IPOPs has generated concern regarding wound breakdown. In a small study on dysvascular amputees, Cohen and colleagues[63] reported problems with wound breakdown when using IPOPs. Burgess and associates[53] operated on 159 dysvascular patients and utilized either a rigid dressing or an IPOP; they reported failure to heal with revision to a higher level in 12 patients. Seven of the 12 were felt to have failed secondary to improper level selection and 5 failed from inadequate postoperative management. The time to definitive prosthesis wearing was significantly shortened to less than 60 days in 123 of the 150 BKAs.[53] Malone and associates,[20] when comparing IPOP and the team concept to soft or rigid dressing without weight bearing or team concept, reported improved healing and markedly shorter rehabilitation times and hospital stays. However, Baker and associates[65] reported no significant difference in healing rates

between soft and rigid dressings, although the rigid dressing patients' rehabilitation times were shorter. Mooney and associates[21] prospectively compared soft dressings, rigid dressings, and IPOPs in diabetics undergoing amputation. In general, they found healing times were shortest for the rigid dressing patients and slowest for the IPOP patients. Surprisingly, soft dressings had the highest percentage of nonhealing. The amount of time before receiving a definitive prosthesis was shortest when rigid dressings were used and longest when the soft dressings were used.[21]

Recommendation. To obtain the benefits of a rigid dressing without the risk of wound breakdown from excessive weight bearing, an IPORD may be used. The dressing is constructed as described earlier but without the pylon-foot assembly. The dressing is changed weekly until the wound has healed. Once sutures are removed, a short, removable rigid dressing (temporary prosthesis) is made.[64] Fiberglass casting is used for reinforcement (see Figure 4-21). The anterior trim line is to the midpatella with medial and lateral trim lines cut back posteriorly to the popliteal space to allow comfortable knee flexion. Suspension is through the waist belt (Figure 4-22). Alternatively, a supracondylar cuff suspension can be used with auxiliary waist belt suspension. A pylon and SACH foot assembly is attached as with the IPOP. Usually, for the first day, the dressing is not removable, but with stump shrinkage, the dressing becomes removable. The dressing is remade when the amputee is using 10 to 15 total ply stump socks. This temporary prosthesis allows early weight bearing. Temporary prefabricated prostheses, after initial rigid dressing or soft dressing, can be used for early weight bearing (Figure 4-23).[66–68]

Fig. 4-22. Soldier with temporary fiberglass/plaster prosthesis with waistbelt suspension.

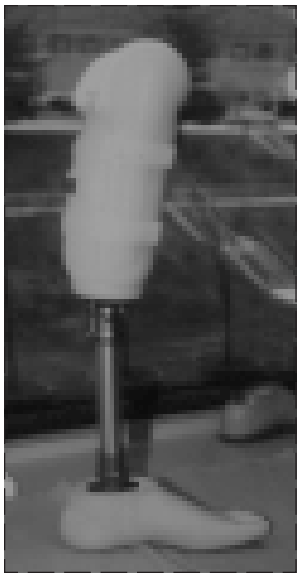


Fig. 4-23. Temporary, prefabricated, adjustable prosthesis with pylon and foot.

Rehabilitation of the Below-knee Amputee

Preoperative. Ideally, the amputation team should see the patient in the preoperative phase when the patient is awaiting amputation or has an open amputation that requires closure or a definitive procedure. For the war-injured amputee this usually involves awaiting closure of the open wound. The primary goal at this time is to prevent skin and soft tissue retraction by using skin traction (see Figure 4-14). Other important goals in the preoperative phase are (*a*) physical evaluation, (*b*) patient education, (*c*) prevention of medical complications, (*d*) physical conditioning, and (*e*) functional training.

A thorough patient evaluation must be obtained in order to plan the rehabilitation program. Medical problems affecting the musculoskeletal, neurologic, cardiac, pulmonary, vascular, and dermatologic systems may directly impact on both short-term and long-term functional goals and prosthetic usage. Besides a general examination, the following should be assessed:

- joint range of motion,
- joint stability,
- arthritic changes,
- amount of pain,
- relative strength,
- unusual sensations,
- patient coordination and balance,
- vision,
- cognition,
- pathologic reflexes and abnormal tone,
- vascular status in the involved limb, and
- dermatologic condition of the residual limb.

Knowledge of the patient's preinjury and current functional status is important. The preinjury functional status helps in establishing long-term goals for functional restoration. The current functional status provides the baseline for initiating therapy and developing short-term goals. The functional evaluation should assess ambulation, transfers, dressing, eating, bathing, and personal hygiene, and determine whether assistance in performing an activity or assistive equipment is needed. In particular, the initial functional examination should include bed mobility, sitting balance, standing balance, and transfers.

The psychological status and available personal support systems of the amputee and amputee's family are important factors affecting rehabilitation. A young amputee may express difficulty or concern with self-identity, body image, social acceptance, loss of function, employment, loss of income, peer acceptance, and sexual function. The psychological aspects of amputation or lack of a social support system may further increase distress.[19] The amputee may experience bereavement over the loss of a limb. This may include anxiety, despair, and anger. Often, a formal psychological or psychiatric consultation is not required. However, if the amputee's psychological status is affecting participation in rehabilitation, formal consultation should be obtained. Also, the effect the amputation has on the amputee's family members and the amputee's relationship with family members needs to be addressed.

Educational and vocational history is important in planning a return to gainful employment. Specific considerations may include (*a*) highest level of education attained; (*b*) jobs held, including dates and duration of employment; (*c*) specifics regarding physical tasks of the jobs; (*d*) job satisfactions and dissatisfactions; (*e*) relationship with coworkers; and (*f*) future employment plans and goals. Other factors that need to be addressed are recreational interests; financial status; location of residence; and availability of health care, prosthetic, and rehabilitation professionals in the patient's community. It is rare for amputees to remain on active duty.

Involvement of the patient as an active member in the rehabilitation process is important. To facilitate involvement, patient education is crucial. Specific explanations should be provided regarding the surgery, postoperative care, phantom sensation, phantom pain, the rehabilitation process, and prosthesis use. The amputation should be viewed as a reconstructive procedure instead of a destructive procedure. The surgeon should address questions about the surgery and postoperative surgical management. The physiatrist may explain other areas of amputee care. Other team members may provide further specific education regarding their areas of expertise. Meeting with a successful amputee, who has been screened by the amputation team, allows the patient to see the functional level that an amputee can achieve. Additionally, the successful amputee may further explain the process from the amputee's perspective and provide psychosocial support. For the prosthetic candidate, education regarding the prosthetic process and prescription is important. The potential prosthetic candidate should be educated regarding the differences between preparatory, intermediate, and definitive prostheses. The prosthetist can further describe the process and provide sample prostheses and components for viewing.

Prevention of complications during the preoperative phase is crucial, because complications may drastically slow or adversely affect the rehabilitation outcome. Complications of immobility include deep vein thrombosis, pulmonary embolism, pneumonia, atelectasis, orthostatic hypotension, decubitus ulcers, loss of muscle mass, osteopenia, and contractures.[18]

In particular, the transtibial amputee is at risk of developing knee flexion, hip flexion, hip abduction, and hip external rotation contractures.[58] Contractures may develop from poor bed positioning and stump pain. The risk of contractures can be further increased by flaccid paralysis, spasticity, edema, ischemia, and bleeding.[18]

A hip flexion contracture can result in decreased contralateral step length from limited hip extension.[58] A knee flexion contracture can decrease step length, create abnormal forces at the involved knee, and prohibit prosthetic fitting.

Contractures can be prevented by frequent position changes, prone lying, and a daily range-of-

motion program.[18] Bed position changes should be done on a scheduled basis. Prone lying is not always needed in the transtibial amputee[59]; however, if the patient is at risk for or is developing a hip flexion contracture, prone lying should be encouraged. The risk of developing a hip flexion contracture is increased with prolonged sitting and use of a soft mattress. Hip abduction contracture risk is increased with use of a pillow between the legs. Hip external rotation contracture is increased in the bedbound patient but the risk is reduced with use of a trochanteric roll. In transtibial amputees, placing a pillow under the thigh or knee should be prohibited to avoid a knee flexion contracture. Also, for sitting in a wheelchair, a knee extension board for the residual limb should be used to avoid knee flexion contracture.[59] Lastly, early ambulation will maintain joint range of motion. If a contracture occurs, an active and passive range-of-motion program with terminal stretch should be instituted.

With bedrest, normal individuals can lose 10% to 15% of their strength per week, or 50% of their strength in 3 to 5 weeks. The quadriceps and back extensors are particularly affected.[18] Weakness in these muscles can adversely affect the transtibial amputee's ability to ambulate.[59] To prevent loss of strength, the patient can perform 20% to 30% maximum isometric contraction for several seconds each day, or 50% maximum for 1 second.[18]

For the transtibial amputee in the preoperative phase, a more specific exercise program should be initiated. Isometrics of the back extensors, hip abductors, hip adductors, abdominals, gluteals, quadriceps, shoulder depressor, elbow extensor, and wrist extensor muscle groups should be performed for 10 seconds at regular 1-hour intervals in the daytime.[58,59] Once the patient is doing well with isometrics, he can progress to concentric and eccentric isotonic exercises. General conditioning can be performed using an arm ergometer. Arm strength is particularly important because amputees use crutches and canes initially for ambulation.

If medically feasible, the physical therapist can begin mobility training in the preoperative phase. Stand pivot transfers should be taught to allow independent transfers between the bed and wheelchair. Once bed transfers are mastered, toilet, tub, and car transfers should be taught. Use of a trapeze should be avoided unless clinically necessary, because the trapeze promotes use of the biceps rather than the triceps, which are normally used for transfers.[59] Also, the patient is unlikely to have a trapeze for use at home, whether visiting on pass or after discharge from the hospital. Wheelchair skills should also be taught. These skills will promote independence while in the hospital before crutch ambulation is mastered. Additionally, this activity helps maintain general conditioning and prevent complications of immobility. The patient will also need these skills in the postoperative phase, and the skills are easier to teach preoperatively than immediately postoperatively. Additionally, some amputees may require wheelchair mobility for long-distance mobility, or for times when they are unable to wear their prostheses. Prolonged wheelchair sitting should be avoided, however, because it leads to hip flexion and knee flexion contractures.

Crutch ambulation should also be instructed preoperatively. First, the patient should master sitting, and then standing balance. Gait training can then begin in parallel bars, advancing appropriately until crutch ambulation is achieved. Once crutch use is mastered on the level surface, training moves toward independent crutch ambulation on stairs and rough terrain.

Independence in basic ADLs should be promoted based on the patient's current function in the preoperative phase. An occupational therapist can evaluate and provide training in feeding, toileting skills, bathing, dressing, and orofacial hygiene. Additionally, the preoperative evaluation and training will help to delineate postoperative capabilities and rehabilitation needs.

Postoperative. The postoperative phase of rehabilitation would be for those individuals who have undergone their definitive amputation procedure. The main goals during this phase are (*a*) wound healing, (*b*) early limb maturation, (*c*) prevention of complications, (*d*) functional independence, and (*e*) prosthetic fitting. Some amputees achieve independence in functional activities sooner than others. The following discussion is a general overview of a typical postoperative rehabilitation program.

Healing is crucial, because delayed healing results in delayed rehabilitation. Causes of delayed healing may include an improperly applied rigid dressing, or excessive weight bearing. If a rigid dressing is poorly fitted, the dressing should be remade. If wound breakdown occurs from excessive weight bearing, weight bearing should be discontinued until healing is complete. The complications of immobility and methods of prevention need to be followed as in the preoperative phase. The postoperative program includes the previously described dressings.

During the first postoperative week, exercises, functional training, and education will continue. On postoperative day 1, isometric gluteal and quadri-

ceps exercises along with gentle range-of-motion exercises of the residual limb are started.[58] Active range-of-motion and progressive resistive exercises are begun on the contralateral limb and upper extremities. The patient should begin standing at bedside. The physical therapist will assist the patient in sit-to-stand transfers and standing balance at bedside.

As the week progresses, wheelchair transfers, wheelchair mobility, and crutch ambulation are begun. The benefit of preoperative training is now apparent, as the patient is already prepared for transfers and crutch ambulation. Crutch ambulation is important to prevent complications of immobility. If the patient is unable to crutch ambulate independently or for long distances, wheelchair mobility should be encouraged. As in the preoperative phase, an extension board for the residual limb should be utilized.

Basic ADL training continues through week 1, based on the patient's current functional and medical states. If the patient was not seen preoperatively, education regarding the rehabilitative process and prosthetic process should now be given. Additionally, education regarding phantom limb sensation and pain should be provided. At the end of week 1, the first IPORD change occurs. The limb is examined and if no complications are present, the second IPORD is applied.

During week 2, gentle resistive exercises for the residual limb begin, and functional training continues. At the end of the second week, if the wound is healing well, the patient is changed to a short, removable rigid dressing with a pylon/foot assembly (temporary prosthesis). If the residual limb shows any areas of nonhealing, the rigid dressing can be continued, or replaced with elastic wrapping to allow close skin monitoring; the pylon/foot assembly is kept in physical therapy to be used for controlled gait training, thereby preventing the patient from harm due to inappropriate weight bearing.

Exercises to increase strength and endurance continue. After the residual limb is inspected for any skin breakdown, functional training in physical therapy advances to progressive gait training with the temporary prosthesis. First, the residual limb is inspected for any skin breakdown. If there is breakdown, the physician should be consulted before any weight bearing is allowed. Second, the amputee is instructed and assisted in sit-to-stand transfer in the parallel bars. Third, the amputee is allowed to weight bear 40 lb through the residual limb.[69] The amount of weight is measured by having the amputee stand on scales under each limb. The prosthetic side is limited to roughly 40 lb initially, then increased as the limb tolerates. Fourth, alignment is checked and should be similar to a patellar tendon-bearing prosthesis (Figure 4-24). The foot

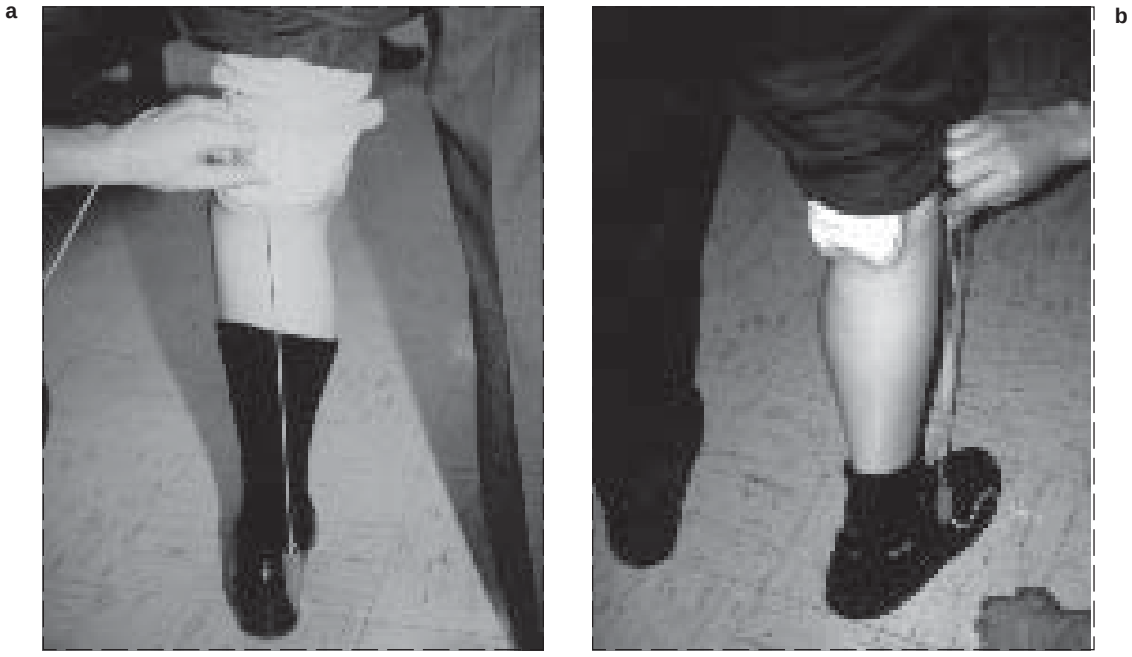


Fig. 4-24. Static alignment is done using a plumb line, (**a**) posterior and (**b**) lateral.

should be flat on the ground and iliac crest height symmetric, suggesting equal leg lengths. The socket should be in 5° to 10° of flexion and 2° to 5° of adduction. A plumb line dropped posteriorly from the middle of the socket should fall approximately 0.5 in. medial to the heel center (see Figure 4-24).[70] A plumb line dropped laterally from the middle of the socket should fall approximately 0.5 in. anterior to the breadth of the heel. The foot is externally rotated 5° to 7° and is symmetric when compared to the opposite limb. Static alignment may only be approximate until the amputee is able to bear equal weight through both legs. Dynamic alignment is evaluated later. Static alignment corrections should be made before proceeding with ambulation. Fifth, standing is done with a bathroom scale under each limb. Only 40 lb of weight bearing is allowed on the amputated side. After standing for a short period, the amputee sits down and the rigid dressing is removed. The residual limb is inspected for evidence of total contact, usually seen by the presence of sock print left on the limb, and for any excess pressure areas and pain caused by the rigid dressing. Any malalignment that has caused pain and excess pressure is corrected. If the problem is inherent in the dressing, a new rigid dressing is fabricated.

If no complications have occurred, gait training continues. Ambulation with assistance in the parallel bars begins with a 40-lb weight bearing limit on the amputated side. (In reality, weight bearing during ambulation cannot be controlled very well without measuring directly the force in the pylon and providing audio feedback when the limit is exceeded.) After the patient ambulates one length of the parallel bars, the residual limb is inspected. Additionally, dynamic alignment (discussed later) is assessed with ambulation. If no complications are noted, another session occurs in the afternoon. If complications attributed to the rigid dressing occur, corrections are made. If the dressing or malalignment was not the cause, the amount of weight bearing is decreased until signs of excessive pressure are resolved.

Advancement of the gait training program includes daily distance increases in parallel bar ambulation. Also, every 2 to 3 days the weight bearing may be increased by 40 lb until full weight bearing is achieved.[69] With each increase in weight bearing, proper weight shifting and standing balance should be mastered before advancing gait training. Proper arm, trunk, pelvic, intact leg, residual limb, and prosthetic movements are practiced.[59] Once parallel bars are mastered, the amputee is progressed to bilateral forearm crutch ambulation within the weight bearing restrictions.

Transfer training is continued with the goal of independence in sit-to-stand transfers with the prosthesis. Because the amputee will not initially have the pylon-foot complex, he or she must first become independent in one-legged stand-pivot transfers.

Basic ADL training with occupational therapy is addressed. In dressing, attention is especially paid to LE dressing along with donning and doffing of the rigid dressing and stump socks. While the amputee with good balance may stand for most bathing procedures, washing of the intact leg and foot usually requires sitting and the use of a tub bench and hand-held shower. In some complicated cases where there are other coexistent injuries, toileting, eating, and personal hygiene may need special training or adaptive equipment.

The purpose of the rigid dressing for limb maturation, edema reduction, protection, and early ambulation is taught. The amputee must be aware of edema development when the rigid dressing is off, especially after showering with the limb dependent. Proper donning of socks is performed with the seams not crossing the incision and without wrinkles. Stump socks give a proper fit to the rigid dressing as the limb shrinks. The amputee maintains steady tension on the socks to prevent excess stress to the incision when donning the rigid dressing. The amputee may wear a nylon stump sheath to decrease shear. Both sheaths and socks should be cleaned and changed daily. The amputee must regularly examine the limb for any skin breakdown, with initial skin checks done in the morning, after any ambulation, and before bedtime.

Another method of edema control, particularly if a temporary prosthesis cannot be used, is elastic wrapping of the residual limb. Elastic wrapping incorporates a figure-8 configuration to avoid the tourniquet effect of circular wraps (Figure 4-25). The wrap begins distally, taking tension off of the incision line and properly shaping the limb. The elastic wrap extends proximally to include the distal third of the thigh. The elastic wrap should be firm but not tight, removed every 4 hours for inspection, and worn at all times when the rigid dressing is off. The residual limb should not be dusky-colored or develop blisters from wrapping.[59]

When applying a shrinker sock, the seam should not cross the incision, no pocket should be formed at the end, and the proximal band should not be too tight (Figure 4-26). A shrinker sock is an elastic sock designed to compress the residual limb and

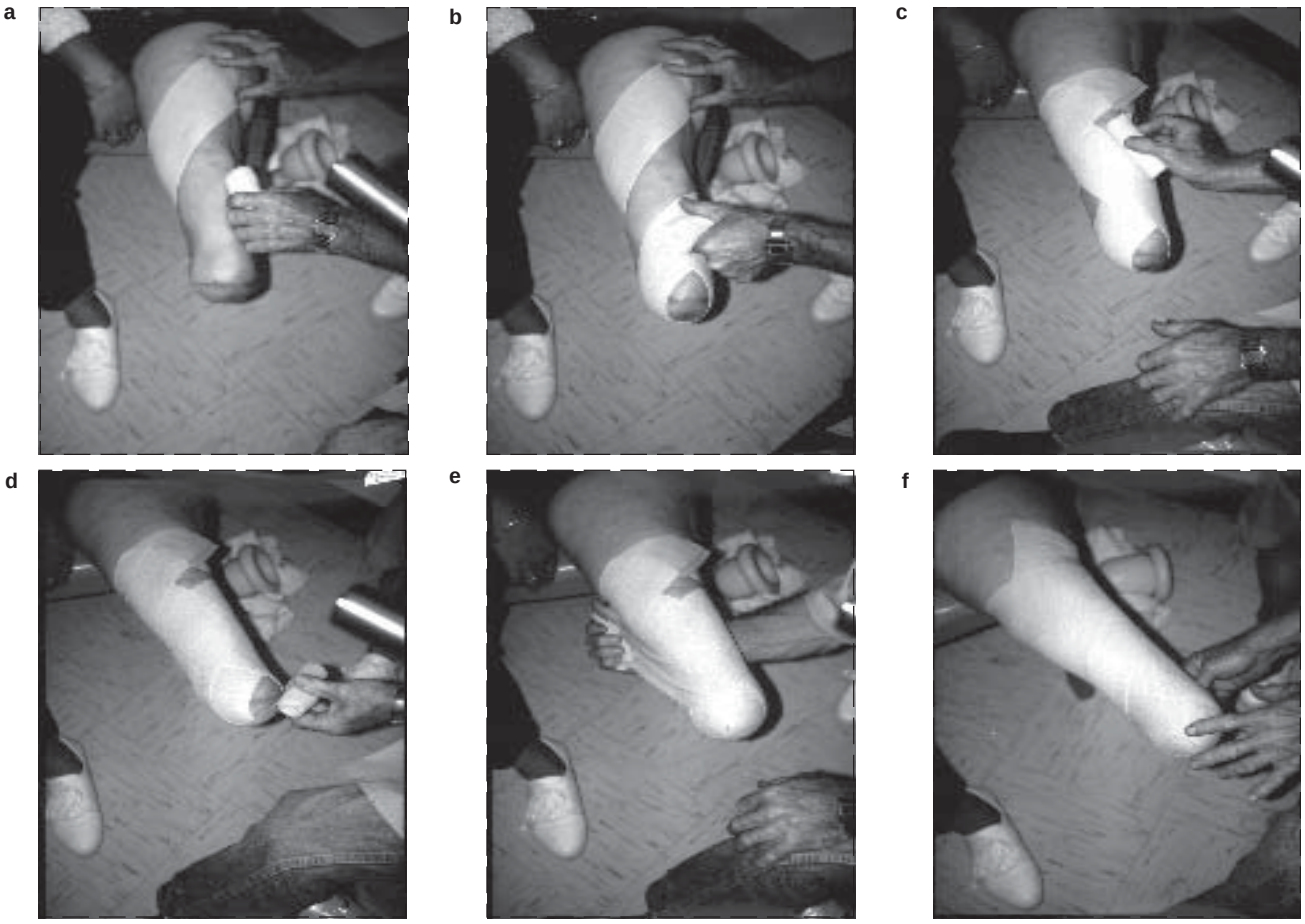


Fig. 4-25. Elastic wrapping is done in a figure-8 configuration. Note how tension is taken off the suture line and shaping of the residual limb is affected by the wrap.

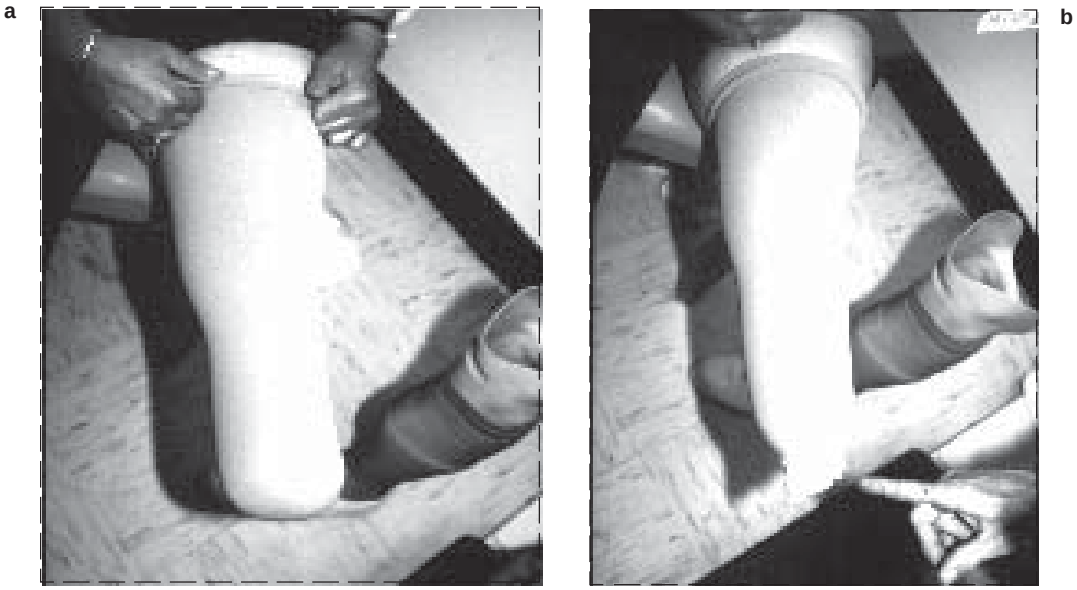


Fig. 4-26. Shrinker with (**a**) proper application and (**b**) improper donning with a distal pocket that allows edema accumulation.

reduce edema. The prosthetist can provide the appropriate size.

As the limb shrinks and matures with proper shaping, the rigid dressing may be substituted with a shrinker or an elastic wrap while sleeping at night. Again, the residual limb must be in the rigid dressing, an elastic compressive wrap, or a shrinker sock to prevent edema accumulation, which can occur rapidly.

Controversy exists over using a shrinker or an elastic wrap. The advantage of the elastic wrap is that it conforms to the residual limb. The disadvantages include tourniquet risk, stump damage with excessive compression, difficult application, slippage, and variation in tension with repeat applications.[59] The shrinker is easier to apply, but may slip down, causing a distal pocket of edema. This can be solved with use of a garter belt. One study[71] of 12 LE amputees evaluated the effectiveness of edema reduction utilizing either an elastic wrap or shrinker. The shrinker was more effective. However, the study can be criticized for small sample size and large standard deviations in edema measurement results. Whether an elastic wrap or shrinker, proper usage and application are most important, and the two items should not be concomitantly used on the same limb, as pressures may become excessive. Once the amputee has stable residual limb volumes throughout the day, an overnight trial without a shrinker or elastic wrap may be tried. If there is no difficulty donning the prosthesis the next day, the shrinker or elastic wrap may be discontinued.[59]

During the second and subsequent weeks, gait training continues with the goal of independent ambulation without assistive devices. The amputee should have forearm crutches for use when unable to wear a prosthesis. Once level surfaces are mastered, advanced gait training should begin. The amputee should become independent in stairs, uneven terrain, and ramps. If feasible, for the amputee living in an area with snow or ice, training on these surfaces should be addressed. The amputee is also trained in more advanced transfers, such as car and floor-to-standing transfers.

Once basic ADLs are mastered, advanced ADL training ensues. Advanced ADL training is individualized toward the patient's home, community, and work environments. Common areas include independence in the kitchen, grocery shopping, and driving. Adaptive equipment needed to perform ADLs is provided prior to discharge from the hospital. These may include tub benches, hand-held showers, wheelchair, and forearm crutches. Necessary equipment should be ordered early in the hospital course to avoid delaying discharge from the hospital secondary to pending equipment. For right transtibial amputees, adaptive pedals are available for operating the car gas and brake pedals. Some states require these adaptations. The amputee should consult his local driver's license bureau and insurance company regarding special requirements for amputees.

The amputee should meet with a vocational counselor regarding future vocation. This will often be handled through the VA vocational counselor. A small percentage of highly motivated soldiers will want to remain on active duty.

Once residual limb volumes are fairly stable with use of the removable rigid dressing, usually about 6 weeks postoperative in the uncomplicated patient with the above protocol, an intermediate prosthesis should be fitted. The intermediate prosthesis consists of a socket, liner, suspension, endoskeletal pylon, and ankle/foot assembly. The advantage of the endoskeletal pylon is the ability to adjust alignment as the patient's gait and function changes in the intermediate period.[72] Once the prosthesis is complete, the amputee is evaluated in the new prosthesis for fit and alignment. Any malalignment is corrected before accepting the prosthesis and allowing ambulation. An improperly aligned prosthesis can result in harm to the residual limb and a less functional outcome. If the prosthesis fits and is aligned correctly, basic and advanced gait training in the prosthesis is taught by the physical therapist. This process is rapid if the amputee has already mastered ambulation skills prior to receiving the intermediate prosthesis. The amputee is thoroughly educated in maintenance and care of the prosthesis. The socket requires daily cleaning. The intermediate prosthesis can often be used for many months.

Many amputees will require at least socket changes during the first year, if not a completely new prosthesis. The amputee is checked in 4 weeks and every few months afterwards if no complications necessitate more frequent follow-up. Over the first year, there will be continued residual limb volume shrinkage with potential need for a new prosthesis. After the first year, the amputee should be followed at least every 6 to 12 months indefinitely.[62]

Prosthetic Prescription

The prosthetic prescription is based on the medical condition, residual limb characteristics, and functional goals of the patient. The patient's overall health and medical condition, especially cardiopulmonary, neurologic, and musculoskeletal must be considered. Residual limb factors include length,

shape, skin condition, soft tissue coverage, joint stability, muscular strength, edema, and presence of contractures. The patient's vocational goals, avocational goals, cosmesis, home and work environment, climate, cultural background, and the availability of prosthetic services also impact the optimal prosthetic prescription.

A knowledge of different prosthetic components is necessary to achieve the appropriate prescription. The prescription requires a team effort with active participation of the patient, physiatrist, therapists, and prosthetist.

Sockets. The socket contains the residual limb and forms the union between the residual limb and prosthesis.[14] The function of the socket is to provide stability, transmit forces, support, and contain the residual limb.

The standard socket in use for below-knee amputees is the patellar tendon bearing, total contact socket (PTB-TCS).[17] This socket allows improved transmission of forces compared to the bucket fitting sockets, which transmit forces primarily through the end of the residual limb. The PTB-TCS, by being a total contact socket, distributes forces over a wider surface area than the bucket fitting sockets, and thus, results in decreased pressures to the residual limb. (Pressure is defined as force per unit of area.) However, not all areas of the residual limb tolerate pressure equally. For example, excess pressure to a bony prominence, such as the tibial crest, may lead to skin breakdown or pain, while a soft tissue region, such as the anterior compartment muscles of the leg, may accommodate the same pressure without difficulty. These areas are termed *pressure intolerant* and *pressure tolerant*, respectively. Pressure intolerant areas in the socket are the distal tibia, distal fibula, fibular head, tibial crest, lateral tibial flare, tibial tubercle, and peroneal nerve.[14,72] These will require pressure relief. The pressure tolerant areas of the socket include the patellar ligament, medial tibial flare, medial tibial shaft, lateral fibular shaft, and anterior compartment muscles.

The anterior wall of the PTB-TCS extends proximally and covers the distal one third of the patella, with an indentation pushing on the patellar tendon (Figure 4-27). The indentation, termed the patellar bar, distributes pressure over a pressure tolerant area. The posterior aspect flares outward to provide relief for the hamstrings. The proximal aspect of the posterior wall may indent to provide a counterforce, which maintains patellar bar contact. The posterior wall must be trimmed to provide relief for the hamstring tendons and allow comfortable knee flexion. The medial and lateral walls extend to the level of the adductor tubercle. Together, the walls provide mediolateral and rotational stability. The medial wall contacts the medial tibial flare for weight bearing. The lateral wall has a pressure relief area for the fibular head.[72]

Fig. 4-27. Patellar tendon bearing, total contact socket (PTB-TCS) endoskeletal design. Lateral view showing the indentation anteriorly below the patella, which represents the patellar bar.

The soft PTB-TCS refers to sockets utilizing a compressible liner. The liners are made from a variety of materials: different types of soft foam padding, leather, silicone gel, etc. The liners are designed from the positive mold of the patient's residual limb, and are worn inside the socket to provide shock absorption, decrease shear forces, and provide comfort. The soft PTB-TCS socket is indicated for amputees with fragile skin, insensate or tender limbs, limbs with excessive scar or sharp bony prominences, skin grafts, and peripheral vascular disease; for bilateral transtibial amputees, to reduce shear force; and for the highly active amputee.[17,72] The disadvantages of liners are decreased hygiene (with absorption of sweat and dirt), increased weight, increased bulk at the knee, and deterioration of the liner over time, leading to loss of prosthetic fit.[72,73]

The hard PTB-TCS does not incorporate a liner. Usually, a distal pad is placed in the hard socket to provide padding for the distal residual limb. A hard socket may be used when there is a mature, cylindrical residual limb with good soft tissue coverage. The hard socket should not be used in the presence of peripheral vascular disease, diabetes mellitus, thin skin, skin grafts, skin predisposed to breakdown, excessive scar, or by new amputees. The main advantages of the hard socket are better hygiene (it is easy to clean and produces less odor compared to liner use) and the capability to make precise socket modifications.[72] The distal pad in the hard socket provides increased comfort and helps prevent distal edema.[14,72] The main disadvantage is that it causes difficulty in fitting bony or sensitive residual limbs. The hard socket with distal pad may be helpful for those with distal skin problems, edema, and impaired proprioception.

The supracondylar PTB-TCS is a variant of the PTB-TCS socket (Figure 4-28). The anterior and posterior walls are essentially unchanged; however, the medial and lateral walls extend proximally over the femoral condyles. The higher medial and lateral walls provide more mediolateral and rotational stability along with increased surface area for pressure distribution. Additionally, suspension is provided by purchase above the femoral condyles. The socket is used with a soft liner with buildup over the medial femoral condyle to provide suspension and still allow the amputee to enter into the socket.

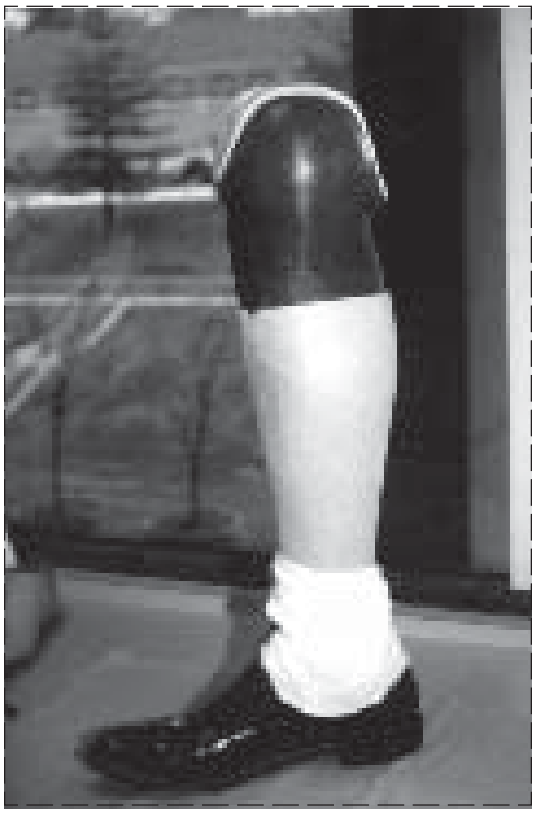


Fig. 4-28. Supracondylar thermoplastic patellar tendon bearing, total contact socket (PTB-TCS). The medial and lateral walls extend above the condyles and indent to provide suspension, mediolateral stability, and rotational control.

Fig. 4-29. Supracondylar exoskeletal PTB-TCS with removable medial brim.

There are two variants for this form of supracondylar suspension: (1) the removable medial brim, which allows donning with suspension provided by reattachment of the brim (Figures 4-29 and 4-30), and (2) the removable medial wedge, which is a foam wedge that is inserted between the socket and medial femoral condyle to provide suspension. The supracondylar PTB-TCS is indicated in patients with a short residual limb, mild knee instability, or as an optional suspension. It is relatively contraindicated in obese and some muscular individuals whose thigh shape may preclude the ability to achieve purchase on the femoral condyles. Individuals with moderate laxity may need the extra stabilization of a thigh corset with side joints.[72]

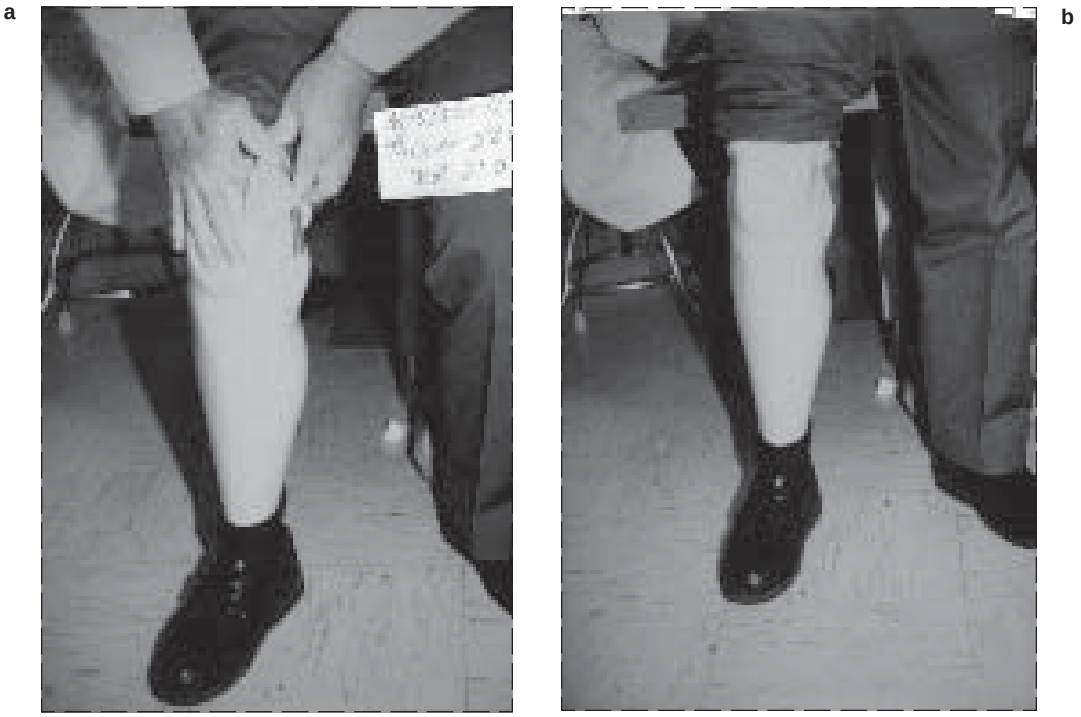


Fig. 4-30. Donning supracondylar patellar tendon bearing, total contact socket (PTB-TCS) with removable medial brim. (**a**) Insertion of medial brim and (**b**) medial brim in place.

The suprapatellar-supracondylar patellar tendon bearing socket is similar to the supracondylar PTB-TCS but with the extension of the anterior wall which covers the patella. Additionally, there is an indentation just proximal to the patella that is termed the quadriceps bar. The quadriceps bar provides a knee extension stop. Compared to the supracondylar variant, this socket provides better distribution of mediolateral, torsional, and surface area pressure. Furthermore, the supracondylar-suprapatellar variant provides anterior-posterior stability and resistance to genu recurvatum.[72] The socket has the same indications as the supracondylar PTB-TCS and the addition of patients with genu recurvatum. The relative contraindications of the suprapatellar-supracondylar socket are the same as those of the supracondylar socket; also, the suprapatellar-supracondylar socket should not be used for patients who frequently kneel.[72]

Thermoplastics now allow fabrication of flexible inner sockets that can be held in a rigid outer frame (Figure 4-31). Flexible sockets may be made from polyethylene, copolymer polypropylene, and polypropylene homopolymer.[72,74] The rigid frame is placed over primary weight bearing areas.[72] The flexible socket covers the bony prominences, providing relief from the rigid frame.[72,75] The degree of socket flexibility can be further increased for improved flexibility and comfort.[75] The advantages include decreased weight, increased comfort, increased heat loss, and the ability to replace the flexible socket for residual limb–socket interface changes.[72,75] The potential disadvantages are decreased cosmesis and difficult fabrication.[72]

Fig. 4-31. Thermoplastic socket with flexible inner socket and rigid outer frame.

Thermoplastics also allow fabrication of below-knee prostheses composed entirely of thermoplastic materials (see Figure 4-28).[76,77] The socket and shank are thermoplastic, with the prosthetic foot incorporated into the shank(see Shank section below). The thermoplastic shank and foot may provide some dynamic response. An average thermoplastic below-knee prosthesis weighs approximately 1.5 to 2.3 lb. The main advantages are decreased weight, dynamic response, low inertial mass, cosmesis, comfort, and ease of suspension secondary to the reduced weight.[76,77] The main disadvantage is that only minor socket modifications and alignment changes can be made after fabrication; however, refabrication is simple.[76]

Suspension. The main goal of suspension is to keep the prosthesis securely attached to the residual limb without causing excessive pressure or shear forces, discomfort, apprehension, impaired function, or choking of the residual limb.

Supracondylar cuff suspension is the most common form of suspension used in the transtibial amputee (Figure 4-32).[7,17,78] The cuff is made from leather-lined Dacron. The cuff wraps around the thigh just proximal to the patella and femoral condyles. The cuff utilizes the patella and condyles for suspension. It is closed by an anterior Velcro strap or buckle and attaches to the socket just posterior to the midsagittal line. This placement assists in resisting knee hyperextension forces, and allows the knee to be slightly withdrawn in knee flexion. The cuff is indicated in most transtibial amputations that exhibit good knee stability. The cuff should not be used in short or ultrashort residual limbs,[72] which need supracondylar/suprapatellar or supracondylar types , or sockets with thigh lacer and side joints. Also, sensitive skin or excessive scarring in the region of the cuff may preclude use of the cuff. The reported advantages of the cuff include ease of donning and doffing, adjustability, easy replacement, adequate suspension for most transtibial amputees, and moderate knee extension control. The disadvantages may include socket pistoning, no mediolateral stability, pinched soft tissue posteriorly between the cuff and socket with knee flexion, and restricted circulation from too tight an application.

A thigh corset with side joints consists of a leather corset with anterior laces encircling the distal two-thirds of the thigh, and metal side joints (single axis or polycentric) attaching the corset to the socket (Figure 4-33). The single axis joints must be precisely located to avoid excessive movement between

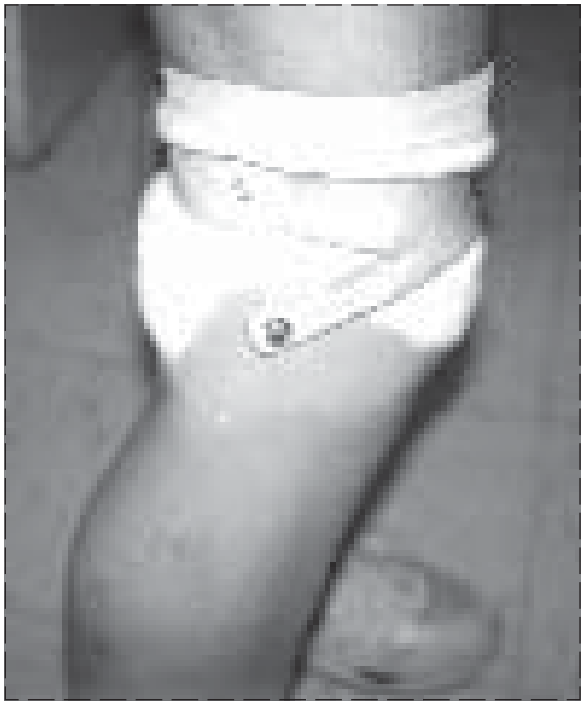


Fig. 4-32. Types of suspension. Supracondylar cuff suspension.

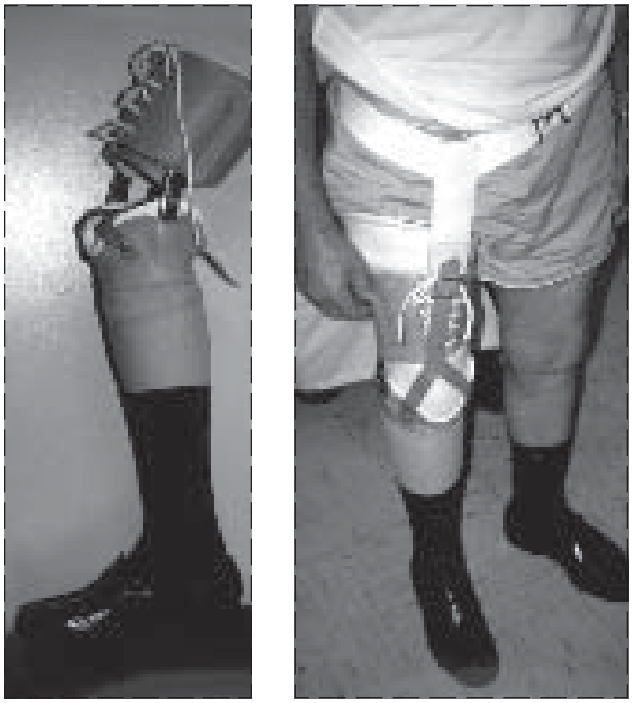


Fig. 4-33. Types of suspension. Thigh corset with side joints (left) and with waist belt suspension (right).

the socket and residual limb. With the knee in full extension, the joints are located slightly posterior and superior to the anatomical joint center. To limit knee extension or control genu recurvatum, a posterior check strap may be attached from the posterior socket to the corset. The thigh corset and side joints provide not only suspension, but also shared weight bearing.[72] Often, this suspension is accompanied by waist belt suspension.

A thigh corset with side joints is indicated when maximal anterior-posterior or mediolateral stability is needed, as in an unstable knee joint. It is also indicated for amputees engaged in very heavy work, or for those who need shared weight bearing to partially unload the residual limb in the socket.[72] Additionally, it may be of benefit to individuals who are obese, lift and carry weights, or have knee joint arthropathy.[14]

Other reported advantages of the thigh corset and side joints are the shared torque and weight bearing, maximum mediolateral stability, maximum reduction of genu recurvatum, and increased proprioceptive feedback.[72] Drawbacks include thigh atrophy, proximal constriction, increased weight, bulk, decreased cosmesis, increased fabrication time, nonhygienic attributes of leather, discomfort in hot weather,[14] and excessive wear on clothing. Also, single axis joints need precise placement to avoid excess movement between the prosthesis and residual limb, and there is the occasional need for auxiliary waist belt suspension (see Figure 4-33).

Waist belt suspension consists of a belt worn above the iliac crests or between the crests and greater trochanter, with an anterior strap suspending the prosthesis. The anterior strap is elastic and connects to a buckle at midthigh. The buckle is connected to another strap attached to a supracondylar cuff or inverted Y-strap. The midthigh buckle is used to adjust the suspension. The waist belt suspension may be used by itself or as auxiliary suspension. Waist belt suspension is indicated in the initial management of a postoperative patient using an IPOP or a temporary prosthesis. Suspension is maintained despite the fluctuations in volume during maturation. Other indications include the need to eliminate proximal constriction, provide auxiliary suspension in sports, and patient preference.[72] The waist belt is contraindicated if there is a scar and sensitive skin in the region of the belt. Problems with waist belt suspension include discomfort in wearing a belt, uneven suspension during swing phase, and no resistance to knee extension.[72] In patients with axillary to femoral or iliac artery grafts, waist belts can occlude the graft.

Sleeve suspension consists of a latex or neoprene sleeve.[14] The sleeve extends from the prosthesis to 2 to 3 in. proximal to the end of the residual limb socks. Suspension is created by negative pressure during the swing phase from the seal created by the sleeve. Suspension is also created from friction between the sleeve and the skin or prosthesis and the longitudinal tension of the sleeve. The latex sleeve provides the best seal and resultant suspension. The neoprene sleeve provides an acceptable seal, while the cloth-lined neoprene sleeve does not work well. Sleeve suspension may be used alone, or as auxiliary suspension for the suction below-knee prosthesis and with cuff suspension during recreational or sporting activities. Sleeve suspension should not be used as the sole suspension with short transtibial residual limbs. Amputees who kneel frequently, or who live in hot, humid climates may be unable to use sleeve suspension because the sleeve is not as durable as other suspensions, and allows perspiration buildup. Reported advantages of sleeve suspension are decreased pistoning, good auxiliary suspension, simplicity, and cosmesis.[73] Disadvantages include decreased suspension if the sleeve becomes torn; lack of durability, causing frequent replacement; skin irritation; contact dermatitis; possible decrease of full knee flexion; undesirable odors; and the need for good hand function to don and doff the sleeve.[14,72,73]

Suction transtibial prostheses are available, but the need for precise fit makes fabrication difficult. The prosthesis is fitted exactly to the shape of the limb with weight bearing over the entire distribution of the residual limb. Two check sockets are utilized to give precise fit. Suction is provided by making the volume of the socket smaller than the residual limb. The smaller socket creates tension on the skin, and increases friction between the limb and socket interface. This process creates suction. The residual limb is lubricated with powder, cream, or lotion to get it into the socket. A valve on the socket allows air to escape while donning the socket. A clear rigid check socket allows the limb to be viewed for exactness of fit and for any discoloration, which would indicate too tight a fit. The residual limb should maintain a normal color while in the socket. The hard socket is adequate for walking, but not for more vigorous activities because of discomfort. For more active amputees, prostheses should be made with soft liners, which have a distal valve to allow suction suspension. For both hard and soft suction sockets, auxiliary suspension with a sleeve is recommended in case of failure of the suction suspension.[73]

Another suction method is the silicone suction socket (3S). The 3S uses a silicone suspension liner fabricated over a mold of the residual limb. At the end of the silicone liner is a small protruding notched pin that secures the silicone liner to the prosthesis (Figure 4-34). Socks may be utilized to provide cushioning. The reported advantages of this prosthesis include decreased shear forces, improved knee range of motion, and no need for an auxiliary suspension.[79] Disadvantages cited are difficulty in donning the liner and loss of suspension if the liner should become punctured.[72] Use of this prosthesis is indicated for individuals with a cylindrical residual limb who desire a suction suspension and are highly motivated, compliant, have an understanding of the function of the valve and liner, and are willing to undergo multiple fittings and modifications. Difficulty in fit may occur with conical shaped limbs.[73] Relative contraindications are fluctuating residual limb volumes, skin hypersensitive to touch, poor hand function, excessive distal soft tissue redundancy, and patients who have never been satisfactorily fitted with any prosthesis.[79]

Overall, reported advantages of any suction suspension system are decreased pistoning, improved circulation from intermittent pressure changes with walking, better prosthetic control, improved sensory feedback, improved cosmesis, improved comfort, and improved mobility. Disadvantages reported are the more difficult fabrication of the socket, the risk of proximal constriction or excessive unremitting negative pressures with resultant distal edema, difficulty in maintenance of suction, possible skin irritation, and the frequent need for liner replacements. Relative contraindications are new amputees with fluctuating volumes, noncompliant patients, and limbs with less than 5 in, of length.[80]

Shank. The shank provides the length of the prosthesis from socket to foot. The shank is the connec-

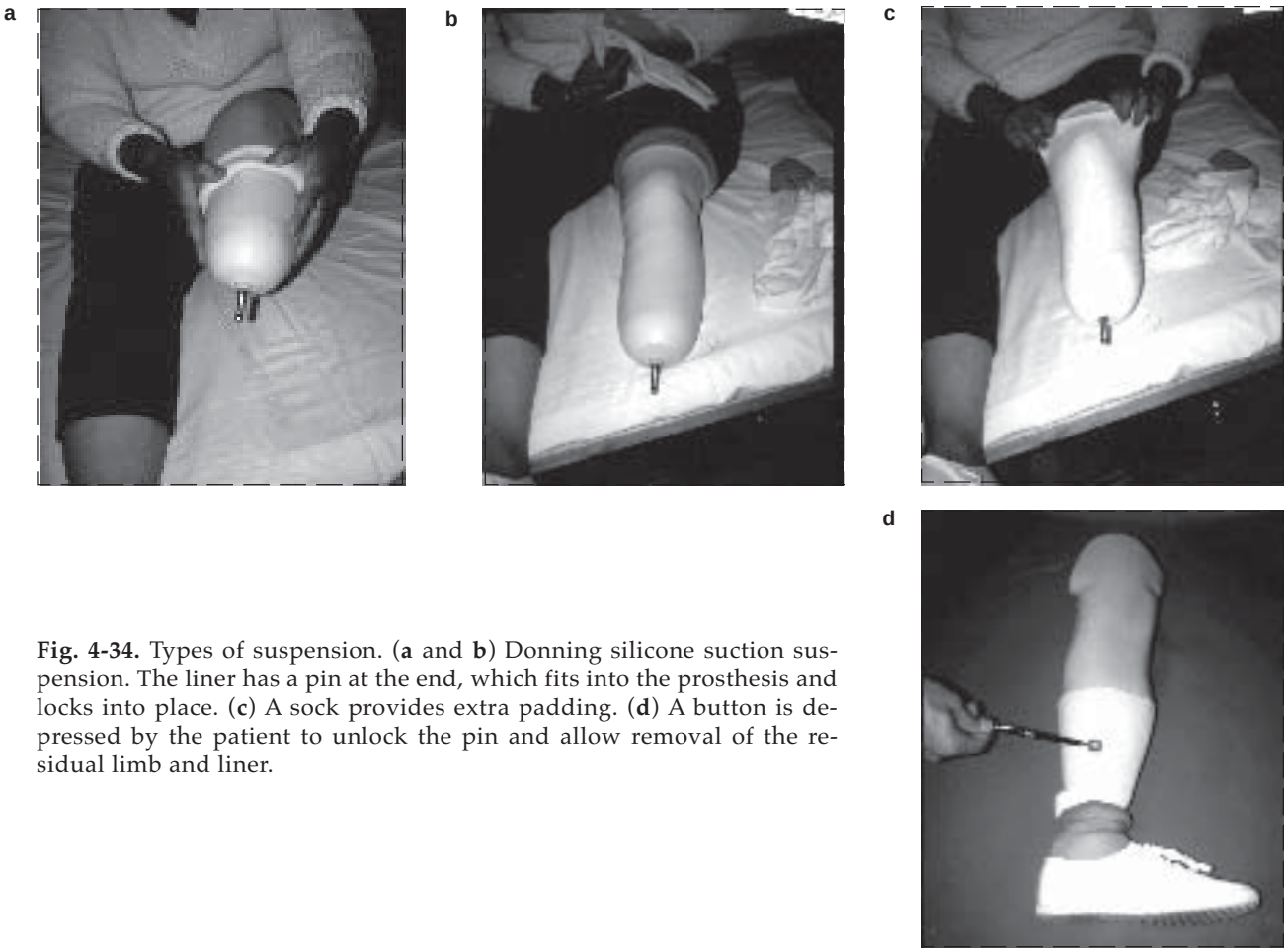


Fig. 4-34. Types of suspension. (**a** and **b**) Donning silicone suction suspension. The liner has a pin at the end, which fits into the prosthesis and locks into place. (**c**) A sock provides extra padding. (**d**) A button is depressed by the patient to unlock the pin and allow removal of the residual limb and liner.

tion between the socket and ankle/foot mechanism. The shank may be of *endoskeletal* or *exoskeletal* construction. The endoskeletal construction consists of metal or composite pieces connecting the socket to the foot (see Figure 4-27). Endoskeletal prostheses often have a cosmetic foam cover and skin tone nylon stockinettes. The reported advantages of the endoskeletal construction is the interchangeability of components for trial or replacement, its lightweight construction, adjustable alignment after fabrication, and cosmesis. The main reported disadvantage for the endoskeletal design is that the foam cover may become torn and need regular replacement.

The exoskeletal shank is made of wood or polyurethane with rigid plastic lamination (see Figure 4-29).[81] This durable construction is its advantage.

A rotator unit can be placed in the shank to allow rotational movement. This may be helpful for those amputees involved in activities requiring rotation, such as golf. Also, a rotator unit may help decrease shear at the socket–residual limb interface.[13]

The Prosthetic Foot. The goal of the prosthetic foot is to simulate normal ankle/foot function. In meeting the demands of a normal ankle/foot complex, the prosthetic foot should provide a stable base of support, simulate ankle/foot joints and muscles, provide shock and torque absorption, and be cosmetic. Different types of the prosthetic foot meet these goals to varying degrees. The prosthetic foot can be divided into four types based on function: (1) SACH, (2) single axis, (3) multiaxis, and (4) dynamic response.

SACH. The SACH foot consists of a wood or aluminum keel, and a molded foam cover (Figure 4-35). At heel strike, the heel cushion provides shock absorption, thus reducing the knee flexion moment. Different heel densities are available and need to be selected to balance the above functions.

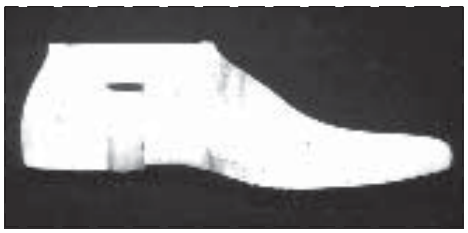


Fig. 4-35. SACH foot. A: keel, B: cushioned heel, and C: belting to prevent end of keel piercing through foam at the toebreak.

When combined with the solid ankle, the keel simulates plantar flexion at push off.[72] The length and width of the keel influences stability. Keel width contributes to mediolateral stability, with a wider keel giving more mediolateral stability, although it may make shoe fit more difficult. A longer keel provides more plantar flexion simulation, greater knee hyperextension force in late stance, and increases stability from falling forward. Too short of a keel results in less forward stability, less plantar flexion simulation, early heel off, and decreased step length.[72] The goal of keel length is to simulate push off without creating a knee hyperextension force in late stance, provide forward stability, and a toe break symmetric with the intact side. The molded rubber sole provides minimal dampening of inversion and eversion forces. The molded foam rubber foot with toes provides the foot shape and cosmesis.

The reported advantages of the SACH foot are moderate weight, durability, low maintenance, low cost, improved late stance stability, and accommodation of different heel heights and shoe styles.[82] The disadvantages cited are heel cushion deterioration over time, lack of adjustability of dorsiflexion and plantar flexion, difficulty ascending inclines, and rigid forefoot with poor shock absorption for vigorous activities.[72,83]

Those amputees requiring increased knee stability may benefit from multiaxis feet or dynamic response feet, which provide greater absorption at heelstrike.

Single-Axis Foot. The single-axis foot consists of a metal, single-axis joint, rubber plantar flexion bumper; rubber dorsiflexion wedge; wooden keel; and rubber foot (Figure 4-36). The single-axis joint allows true plantar flexion and dorsiflexion joint movements. Conceptually, this is its main difference from the SACH foot. The plantar flexion allowed at heel strike provides a smoother transition to foot flat, decreasing the knee flexion moment and increasing stance phase stability.[72]

The rubber dorsiflexion wedge limits the degree of dorsiflexion movement through the single-axis joint. The amount of limitation is dependent on the firmness of the rubber wedge. Forward stability is influenced by the dorsiflexion wedge and keel length.

The advantages are improved knee stability, adjustable plantar flexion, and less difficulty with descending inclines. The disadvantages are a tendency for noisy joints, breakdown of the rubber bumper, lack of mediolateral movement, increased maintenance, increased weight, and less cosmesis.[72,82]

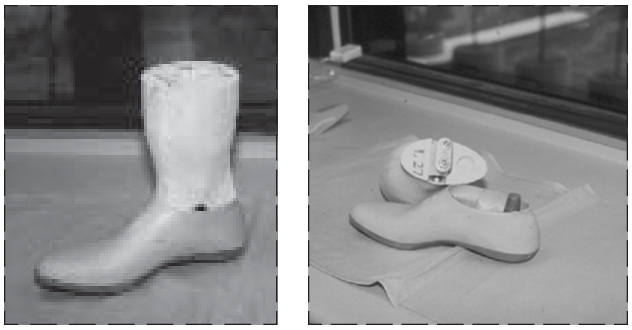


Fig. 4-36. Single-axis foot. External view (left). Components: metal, single axis joint and plantar flexion bumper demonstrated (right). The wood keel is not shown.

The foot may be indicated in amputees with weak quadriceps, which require a reduction of the knee flexion moment at heel strike, or those who have difficulty walking on inclines. The foot has more of a role in transfemoral amputees and is not frequently used in transtibial amputees.[72]

Multiaxis. The multiaxis foot is for amputees who walk on uneven terrain. This foot provides plantar flexion, dorsiflexion, inversion, and eversion joint simulation.

The *Greissinger* is a multiaxis foot consisting of a planter flexion rubber bumper, wooden keel, rubber heel, rubber rocker block, and metal rocker insert (Figure 4-37). The plantar flexion bumper and keel serve the same function as in the single-axis foot. The rubber heel serves the same purpose as in the SACH foot.

The main components that give the foot multiaxial function are the oval-shaped, rubber rocker block and the metal rocker insert. These components simulate inversion and eversion.[72] The degree of simulation is dependent on the various resistances of rubber inserts that can be provided. Shock is absorbed through the rubber heel, plantar flexion bumper, and rocker. While walking stability on uneven surfaces is increased, standing stability may be decreased by the greater movement allowed.

The advantages are multiaxis capability, improved stability on uneven terrain, adjustability, and reduced limb torque.[72,82] Disadvantages are increased maintenance, possible frequent replacement of rubber components, increased weight, reduced static mediolateral stability, and poorer cosmesis.[8,72,84] The foot may be used for individuals who often ambulate on uneven surfaces and have access to a prosthetist for maintenance.

The *multiflex ankle/foot* utilizes a ball-and-joint configuration with rubber components. The foot allows controlled plantar flexion, dorsiflexion, inversion, eversion, and transverse rotation. Advantages are multiaxis function, lightweight, and adjustability for different heel heights.[84] The disadvantage is that the foot can only be attached to an Endolite prosthesis by a certified Endolite prosthetist.

SAFE stands for stationary attachment, flexible endoskeleton. Although the SAFE foot has some dynamic response, the foot may be classified in the multiaxis category.[82] The foot consists of a flexible endoskeleton keel, a plastic bolt block, two polyester bands, a cushioned heel, and a molded foam cover (Figure 4-38). The polyester bands attach posteriorly and then run along the plantar surface. One

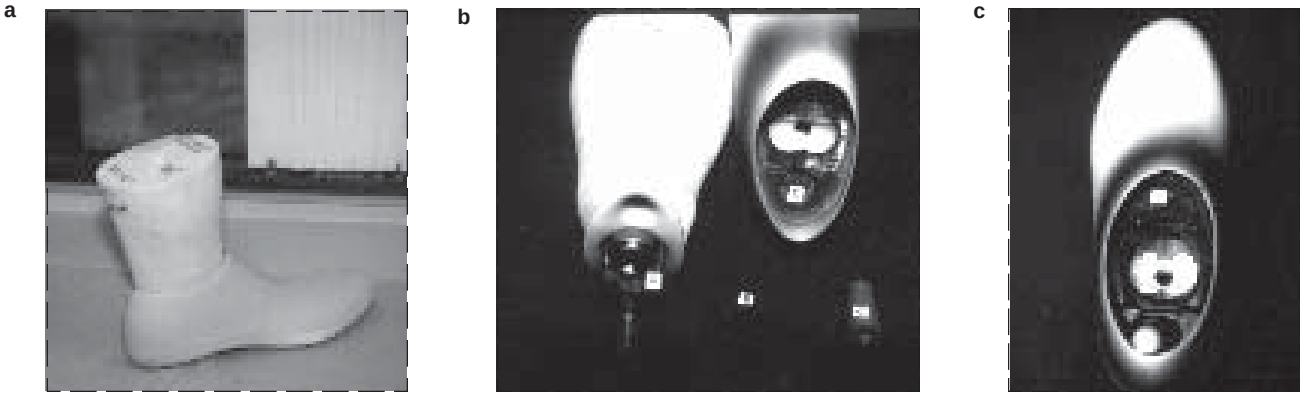


Fig. 4-37. Greissinger foot. (**a**) External view. (**b**) Components: A: metal rocker insert, B: rubber rocker block, C: plantar flexion bumper, and D: location of plantar flexion bumper in foot. (**c**) Superior view of foot with rubber rocker block inserted.

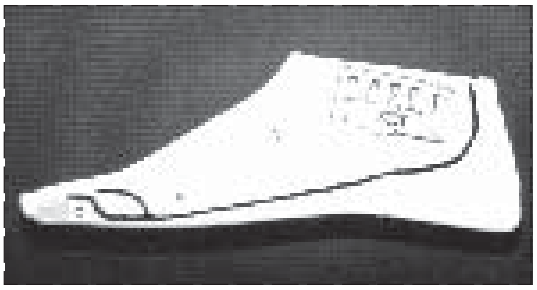


Fig. 4-38. SAFE foot, sagittal view. A: flexible endoskeleton keel, B: plantar ligament band, C: plantar fascia band, D: bolt block.

band attaches to the metatarsal head region and simulates the long plantar ligament. The other band attaches into the toe region to simulate the plantar fascia. The half dome simulates the normal foot bony arch at ground contact.[85]

The flexible endoskeleton simulates the transverse tarsal joint by absorbing torque along the foot axis created during the stance phase. Furthermore, the flexible endoskeleton simulates inversion and eversion joint motions. Using the windlass mechanism, the plantar fascia band simulates the plantar fascia at push off by converting the flexible endoskeleton to a semirigid lever arm.[85]

The advantages cited are some ability to absorb torque forces, multiaxis function, improved stability on uneven terrain, improved dynamic response compared to the SACH foot, improved ascent of inclines, and availability of different heel heights.[82–84,86] The disadvantages cited include increased weight, decreased cosmesis, and not as much dynamic response compared to some of the dynamic response feet.[82,86] Weight can be decreased with a nonwaterproof oak bolt block instead of plastic.[84] Also, cosmesis can be improved with the SAFE II foot, which has cosmetic toes.

The multiaxis *Graph-lite* is a lightweight foot, with a carbon graphite multidirectional pin and four rubber bumpers that also provide dynamic response.

Dynamic Response. The *Dynamic Response* foot, also known as an energy storing foot, is an attempt to simulate the push off phase of gait. The concept is for the foot to store energy created by dorsiflexion that occurs during stance and to release the energy at push off. Most prosthetic feet utilize a spring mechanism that deforms proportionally to the load created at stance. Hence, the foot responds dynamically, based on load. In comparison to normal push off generated by the ankle plantar flexor muscles, the dynamic response foot does not generate force, it only releases stored energy.[87] Some of the more common prosthetic feet in this category will be discussed.

The *Seattle Foot* was designed to provide push off during walking and running.[88] It consists of a leaf spring keel, cushioned heel, toe reinforcement pad, and polyurethane cosmetic molded foam cover (Figure 4-39). The leaf spring keel simulates plantar flexion at push off by compressing at foot flat and then extending at push off.[86,88,89] Additionally, the keel absorbs energy at heel strike. The length

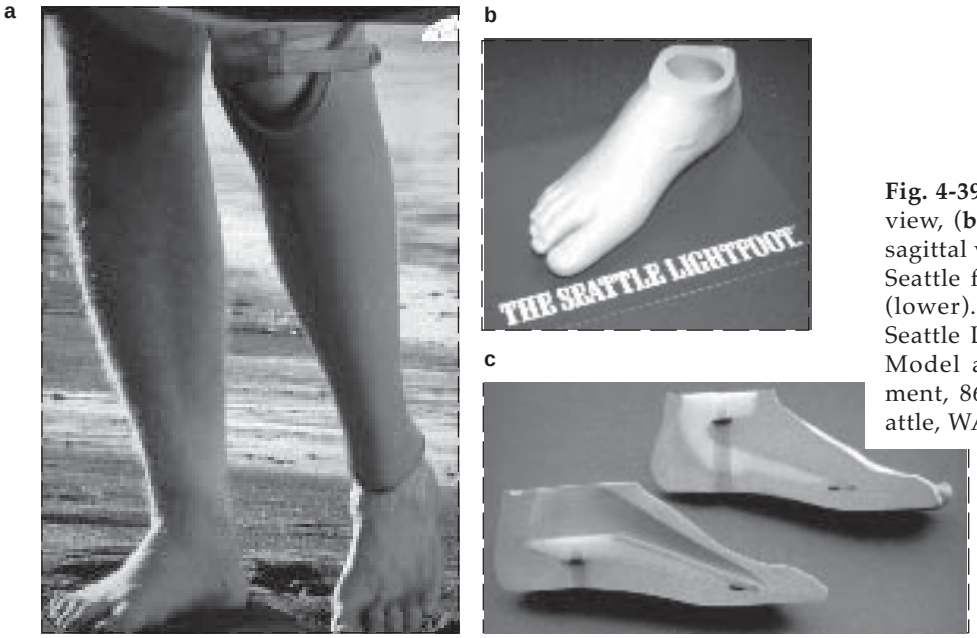


Fig. 4-39. Seattle Foot: (**a**) External view, (**b**) Seattle Lightfoot, and (**c**) sagittal view demonstrating keel of Seattle foot (upper) and Lightfoot (lower). Photograph: Courtesy of Seattle Lightfoot and Seattle Foot, Model and Instrument Development, 861 Poplar Place South, Seattle, WA 98144.

and width affect mediolateral and anterior-posterior stability, as described with other keels.

The cushioned heel serves a similar function as with the SACH. The molded foam cover was developed from male and female molds for excellent cosmesis.[88]

The advantages cited are enhanced performance with running and fast walking, improved ascent of stairs and inclines, completely waterproof, better cosmesis, availability of a wide range of sizes and keel stiffnesses, and smooth roll-over.[82,88,89] The disadvantages cited are that the wide foot may be difficult to fit into narrow shoes, the keel may break during activity, increased weight, and foam breakdown with barefoot use. Keel breakage is felt to be less of a problem if manufacturer recommendations for amputee weight and activity level are followed along with use of a heavier keel for more active users.[82,88] The cosmesis of the foot is felt to have led to barefoot use and resultant foam breakdown.[88] This has been addressed with a reinforced toe pad and the recommendation to run only while wearing shoes.

The *Seattle Lightfoot* has addressed some of the disadvantages cited. The Seattle Lightfoot is approximately half the weight with similar performance of the Seattle Foot. Additionally, the Lightfoot is slimmer with a decreased medial arch prominence, allowing easier shoe fit. The Lightfoot can be used with the Seattle Ankle with enhanced plantar flexion and dorsiflexion abilities.[84] Indications for the Seattle Foot or Lightfoot may include an amputee who desires enhanced activity performance, improved cosmesis, or waterproofness.

With the *Carbon Copy II* (Figure 4-40), the main design principle is the utilization of two carbon deflection plates to deform with loading and release at push off. This foot consists of the two carbon deflection plates, Kevlar sock, cushioned heel, thermoplastic nylon-Kevlar keel, and urethane foam molded cosmetic cover.[90]

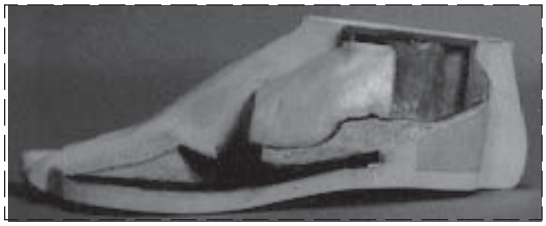


Fig. 4-40. Carbon Copy II, sagittal view demonstrating the two carbon deflection plates. Photograph: Courtesy of Carbon Copy II and III, Ohio Willow Wood Company, 15441 Scioto-Darby Road, Sterling, OH 43143.

The primary deflection plate extends from the heel to the toe region. Arbogast and Arbogast[90] postulate that plantar flexion contraction at push off is simulated by the plate storing energy obtained after midstance and releasing the energy at the last few degrees of toe off. The extension of the plate into the toe region provides a stiffer forefoot, resisting the drop off at push off seen with conventional keels.

The secondary deflection plate is shorter and deflects at higher loads, such as in fast walking and running, or when descending stairs. The plate also provides stability from falling forward by resistance to bending at midstance until the primary deflection plate bends.[90]

A Kevlar sock incorporates the plates to prevent knifing of the plates through the polyurethane mold.[90] The cushioned heel is similar to the SACH heel.[86] The thermoplastic keel was successfully designed to prevent bolt breakage, which is often seen with deformation of wooden keels. Additionally, the thermoplastic keel is reported to dampen vibration at impact, assisting in shock absorption.[90] The foot uses live foot molds for cosmesis and is flat on the bottom to assist with mediolateral stability.

The advantages are graded and enhanced performance, lighter weight, better cosmesis, and mediolateral stability.[86,90] The disadvantage is the width, which causes a difficult shoe fit.[82] Also, the investigations of Barth and associates[83] suggest dysvascular amputees should use caution with this device secondary to increased loading of the contralateral leg. The foot may be used for those desiring enhanced performance.

The *Carbon Copy III* system consists of a carbon fiber and Kevlar shank-ankle-foot unit (Figure 4-41). The foot contains three deflection plates instead of two. The plates come in different levels of stiffness for different amputee needs. The shank absorbs rotational forces and can be aligned by heating and deforming it. The system is light and modular.[84]

The main design difference in the *Flex Foot* is the incorporation of the shank and foot into one carbon graphite unit (Figure 4-42). The foot consists of two broad carbon graphite leaves and a soft cover.

The primary leaf extends from the inferior aspect of the socket to the toe region. The leaf provides dynamic compression in midstance with rebound plantar flexion at push off.[87] The leaf allows dynamic dorsiflexion in late stance. The posterior leaf at heel strike provides shock absorption, propels the prosthesis forward, and simulates plantar flexion joint motion.[86]

Fig. 4-41. Carbon Copy III, sagittal view showing the three deflection plates and Kevlar shank. Photograph: Courtesy of Carbon Copy II and III, Ohio Willow Wood Company, 15441 Scioto-Darby Road, Sterling, OH 43143.

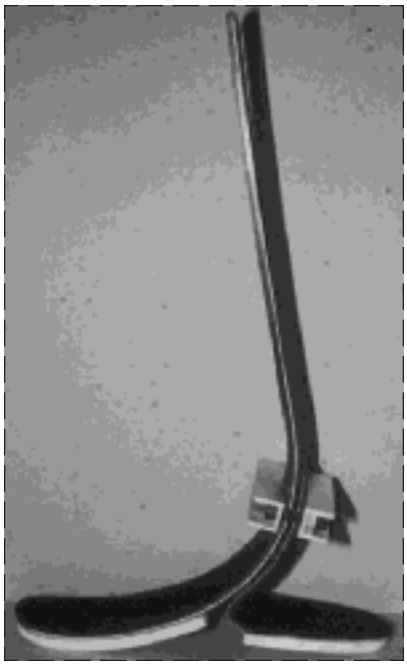
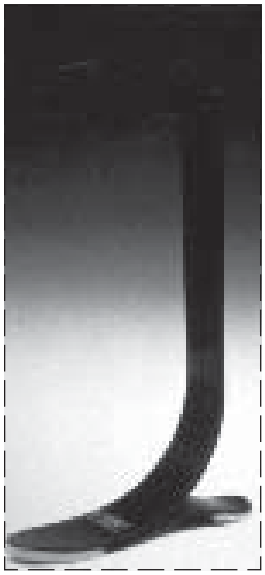


Fig. 4-42. Flex Foot. Photograph: Courtesy of Flex Foot Inc., 27071 Cabot Road, Suite 606, Laguna Hills, CA 92653.

Fig. 4-43. Flex Foot, with cosmetic foot cover. Photograph: Courtesy of Flex Foot Inc., 27071 Cabot Road, Suite 606, Laguna Hills, CA 92653.

The advantages cited are its high level of enhanced performance, lighter weight, smoother running, improved jumping, improved ambulation on inclines and stairs, mediolateral stability, availability of junior sizes for adolescents, and adjustability of the leaves.[82–84,86,91] The disadvantages are difficult alignment, cost, and minimal inversion and eversion.[82,86] Cosmesis has been improved with a cosmetic foot cover (Figure 4-43). The foot may be indicated in those desiring a high level of enhanced performance, or involvement in vigorous sporting events, such as running and jumping.[82,86,88]

Variants of Flex Foot are the Modular Flex Foot, Split Toe Flex Foot, Flex Walk, Flex Foot Symes, Flex Sprint, Vari-Flex, Air-Flex, and Vertical Shock Pylon (Figures 4-44 through 4-51). The Modular variant makes alignment, fabrication, and changing of sockets easier by use of bolt attachment instead of bonding.[79,82] This is an advantage for new amputees and adolescents who may need to replace sockets. The Split Toe model allows some inversion and eversion. The Flex Walk does not include the carbon graphite shank, which allows fit onto long transtibial amputees and may be of more benefit for less active amputees.[84] The Flex Foot Syme's is for Syme's amputees. The Flex Sprint is modified with plantar-flexed spring for sprinting. The Vari-



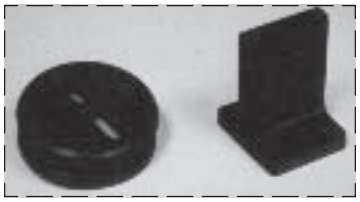


Fig. 4-44. Modular Flex Foot III. Foot (left) and T-system for modular attachment of foot to socket (right). Photograph: Courtesy of Flex Foot Inc., 27071 Cabot Road, Suite 606, Laguna Hills, CA 92653

Fig. 4-45. Split Toe Flex Foot. Photograph: Courtesy of Flex Foot Inc., 27071 Cabot Road, Suite 606, Laguna Hills, CA 92653

Fig. 4-46. Flex Walk II. Photograph: Courtesy of Flex Foot Inc., 27071 Cabot Road, Suite 606, Laguna Hills, CA 92653.

Fig. 4-47. Flex Low Profile Syme's. Photograph: Courtesy of Flex Foot Inc., 27071 Cabot Road, Suite 606, Laguna Hills, CA 92653.

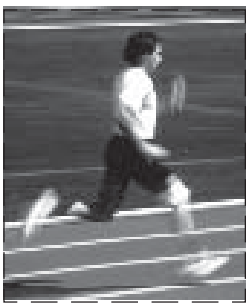


Fig. 4-48. Flex Sprint. Flex Sprint (left) and individual sprinting with Flex Sprint (right). Photograph: Courtesy of Flex Foot Inc., 27071 Cabot Road, Suite 606, Laguna Hills, CA 92653

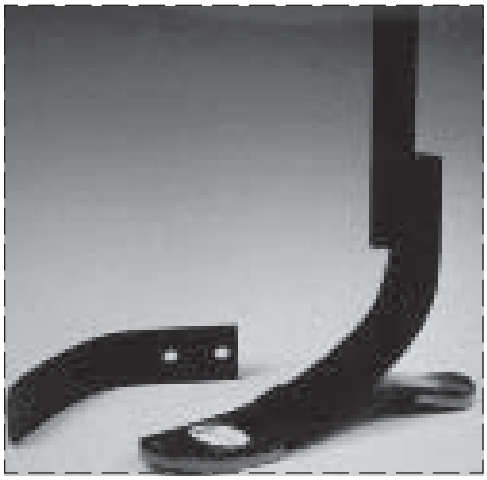


Fig. 4-49. Vari-Flex Foot. Addition of plate allows variable resistance. Photograph: Courtesy of Flex Foot Inc., 27071 Cabot Road, Suite 606, Laguna Hills, CA 92653

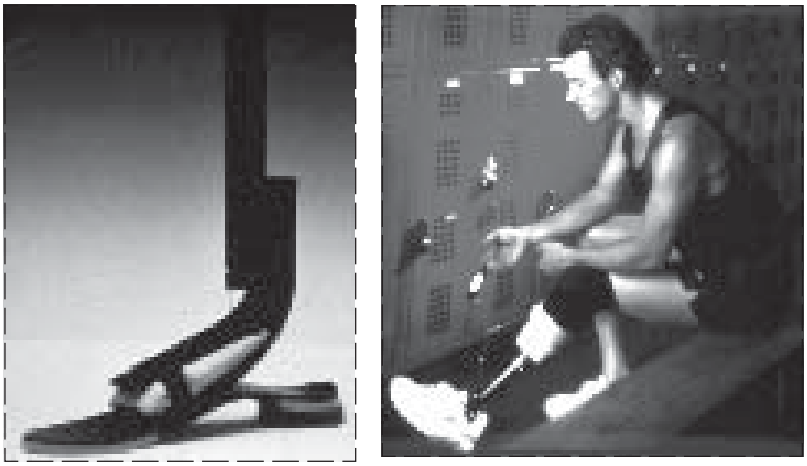


Fig. 4-50 Air-Flex Foot. Air-Flex (right) and Pumping-up the Air-Flex to increase resistance (far right). Photograph: Courtesy of Flex Foot Inc., 27071 Cabot Road, Suite 606, Laguna Hills, CA 92653.

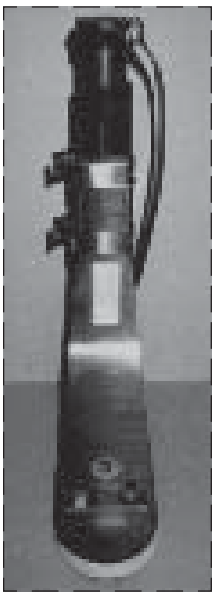


Fig. 4-51. Flex Vertical Shock Pylon. Photograph: Courtesy of Flex Foot Inc., 27071 Cabot Road, Suite 606, Laguna Hills, CA 92653.

Fig. 4-52. Springlite with cosmetic foot cover. Photograph: Courtesy of Springlite, 97-E Chinook Lane, Steilacoom, WA 98388.

Flex and Air-Flex are two models that allow the amputee to vary resistance in the foot and thereby meet performance or growth needs. The Vari-Flex resistance is changed by the prosthetist to match a change in activity level or growth in an amputee. This makes the Vari-Flex useful for the growing child. The Air-Flex allows the amputee to vary resistance by filling an air bladder in the foot during more demanding activities. The Vertical Shock Pylon is under development and will provide increased shock absorption.

Springlite II is similar to the Flex Foot concept that utilizes a carbon fiber and epoxy composite shank, heel, and foot. The main differences are that the Springlite II has one-piece construction, no foot clamp, and a removable rubber heel plug (Figure 4-52).[84] Also, its cosmetic foot cover, improved resistance to breakage, and cosmesis are benefits of the one-piece construction with no foot clamp.[84,92] The removable rubber plug, available in different densities, allows the amputee to select heel resistance according to activity level. The rubber plug varies the heel lever arm, affecting eccentric dorsiflexion simulation. Clamp fitting of shank to socket allows easier alignment and changing of sockets.[92] The stiffness of the foot is customized to the amputee's weight and activity level.

The advantages cited are enhanced dynamic response, cosmesis, availability of junior sizes, ability to change sockets and to tailor heel resistance for activity level.[84,92] Disadvantages are the inability to fit long transtibial amputees, and the higher

cost because of custom fabrication and difficult alignment.[84] The foot may be indicated for those amputees desiring enhanced performance and the ability to select heel resistances, or for those who require socket changes.

The *Sabolich* foot consist of a Delrin longitudinal arch. It absorbs energy at heel strike with further absorption at midstance by the arch and release at push off.[84]

With the *STEN* (STored ENergy) Foot, the main design feature is the use of an articulated keel. The foot consists of an articulated keel, rubber block articulations, dual plantar belting, cushioned heel, polyurethane foam covering, and rubber sole (Figure 4-53). The keel is three wood blocks separated by rubber blocks with plantar fabric belting connections. The articulations correspond with the metatarsophalangeal and tarsometatarsal joint articulations.[82] The articulated keel allows inversion, eversion, smoother roll-over compared to a SACH foot, and energy dissipation with compression, but it has little energy storage and release.[82,84,86] The heel, foam cover, and rubber sole are similar to the SACH and serve the same function.[82,86]

Advantages include smooth roll-over, mediolateral stability, and a wide range of shoe sizes and heel heights.[82,84] Disadvantages are increased weight and decreased anterior stability for higher level amputees. The foot may be indicated for those desiring a smoother roll-over compared to what the SACH provides.[82] The STEN does not have as much inversion and eversion capabilities as the SAFE foot, but it is lighter.[86]

The *Otto Bock 1D10 Dynamic Foot* consists of a wooden keel extending to the midfoot with a flexible plastic surrounding the keel and extending into the toe region, a cushioned heel, and a polyurethane foam cover with a split first web space.[84,86] The foot provides good shock absorption, but with minimal dynamic response.[84] Its advantages are that it is lightweight, has good shock absorption, provides accommodation to uneven terrain, and gives a smooth roll-over.[84,86] A disadvantage is its minimal dynamic response.[84]

Summary. In deciding which type of prosthetic foot to use, the functional properties of the foot should be matched with the amputee's medical and functional status, and goals. For new amputees undergoing training in the immediate postoperative period, a SACH foot may be used. A SACH foot may also be a good choice in less active amputees involved in mainly level surface household or community ambulation.

Amputees who ambulate on uneven surfaces may benefit from multiaxis feet, such as the Greissenger or Multiflex foot. However, if moving parts and maintenance are concerns, a multiaxis foot without moving parts (such as the SAFE, SAFE II, or Otto Bock 1D10 Dynamic Foot) may be good choices. If multiaxis and dynamic response is needed, the Flex Foot Split Toe variant or Graph-Lite may be good choices.

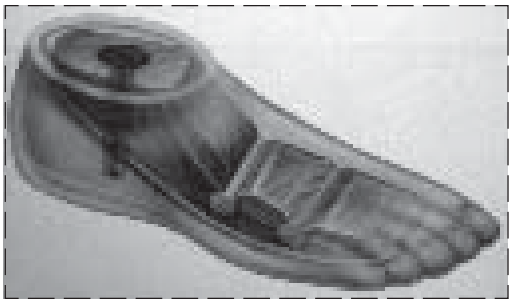


Fig. 4-53. STEN Foot. Photograph: Courtesy of STEN Foot, Kingsley Manufacturing Company, Costa Mesa, CA.

For active amputees who walk at varying velocities or are involved in athletics, a foot with dynamic response may be beneficial. Good choices include the Seattle Foot or Lightfoot, Carbon Copy II or III, Flex Foot, and Springlite II. For a newer amputee or a growing adolescent, modular components are beneficial for reusing the foot on remade sockets.

As newer prosthetic feet increase choices, it is important to analyze the biomechanical aspects of the foot or ankle/foot complex to meet the functional and cosmetic needs of the amputee. After this, the most important test is amputee satisfaction in maximizing quality of life.

Computer Aided Design/Computer Aided Manufacturing

Computer aided design (CAD) and computer aided manufacturing (CAM) utilize surface topography measurements of the residual limb, which are entered into a computer to generate a quantitative positive mold for socket manufacturing. Computer aided socket design may be beneficial for (*a*) quantitating successful socket designs, (*b*) ease of replicating sockets for amputees needing socket replacement, and (*c*) reducing the time needed to make a socket.[93–95]

To understand the differences between CAD/CAM and traditional prosthetic socket fabrication, the traditional method will be reviewed. The traditional process can be broken down into a series of steps as described by Staats and Lundt[73]: First, the

residual limb is measured anterioposteriorly, mediolaterally, and circumferentially; then a plaster cast impression is made of the residual limb. The cast is removed and placed in a gimbal-ring casting stand for alignment. Alginate is placed into the cast and to give an intimate fit, the amputee puts on the cast and bears weight while in the casting stand. The residual limb is then removed and the cast is filled with plaster of Paris. A metal pipe is placed in the middle with which to grasp the inner cast. The outer cast is removed from the plaster of Paris positive mold and modifications are done on the positive mold as needed. A clear check socket is made over the positive mold and fitted to the amputee. Any modifications are noted and added to the plaster positive mold. The process of check sockets and plaster positive mold modifications are repeated until correct fit and alignment are achieved.

By comparison, computer aided socket design (CASD) consists of entering the topographical measurements of the surface of the residual limb into a computer, which then generates and displays the image developed from those measurements and existing templates of residual limb shapes. The positive mold image can be modified based on the computer software capabilities. This quantitative information of the completed positive mold is sent electronically to a computer milling system that generates a polyurethane positive mold. Computer aided manufacture of the socket is done by a vacuum forming machine that makes a thermoplastic socket over the positive mold. The polyurethane mold is broken out of the socket. The check socket is then tried on by the amputee and any socket modifications are made via the computer. From these modifications, another check socket is fabricated. The process is repeated until proper fit and alignment are achieved.

The way in which surface topography is measured varies. One method uses manually obtained measurements of the anteroposterior, mediolateral, and circumferential dimensions of the residual limb, which are then entered into the computer.[95,96] These measurements, along with preexisting templates of nine socket shapes (small, medium, and large of cylindrical, conical, and bulbous shapes) are combined to create the positive mold computer image.[96]

Another method uses multiple cameras and laser beam scanning of the residual limb with the measurements sent directly to a computer.[93] From these data, a computer generated image of the residual limb is obtained for creating the positive mold. Another method uses the electromechanical digitization of plaster cast molds of the residual limb.[97] These data are combined with preexisting templates of socket designs to create the positive mold computer image. Other methods include computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound measurements.

Clinical trials[96,97] comparing below-knee prosthetic sockets created by traditional methods and those created with CAD/CAM have been conducted. One study evaluated the CANFIT system in 48 below-knee amputees.[96] The CANFIT system utilizes hand measurements of the residual limb along with preexisting templates of socket shapes to create the computer generated positive mold image. Utilizing two check sockets for the traditional method and CANFIT method, only 21% of the amputees preferred the CANFIT socket. When five check sockets for the CANFIT system were compared to two check sockets for the traditional method, 54% preferred the CANFIT method. Topper and Fernie[96] concluded that in its current form, the CANFIT system was useful only in limited cases, with a more accurate residual limb measurement method needed and more trials to produce an adequate fit. They also concluded that CANFIT was more time consuming, and not cost effective; however, with resolution of these limitations, the system could be beneficial.[96]

In a multicenter study[97] utilizing prototype equipment from University College of London Bioengineering Center (UCL-BC), computer-made sockets were compared to traditional socket fabrication. The modified UCL-BC system used electromechanical digitization of plaster casts from the residual limb along a with preexisting template of socket design to create the positive mold computer image. The system provided for residual limb lengths between 8.0 and 38.5 cm. In regards to overall satisfaction, 94% of computer made socket users rated the socket from very good to fair. In comparison, 80% of traditional users rated their sockets in the very good to fair range. However, more subjects rated the traditional method highest in terms of comfort, fit, and function. Also, the computer generated method had difficulty in fabricating sockets for those users who were highly active or had short limbs, and required significantly more time to do so. Based on the study, a number of recommendations were made for further developing CAD/CAM into a useful prosthetic tool. Overall, CAD/CAM for below-knee prosthetic socket fabrication is in the developmental stages, but it holds promise as a prosthetic tool of the future.

THROUGH- AND ABOVE-KNEE AMPUTATIONS

Amputation does not signify a surgical failure and should never be viewed as such, but rather as the means to return the patient to a functional level. The value of approaching amputation positively and with a reconstructive philosophy cannot be overemphasized. As discussed earlier in the Partial Foot and Syme's Amputations section, the decision to amputate is an emotional process for the surgeon, the patient, and the family, and the rehabilitation team should stand ready to respond and assist them. Once healing has occurred, it is critical to avoid scar tissue adhesions.

General Surgical Techniques and Definitions

Above-knee amputation includes amputations through the knee (knee disarticulation) and above the knee (transfemoral). These two amputation levels require the same type of rehabilitation interventions and similar prosthetic components. In the general population, the transfemoral level of amputation for dysvascular disease is the second most common level for lower limb amputees. Advantages of knee disarticulation include the surgical technique, which lessens the operating room time and blood loss and provides direct end bearing characteristics for the residual limb and improved self suspension with a decrease in socket length. It also maintains the integrity of the thigh musculature, which results in better biomechanics for walking. One major disadvantage to be considered, mostly for the young female, is the created thigh/shank length disproportion, which results in a marginal cosmetic appearance that, in the long run, may outweigh the above-mentioned advantages. For the bilateral transfemoral amputee, the knee disarticulation is a more desirable level when possible.

In surgical techniques, soft tissue handling is especially critical to wound healing and functional outcome. The tissues are often dysvascular or traumatized, and the risk of wound failure is high. Flaps should be kept thick, and unnecessary dissection between the skin, subcutaneous, fascial, and muscle planes should be avoided. All bone edges should be rounded, and prominences beveled for optimal prosthetic use.

When the skeletal attachments are divided during amputation, muscle loses its contractile function. Stabilization of the distal insertion of muscle can improve residual limb function. Myodesis is the direct suturing of muscle or tendon to bone. This technique is most effective in stabilization of the strong muscles, which are needed to counteract strong antagonistic muscle forces, as found in the transfemoral and knee disarticulation levels. Myoplasty involves suturing muscles to periosteum, or muscle to muscle over the end of the bone. Myoplasty does not provide as secure a distal stabilization of the muscle as does myodesis. A mobile sling of muscle over the distal end of the bone may result in a painful bursa and should be prevented. The excessive shortening of hip flexor musculature at the time of the amputation is not uncommon due to the position of the limb during surgery; if not accounted for, this may result in a hip flexion contracture.

All transected nerves will form neuromas. Careful retraction and clean transection of a nerve to allow the cut end to retract into the soft tissues, away from the scar and prosthetic pressure points, should always be attempted. STSGs are generally discouraged except as a means to save essential residual limb length. Skin grafts do best with adequate soft tissue support, and are least durable when closely adherent to bone.

Trauma. The absolute indication for amputation in trauma is an ischemic limb with unreconstructible vascular injury. As vascular reconstruction techniques improved, many limbs were initially salvaged, only to be amputated after multiple surgical procedures, and substantial investments of time, money, and emotional energy. Massively crushed muscle and ischemic tissue release myoglobin and cell toxins, which can lead to renal failure, adult respiratory distress syndrome, and even death. Recent studies show the value of early amputation (when salvage is unlikely to result in a functional limb), not only in saving lives, but in preventing the emotional, marital, financial, and addictive disasters that can follow unwise and desperate attempts at limb salvage.[98–100]

Salvage should be based on providing an extremity that can tolerate weight bearing, have enough sensation to provide protective feedback, and have a durable soft tissue cover. A lower limb functions poorly without sensation, and unless the limb can tolerate full weight bearing, is relatively pain free, and has durable skin and soft tissue coverage that does not break down whenever walking is attempted, it will often function worse than a modern prosthetic replacement. Recently, several scales for grading mangled lower limbs have been developed and should serve as guidelines to help the surgeon realize the gravity of the injury and the

subsequent risks of salvage (see Management of New Amputee section).[98–100]

For patients with multiple injuries and elderly individuals, salvage of a mangled limb, even though technically possible, may be life threatening; these patients may be best served by early amputation. This is a truly difficult but extremely important decision.

Tissue injury from cold exposure can involve both direct freezing of tissue, and a related vascular impairment from endothelial vessel injury and increased sympathetic tone. If the skin is wet, or directly exposed to the wind, cold injury can result even in above-freezing temperatures. The immediate treatment involves restoring the core body temperature, and then rewarming the injured body part in a 40°C to 44°C water bath over a 20 to 30 minute time period. Rewarming can be painful, and often requires strong analgesia. Care should be taken to maintain skin integrity.

It may not be unusual to wait several months before definitive surgery. A zone of dry gangrene develops distally, and a zone of intermediate tissue injury forms just proximal to this. Even at the time of clear demarcation, the tissue just proximal to the gangrenous zone continues to heal from the cold insult, and although the outward appearance is often pink and healthy, this tissue is not normal. Delaying amputation surgery, even after clear demarcation, can improve the chance of primary wound healing. In spite of having mummified tissue, infection is rare if the tissue is kept clean and dry.

The Rehabilitation Program

The amputee rehabilitation program should be designed to cover the wide spectrum of care beginning with preamputation and continuing to the patient's reintegration into the community and long-term follow-up.

Preamputation Counseling. In the case of war related amputations, it is not often possible to address prosthetic issues until the soldier arrives at a medical center. At the earliest opportunity, it is essential to develop a communication flow between the patient, the family, and the physician regarding the need for amputation and the expected surgical outcome. Communication with the physiatrist, therapist, and other members of the team should be facilitated. At this point it is appropriate to have initial discussions about phantom limb sensation and expected functional outcomes. Prosthetic devices and their fitting should be discussed, and when possible, an arranged demonstration of such devices by a trained volunteer with a similar level of amputation should be part of the discussion. If a rigid dressing is being considered, an explanation should be given for its use, and the pros and cons should be discussed. Family involvement throughout this process should be encouraged. For any level of amputation the program should include strengthening exercises for the thighs, hips, and upper limb musculature and stretching of hip flexors and adductors, when possible.

Amputation Surgery

Selection of a transfemoral level amputation over a transtibial amputation presents a number of important dilemmas in rehabilitation. The lack of an anatomical knee joint will require increased energy consumption during ambulation[45] and increased effort and cost for prosthetic restoration. The value of preserving the knee is so important that with current surgical techniques, it is advisable to attempt a transtibial amputation even when there may be a risk of requiring revision to a higher level amputation at a later time. Transtibial amputees will be more likely to accept and utilize a prosthesis than will transfemoral amputees. In most instances, transfemoral amputations do heal.

Knee Disarticulation. For the critically ill patient, knee disarticulation is a less traumatic procedure than other amputation levels. For patients who are expected to walk, the advantages over a transfemoral amputation include improved socket suspension by contouring above the femoral condyles, the added strength and prosthetic control of a longer lever arm, the retained muscle balance of the thigh, and most important, the end bearing potential for direct weight transfer to the prosthesis, thereby obviating the need of ischial weight bearing transmission. Additionally, there is usually a lower surgical risk because no bone and muscle transection is required and minimal blood loss results. Until recently, clinicians have discouraged the use of this level of amputation, finding it difficult to fit with a satisfactory, functional, and cosmetic prosthetic substitute. However, the past few years have shown significant improvements in knee disarticulation prosthetics, including new socket fabrication materials and techniques that allow a less bulky interface to be manufactured. Improvement has also been achieved because of refined polycentric (four bar linkage) and multiaxial knee units with hydraulic or pneumatic damping mechanisms that can fold under the socket and improve the biomechanics of walking and the appearance of the pros-

thesis when sitting. Disarticulation through the knee joint is indicated in ambulatory patients when a transtibial amputation is not possible, but when suitable soft tissue is present for a knee disarticulation. This most commonly occurs in trauma. In dysvascular patients, the blood supply to the soft tissues is such that most patients who would be capable of recovering from a knee disarticulation will also be able to heal from a short transtibial amputation. Knee disarticulation is indicated in dysvascular patients who are nonambulatory, especially if knee flexion con-tractures are present. Sagittal flaps appear to improve healing compared to traditional anterior-posterior flaps.[34] The patella is retained and the patellar tendon is sutured to the remaining cruciate ligaments to stabilize the quadriceps complex. The hamstring tendons should also be stabilized to the crutiate ligaments. Although many techniques have been described to trim the condyles of the femur, radical trimming should be avoided because this can decrease some of the suspension advantages of the knee disarticulation.[101]

Transfemoral Amputation. The above-knee amputation is usually performed with equal anterior and posterior fish-mouth flaps. Since increased prosthetic control and function are directly proportional to length of the residual limb, atypical flaps can and should be used to save all possible femoral length in cases of trauma. Muscle stabilization is more important in transfemoral amputation than in any other major limb amputation.[102]

The major deforming force is into abduction and flexion. Myodesis of the adductor muscles through drill holes in the femur can counteract the abductors, improve prosthetic control, and prevent the adductor tissue roll in the groin. Without muscle stabilization, the femur commonly migrates laterally through the soft tissues, much like a stick would migrate in a bowl of gelatin. Newer transfemoral socket designs attempt to better control the position of the femur, but they are not as effective as muscle stabilization.[102] Careful balance of the hip flexors and extensors during the surgical procedure should be achieved to avoid promoting contractures of this joint.

In the traumatic transfemoral amputation it is not uncommon to find a pelvic or proximal femoral fracture, which may, temporarily or in a permanent way, interfere with the optimal gait pattern because of changes in the normal biomechanics of the hip and pelvis and their musculature.

Hip Disarticulation and Hemipelvectomy. The traditional racquet shaped hip disarticulation incision with an anterior apex is used in dysvascular patients and, when possible, in trauma cases. In tumor surgery, creative flaps based on the uninvolved anatomic compartments must be designed.[103] Prosthetic replacement can be successful in healthy, young patients who require hip disarticulation because of trauma or malignancy, but its success is limited in elderly dysvascular patients. Because residual limb capabilities to propel an artificial limb are lost, compensatory trunk movements are required at an energy cost that is significantly higher than crutch walking with a swing-through gait (see Table 4-1).[43]

Hemipelvectomy is performed infrequently, but may be required for trauma or malignancy involving the pelvis. Prosthetic use is extremely rare after this procedure, as the body weight must be transferred from the sacrum and thorax to the prosthetic socket. To maintain a leveled pelvis, special considerations for seating, such as a prosthetic bucket, may be required.

Acute Postamputation Rehabilitation

Pain control, maintenance of range of motion and strength, and promotion of wound healing are the goals of this stage, which begins with the surgical closure of the wound and culminates with healing after the sutures are removed. In war injured amputees, who by necessity have open wounds, maintenance of skin traction during transport from the battlefield to the medical center and until the wound is closed (operatively or by secondary intention) is of paramount importance. Figure 4-54 shows a skin traction device that allows the amputee crutch mobility.

Pain control and residual limb maturation should be aggressively pursued. The IPORD or soft elastic bandage and subsequent pneumatic compression are indicated for edema control. For selected amputees, the IPOP can also be used. An increasingly popular method of wound protection for early shaping and shrinking is the removable rigid dressing as proposed by Burgess and associates.[34] This dressing is easily changed to accommodate stump shrinkage until the residual limb stabilizes in size and is ready for the first prosthetic casting. In addition, this dressing protects the residual limb while the patient practices ambulation training with gait aids and other essential mobility skills. IPORDs are more difficult for the transfemoral amputee to apply and keep positioned than for those patients who experienced more distal amputations. IPORD techniques

Fig. 4-54. Skin traction device. 1: plaster attachment to the body, 2: outrigger, and 3: stockinette attached to skin of residual limb placed under tension and secured to outrigger.

do offer the advantages of early rehabilitation and control of edema and pain, and are preferred if the patient has no history of chronic arterial compromise and the expertise to apply it is available. Soft compressive dressings alone are used in many centers.[21] This dressing should be extended proximal to the hip and around the pelvis to prevent the development of an adductor soft tissue roll, and to improve suspension of the dressing. Proper postoperative positioning and rehabilitation are essential to prevent hip flexion and abduction contractures.

Acute Pain Management

Initial pain control can best be achieved with a PCA system followed by the use of scheduled oral analgesia. This is also a time for the patient to adapt emotionally to the new body image and learn to function without the prosthesis, which is essential for later life experiences when there are times that the prosthesis will not be worn. A skin desensitization program that includes massage, soft tissue and scar mobilization, and lubrication is recommended for the patient who is managed with a removable, soft or elastic dressing.

When the patient is medically stable, early mobilization, general endurance and strengthening exercise (with emphasis to the gluteus medius and maximus and the avoidance of joint contractures), and improvement in sitting and standing balance are initiated. Strengthening of upper limb musculature is essential for wheelchair propulsion, transfers, walker, and crutch ambulation and are also aggressively pursued. It is important physically to emphasize the strength and function of the remaining limbs. For this purpose the Universal Below-the-Knee Bicycle Attachment was developed.[104] This device permits early endurance exercise with controlled weight bearing, using a stationary bicycle. Whenever possible, patients should be placed in a cardiovascular conditioning program before the amputation. At this time emotional counseling to the patient and the family, with special attention to the significant other and children, should be implemented.

Psychosocial evaluation of the patient and family should be initiated to assess and manage the existence of depression and anxiety or both. It is important during this phase to assure and promote patient participation in the decision-making process to encourage independence. This is a time for introspection and reassessment of goals in life. This process can result in the individual's taking a more mature approach toward life plans and pursuit of goals. Occasionally, however, a patient may become so emotionally disturbed by the limb loss that there is failure to cope. This will affect the rehabilitation outcome.[105]

Postoperative Care

Postoperative edema is common following amputation; if soft dressings are used, they should be combined with stump wrapping to control edema, especially if the patient is a prosthetic candidate. The ideal shape of a residual limb is cylindrical, not conical. The major complication from stump wrapping results from applying the elastic wrap too tightly at the proximal end, which actually causes congestion and worsening edema, and results in a dumbbell shaped residual limb. A figure-8 wrapping technique should be reapplied every 4 to 6 hours (see Figure 4-25).

The preferred treatment approach is to use an IPORD to control postoperative edema, protect the limb from trauma, decrease postoperative pain, desensitize the limb, and allow early mobilization and rehabilitation. In selected patients, usually

young traumatic amputees where the amputation was performed above the zone of injury, immediate, limited postoperative weight bearing can be safely initiated. An IPORD and immediate IPOP need to be applied carefully, but their application is easily learned and well within the scope of interested physicians and prosthetists.[21,34]

Phantom Limb. As discussed earlier, phantom limb sensation is the feeling that all or part of the amputated limb is still present. This sensation is felt by nearly all acquired amputees, but is not always bothersome.[35] The traditional explanation for phantom limb sensation and its associated pain is that the remaining nerves in the amputated limb continue to generate impulses that flow through the spinal cord and the thalamus to the somatosensory areas of the cerebral cortex. Another theory suggests that the phantom arises from excessive, spontaneous firing of spinal cord neurons that have lost their normal sensory input from the missing body part, while yet another suggests that the phantoms are caused by changes in the flow of signals through the neuromatrix in the brain.[35] (For more detail, see the Medical and Surgical Complications section below.) The management of phantom limb sensation should include prosthetic socket revisions, desensitization techniques, transcutaneous electrical nerve stimulation (TENS), neuropharmacological intervention, and the voluntary control of the phantom limb. For severe cases, nerve blocks, steroid injections, and epidural blocks may be useful. Nonsurgical interventions are far more successful than surgical ones. Clearly, the source of phantom limbs is more complex than any of the theories presented here would suggest, and treatment is complex. Important issues to discuss with the patient are normal phantom sensation; phantom pain; and the relationship between tension, anxiety, stress, and pain perception.

Joint Contractures

Joint contractures usually occur between the time of amputation and prosthetic fitting. In the transfemoral amputee, the deforming forces are due to flexion and abduction. Adductor and hamstring stabilization during surgery can oppose the deforming forces. As with BKA, after surgery, patients should avoid propping the leg up on a pillow and prolonged seating. The patients should also be started on active- and passive-motion exercises early, including lying prone to stretch the hip. Strengthening of the hip extensors and abductors should be encouraged. Efforts should be directed at prevention with aggressive rehabilitation, beginning soon after surgery.

Preprosthetic Rehabilitation

In the last two decades, with the advent of specialized treatment teams and new prosthetic devices, the outlook for the lower limb amputee has improved. The patient who has undergone a lower limb amputation may quickly become deconditioned and most likely depressed. A preprosthetic rehabilitation program must be initiated as soon as possible. Pain control and residual limb maturation should be continued during this phase.

Preparatory Prosthetic Fitting. The use of a preparatory or temporary prosthesis should be implemented at this stage. The prosthetic device is intended to serve as a short-term, gait training tool. In most instances the prosthesis is simplistic in its components, but when possible, it should permit the same function as that of the expected permanent prosthesis so that retraining can be minimized. The device should allow for easy adjustability of the socket fit and alignment. The alignment will change as training and increased weight bearing progress and as patient confidence increases.

The preparatory prosthesis should promote residual limb maturation and desensitization and allow the patient to build up wearing tolerance. The patient must develop the understanding of normal residual limb volume fluctuations and how to accommodate for them.

At times, delayed wound healing or other soft tissue or bone injuries will prevent weight bearing on the residual limb, complicating the rehabilitation process. Because of architectural limitations, ambulation may be a requirement for a patient to return home or, because this function is viewed by many patients as the primary goal in their rehabilitation, it may be necessary to allow bipedal ambulation with a modified preparatory prosthesis. It is possible to use a bypass prosthesis, which transfers most of the weight bearing function to a more proximal segment by using a thigh corset or ischial weight bearing and a modified socket.[106] This is used when there are healing limb fractures as well as the amputation.

The use of upper limb support for balance will be necessary for most amputees in the preparatory prosthesis fitting stage. Usually a cane or single crutch on the opposite side will be sufficient for the unilateral amputee. All unilateral transfemoral amputees should be safe ambulating with bilateral crutches without prosthesis as there may be times when the

artificial limb may not be used. In some cases, due to other injuries, a walker may be used, which admittedly makes for a poor gait pattern, but this is preferable to not being able to ambulate at all.

Pneumatic compression can be used to further promote residual limb maturation, decrease swelling, desensitize the tissues and increase tolerance to pressure. When, over a period of 2 months, no significant volume fluctuation is noted in the residual limb, consideration should be given to the fitting of the first permanent prosthesis. Serial circumferential measurements of the limb at preestablished locations, and weight of the patient are the simplest techniques to determine residual limb size stability.

Prosthetic Fitting

In view of the variety and complexity of new prosthetic components (feet, ankles, and knees); socket fabrication techniques; suspension systems; and available materials, the selection of the most appropriate components for prosthetic restoration of the lower limb amputee is an extremely challenging task. Ideally, this task should be accomplished by an expert team of professionals in close communication with the patient. Members of the team may include a surgeon; a physiatrist knowledgeable in amputee rehabilitation and prosthetics; a certified prosthetist; occupational, physical, and recreational therapists; a psychologist; a social worker; and the patient and his family.

Prosthetic Feet. The human foot is a complex anatomical structure that provides a stable weight bearing base, absorbs impact, and generates dynamic propulsion essential for normal locomotion. Most patients who suffer a lower limb amputation and undergo prosthetic restoration will require ankle/foot mechanisms for their prostheses.

A variety of new prosthetic ankle/foot components has developed from a better understanding of the biomechanics of human locomotion and through progress in materials technology, including new plastic resins, carbon graphite composites, and synthetic fibers. These changes have resulted in improvements in the durability, weight, mass distribution, and cosmesis of the components and have provided dynamic characteristics to the prosthetic ankle/foot (see Figures 4-39 and 4-41 through 4-53).[84]

Some of the new dynamic response prosthetic feet have been based on the widely used SACH concept, which is known for its reliability. Other, more assertive designs attempt to use the foot, ankle, and part of the shank to improve the dynamic response. Not all of the newer ankle/foot systems provide a dynamic response; some have been improved by increasing energy dissipation characteristics, incorporating the capability for adjustment to uneven terrain, achieving a major reduction in weight, decreasing requirements for maintenance, and by providing an easy adjustment to different heel heights (see preceding sections on Below-Knee Amputation for details regarding prosthetic feet).

With the availability of more scientific information regarding the biomechanical performance of the many dynamic response feet,[84] their effects on human function, and the nature of the energy exchanges which occur at the ankle joint,[107] it has become evident that replacement of the normal ankle/foot function cannot be accomplished with the prosthetic foot.

Available data seem to indicate differences in metabolic energy consumption at high walking velocities, walking up inclines, and running using different feet.[108,109] For sedentary amputees, weight reduction and improved cosmesis appear to be the major contributors to the improvement in their quality of life. A recent, well-organized description of the most widely utilized and better known ankle/foot devices has been summarized by Esquenazi and Torres.[84]

Prosthetic Knees

A great many prosthetic knees are available for use in the treatment of the transfemoral amputee. Important to the selection of these components are the features of knee stability during stance, control of knee flexion and extension during swing, weight, and cosmesis. Knee stability during stance can be provided with a stable static alignment that keeps the knee axis posterior to the line of gravity when it traverses from the greater trochanter through the knee to the ankle—the trochanter, knee, ankle (TKA) line.

Other available choices are the use of a weight activated brake mechanism which allows knee flexion during swing phase, but prevents accidental knee flexion during stance. Several options are available for this purpose. One system is a knee with a brake mechanism that is activated during the weight bearing portion of walking while the knee is in no more than 20° of flexion (the safety knee). The brake is disengaged when weight bearing is removed, as occurs in the swing phase of walking. This knee can be very light when endoskeleton components are manufactured from titanium or other

lightweight alloys. This type of knee joint will prevent knee flexion during weight bearing but, if not well adjusted, will require that the weight be completely off the prosthesis before swing phase knee flexion occurs. It will also interfere with stand-to-sit transfers and step-over-step stair descent.

A different approach to the same problem of knee control is the use of a biomechanically stable knee joint. This system uses a polycentric knee that has two axes of rotation ("four bar linkage") (Figure 4-55). From early to midstance, the TKA line will be maintained in a position anterior to one or the other mechanical axes of rotation of the prosthetic knee. These knees have a smooth motion and allow better cosmetic accommodation for longer residual limbs. Because they are heavier, they may give a sense of instability when walking. This problem is caused by the desirable mechanical tendency of the knee to attain an extended position. When the knee is in slight flexion (10°–20°) and the foot contacts the ground, the knee is thrust into extension instead of retaining a given flexed position. This sudden push of the knee into extension can produce a sense of instability.

The use of fluid control for the knee can be compared to the fluid-filled cylinders used in shock absorbers of an automobile. As increased velocity is generated by the walking patient, the fluid- (air or oil) filled cylinder used in the prosthetic knee will become more resistant to motion. This unique characteristic allows some degree of cadence response (velocity dependent) by the device. In some instances it will permit stair descent, step over step. In swing phase, caution should be taken to not underestimate the force generated by the residual limb, which may not be controlled by a small cylinder. These types of knees, when appropriately adjusted, best resemble the function of the normal human knee, but they have the disadvantage of significantly increased cost, weight, and maintenance.

Some patients, because of weakness, short residual limb, or other complications, may need a manual knee lock to use during ambulation. This produces the undesirable, energy consuming side effect of a stiff knee during the swing phase of ambulation; this mechanism should be avoided whenever possible. It is useful for people who must stand for long periods in their vocational or avocational activities.

Some prosthetic knees have combined two of the features described above to permit both braking during stance phase and fluid control during swing phase. The two major classifications of prosthetic knees are shown in Table 4-2

Fig. 4-55. Four-bar hydraulic knee joint.

TABLE 4-2

CLASSIFICATION OF PROSTHETIC KNEE CHARACTERISTICS

Classification	Characteristics
Single Axis	Weight activated extension lock
	Fluid control (swing-only or swing-and-stance)
	Mechanical extension assistance
	Constant friction
	Hybrid (weight activated knee lock and swing-only fluid control)
	Manual lock
Polycentric (4-bar)	Weight activated stabilization
	Fluid control (swing-only)
	Mechanical extension assistance
	Friction

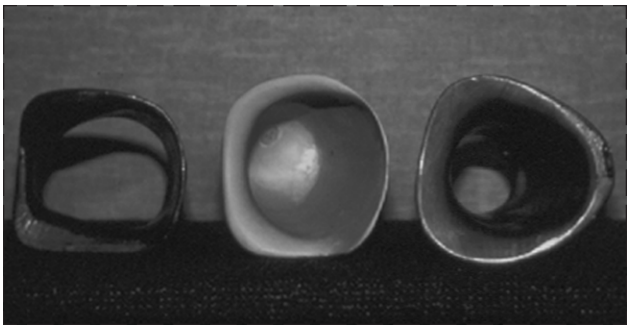


Fig. 4-56. Quadrilateral ischial weight bearing socket (left), ischial containment socket (center), and plug fit (right). The top of the illustration represents the anterior direction.

Prosthetic Sockets. Socket configuration and materials are another area of recent development. The key functions of a prosthetic socket include the comfortable total contact interface with the residual limb, efficient energy transfer from the residual limb to the prosthetic device, secure suspension, and good appearance A patient may fail to accept a prosthesis if the socket does not provide most of the above mentioned characteristics.

In the past, the plug fit socket (Figure 4-56) was carved out of wood. As the patient wore the device, blisters or other forms of skin irritation formed, indicating to the limb fitter where it was necessary to carve out more wood until a nonirritating fit was achieved. These sockets had the disadvantage of being open ended, which promoted residual limb swelling in the distal end with the potential development of chronic edema and trophic skin changes. In the mid 1950s, a federally funded research group developed the concept of total socket-residual limb contact.[110]

The quadrilateral socket was designed to match the anatomic shape of the transfemoral residual limb cross section, with appropriate relief for the muscles and tendons of the proximal thigh. The socket had a total-contact design, as opposed to the earlier open ended, plug fit socket. The weight of the bony skeleton of the body was intended to be transferred to the exoskeleton of the prosthesis from the ischial tuberosity as it sat on the horizontal ischial seat of the posterior brim of the socket. In this socket design, the ischium was not contained in the socket. To maintain the ischium on the prosthetic seat, an anterior counter force was required. Most of the pressure was applied over the Scarpa's triangle and often produced discomfort, skin irritation, and interference with the venous and arterial circulation of the limb. High temperature rigid plastic materials, such as polyester resin, are currently used instead of wood, thus decreasing the overall weight of the prosthesis and increasing the durability. More recently, acrylic lamination has replaced polyester in socket manufacturing.

In the 1970s, Long[111] noted the fact that radiographs of patients wearing conventional quadrilateral sockets demonstrated that the femur was always abducted. This led him to develop the theory that the wide mediolateral dimension of the quadrilateral socket did not provide adequate femoral control, and permitted femoral abduction to occur. He experimented with altering the dimensions of the quadrilateral socket design to improve the fit, control, and function of transfemoral prostheses, which ultimately resulted in the normal shape, normal alignment (NSNA) socket design. This socket is characterized by having a narrow medio-lateral dimension and containment of the ischial tuberosity, rather than having it rest on top of the posterior brim of the socket as occurs with the quadrilateral socket. Long's concepts also resulted in a specific static alignment for the NSNA socket/prosthesis, which came to be known as Long's Line.[111,112] Sabolich's[113] modifications to the concept of the narrow mediolateral ischial containment socket has become known as the Contoured Adducted Trochanteric/Controlled Alignment Method (CAT/CAM) socket (see Figure 4-56). In current practice, the ischial containment socket has become a very common design for transfemoral sockets. The original concepts of Long and Sabolich have undergone several modifications by the many prosthetists who adopted the use of these designs. The common variations appear to be the use of ischial containment, and narrowing the mediolateral diameter of the quadrilateral socket.[113–116]

Ischial containment has the advantages of maintaining a controlled anatomic relationship between pelvis and femur, as both are captured inside the socket. Many advocates of this socket design believe that the relationship between femur and pelvis maintains the proper femoral adduction in the socket, placing the hip abductors at a mechanical advantage, and thus giving narrow mediolateral ischial containment sockets a functional superiority over the quadrilateral sockets. Gottschalk and colleagues[102] have shown that the narrow mediolateral socket does not provide improved femoral abduction control, and that this control does require surgical reattachment of the adductor musculature for this to occur.

The ischial containment sockets have provided definite advantage for transfemoral amputees with

short residual limbs. Previously, fitting a transfemoral patient with a quadrilateral socket necessitated that the residual femur extend beyond the level of the ischial tuberosity. Ischial containment sockets have greater proximal extension, and thus contain more of the femur inside the socket. Amputees with shorter residual femurs have been successfully fitted with ischial containment transfemoral prostheses, rather than resorting to a hip disarticulation style prosthesis.

Sockets are custom made by obtaining a plaster of Paris wrap negative impression of the residual limb. This is then converted to a positive mold, which is modified by the prosthetist to distribute forces appropriately throughout the entire surface of the residual limb. Routinely, a transparent plastic socket is manufactured to permit direct visualization of the soft tissues during controlled weight bearing. The transparent socket (a check socket) is modified to assure comfortable total contact, and then a final socket is fabricated. Prior to the availability of transparent plastics, radiographic or xeroradiographic evaluation of the socket fit during weight bearing was used. This imaging technique continues to be used for the patient with very prominent bony structures, poor soft tissue coverage and painful residual limb during weight bearing.[117] More recently, the concepts of CAD/CAM have been adapted to prosthetic fabrication. Direct surface photographic imaging of the residual limb; ultrasound or MRI, or both; and direct digitization from a plaster of Paris mold are being used in some centers as sources of digital data to be manipulated in a computer environment. From there, a computer controlled carver can create a positive mold on wax or plaster from which a socket can be manufactured from vacuum formed thermoplastics. These systems promise significant time savings and the possibility of rapid socket duplication with or without further modifications (See Prosthetic Prescription section above).

New flexible plastic materials have made sockets lighter and more comfortable and energy efficient.[118] The utilization of these new materials in prosthetics has resulted in the development of improved socket construction techniques such as the Icelandic-New York (ISNY).[114,119–122] The inner socket provides total contact with the residual limb and is the interface that, if desired, provides suction suspension. The outer socket, or frame, is made of a more rigid material, thermoplastic or resin, and provides the structural integrity and weight bearing feature of the socket (Figure 4-57). When double sockets are used, windows can be cut out of the exterior frame so muscles can expand during contraction; this will improve comfort and sensory

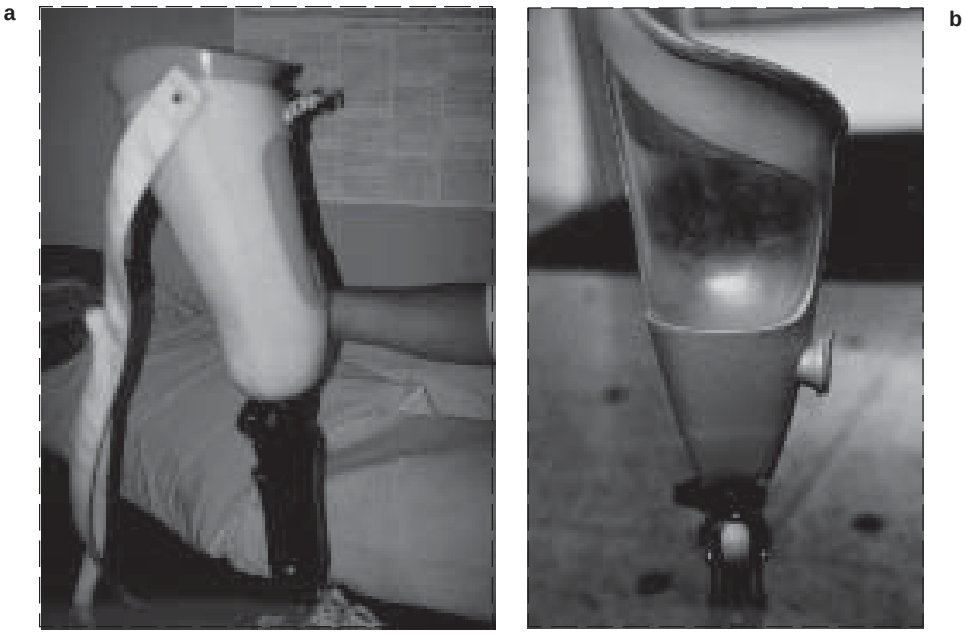


Fig. 4-57 ISNY (Icelandic-New York) transfemoral suction socket and flexible interface. (**a** and **b**) Two examples of the type of socket design.

feedback during sitting.[119] The flexibility of thermoplastic material results in a more comfortable fit at the proximal socket brim during standing. Although it is more costly and time consuming to fabricate, the frame socket design has the added advantage of allowing replacement with a new, inner socket to accommodate for residual limb changes without changing the external frame, knee, or foot units. This is an optimal design for war injured amputees, because it allows early ambulation with a prosthesis with definitive knee, shank, and foot components. As the limb changes shape, the inner socket can be changed without changing these components. It is a rather simple process to pull out the old liner and slip in the new one. The inner socket is usually held in place by Velcro or other removable fastener. The frame can be made for quadrilateral or ischial containment designs, and suction or nonsuction suspension systems.

Suspension Systems. Prosthetic devices need to be secured to the body. The more secure the suspension system, the better the prosthetic control and comfort the patient will experience. In the past, the lower limb amputee has been provided with suspension systems that consisted mostly of leather straps with metal attachments; those systems limited mobility and were ineffective and heavy.

The traditional nonsuction suspension mechanisms for transfemoral sockets included either a Silesian belt or a waist belt. The Silesian suspension attaches laterally to the outside of the socket and passes posteriorly around the pelvis over the opposite iliac crest and then attaches to the anterior socket. A significant problem with this suspension is its tendency to allow the socket to rotate internally on the residual limb. A modified Silesian suspension adds an additional strap that goes around the ipsilateral iliac crest. The waist belt suspension usually has a hip joint and pelvic band and is fabricated from leather and metal components. Lighter, plastic components are available, but plastic pelvic bands and hip joints do not control the tendency of internal rotation as well as do the metal pelvic bands. For the transtibial prosthesis, the total elastic suspension (TES) belt is a neoprene waist belt that is slipped over the prosthesis like the sleeve suspension and then encircles the waist to suspend the prosthesis. It has an anterior Velcro closure. Like the Silesian belt, the TES belt does not control rotation well.

The hypobaric suction suspension provides an excellent transition between nonsuction and suction suspension. This suspension system utilizes a sock that has a special silicone band in its proximal portion. This band creates a seal between the socket and the skin of the residual limb, and the resulting suction can be used for suspension. However, experience suggests that the suction suspension is often only partial, and an auxiliary suspension system, such as a Silesian belt, is necessary. The advantage of this suspension system is that changes in residual limb volume, which cannot be easily accommodated with a standard suction socket, can be managed simply by altering the number of ply (thickness) of the special residual limb socks. Another use for these socks is to control the internal rotation of the transfemoral socket. The friction of the band against the socket and the skin works well for this purpose.

In most cases, socks are used as an interface between the residual limb and the socket to adjust for the physiological volume changes that occur through out the day and from day to day. The only exception is when a suction socket is used. Patients must be educated about the need to appropriately alter sock thickness.

In some patients with very short and bulbous residual limbs, shoulder suspension (a type of suspender) may be used to improve suspension. This technique may be applicable to those patients who are unable to tolerate pressure over the abdominal wall.

For the transfemoral amputee, suction suspension (negative pressure) without the use of straps is preferred; this allows the patient optimal function, balance, strength, endurance and coordination. The socket is made small enough and is provided with a one-way valve that permits the expulsion of air during donning. The amputee dons the socket using a pull sock, Ace bandage, or wet fit (using a lubricant lotion on the skin of the residual limb). The intimate fit, especially proximally, results in a tight seal between the socket and skin. For doffing, it is necessary to break the vacuum seal and the valve is designed to allow this. To maintain proper suspension over time, the residual limb must be mature and volume stable.[43]

A novel idea infrequently used in this country is the TC-3[123] socket, developed in Japan at the Tokyo Metropolitan Rehabilitation Center. It permits the patient to handle the socket with the suspension system of choice separate from the prosthesis (Figure 4-58). This gives the patient the advantage of handling a smaller section of the prosthesis, with decreased bulk and weight for optimal application. The socket and residual limb are inserted into a thin, lightweight receptacle that has the knee, shank, and foot, and attaches with a Velcro strap. This system permits donning and doffing in the seated position

Fig. 4-58 TC-3 (Tokyo Rehabilitation Center) transfemoral socket. (**a**) Patient dons internal socket. (**b**) Velcro strap on the lateral aspect allows firm suspension of internal and external sockets.

and also is very helpful for the patient with limited hand function.

Cosmetic covers should be considered an integral part of the prosthesis, and for many patients it could be the catalyst that promotes success or failure. Exoskeleton prostheses do not afford as good visual or tactile cosmetic results, whereas the use of endoskeleton components allow the fabrication of a soft foam external cover. The shape of the cover may be derived from a mirror image of the sound limb and finished with one of the many plastic materials that resemble skin and is water resistant. Lifelike, custom made covers that use silicone or other materials can provide excellent cosmetic results, but may be very expensive and deteriorate rapidly (Figure 4-59).

For the through-knee amputee with a weight bearing bulbous end, a socket with a trap door, similar to a Syme's prosthesis, can be opened to allow donning, then closed, providing suspension by grasping the femoral condyles (see Figure 4-59). In this case, the socket does not have to extend proximally to the ischial tuberosity.

Hip Disarticulation and Transpelvic Amputation. Little change has occurred in the basic socket design for hip disarticulation or transpelvic amputations since the Canadian design was introduced in the 1950s. The major changes have been in slight modifications of the socket trim lines, suspension, and materials used to construct the socket. The standard Canadian socket design consists of a bucket-type socket, which encloses the residual pelvis and

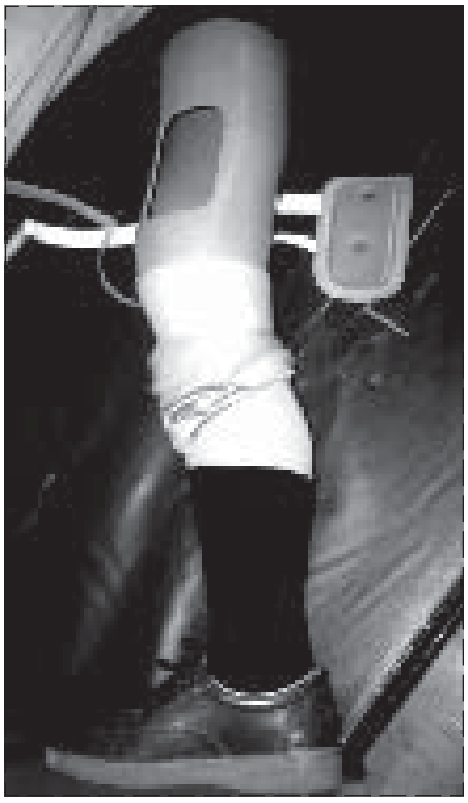


Fig. 4-59. Through-knee amputation endoskeletal prosthesis, showing lifelike foam cover and skin tone nylon stocking (turned down). Suspension via trap door closure over a bulbous end is similar to that of a Syme's prosthesis.

extends around the pelvis of the uninvolved side, leaving an opening for the nonamputated leg. The socket extends over both iliac crests and has an anterior opening to allow donning and doffing. By fastening the anterior opening, the socket is securely self-suspended from the iliac crests. The external appearance of the socket is similar for both levels of amputation. The interior surface contour is altered to provide total contact for the different levels of amputation.

As with sockets for other levels of amputation, thermoplastic materials are being used to provide lighter and more comfortable sockets. An obliquely trimmed socket brim (below the iliac crest on the nonamputated side), together with a Silesian belt over the nonamputated iliac crest for suspension, can be used to reduce the size of the traditional socket design. In the oblique modification, the socket still extends over the ipsilateral iliac crest of the amputated limb. For this high level of amputation, most of the advances in prosthetic design have occurred with new lighter components.

Prosthetic Training

Gait training is integral in the rehabilitation process. A new amputee or an experienced one who receives a prosthetic device that has different components should participate in such training. This program should be a coordinated effort between the physical, occupational and recreational therapists, and the prosthetist with frequent physiatric input. Each team member will use different techniques to teach to, and review with, the amputee all of the important topics that need to be learned.

The patient should learn the basic principles behind the function of each of the components in the prosthesis, its maintenance and care, and other points of prosthetic management. The patient should practice how to put on and take off the prosthesis, how to determine the appropriate sock thickness to wear, and techniques to adjust them. Skin care and inspection techniques are also reviewed. Weight shift techniques should be encouraged, including the use of stepping and a balance board. It is essential that gait training initially address proper technique, and after that training, addresses velocity on flat surfaces. Progress to uneven surfaces and elevations is then included. A review and practice in the use of the prosthesis in transfers, driving, sports, and other activities should always be part of the training.

Special Considerations for the Bilateral Transfemoral Amputee. For the bilateral transfemoral amputee, training should be initiated with very short nonarticulated prostheses (stubbies). As the patient gains balance and strength, first one and then the other knee can be articulated. After articulation of the bilateral knees has been achieved, then the length of the prosthesis may be increased. The goal of the prosthetic restoration should be to maintain the patient at a slightly lower-than-preamputation height to decrease the energy requirement of balance during standing, and to decrease the effort in energizing the prosthetic devices while walking. Alternate techniques for putting on the prosthesis are frequently required. These include using the bed for support of the prosthetic devices, or leaning against the wall.

For bathing activities, the patient with bilateral transfemoral amputations will require a shower and a shower wheelchair. In some cases, shower prostheses (devices that are waterproof and have nonslip feet) are indicated, and should be considered a medical necessity because they will allow the patient to safely perform this necessary activity.

Reintegration Into the Community

The reintegration into the community is best done as a gradual process. This process can be initiated early in the rehabilitation program with supervision by team members during organized trips for shopping, recreation, work, or school. When possible, a good system to foster community reintegration is the use of "day hospital" rehabilitation programs in which the patient participates in rehabilitation for 6 hours a day, 5 days a week and returns home every evening and weekend. When safe, the patient may return to work. Initially, modified or restricted work should be provided, but the patient should not be discouraged from returning to the premorbid work level, if it is safe to do so.

Long-Term Follow-up

The patient who has successfully completed a rehabilitation program should be seen for follow-up by a one or more team members at least every 3 months for the first 18 months. These visits may need to be more frequent and include more members of the team if the patient is having difficulties with prosthetic fitting, the residual limb, specific activities, or psychosocial adjustment. After this critical period, the patient should be seen at least every 6 months to assure adequate prosthetic fit and function, and attend to any prosthetic maintenance. It may be necessary to replace a prosthesis or parts

of it every 2 to 3 years.

Support Groups. For many patients, support groups are a source of information, peer counseling, and motivation, and ideally, these groups should be one more component of the comprehensive rehabilitation approach. Patients who have recently suffered an amputation will benefit from exposure to group members who are experienced amputees, while at the same time, the veteran amputee will appreciate being used as a resource.

Monitoring Residual Limb Problems. The skin of a patient who wears a prosthesis is subject to many stresses. Most prosthetic sockets prevent air circulation, thereby trapping perspiration. This can result in a variety of problems, such as hyperhydrosis, folliculitis, allergic dermatitis, and, where adherent scars are present, skin breakdown. Poor hygiene is frequently the cause of some of these problems; for this reason the patient should be carefully trained in proper washing technique for the leg, socks, and the socket and liner. A daily routine of washing the skin and the internal wall of the socket with a mild soap may suffice. At times it may be necessary to use concentrated antiperspirants, bacteriostatic or bactericidal soaps, antifungal powders, or antibiotics.

Stump edema syndrome is a condition with edema, pain, and increased pigmentation. It is commonly caused by proximal constriction, and usually responds to stump elevation, compression, prosthetic modifications, or temporarily discontinuing use of the prosthesis. Verrucous hyperplasia is a wartlike overgrowth of skin caused by a lack of distal prosthesis contact by the residual limb. Prosthetic modifications to improve distal contact must be made to address this problem and prevent recurrence (see Medical and Surgical Complications below).

Summary

The rehabilitation process for the patient with a transfemoral amputation is complex and is best accomplished by the patient who is able to cooperate with a comprehensive interdisciplinary specialized team. The team should be able and ready to assist the patient throughout the rehabilitation program, from preamputation to community reintegration. If no other significant comorbidity exists and optimal rehabilitation is provided, the otherwise healthy, war injured transfemoral amputee can be expected to return to a high functional level.

MEDICAL AND SURGICAL COMPLICATIONS OF AMPUTEES

Postoperative Complications

Mortality. In the Civil War, the mortality rates of above-knee and below-knee amputations were 54.6% and 32.3%, respectively. In World War I the mortality rates were 40% and 18% for above-knee and below-knee amputations, respectively. In World War II, the mortality rate for amputations in one hospital was 9%. Mortality rates decreased in the Korean and Vietnam conflicts.[47] In general, more proximal traumatic amputations yield higher morbidity. Mortality in traumatic amputations is also dependent on associated injuries and their severity.

Pain. Immediately postoperative, residual limb pain is severe; however, as healing occurs, the pain subsides significantly within the first week. Postoperative analgesics should be used to provide pain relief. However, if the pain worsens, a cause should be sought. Possible etiologies of increased pain include infection, ischemia, hematoma formation, improperly fitted rigid dressing, and excess weight bearing with an IPOP.

Infection. Infection is a major risk in traumatic amputations during wartime. Peterson[6] states that during World War II, infection was common in modified amputations with irregular skin flaps. Osteomyelitis was almost always present under these circumstances. In World War II, 1,670 patients who underwent primary stump closure reportedly had an infection rate of 8.9%. This amounted to about 149 patients. For the above reasons, primary closure was contraindicated, and the open circular technique was chosen as the amputation of choice.

After late revision and closure of the open amputation, the residual limb is monitored for signs of infection, particularly when there is increased pain in the immediate postoperative period. In cases of residual limb erythema with cellulitis, treatment should be initiated with systemic antibiotics. If deep wound infection occurs with abscess formation, wide drainage and antibiotic therapy is instituted.[124]

Hematoma. Hematoma formation may occur from inadequate hemostasis or drainage. Hematoma serves as a medium for infection. Additionally, hematoma formation may result in pain and skin edge necrosis. For small hematomas, aseptic drainage followed by compressive wrapping suffices.[47] For larger hematomas, evacuation in the operating room is usually required.

Delayed Healing

Delayed healing may be the result of improper amputation level selection, poor operative technique, infection, or poor nutrition. Improper amputation level selection mainly refers to the presence of inadequate circulation for healing. Circulation may be assessed preoperatively with vascular studies, including transcutaneous oxygen pressure ($TcPO_2$) determination, Doppler studies, and Xenon 133 isotope clearance. $TcPO_2$ determination may also be used postoperatively to assess vascular status. In one study[125] of postoperative amputations, amputees with a $TcPO_2$ equal to or greater than 40 mm Hg all healed. Those requiring revision related to nonhealing without infection had a $TcPO_2$ less than 40 mm Hg.

Poor operative techniques that may cause delayed healing include handling the skin with forceps, closing the skin under tension, and excess tension when closing muscle.[126] Obviously, infection requiring further debridement will delay healing. Nutrition is correlated with healing. Albumin levels of less than 3.5 g/dL and lymphocyte counts of less than 1,500 /mm^3 are associated with delayed healing; therefore, these parameters should be monitored, and proper nutrition provided via food supplements, if necessary.

Necrosis. Excess skin tension can result in necrosis. Improperly applied rigid dressings, particularly in patients with decreased sensation, can also result in skin necrosis (Figure 4-60). Additionally, an improper amputation level selection that results in inadequate circulation may lead to necrosis. Furthermore, proximal vessel thrombosis may lead to circulatory compromise and resultant necrosis.[47] If the area of necrosis is small, it may heal if all pressure is removed. Rigid dressing may continued without weight bearing.[124] If the area is large, revision may be required.[47]

Wound Dehiscence. Wound dehiscence may occur from one or several of the following: infection, premature suture removal, excess early weight bearing, or a fall onto the residual limb. Before treating, the cause needs to be determined. Treatment is based on the underlying cause. For example, in the case of infection, wide drainage and antibiotic therapy is the treatment. In uncomplicated cases, the wound may be closed in the operating room. However, if more than 6 hours had elapsed, the wound is left open with delayed closure performed.[47]

Contracture. The lower limb amputee is at increased risk for developing joint contractures secondary to immobilization, poor positioning, pain, and changes in agonist–antagonist muscle balance. Additionally, the risk of contracture is increased in the presence of flaccid paralysis, spasticity, edema, ischemia, and bleeding.[18] Contracture can prohibit prosthetic fitting, affect step length, place abnormal forces at joints, and increase energy expenditure during ambulation.[2]

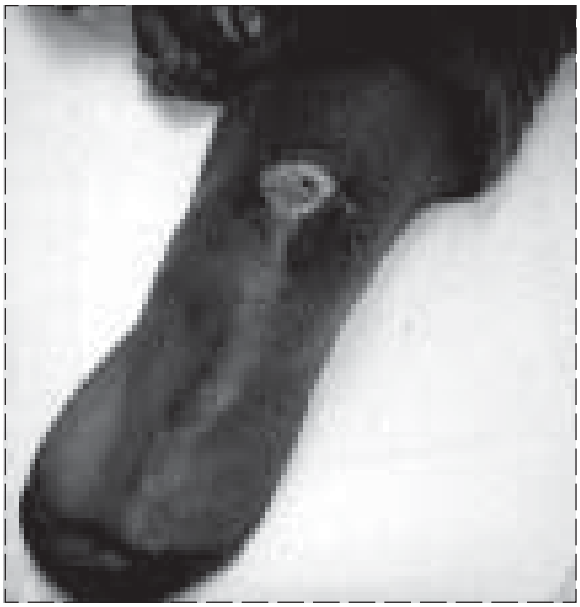


Fig. 4-60. Skin necrosis from excess rigid dressing pressure over patella.

Joint contractures may be prevented by proper positioning and exercise. Frequent bed position changes should be scheduled.[18] Prone lying should be encouraged to prevent hip flexion contractures. In the bedbound patient, a trochanteric roll may be helpful in preventing hip external rotation contracture. Prolonged sitting and use of a soft mattress should be avoided to prevent hip flexion contractures. A pillow between the knees or thighs can cause hip abduction contracture, especially in the above-knee amputee, and should be avoided.[59] A pillow under the thigh or knee should also be avoided to prevent knee flexion contracture. When sitting, the below-knee amputee should use a knee extension board.

Range-of-motion exercises should be done on a scheduled basis with a frequency of four times daily.[14] Early mobilization in a rigid dressing with pylon is very valuable in the prevention of contracture.[2,18] If a contracture occurs, an active and passive range-of-motion program with terminal stretch should be prescribed.

Skin Complications

Skin serves as an environmental barrier to infection, solutes, and water, and through sweat evaporation, is involved in temperature regulation. Other functions include sensation, pigmentation, and vitamin D synthesis. In the amputee, skin provides soft tissue protection to the mechanical forces induced by the prosthesis. To serve these functions, skin must be healthy.

In the amputee, residual limb skin would ideally be normal healthy skin with good elasticity, freely moveable, and adequately padded with subcutaneous tissue. Increased skin tension, which may subject the skin to an increased risk of breakdown, can result from discontinuing skin traction too soon or surgical closure with insufficient skin present. Tenting of skin over the end of bone or redundant skin folds should be avoided because of the increased risk for skin breakdown.[127] Skin tension can be further increased by skin stretching when donning a socket.

Scar location is important. Ideally, a surgical scar is linear and avoids bony prominences, the cut end of bone, and socket pressure points. Scar should be freely moveable, soft, pliable, and insensitive. Skin grafts present problems secondary to adhesions, inelasticity, and atrophy, making the graft susceptible to mechanical forces.[127] Scar adhesion to bone renders the scar immobile and increases the risk of skin breakdown due to excessive shear forces from the prosthesis. The adhered scar often undergoes breakdown, which necessitates discontinuance of the prosthesis. Treatment of adhered scar consists of friction massage to mobilize the scar from bone; care must be taken to avoid tension at the incision line with the massage. Additionally, a nylon sheath or liner may be used to decrease shear forces to the area.

Skin is challenged by shear and loading forces delivered by the prosthesis to the residual limb during ambulation. Furthermore, skin is taxed by the closed socket, which affects temperature regulation and creates a moist environment from sweat accumulation. When a socket is poorly fitted, pathologic skin changes may occur. Some of the more common skin disorders seen in amputees are discussed below.

Mechanical Injury. Mechanical forces imposed by the prosthesis on the residual limb are pressure, shear, and friction.[127] Excessive forces may result in skin destruction or proliferation.

Tissue Destruction. Maceration occurs because of rubbing over pressure points combined with moisture from sweat accumulation in the socket. Maceration can be treated with proper prosthetic fitting with appropriate pressure relief. Additionally, optimal residual limb hygiene should be followed.[127]

Blisters occur from the combination of friction and pressure, secondary to a loose or excessively tight fitting socket.[127] Inadequate suspension may also lead to blister formation. Risk areas include the socket brim and distal end of the residual limb. The socket should be checked for proper fit and suspension, and modified as necessary. Abrasions and erosions result from rubbing and pressure. Pressure points in the socket are risk areas. Socket fit, alignment, and suspension should be evaluated.

Ulcerations occur from excess mechanical forces. Risk factors include ischemia, edema, lymphatic blockage, adherent scars, impaired sensation, and systemic disorders such as diabetes.[127] Treatment includes proper prosthetic fit and alignment. Also, the prosthesis should not be worn until ulceration has healed.

Tissue Proliferation. Lichenification occurs secondary to excess mechanical forces. This is a common problem presenting with thickened, leathery skin. Exaggeration of the normal skin markings with a crisscross pattern is visible on examination. Furthermore, physical examination may reveal scaling, erythema, edema, fissuring, erosion, and pigmentation (Figure 4-61). The location of the lichenification is usually over pressure areas in the socket

Fig. 4-61. Lichenfication demonstrating thickened, leathery skin with hyperpigmentation.

or along the brim. The patient may complain of burning, itching, or soreness. Treatment consists of realignment of the socket to decrease pressure, or modification of the socket to reduce pressure over these areas. If the problem continues, a Teflon brim may be used for lichenification at the brim location. Temporary relief may be provided by corticosteriod cream.[127]

Callosities are circumscribed hyperkeratosis lesions, which usually occur over bony prominences and are secondary to chronic mechanical forces.[127] Treatment consists of pressure relief and good skin hygiene.

Follicular hyperkeratosis and coiled hairs result from mechanical forces that cause enlarged keratin plugs that trap and coil hair. The plugs may act as a foreign body with secondary infection. Some plugs may require manual expression. Treatment consists of proper fit, alignment, and, if needed, changing socket materials.[127]

Epidermoid Cysts. Epidermoid cysts are associated with the development of a tissue roll that is subjected to mechanical shear from the socket. Skin then undergoes invagination of surface keratin and epidermis, thereby forming a cyst. The cyst may enlarge and act as a foreign body with secondary infection with later sinus tract development. With prosthetic usage, cysts develop over months to years and may become painful with local inflammation or infection. In above-knee amputees, cysts tend to occur in the adductor region, the inguinal fold, and the ischium. In below-knee amputees, cysts tend to develop over the anterior tibial surface and popliteal region (Figure 4-62). Amputees may experience discomfort with prosthetic use, leading to eventual discontinuance of prosthetic usage. Individuals at increased risk for development of epidermoid cysts include those with cysts elsewhere, acne, and seborrheic dermatitis. Cyst development is prevented through good prosthetic fit, proper alignment, appropriate pressure relieves, smoothing of rough areas in the socket, use of a liner or socks, and good hygiene.[127]

Treatment consists of optimizing pressure relief and skin hygiene. The fluctuant cyst must be incised and drained. If there are signs of infection or purulent drainage, cultures are obtained and antibiotic therapy initiated.

Circulatory Complications

These disorders result from circulatory disturbance in either venous or arterial systems, or both. Selected disorders will be presented here.

Reactive Hyperemia. Reactive hyperemia tends to occur in new prosthetic wearers, especially those with poorly fitting sockets. The amputee may complain of tingling, warmth, and tightness. On removal of the prosthesis, the residual limb is noted to be flushed and warm. The flushing and warmth subsides without residual problems. In some cases, venous and lymphatic congestion may occur, leading to edema and small vessel hemorrhage, and causing a bruising discoloration. Later, residual brown pigmentation from hemosiderin deposits may be noted. Prevention consists of compression wraps.[127] A rigid dressing prepares the residual limb for mechanical stresses induced by prosthetic use.

Stasis Dermatitis. When there is insufficiency or stasis of venous flow, stasis dermatitis may develop. Causes of venous insufficiency or stasis include old thrombophlebitis, varicose veins, external pressure, neoplasia, anomalous veins, and arteriovenous fistulas. Additionally, inadequate distal residual limb prosthetic support may result in edema. Distal swelling may result in congestion, cyanosis, and

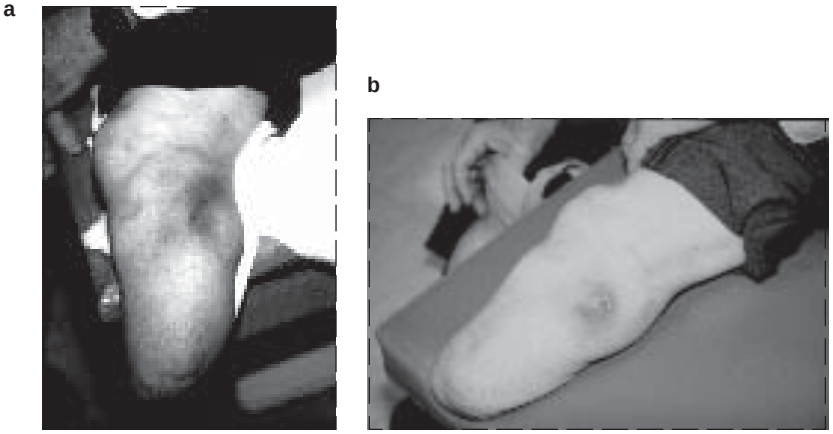


Fig. 4-62 Epidermoid cyst. (**a**) Fluctuant epidermoid cyst requiring incision and drainage and (**b**) same patient after incision and drainage. Socket modifications were required to correct a poorly fitting socket.

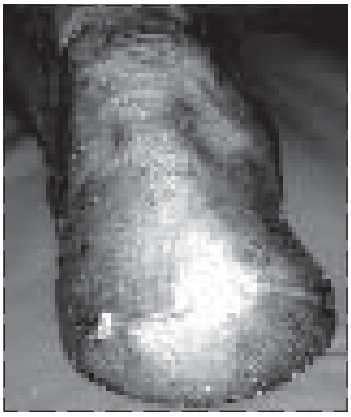


Fig. 4-63. Edematous residual limb.

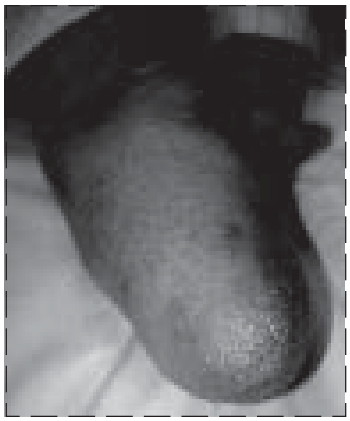


Fig. 4-64. Verrucose hyperplasia.

hemosiderin deposition. With venous insufficiency, the skin becomes pruritic and exhibits scaling. Differential diagnoses include contact dermatitis and fungal or bacterial infection. Stasis dermatitis treatment consists of removing the underlying pathologic condition causing the dermatitis. Prosthetic causes are corrected by ensuring total contact. Additionally, when not in the prosthesis, the limb should be in a compressive wrap or sock.[127] Contact dermatitis will be discussed later.

Stump Edema Syndrome. Proximal constriction of the residual limb from an external source may cause the stump edema syndrome. The external source of constriction may be an improperly wrapped elastic bandage or improperly fitting socket. Also, excess negative pressure from a suction socket may cause distal edema. Early signs may include narrowing proximally with bulbous soft edema distally (Figure 4-63). Over weeks to months the edema may become firm. With firm edema, palpation of a fold of skin between the fingertips reveals thicker and firmer skin compared to the same area contralaterally. Additionally, an orange peel appearance develops. There is an increased risk in overweight and sedentary patients.[127] Treatment consists of proper fitting with relief of proximal socket and suspension constriction and ensuring total socket contact, including the distal residual limb. A rigid dressing, temporary socket, or elastic bandage may decrease the edema. Proper education regarding wrapping and socket fit is important. The residual limb should be in a compression wrap or sock when not in the prosthesis or rigid dressing.

Verrucose Hyperplasia. Verrucus hyperplasia is a warty condition of the distal residual limb (Figure 4-64), caused by proximal constriction and vascular insufficiency from a poor socket fit. Treatment consists of distal compression, total contact socket, and relief of proximal constriction.

Contact Dermatitis

Contact dermatitis may be divided into primary irritant dermatitis and allergic contact dermatitis. The mechanism in each is cutaneous contact with the offending agent. Susceptibility to the offending agent may be increased by mechanical injury to the skin. Skin reaction may be mild and consist of slight erythema, burning, and minor itching; or a fulminant reaction may occur with local inflammation, vesiculation, crusting, and serous oozing[127] (Figure 4-65).

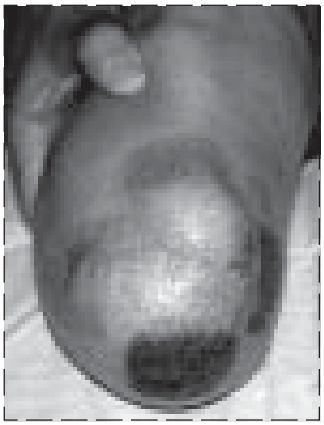


Fig. 4-65. Contact dermatitis from bandage tape. Note the contact pattern corresponding to the tape.

Primary Irritant Dermatitis. In most individuals, a primary irritant is a substance that results in an irritating skin reaction on first exposure (see Figure 4-65). A relative primary irritant requires prolonged contact and mechanical susceptibility of the skin, such as maceration. The patch test, described later, may be normal.[127]

Allergic Contact Dermatitis. Allergic contact dermatitis results from exposure to an allergen by skin antibodies, leading to an allergic reaction. It is an acquired hypersensitive state and requires preliminary exposure to a specific agent. There is a refractory period, incubation period, reaction time, and sensitive state in the development of allergic contact dermatitis. The refractory period is the time course during which potential sensitizers may be in daily contact with skin without sensitization. Once sensitization occurs, there is an incubation period before sensitization is complete. Then there is a variable time between exposure and first manifestations of sensitization, termed the reaction time. This time period is reduced by mechanical stresses (such as maceration) on the skin. With development of an allergic skin reaction, the sensitive state begins. The sensitive state may persist indefinitely. However, the skin reaction may diminish with time.[127]

Certain conditions may affect the severity of the skin response, including a higher concentration of irritant or allergen, and prolonged exposure. Environmental factors such as heat, moisture, and pressure, may predispose to a skin reaction, as well as preexisting irritation or inflammation of the skin.[127]

Diagnosis and Treatment. Diagnosis of contact dermatitis is based on patient history, physical examination, and a patch test. Important items in the history include (*a*) condition onset; (*b*) previous episodes; (*c*) initial site of involvement; (*d*) history of skin allergies; (*e*) recent activities that may have resulted in exposure to an irritant or allergen; (*f*) change in medications, socket, socks, soap, or cleansing agent for the socket, or any of those; and (*g*) all materials contacting the residual limb. Contact dermatitis should present in a recognizable pattern where the area of skin involved corresponds geographically to the inciting contact irritant or allergen (see Figure 4-65). A patch test evaluates the suspected agent. Using a standard size patch, the agent is attached to normal skin. The patch is left in place for 24 to 72 hours and, in some cases, up to 5 days, after which the patch is removed and the skin examined. Skin is reexamined for up to 5 weeks in case there is a delayed reaction. Even with a careful history, physical examination, and patch testing, the offending agent may be difficult to identify. This is especially true in allergic contact dermatitis where the refractory period and incubation time makes identification of the offending allergen confusing. Offending agents to keep in mind are the materials used in making sockets or liners. Additionally, residual limb socks and cleansing materials may be the offending agent.

Treatment consists of removal of the causative agent. Additionally, dermatitis treatment with topical steroids, cold compresses, aspirin, antihistamines, and antipruritic medications may be beneficial. In pruritic cases, scratching may lead to secondary infection, requiring antibiotic agents.[127]

Residual Limb Pain: Intrinsic Causes

Ischemia. Ischemia may present with intermittent claudication recurring at a set distance.[126] The amputee may have pallor and soft tissue breakdown revealed on examination of the residual limb.[128] Doppler, $TcPO_2$ and xenon 133 vascular studies can assess for ischemia.

Ischemia may be caused by dysvascular disease or may result from excess vascular compression by the socket. An excessive popliteal bulge in the PTB-TCS, or Scarpa's triangle in the quadrilateral socket, can result in ischemia.[128]

Initial treatment in the dysvascular patient consists of conservative measures. If soft tissue breakdown is present, appropriate wound care and discontinuance of weight bearing in the prosthesis is usually indicated. Once healing has occurred, a gel socket or appropriate liner is needed. In some cases, a below-knee bypass prosthesis may be utilized if the gel socket is unsuccessful.[128] If conservative measures fail, revision or revascularization should be considered.

If ischemia is caused by excess vascular compression by the prosthesis, socket modification is required. The popliteal bulge can be eliminated.[128] In the quadrilateral socket, Scarpa's triangle pressure can be decreased or replaced with an NSNA socket with reduced pressure over the femoral artery.

Ectopic Bone. Development of bone spurs may result in localized pain, which may become worse with prosthetic use. The amputee may even be unable to tolerate pressure from the socket. Ectopic bone results from periosteal stripping of retained bone either by the original trauma, or by surgical procedure.[126] Ectopic bone may also occur from retained periosteum in the residual limb.[14] Radio-

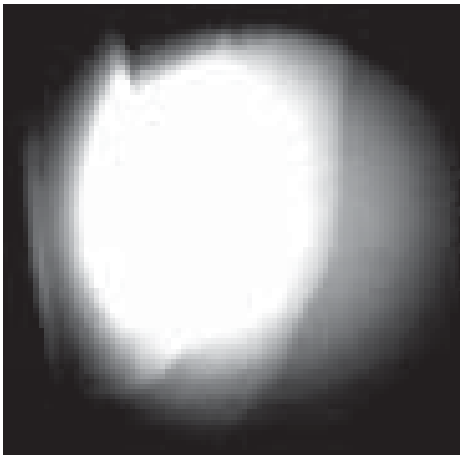


Fig. 4-66. Ectopic bone on plain x-ray.

graphs readily show the ectopic bone (Figure 4-66). Ectopic bone development may be minimized or possibly prevented by not disturbing the periosteum.[126] Once symptomatic ectopic bone is present, initial conservative management is warranted; a socket relief is tried.[126,128] If unsuccessful, a soft liner with a cutout for a gel pad insert over the bony prominence may be helpful. If conservative measures fail, surgical revision is considered. Only symptomatic spurs should be removed.[126]

Excess Fibular Length. When the fibula is longer than the tibia, an amputee may present with pain that is worse on weight bearing. Examination will reveal a prominent fibula longer than the tibia, and there may be soft tissue breakdown at the distal fibula. Radiographs will confirm the diagnosis. Conservative treatment consists of socket modifications, soft liners, or distal pads. If unsuccessful, a revision may be necessary.[126]

Inadequate Tibial Beveling. Inadequate tibial beveling can result in bursal development or pain and soft tissue breakdown over the distal anterior tibia. Treatment consists of socket modifications to relieve pressure over the region or utilization of a liner. A soft foam liner with a cutout for a gel pad over the distal tibia can be used. If conservative methods fail, surgical revision may be necessary.

Hypermobile Fibula. In addition to pain, amputees may complain of symptoms secondary to pressure on the peroneal, tibial, or sural nerves. Examination will reveal an excessively mobile fibula. The hypermobile fibula results from traumatic disruption of the interosseous membrane and the proximal tibiofibular joint. Treatment in symptomatic individuals is fusion of the proximal tibiofibular joint.[126]

Unbalanced Myodesis in Above-Knee Amputation. The amputee may complain that distal, anterolateral residual limb tenderness is worse when wearing the prosthesis. A physical examination may reveal the tenderness on palpation. Skin inspection may reveal ulceration. The problem results from an unbalanced myodesis of the adductor muscles with femoral drift anterolaterally through the soft tissues. Treatment consists of socket relief at the bony prominence. Alternatively, the socket may be built up anteriorly and proximally, thereby reducing pressure over the painful area. If conservative measures fail, surgical revision with adductor myodesis may be necessary.[126]

Neuroma. Individuals with a symptomatic neuroma often present with sharp, shooting pain.[36] Symptoms are brought on or worsened by prosthetic wear or weight bearing. Examination will reveal a positive Tinel's sign with tingling discomfort[36,126] and pain reproduction with direct palpation over the symptomatic neuroma.

Neuromas normally develop after nerve transection and may generate pain when mechanically irritated. Less commonly, a neuroma may become a spontaneous pain generator.[36] If a neuroma becomes adherent to scar located in weight bearing regions or in areas of socket pressure, it is subjected to increased mechanical irritation with subsequent pain generation.

Neuromas located by MRI will reveal hetergenous ovoid structures of intermediate intensity surrounded by a rim of low signal intensity on T1 images. T2 images demonstrate ovoid structures of increased signal intensity intermingled with strand-like areas of low intensity surrounded by a low signal intensity rim.[129]

Singson and associates[129] postulate that the hetergenous ovoid structures represent low signals from collagen matrix and intermediate to high signals from cellular nerve fascicles. They further postulate that the rim of low intensity signal represents collagen tissue. MRI may also differentiate neuroma from scar tissue, abscess, osteomyelitis, and hematoma as causes of residual limb pain.[129] Anesthetic nerve blocks or a neuroma injection can determine if a neuroma is the cause of residual limb pain. Injections are not only diagnostic, but may provide therapeutic analgesia.

The best treatment for symptomatic neuromas is prevention. Avoidance of neuroma formation in the wound scar, weight bearing location, or socket pressure point should be considered at the time of surgery. Nerves should be sharply transected under

tension to allow retraction into healthy muscle, which provides protection from mechanical irritation or pressure with prosthetic use.

Initial conservative management of symptomatic neuromas consists of socket modifications to decrease pressure or shear. Pressure may be decreased with socket reliefs, total contact sockets, or liners. Shear may be decreased with a nylon sheath and optimal suspension to minimize pistoning. A TENS unit trial is performed. Also, a trial with anticonvulsant medications, such as carbamazepine or phenytoin, may be concomitantly tried or used alone.[36] If the neuroma becomes refractory to socket modifications, and if temporary anesthetic injection of the neuroma relieves the pain, Davis[36] recommends chemical obliteration of the nerve with CT-guided phenol injection of the neuroma. However, Friedman[14] states that chemical obliteration is of no benefit. If conservative management fails, surgical revision may be necessary. The neuroma is resected and moved to a deeper site by transection under tension and retraction into muscle. Another method consists of burying the nerve end in bone,[126] although Friedman[14] believes this procedure has no benefit.

Reflex Sympathetic Dystrophy. Reflex sympathetic dystrophy (RSD) is a symptom complex that may occur after major or minor trauma, including amputation.[130] It may occur in any age group. Major symptoms are pain, swelling, and tenderness in an extremity. Physical examinations may reveal edema, tenderness, contractures, mottling, and dusky red or blue color. There may be skin atrophy, or increased or decreased skin temperature, nail growth, hair growth, and sweating. The symptoms and signs are partly dependent on the stage of RSD. RSD is divided into three stages:

1. Stage I (acute phase) may consist of pain, local edema, local increased temperature, increased blood flow, joint stiffness, increased hair growth, and increased nail growth. This stage lasts for a few weeks to several months.[131]
2. In stage II (dystrophic phase), there is decreased hair growth, nail growth, and local temperature. Additionally, muscle and subcutaneous atrophy begins and nails become brittle. The skin may be pale or cyanotic. This stage may last 3 to 6 months.
3. In stage III (atrophic phase), contractures, marked muscle wasting and subcutaneous atrophy occur, with the skin developing a glassy appearance.[131]

Diagnosis of RSD is mainly clinical. Other disorders that cause pain must be eliminated from the differential diagnosis. Kozin[132] developed a classification to help in the diagnosis of RSD. The probability of having RSD was divided into *definite, probable, possible,* and *doubtful* groups. The *definite* group exhibits pain, edema, vasomotor, and sudomotor changes in the involved extremity. The *probable* group exhibits pain, vasomotor, and sudomotor or edema, but not both, in the involved extremity. The *possible* group exhibits vasomotor or sudomotor changes. The *doubtful* group exhibits pain only.

Diagnostic tests mainly include radiographs, bone scans, and sympathetic blocks. Radiographs may demonstrate patchy osteopenia in the involved limb after 6 weeks.[131] Prior to 6 weeks, radiographs may be normal. Three-phase bone scans with technetium 99m methylene diphosphonate are reported as abnormal in 83% of individuals with definite RSD.[132] In stage I, bone scans reveal increased velocity, blood pooling, early fixation, and delayed fixation. In stage II, there is increased early fixation and delayed fixation with normalization of velocity and pooling. In stage III, there is decreased velocity and blood pooling with normalization of early and delayed fixation.[133,134]

Response to anesthetic sympathetic blockade is supportive of the diagnosis. However, nonresponsiveness does not preclude the diagnosis. Additionally, radiographic and bone scan findings are not entirely specific to RSD. The diagnosis is clinical with supportive diagnostic tests.

Prevention is the key, because once RSD develops, it may be very difficult to treat. Early mobilization is the mainstay in prevention. Once RSD develops, early physical therapy and occupational therapy should be initiated. Active and passive range-of-motion exercise of the involved extremity is performed. Modalities such as heat, cold, or TENS units may be tried for local pain control to help promote function or allow exercise to proceed. Desensitization therapy with contrast baths and friction massage may be started.

Tricyclic antidepressants such as amitriptyline may be used for analgesia. Sedative effects may improve sleep. Also, if depression is present, these medications will be of benefit. If this regimen is unsuccessful, a series of anesthetic sympathetic blocks may be tried. If improvement occurs, blocks may be periodically continued. If no improvement occurs after three to five blocks given daily or every other day, then sympathetic blockade is stopped.[132] Oral prednisone may be tried with 2- to

4-day intervals at dosages of 60 to 80 mg, 40 to 60 mg, and 30 to 40 mg in four divided doses.[133] This is followed by tapering as follows: 2- to 3-day intervals of 40, 30, 20, 10, and 5 mg given each morning and then discontinued. Kozin and associates[133] studied 64 patients with RSD and reported good results with an oral prednisone bolus in 90% of those with RSD and a positive bone scan.

Other medical treatments have included nitroglycerin, nifedipine, phenoxybenzamine, prazosin, propranolol, and intranasal calcitonin.[135] If the problem becomes chronic, a multidisciplinary pain approach is necessary. Treatment goals should be improved function and quality of life.

Other Medical Causes. Amputees may suffer from medical conditions that affect the general population. Degenerative arthritis or other rheumatologic disorders may present with symptoms in the residual limb. These conditions should be considered in the differential diagnosis of residual limb pain.

Referred Causes. In addition to local causes, conditions referring pain into the residual limb are in the differential diagnosis of residual limb pain. Radiculopathy has been reported as a cause of residual limb pain in an amputee.[136] Diagnosis may become difficult secondary to inability to test sensation, strength, or reflexes for a particular neurologic level because of the amputation. Electrodiagnostic studies and radiographic imaging may be required. Residual limb pain has been reported[137] after epidural anesthesia. The pain occurred in the residual limb at the time of epidural anesthesia. With increased anesthetic medication given epidurally, the pain subsided. However, the pain returned postoperatively. A TENS unit was helpful in reducing the pain, and complete pain resolution occurred in 3.5 hours after onset. Other referred causes in the differential diagnosis include spinal stenosis, facet syndrome, piriformis syndrome, and myofascial pain syndrome.

Residual Limb Pain: Extrinsic Causes

Extrinsic causes of residual limb pain refer mainly to residual limb socket interface problems. These problems result from poor socket fit or alignment. Poor prosthetic fit is the most common cause of residual limb pain.[36]

Localized Increased Pressure. A poorly fitted prosthesis with increased pressure may cause focal pain. Examination of the residual limb often will reveal abrasions, ulceration, or callosities. Increased pressure over a bony prominence may result in bursal development.[126,128] In these cases, pain is worsened with prosthetic use. Upon removal of the prosthesis after ambulating, prolonged erythema may be present. Treatment consists of socket relief or, if unsuccessful, fabrication of a socket.

Bursal Enlargement or Inflammation. A socket with increased pressure, or with inadequate suspension with increased shear over a bony prominence, may result in development, enlargement, or inflammation of a bursa. The amputee will complain of pain during prosthetic use. Examination will reveal signs of localized increased pressure or shear and a tender, fluctuant mass is palpated. Usually, the bursa is warm and erythematous. Treatment consists of correcting the cause and symptomatic treatment of the bursitis. If the bursa developed from increased pressure or poor suspension, a socket relief is made or suspension is corrected, respectively. Symptomatic treatment involves pain relief and antiinflammatory measures. In most cases, symptoms are relieved with cessation of prosthetic irritation. Local ice treatment over the inflamed bursa may be helpful; in some cases, a corticosteriod bursal injection may be required.

Loose Socket. A loose socket may cause pain over bony prominences or skin breakdown.[126,128] The above-knee amputee often presents with pubic ramus and distal pain. The below-knee amputee may present with pain at the hamstrings, inferior patellar pole, fibular head, and distal residual limb, which is worsened with prosthetic ambulation.[128] Examination after ambulation often will reveal areas of increased pressure or shear. The loose socket may occur with residual limb volume changes from generalized weight loss, local muscle atrophy, or normal residual limb maturation. Treatment consists of proper education and use of stump socks to provide an optimal prosthetic fit. Socket modifications, a new socket, or liner may be necessary.

Bell Clapping. Bell clapping results from distal residual limb shrinkage and presents with poorly defined residual limb pain and choking.[126] With ambulation, the residual limb strikes the anterior socket wall during swing phase, and choking occurs from proximal constriction. This may be precipitated by use of stump socks to improve distal fit. However, because the proximal residual limb has not proportionately changed, the addition of extra stump socks causes proximal constriction.

Often, careful questioning can reveal a history of residual limb changes with pain on prosthetic use. The amputee may complain of a loose socket distally with the residual limb hitting the socket walls with ambulation. Physical examination will

reveal a conical residual limb with signs of distal edema, skin irritation, and proximal constriction. When bell clapping is suspected, a xeroradiograph can be obtained to evaluate socket fit. A xeroradiograph is a special radiograph that provides a clear picture of the socket and soft tissue interface. With bell clapping, xeroradiography of the residual limb in the socket demonstrates the proximal constriction and distal socket looseness and confirms the clinical diagnosis.

Treatment requires socket modification. Posterior filling of the socket to eliminate the distal looseness may help.[126] If unsuccessful, or socket modifications are impractical, a new socket should be fabricated.

Choking. Choking results in proximal constriction and distal edema. The amputee may complain of pain. There may be skin breakdown. Treatment consists of socket modification or refabrication to provide total contact without excess proximal constriction.

Phantom Limb Sensation

Phantom limb sensation is defined as any sensory phenomenon except pain, referred to an absent limb or portion of a limb. After amputation, 80% to 100% of amputees experience phantom sensations at some time in their lives.[138] A recent study revealed that within the first 24 hours postoperatively, about one third of new amputees experience phantom sensations. The percentage who experienced phantom sensation at 8 days, 6 months, and 2 years postoperatively were 84%, 90%, and 71%, respectively.[139]

While a wide variety of sensations are described, phantom sensations may be divided into three categories:

1. Kinesthetic sensations are those related to the phantom limb posture, length, and volume.[138,140] The phantom limb may be perceived as behaving just like a normal limb, such as bending at the knee with sitting.[35] Eight days postoperatively, normal volume and length were perceived in 48% and 55% of amputees, respectively.[138]
2. Kinetic sensations are sensations of movement. Both willed and spontaneous movement may be perceived as occurring.[138,140] Willed movements are often simple flexion and extension movements of a phantom foot or hand. The incidence of kinetic sensations at 8 days, 6 months, and 2 years are reported as 36%, 37%, and 24%, respectively.[138]
3. Exteroceptive sensations are surface sensations such as touch, temperature, pressure, and pruritus. Amputees may report a wide variety of sensations, such as itching, tingling, and heat. In one study,[139] the incidence of having only exteroceptive sensations were 13% and 14% at 8 days and 6 months postamputation, respectively.

Phantom sensations often undergo telescoping. Telescoping is the perceived shortening of the phantom limb. Often, more distal portions of the limb are perceived to remain with disappearance of the more proximal.[140] Hence, an above-knee amputee may experience telescoping with a phantom foot perceived directly at the end of the residual limb. Telescoping is usually completed in one year and occurs in 25% to 75% of cases.[138] Usually, only a painless phantom shortens; conversely, phantom pain attacks may perceptually lengthen a phantom limb.

Phantom limbs often undergo fading. Fading refers to a change in the frequency and intensity of phantom episodes. Approximately 70% of amputees have marked phantom sensations immediately postoperatively. After 10 years, only 29% of amputees experience marked phantom sensations.[138]

There is no specific treatment for phantom sensations, which may be regarded as a normal occurrence after amputation. However, the experience may be frightening to the new amputee who is not educated regarding this phenomenon. Some amputees may think they are becoming insane. To avoid psychological discomfort, preoperative education regarding phantom sensations should be provided. Additionally, having experienced amputees relate their own phantom sensation experiences is beneficial.

Phantom Limb Pain

Phantom limb pain is defined as pain in an absent limb or portion of a limb. Phantom limb pain must be differentiated from phantom sensation and residual limb pain. Problems with the definition are differences in pain tolerance, threshold, and individual interpretation. Furthermore, past studies have not always differentiated residual limb pain from phantom pain and phantom limb sensation, thereby creating problems in evaluating the incidence and treatment of phantom pain.

The incidence of phantom limb pain ranges from 2% to 97%.[140] In a survey of 5,000 American veteran amputees with 55% responding, 78% reported

phantom limb pain.[141] Another study evaluated the incidence of phantom limb pain at 1 week, 6 months, and 2 years and found the incidence of phantom pain was 72%, 65% and 59%, respectively. Duration and frequency of phantom pain episodes decreased with time, with 21% experiencing pain daily, but none constantly at 2 years.[138] Phantom pain episode durations are usually seconds to hours and rarely last weeks to months.[142] Jensen and Rasmussen[138] speculate that 5% to 10% of amputees have severe, persistent phantom limb pain. While phantom pain may persist, some individuals have spontaneous remission.[140]

Phantom pain tends to localize distally.[138,140] All types of painful stimuli have been reported, although at 6 months postoperatively, most individuals with phantom pain reported a burning, squeezing pain.[143] A minority reported a painful distorted position similar to the position prior to surgery.[138] The perceived pain can be intermittent or continuous. A variety of factors can modify the pain. Factors making the pain worse include yawning, micturition, fatigue, sleeplessness, anxiety, weather changes, pain from other body sites, stimulation of another body part, heat, cold, and a poorly-fitted prosthesis.[138,140,142,143] Factors mitigating the pain include mental distractions, rest, emotional pleasure, massage, elevation of the residual limb, electrical stimulation, cold, heat, and a well-fitted prosthesis.[138,139] The presence of preamputation pain correlating to phantom pain is controversial.[140,143] The incidence of phantom pain reported as being similar to preoperative limb pain ranges from 12.5% to 79%.[144] Jensen and colleagues[143] prospectively found 36% of amputees had phantom pain similar to their preoperative pain. At 2 years from amputation, 10% had phantom pain similar to preoperative pain.[143] Additionally, long-term residual limb pain has correlated with an increased risk for phantom limb pain.[143] Factors identified as having no significant effect on the development of phantom limb pain in adults include site and level of amputation, cause of amputation, sex, age, and civilian vs military etiology of amputation.[139,142,145] Furthermore, psychopathologic personalities or psychiatric abnormalities are not the etiology of phantom pain.[146]

Pathophysiology. The pathophysiology of phantom limb pain is unknown. Current theories have not adequately explained all the observed phenomena seen in phantom pain patients. Current theories may be divided into peripheral, spinal cord, supraspinal, and neuromatrix.

The peripheral theory postulates spontaneous discharges from neuromas traveling through the somatosensory system to the brain.[35] The impulses may be interpreted as phantom sensations or pain. Various observations have suggested a peripheral role. Phantom limb sensation may be modulated by residual limb manipulation. Phantom pain has been temporarily abolished by local residual limb anesthesia. Residual limb revisions or neuroma resection may transiently decrease phantom pain. Tapping of neuromas may increase phantom pain.[138] Additionally, axons in the neuroma generate spontaneous impulses. There is abnormal sensitivity to mechanical and chemical stimuli.[140] However, treatments aimed at peripheral solutions have not given long-term relief. Neuroma resections and dorsal rhizotomies have not been successful in permanent relief, which suggests more proximal structures are involved in phantom pain generation.

Changes in the spinal cord may play a role in phantom pain. After nerve transection, changes take place in the dorsal horn. There is atrophy of primary afferent terminals, postsynaptic inhibition, and changes in concentrations of neuropeptides.[138] Decreased postsynaptic inhibition by loss of afferent fibers may lead to pain enhancement at the spinal level.[36] Dorsal horn neurons that have lost afferent input may begin to respond to nearby intact afferents, resulting in an expanded receptive field. An expanded receptive field is thought to explain stimulation of the residual limb, which modifies phantom sensation or pain.[138] Additionally, phantom pain or sensation may be related to spontaneous discharges from dorsal horn neurons that have lost primary afferent input.[35] While dorsal horn changes may have a role in phantom pain, these changes cannot adequately explain the onset of phantom limb pain in a low thoracic (T-11) paraplegic with complete sensory loss who underwent leg amputation. Supraspinal areas may play a role in phantom pain. Nociceptive specific neurons exist in the cortex, and the plasticity that occurs in nociceptive neurons in the periphery and spinal cord are postulated to ascend centrally to nociceptive and antinociceptive systems.[138] Observations suggesting a central role have been described. Bursting activity in thalamic neurons was demonstrated in a paraplegic suffering from pain in regions innervated below the level of complete spinal cord injury.[35] Additionally, thalamic stimulation has mitigated phantom pain in some individuals. These observations suggest a thalamic role. The parietal lobe may also be involved, as in one case, a right hemisphere lesion resulted in disappearance of phantom pain. Supratentorial areas are further postulated to play a role secondary to the need for in-

tegration of complex afferent input and the wide variety of sensations and pain described in phantom phenomena.[138] However, ablative treatments of the thalamus and cortex have not been successful in treating phantom pain.[35]

Melzack[35] has a newer theory to explain phantom sensation and pain. He hypothesizes there exists in the brain a network of neurons that responds to sensory stimulation based on previous sensory experiences and generates a perception of the body and its limbs. Melzack terms the neuron network as the neuromatrix and based on the report of phantom sensations in congenital amputees, he postulates that the neuromatrix is genetically determined and "prewired" before birth. For example, the existence of a phantom hand in a congenital amputee would suggest that the neuromatrix was prewired with the body having a hand, although physically the hand was congenitally absent. Sensory phenomena are interpreted not only by the pure sensory impulses through the somatosensory system, but also emotionally and on past experiences to the same or similar phenomena.

The wide interpretation of sensory phenomena suggests the neuromatrix is influenced by experiences and consists of more than the somatosensory system. Melzack[35] states that the neuromatrix is composed of at least three parallel systems: (1) somatosensory, (2) limbic, and (3) cortical. The limbic system provides emotional and motivational responses. The cortex, with probable involvement of the parietal lobe, provides recognition of the body as one's own. Support for parietal lobe involvement are cases of neglect of the hemiparetic body in persons with right parietal lobe cerebrovascular accidents. The right parietal lobe infarct patient may not recognize the left leg as self or not shave the left side of his face. Conversely, in the amputee with phantom limb sensation, the neuromatrix may still have a body concept that includes the amputated leg.

The generated pattern providing the body image and as one's own is termed the neurosignature. The pattern that is "prewired" to give the body image is influenced by experience,[35] which may influence the strength of synaptic connections. The pattern of connectivity among the neurons of the neurosignature is affected by which neurons are involved, the number of synapses, and the types and strength of the synapses.[35]

Normally, afferent input would enter the neuromatrix. The neuromatrix would place its neurosignature on the input along with the integration of the sensation, perception, and emotional response. This information travels to another part of the brain for transformation into a conscious experience.[35] In phantom sensation or pain, the neurosignature still incorporates the amputated limb as part of one's own body. Spontaneous impulses from transected nerve axons, dorsal horn neurons, or thalamic neurons may enter the neuromatrix, resulting in a wide array of conscious sensory interpretations. Pain may occur from strong synaptic connections resulting from pain prior to amputation. Severe pain continuing to the time of amputation has been associated with phantom pain.[144] Pain may also occur as the neuromatrix fires in impulse bursts from the loss of usual sensory input from the amputated limb. The bursts may create a burning sensation. Furthermore, the neuromatrix may send impulses for the amputated limb to move. The lack of response may result in more frequent, stronger impulses perceived as cramping or shooting pain.[35]

The neurosignature may change as neurons in the neuromatrix previously associated with the amputated limb reassociate with other synapses. The changed pattern may explain "fading" of the phantom limb.[35] The neuromatrix and neurosignature theory provides a plausible explanation of phantom pain and sensation but requires further research to develop and test the hypothesis.

Treatment. Overall treatment of phantom limb pain has been unsuccessful for long-term relief. In the treatment of over 8,000 amputees with phantom pain, Sherman[147] reported that only about 7% of them received some benefit from treatment. Problems in evaluating treatment have arisen secondary to research studies not differentiating residual limb pain from phantom pain, and other study design flaws.[140] Current treatments consist of a variety of methods that may be used alone or in combination. Sherman, Sherman and Gall[37] reported that their surveyed care providers prescribed 50 different treatment methods.

Medical. Ideally, the selection (and use) of a medication is based on knowledge of how the pharmacologic characteristics of the medication will affect the pathophysiology of the disorder. Unfortunately, since the pathophysiology of phantom limb pain is unknown, there is an ambiguous direction in pharmacologic treatment with many agents rarely exceeding the effects of placebo administration.

Nonsteroidal antiinflammatory drugs, (NSAIDs) the most commonly used analgesics in chronic pain,[140] are usually tried first.[148] Based on the benefit in arthritic patients whose pain is made worse with changes in barometric pressure, NSAIDs may be useful in the treatment of phantom limb pain that is worsened by weather changes.[147]

Narcotics are not recommended in the treatment of phantom limb pain except in very limited circumstances, because they usually result in increased dosage requirements, increased dependence, poorer control, and increased depression.[140] But low dose narcotic agents, in combination with an antidepressant, may be helpful in increasing function.[148] Patient selection is based on refractoriness to other treatments, no drug dependence behavior, no past history of drug dependence, and increased function with narcotic treatment. Sedative/hypnotic medications have no role in the treatment of phantom pain, as they are habit forming and may increase depression.[140]

Anticonvulsants, based on membrane stabilizing properties, have been used in the treatment of phantom pain.[148] Most commonly, carbamazepine has been used with a 22% to 77% success rate. Other agents tried include phenytoin, valproate, and mephenytoin.[140] Anticonvulsants may be useful for a cramping, shooting pain.[148]

Tricyclic antidepressant medication has been used based on an analgesic serotonergic mechanism.[149] Serotonin is postulated as having a central pain inhibitory mechanism.[148] Additionally, the antidepressant effects are beneficial for those individuals who are concomitantly depressed.

Neuroleptics, such as butyrophenone, phenothiazines, and benzamides have been used. Frequently, chlorpromazine has been used. Neuroleptics are postulated to alter centrally the projection and interpretation of pain stimuli.[148]

Sympathetic beta-blockers such as propranolol and atenolol may be given. Propranolol at 40 mg/d has been used.[140] These medications are hypothesized to centrally increase serotonin concentrations.[148] Beta-blockers may be beneficial for a burning, throbbing pain.[147]

Baclofen is a gamma aminobutyric acid agonist and has been shown to depress trigeminal spinal nucleus activity in cats, and has been used successfully in the treatment of trigeminal neuralgia. In phantom pain, baclofen may be useful alone, or in combination with phenytoin or propranolol.[148]

Mexiletine, a sodium channel blocker and cardiac antiarrhythmic, has been reported to provide relief from phantom pain. Davis[36] reports an 87% success rate in significantly improving phantom pain in 45 patients.

Intravenous calcitonin has met with success in treating phantom pain. Jaeger and Maier,[150] in a double blind, crossover study, obtained greater than 50% relief of early postoperative phantom pain in 19 of 21 patients, and complete relief in 16 of 21 patients treated with intravenous calcitonin. At 1 year postamputation, 8 of 13 surviving patients had greater than 75% pain relief. Side effects were transitory and included headache, vertigo, nausea, vomiting, enhanced phantom sensation, drowsiness, and hot or cold flashes. The mechanism of pain relief is unknown. Jaeger and Maier postulate that a central, sertonergic mechanism exists and conclude that intravenous calcitonin may be beneficial in the early postoperative relief of phantom limb pain.

Local Anesthetic Blocks. Local anesthetic blocks are mainly used as a diagnostic tool to evaluate for neuromas, sympathetically mediated pain, and pain from referred sources. Trigger point injection for myofascial pain syndrome with radiation into the phantom extremity may be helpful. For phantom pain resembling RSD symptoms, a sympathetic block may be helpful.[138,147]

Surgical. Neuroma resection is performed only for symptomatic neuromas. It may give temporary relief for approximately 3 weeks until the neuroma regrows with subsequent return of symptoms.[140] Neuroma resection is not done to alleviate phantom limb pain. The indications were previously discussed under Residual Limb Pain: Intrinsic Causes.

Dorsal root entry zone (DREZ) lesioning is a new method. The procedure may utilize selective thermocoagulation or electrocoagulation of the substantia gelatinosa. Saris, Iacono, and Nashold[151] performed DREZ on 22 patients and had good results in 36%. For 9 patients with phantom pain alone, good results were obtained in 6. Five of 6 patients with root avulsion had good results. Poor results were obtained in those with phantom pain and residual limb pain or residual limb pain alone. DREZ will require further investigation before conclusions regarding its efficacy in phantom limb pain can be determined.

Two sites of stimulation have been used in deep brain stimulation: (1) the periaqueductal and periventricular gray and (2) the lateral thalamic and internal capsule. Periaqueductal and periventricular gray stimulation is postulated to decrease pain by increased endorphin release. This site of stimulation has not been effective for long-term relief with chronic deafferentation pain.[148] Lateral thalamic and internal capsule stimulation has resulted in satisfactory pain relief in some patients.[140] This site has been used along with TENS unit stimulation.[148] Further study is needed to evaluate the effectiveness of this method.

Dorsal cord stimulation is based on the gate theory of pain relief. This method has resulted in an immediate 80% success rate, which dropped to

20% after 1 year.[148] Further study is needed. Ablative procedures that have been tried include dorsal rhizotomy, anterolateral chordotomy, thalamotomy, and cortical resections. Overall, these procedures have not been successful.[138,140,147] Ablative procedures are not recommended in the treatment of phantom limb pain.

Psychological. Hypnosis has been utilized in chronic pain, but its efficacy is unknown.[148] Multidisciplinary pain clinics that utilize an operant behavioral approach have not usually been necessary for the treatment of phantom pain.

Biofeedback requires prolonged training and has not been widely used. Biofeedback may be useful for a cramping phantom pain associated with increased muscle tension.[147] A valuable technique used in conjunction with other treatments is relaxation. Relaxation techniques may be helpful for phantom pain made worse with stress or increased muscle tension.

Preoperative education helps in preventing maladaptive behavior and decreases patient stress.[152] Preoperative education should include discussion of the preoperative, operative, postoperative, rehabilitative, and prosthetic processes. Potential complications should be explained. Postoperative pain, phantom sensation, and phantom pain issues and management should be discussed. Often, an experienced amputee screened by the multidisciplinary team may provide the patient with valuable education and insight.

Phantom pain is not due to a psychologic disorder. Any psychotherapy for the patient with phantom pain should be for preexisting psychologic disorders. Psychotherapy is not used for phantom pain alone.

Other. Range-of-motion exercises and massage of the residual limb may provide temporary relief. Additionally, the use of heat or cold modalities may provide temporary relief.

Acupuncture may provide pain relief by increasing endorphins or serotonin.[148,153] The successful use of acupuncture along with TENS and electroacupuncture to decrease phantom pain has been described.[153] However, the relief may be only temporary.[36,142] Further trials are needed to establish acupuncture's efficiency.

TENS has good success up to 6 months, but only 25% of those treated have relief at 12 months.[140] The main complication related to TENS is allergic reactions to electrode pads attached to the skin. TENS trials may require use of different electrode placements to achieve analgesia. Electrodes may be tried on the residual limb or contralateral limb.[138] As TENS has minimal complications, a trial should be given.

Vibration or percussion of the residual limb has been tried in the past and may be effective. These methods are not often used today. Early prosthetic fitting and ambulation have been reported to decrease phantom pain.[58] Additionally, rigid dressings have also been reported to reduce phantom pain.

Prevention

Overall, treatment has not been very successful; therefore, the prevention of phantom pain is an important measure. Bach and associates[154] controlled pain for 3 days prior to surgery by using a lumbar epidural blockade with bupivacaine or morphine. With this they found a deceased incidence of phantom limb pain. Another method used was insertion of a catheter into the nerve at the time of surgery, which provided pain relief postoperatively by continuous nerve sheath blockade. With this method, the patients in the study experienced no phantom pain.[155] Both of these studies had small sample sizes, which suggests further study is needed on the efficacy of these methods. However, pain control in the preoperative period to prevent phantom pain appears useful.

Treatment Summary

Phantom pain may be a difficult problem to treat in some amputees. The unknown pathophysiology and myriad treatments makes treatment decisions confusing and difficult. As any one treatment has not met with overwhelming success, it is necessary to try a variety of treatments or treatment combinations before a satisfactory result is found for an individual patient. Additionally, because of low success rates, initial treatments should have low morbidity, and treatments that may result in permanent deficits should be avoided. Furthermore, total treatment of the amputee optimizes overall outcome, which may influence chronic phantom pain problems. Early comprehensive multidisciplinary rehabilitation results in better acceptance and management of phantom pain and higher functional outcomes.[36] Additionally, comprehensive treatment may be helpful in prevention of phantom pain. The following guidelines may be helpful in the prevention and management of phantom pain.

Preoperative education is provided when possible. A multidisciplinary team approach to provide comprehensive treatment is utilized. Preoperative pain control is instituted to decrease the risk of de-

veloping phantom pain. If not contraindicated, an IPOP or rigid dressing is utilized. Normal postoperative residual limb pain is treated; and if pain continues, it must be established whether the pain is residual limb pain, phantom sensations, phantom pain similar to preoperative pain, phantom pain, or referred pain. Residual limb pain and referred pain causes are important to identify because treatment is different for each and often has a higher success rate. Range-of-motion exercises, residual limb massage, and desensitization are often helpful. The prosthesis or rigid dressing must fit properly. A TENS trial should be initiated. Pharmocologic therapy starts with an NSAID, and if phantom pain is similar to preoperative pain, the same agents that were successful in the preoperative pain management of the patient may be used. For other cases of phantom pain, carbamazepine or a tricylic may be started. Additionally, other agents may be tried dependent on the type of pain. Mexiletine and IV calcitonin may hold promise, but further studies are needed. For those with phantom pain resembling RSD symptoms, a sympathetic block may provide relief when the above treatments are unsuccessful. The multidisciplinary treatment team must meet and discuss how pain is impacting on functional gains and quality of life issues. Psychologists need to assess for depression, which frequently occurs in individuals with chronic pain, and utilize psychological techniques previously discussed to assist phantom pain control. A log should be kept regarding phantom pain episodes and factors modulating the pain. The log is analyzed for modulating factors to determine efficacy of interventions.[147] In limited circumstances, a narcotic along with an antidepressant may be used, but only if other measures have failed and the earlier-stated indications and contraindications are followed. Ablative surgical procedures should be avoided. Surgical procedures such as dorsal column stimulation and DREZ require further study before conclusions regarding their efficacy can be reached. Usually, multidis-ciplinary pain clinics are not required, except in cases when phantom pain is chronic, unresponsive to standard treatment, and influenced by environmental entities.

THE MULTIPLE AMPUTEE

The amputee with multiple amputations poses challenges to the rehabilitation team. It is important for military rehabilitation professionals to be cognizant of the special needs of these casualties. At Fitzsimons General Hospital during the Vietnam War, over 500 soldiers with major amputations were treated, with 342 sustaining loss of part of a leg and 44 with loss of both legs.[57] Three were triple amputees and 18 of the 44 had bilateral transfemoral amputations. Brown[156] describes these multiple amputees as complicated casualties often with concomitant loss of vision, fractures, and systemic infections. All of these casualties sustained their wounds as a result of high-explosive or high-velocity missile injuries. This report by Brown and the subsequent discussion by Commander Donald Rohren of the Oakland Naval Hospital (described in Brown's paper) provides an excellent insight into military rehabilitation of the multiple amputee.

The traumatic battle casualty who subsequently loses one or more limbs was described as demonstrating an initial shock and sense of relief that he remained alive following his wounding.[156] This was followed by manifestations of depression, anxiety, and hostility. These multiple amputees required substantial multidisciplinary rehabilitation care, which included a physician, prosthetist, physical therapist, and an occupational therapist. These casualties frequently required revision of their residual limbs. Thirty-four of the 88 required skin grafting, and 48 of the 88 required definitive revisions. There were 12 knee disarticulations and 8 of those required revisions to an above-knee level.[156] Seven stumps had persistent drainage that required sinus tract excisions, and 4 stumps had bone spur formation that required excisions. These figures demonstrate that many war-injured amputees require multiple surgical procedures in order to obtain a healed, painless, and functional residual limb.

Brown[156] reported that due to the residual limb problems in his series of bilateral leg amputees, prosthetic fitting and training were often delayed for weeks or months. Therefore, rehabilitation efforts focused on strengthening and maintenance of joint range of motion. Twice-a-day exercises to condition remaining muscles for ambulation were instituted.

At the earliest possible time, prostheses were fitted and ambulation training was begun using crutches and parallel bars. Brown[156] felt that for the bilateral transfemoral amputee, "stubby prostheses" were optimal first devices. These short, rigid prostheses with no knee joints are advantageous in

the early training of amputees, particularly with respect to development of balance.

Functional activities were vigorously addressed. Community ambulation and automobile use were taught. In addition, a skiing program was incorporated into the rehabilitation treatment plan. Over 100 of the amputees who were treated skied during 1968 and 1969 using adaptive skiing aids. These war-injured casualties developed confidence and a sense of accomplishment through pursuit of this activity. In addition, other recreational activities, such as swimming, scuba diving, and water skiing were available.[156] The Fitzsimons General Hospital amputee rehabilitation program stressed treatment of the amputee as a whole individual with the goal of returning to an optional level of function in many functional and vocational areas.

In Brown's report,[156] Rohren discussed the Fitzsimons amputee care, contrasting it with amputee rehabilitation at the Oakland Naval Hospital. Rohren felt that the focus of amputee rehabilitation should be on the gait training and exercise, and he, therefore, felt that allowing amputees time off for recreational training detracted from optimal basic rehabilitation training, and thus prolonged their rehabilitation. Rohren echoed the complexity of the war injured amputee. In addition, he reported that convalescent leave impeded optimal stump wrapping and resulted in excessive weight gain, leading to prolonged rehabilitation.

Dunlap[157] reported a case of a combat wounded, bilateral leg amputee who sustained a right transfemoral amputation and a left hip disarticulation. A long rehabilitation course was required for ambulation training. The difficulties in fitting the amputee with two prostheses were described.

Canty[158] described amputee care during World War II at the United States Naval Hospital, Mare Island, Vallejo, California. This hospital was the first Armed Service Amputation Center established. Over 2,500 amputees were treated and rehabilitated with a reported 90% success rate. The rehabilitation program began with proper stump conditioning by means of surgical procedures, stump wrapping, and exercises. Physical therapists began bed exercises early in the casualty's course of rehabilitation. Occupational therapists provided the soldier with a variety of arts, skills, and hobbies during recuperation. Chaplain support and recreation departments also supported the casualty. Group support was provided by round table discussions, which gave the amputee valuable psychological support. As the casualty improved, aggressive physical training, including swimming, was stressed. Gait training with a prosthesis progressed from posture and balance training to walking. Prevocational training, driving, and dancing and various sports and games were introduced into the rehabilitation program, facilitating optimal adjustment to the new disability. Competition between amputees in ambulation was encouraged, which built a spirit of competition and improved functional outcomes. Canty[158] stated that a prosthetic device must be aligned and fitted properly so that the amputee can wear it without discomfort. Prostheses must also be light, strong, durable, economical in cost, and cosmetically acceptable. Canty also reported a case of a naval pilot who lost a leg, was rehabilitated, and returned to flying duty in the forward area. After the war this amputee was discharged from the service, following which he successfully operated his own commercial airline.

Ambulation for the bilateral leg amputee has been extensively addressed in the literature for the older dysvascular amputee.[159–165] Traumatic bilateral leg amputees have a better outcome in terms of ambulation in general than do elderly dysvascular amputees.[161] Kerstein and associates[164] found that elderly dysvascular amputees with bilateral amputations required an average 30 weeks of rehabilitation, but that 50% of bilateral transfemoral amputees eventually walked, although usually only for short distances and with ambulatory aids. Hamilton and Nichols[166] reported the need for inpatient rehabilitation for these patients. The younger traumatic bilateral amputee is more likely to ambulate; however, the energy cost of ambulation is much higher than for their able bodied counterparts. For the bilateral transfemoral amputee, "stubbie" prostheses are used initially with the feet set in a wider stance to provide a wider base of support.[159]

In the case of a quadruple amputee,[167] ADL training along with special wheelchair drive adaptations were required. This individual was able to become an independent ambulator with prostheses and achieved independence in all ADLs, including driving a car.

In summary, the war injured bilateral lower limb amputee can usually achieve a higher level of ambulatory independence than can older, dysvascular amputees. During an intense, prolonged war, multiple amputees must be anticipated. These soldiers present more difficult rehabilitative needs, and frequently require a prolonged course of inpatient rehabilitation, often due to other coexistent problems, such as nerve injuries, fractures, and so forth.

VOCATIONAL OUTCOMES OF AMPUTEES

The complete rehabilitation of an amputee requires the achievement of all functional goals, so that when possible, the amputee will assume an expected societal role that involves productive work. This is particularly important for the war-injured soldier who will have many productive years remaining after injury.

Statistics regarding amputees who return to active duty are not readily available, but a review of data from a U.S. Army Physical Evaluation Board over a 9-year period (1981–1989), revealed that only 2.3% of all amputees returned to active duty. Historically, during times of prolonged major conflict, amputees were utilized to perform many non-combat tasks. In World War II, amputees were sometimes trained in prosthesis fabrication and utilized in military hospitals.[3] The British Royal Air Force retained amputees on active duty because they found that it was more costly and time consuming to retrain aircraft mechanics and crewmen than it was to retain amputees.[168,169]

The literature concerning vocational outcomes of war-injured amputees is limited.[170–172] In a follow-up study[171] of amputees from the Vietnam War, it was found that when comparing the social and vocational outcomes of these amputees to those of noninjured Vietnam veterans, the amputees fared less well. The amputees showed twice the unemployment rate, earned less money, held more blue collar jobs, and obtained fewer college degrees than their noninjured counterparts. These results underscore the need for emphasis on vocational rehabilitation. Steinbach[170] described the Israeli experience in the rehabilitation of war injured amputees and pointed out the importance of vocational counseling as soon as possible after the injury. He also reported that 96% of their amputees at discharge had vocational plans with 28% returning to their previous jobs. Unfortunately, the percentage who returned to active duty was not specified. Ryan and colleagues[172] conducted a follow-up study of World War II amputees who had been treated by naval physicians. These authors followed 200 amputees and found that 78% were working or pursuing higher education. They again pointed to the need for addressing vocational issues while the injured soldier convalesced at a military hospital, including driving instruction.

The civilian experience relates similar findings and also highlights intervention strategies for improving vocational outcome. The literature[173,174] suggests that amputees do have higher unemployment rates than their able-bodied counterparts. Only a small percentage of amputees return to their previous jobs.[173] The reasons for reduced vocational outcomes were address by Sheikh[175] in a study of limb injuries, including amputations. Surprisingly, Sheik found that the exact type of limb injury (fractures, amputations, or soft tissue injuries) had little if any effect on vocational outcome. However, variables such as motivation, low level of disability, short duration of unemployment, a vocational retraining program, and low unemployment in the general population, strongly influenced return to work. Some of these variables can potentially be modified through appropriate rehabilitation. Millstein et al[49] in their review of 1,010 Canadian amputees, found that 87% of lower limb amputees returned to work. Unilateral amputees were more likely than multiple amputees to return to work. Most of these Canadian amputees were casualties of work related accidents. They found that younger ages, comfortable and routine prosthesis use, and provision of vocational services were associated with return to work. Phantom pain and residual limb pain, along with multiple amputations, negatively impacted on return to gainful employment. Helm and colleagues,[176] in their series of amputees, found that prosthetic fit and pain were important variables affecting amputee function. The above authors, and others, strongly support early vocational intervention.[177–179] Brown[179] notes that simulated work tasks coordinated by occupational therapists and other rehabilitation professionals can help amputees develop skills that will be used in pursuing alternative careers. Part-time return to duty, if this is possible during convalescence, can also be advantageous.[180,181]

REFERENCES

1. Stern PH. The epidemiology of amputations. *Phys Med Rehabil Clin North Am.* 1991;2(2):253–261.

2. Peterson LT. Amputations: Historical note. In: Mullins WS, Cleveland M, Shands AR, McFetridge EM, eds. *Surgery in World War II: Orthopedic Surgery in the Zone of the Interior.* Washington, DC: Office of The Surgeon General, US Department of the Army; 1970.

3. Peterson LT. Administrative considerations in the amputation program. In: Mullins WS, Cleveland M, Shands AR, McFetridge EM, eds. *Surgery in World War II: Orthopedic Surgery in the Zone of the Interior*. Washington, DC: Office of The Surgeon General, US Department of the Army; 1970.

4. Brown PW. Rehabilitation of bilateral lower extremity amputees. *J Bone Joint Surg Am.* 1970;52(4):687–700.

5. Dillingham TR, Spellman NT, Braverman SE, et al. Analysis of casualties referred to army physical medicine services during the Persian Gulf conflict. *Am J Phys Med Rehabil*. 1993;72(4):214–218.

6. Peterson LT. General clinical considerations of amputation. In: Mullins WS, Cleveland M, Shands AR, McFetridge EM, eds. *Surgery in World War II: Orthopedic Surgery in the Zone of Interior.* Washington, DC: Office of The Surgeon General, US Department of the Army; 1970.

7. Malone JM, McIntyre KE Jr, Rubin JR, Ballard J. Amputation for trauma. In: Moore WS, Malone JM, eds. *Lower Extremity Amputation.* Philadelphia: WB Saunders; 1989:320–329.

8. Sanders R, Helfet D. Trauma. In: Bowker JH, Michael JW, eds. *Atlas of Limb Prosthetics: Surgical, Prosthetic, and Rehabilitation Principles.* 2nd ed. St. Louis: Mosby-Year Book; 1992:19–24.

9. Bowker JH. The choice between limb salvage and amputation. In: Bowker JH, Michael JW, eds. *Atlas of Limb Prosthetics: Surgical, Prosthetic, and Rehabilitation Principles.* 2nd ed. St. Louis: Mosby-Year Book; 1992:17–18.

10. Gregory RT, Gould RJ, Peclet M, Wagner JS, et al. The mangled extremity syndrome (M.E.S.): A severity grading system for multisystem injury of the extremity. *J Trauma.* 1985;25:1147–1150.

11. Lange RH, Bach AW, Hansen ST Jr, Johansen KJ. Open tibial fractures with associated vascular injuries: Prognosis for limb salvage. *J Trauma.* 1985;25(3):203–208.

12. Helfet DL, Howey T, Sander R, Johansen K. Limb salvage versus amputation: Preliminary results of the mangled extremity severity score. *Clin Orthop.* 1990;256:80–86.

13. Bowker JH, Goldberg B, Poonekar PD. Transtibial amputation: Surgical procedures and immediate postsurgical management. In: Bowker JH, Michael JW, eds. *Atlas of Limb Prosthetics: Surgical, Prosthetic, and Rehabilitation Priniciples.* 2nd ed. St. Louis: Mosby-Year Book; 1992:429–452.

14. Friedmann LW. Rehabilitation of the lower extremity amputee. In: Kottke FJ, Lehmann JF, eds. *Krusen's Handbook of Physical Medicine and Rehabilitation.* 4th ed. Philadelphia: WB Saunders; 1990:1024–1069.

15. Bowen TE, Bellamy RF. Amputations. In: *Emergency War Surgery.* 2nd revision. Washington, DC: US Government Printing Office; 1988:273–279.

16. Thompson TC, Alldredge RH. Amputations: Surgery and plastic repair. *J Bone Joint Surg.* 1944;26(4):638–644.

17. Leonard JA Jr, Meier RH III. Prosthetics. In: DeLisa JA, Currie DM, Gans BM, Gatens PF Jr, Leonard JA Jr, McPhee MC, eds. *Rehabilitation Medicine: Principles and Practice.* Philadelphia: JB Lippincott; 1988:330–345.

18. Halar EM, Bell KR. Contracture and other deleterious effects of immobility. In: Delisa JA, Currie DM, Gans BM, Gatens PF Jr, Leonard JA Jr, McPhee MC, eds. *Rehabilitation Medicine: Principles and Practice.* Philadelphia: JB Lippincott; 1988:448–462.

19. Racy JC. Psychological aspects of amputation. In: Moore WS, Malone JM, eds. *Lower Extremity Amputation.* Philadelphia: WB Saunders; 1989:330–340.

20. Malone JM, Moore W, Leal JM, Childers SJ. Rehabilitation for lower extremity amputation. *Arch Surg.* 1981;116:93–98.

21. Mooney V, Harvey PJ Jr, McBride E, Snelson R. Comparison of postoperative stump management: Plaster vs. soft dressings. *J Bone Joint Surg.* 1971;53A(2):241–249.

22. Stern PH. Occlusive vascular disease of lower limbs: Diagnosis, amputation surgery and rehabilitation: A review of the Burke experience. *Am J Phys Med Rehabil.* 1988;67:145–154.

23. Harris RI. Syme's Amputation: The technique essential to secure a satisfactory end-bearing stump. *Can J Surg.* 1964;7:53–63

24. Bone GE, Pomajzl MJ. Toe blood pressure by photoplethysmography: An index of healing in forefoot amputation. *Surgery.* 1981;89(5):569–574.

25. Ramsey DE, Manke DA, Sumner DS. Toe blood pressure: A valuable adjunct to ankle pressure measurement for assessing peripheral arterial disease. *J Cardiovasc Surg.* 1983;24:43–48.

26. Robertson PA. Prediction of amputation after severe lower limb trauma. *J Bone Joint Surg Am*. 1991;73B(5):816–818.

27. Pinzur MS, Kaminsky M, Sage R, Cronin R, Osterman H. Amputation at the middle level of the foot. *J Bone Joint Surg Am*. 1986:68:1061–1064.

28. Greene WG, Cary JM. Partial foot amputation in children: Comparison of the several types with the Syme's amputation. *J Bone Joint Surg Am.* 1982;64(3):438–443.

29. Smith DG, Stuck RM, Ketner L, Sage RM, Pinzur MS. Partial calcanectomy for the treatment of large ulcerations of the heel, and calcaneal osteomyelitis: An amputation of the back of the foot. *J Bone Joint Surg Am.* 1992;74A(4):571–576.

30. Syme J. On amputation at the ankle joint. *London Edinbourough Monthly J Med Sci.* 1843:3(XXVI):93.

31. Harris RI. The history and development of Syme's amputation. *Artificial Limbs*. 1961;6:4–43.

32. Harris RI. Syme's amputation: The technique essential to secure a satisfactory end-bearing stump: Part I. *Can J Surg.* 1963;6:456–469.

33. Harris RI. Syme's amputation: The technique essential to secure a satisfactory end-bearing stump. *Can J Surg.* 1964;7:53–63.

34. Burgess EM, Romano RL, Zettl JH. *The Management of Lower-Extremity Amputations.* Washington, DC: US Government Printing Office; TR 10-6. 1969.

35. Melzack R. Phantom limbs. *Sci Am.* 1992;4:120–126.

36. Davis RW. Phantom sensation: Phantom pain and stump pain. *Arch Phys Med Rehabil.* 1993;74:79–91.

37. Sherman AR, Sherman JC, Gall GN. A survey of current phantom limb pain treatment in the United States. *Pain*. 1980;8:85–99.

38. Pinzur MS. New concepts in lower-limb amputation and prosthetic management. *Instr Course Lect.* 1990;39:361–366.

39. Lange LR. The lange silicone partial foot prosthesis. *J Prosthetic Orthot.* 1991;4:56–61.

40. Hayhurst DJ. Prosthetic management of a partial-foot amputee. *Inter-Clinic Information Bulletin.* 1978;17:11–15.

41. Doyle W, Goldstone J, Kramer D. The Syme prosthesis revisited. *J Prosthetic Orthot.* July 1993;5(3):95–99.

42. Esquenazi A, Torres M. Prosthetic feet and ankle mechanisms. *Phys Med Rehabil Clin North Am.* 1991;2(2):299–309.

43. Esquenazi A. Geriatric amputee rehabilitation. *Geriatric Clinics of North America.* Nov 1993.

44. Huang CT, Jackson JR, Moore NB, Fine PR, Kuhlemeier KV, Traugh GH, et al. Amputation: Energy cost of ambulation. *Arch Phys Med Rehabil.* 1979;60:18–24.

45. Waters RL, Perry J, Antonelli D, Hislop H. Energy cost of walking of amputees: The influence of level of amputation. *J Bone Joint Surg Am.* 1976;58(1):42–46.

46. Gonzalez EG, Corcoran PJ, Reyes RL. Energy expenditure in below-knee amputees: Correlation with stump length. *Arch Phys Med Rehabil.* 1974;55:111–119.

47. Epps CH Jr. Principles of amputation surgery in trauma. In: Evart CM, ed. *Surgery of the Musculoskeletal System.* Vol 5. 2nd ed. New York: Churchill Livingston; 1990:5075–5092.

48. Purry NA, Hannon MA. How successful is below-knee amputation for injury? *Injury.* 1989;20:32–36.

49. Millstein S, Bain D, Hunter GA. A review of employment patterns of industrial amputees: Factors influencing rehabilitation. *Prosthet Orthot Int.* 1985;9:69–78.

50. Harris RI. Amputations. *J Bone Joint Surg.* 1944;26(4):626–634.

51. Peterson LT. Amputations of the lower extremity. In: *Orthopedic Surgery in Zone of Interior.* 1970:965–1014.

52. Peterson LT. The army amputation program. *J Bone Joint Surg.* 1944;26(4):635–638.

53. Burgess EM, Romano RL, Zettl JH, Schrock RD. Amputations of the leg for peripheral vascular insufficiency. *J Bone Joint Surg Am .* 1971;53(5):874–890.

54. Persson BM. Sagittal incision for below-knee amputation in ischaemic gangrene. *J Bone Joint Surg Am.* 1974;56(1):110–114.

55. Alter AH, Moshein J, Elconin KB, Cohen MJ. Below-knee amputation using the sagittal technique. *Clin Orthop.* 1978;131:195–201.

56. Latimer HA, Dahners LE, Bynum DK. Lengthening of below-the-knee amputation stumps using the Ilizarov technique. *J Orthop Trauma.* 1990;4(4):411–414.

57. Eldridge JC, Armstrong PF, Krajbich JI. Amputation stump lengthening with the Ilizarov technique: A case report. *Clin Orthop.* 1990;256:76–79.

58. Katz RT, Wu Y. Postoperative and preprosthetic management of the below-knee amputee. *Contemporary Ortho.* 1985;10(1):53–63.

59. Mensch G, Ellis PM. *Physical Therapy Management of Lower Extremity Amputations.* Rockville, Md: Aspen Publishers; 1986.

60. Zettl JH. Immediate postoperative prostheses and temporary prosthetics. In: Moore WS, Malone JM, eds. *Lower Extremity Amputation.* Philadelphia:WB Saunders; 1989:177–207.

61. Malone JM. Complications of lower extremity amputation. In: Moore WS, Malone JM, eds. *Lower Extremity Amputation.* Philadelphia: WB Saunders; 1989:208–214.

62. Bowker JH. Critical choices: The art of prosthesis prescription. In: Bowker JH, Michael JW, eds. *Atlas of Limb Prosthetics: Surgical, Prosthetic, and Rehabilitation Principles.* 2nd ed. St. Louis: Mosby-Year Book; 1992:717–720.

63. Cohen SI, Goldman LD, Salzman EW, Glotzer DJ. The deleterious effect of immediate postoperative prosthesis in below-knee amputation for ischemic disease. *Surgery.* 1974;76(6):992–1001.

64. Wu Y, Keagy RD, Krick HJ, Stratigos JS, Betts HB. An innovative removable rigid dressing technique for below-the-knee amputation. *J Bone Joint Surg Am.* 1979;61(5):724–729.

65. Baker WH, Barnes RW, Shurr DG. The healing of below-knee amputations: A comparison of soft and plaster dressings. *Am J Surg.* 1977;133:716–718.

66. Pinzur MS, Littooy F, Osterman H, Wafer D. Early post-surgical prosthetic limb fitting in dysvascular below-knee amputees with a pre-fabricated temporary limb. *Orthopedics.* 1988;11(7):1051–1053.

67. Kraker D, Pinzur MS, Daley R, Osterman H. Early post-surgical prosthetic fitting with a pre-fabricated plastic limb. *Orthopedics.* 1986;9(7):989–992.

68. Gottschalk F, Mooney V, McClellan B, Carlton A. Early fitting of the amputee with a plastic temporary adjustable below-knee prosthesis. *Contemporary Ortho.* 1986;13(1):15–18.

69. Czerniecki JM. Procedure manual for stamp/rms resident rotation. Seattle: VA Medical Center; 1989.

70. Saunders GT. Static and dynamic analysis. In: Saunders GT, May BJ, Hurd R Jr, Milani J, eds. *Lower Limb Amputations: A Guide To Rehabilitation.* 2nd ed. Philadelphia: FA Davis; 1988:415–475.

71. Manella KJ. Comparing the effectiveness of elastic bandages and shrinker socks for lower extremity amputees. *Orthot Prosthet.* 1982;36(1):30–34.

72. Kapp S, Cummings D. Prosthetic Management. In: Bowker JH, Michael JW, eds. *Atlas of Limb Prosthetics: Surgical, Prosthetic, and Rehabilitation Principles.* 2nd ed. St. Louis: Mosby-Year Book; 1992:453–478.

73. Staats TB, Lundt J. The UCLA total surface bearing suction below-knee prosthesis. *Clin Prosthet Orthot.* 1987;11(3):118–130.

74. Clover W, Jr. Lower extremity thermoplastics: An overview. *J Prosthet Orthot.* 1990;3(1):9–13.

75. Sabolich J. Prosthetic advances in lower extremity amputation. *Phys Med Rehabil Clin North Am.* 1991;2:415–422.

76. Rothschild VR, Fox JR, Michael JW, Rothschild RJ, Playfair G. Clinical experience with total thermoplastic lower limb prostheses. *J Prosthet Orthot.* 1990;3(1):51–54.

77. Valenti TG. Experience with endoflex: A monolithic thermoplastic prosthesis for below-knee amputees. *J Prosthet Orthot.* 1990;3(1):43–50.

78. Staats TB. Lower extremity prosthetics. In: Moore WS, Malone JM. eds. *Lower Extremity Amputation.* Philadelphia: WB Saunders; 1989:274–294.

79. Fillauer CE, Pritham CH, Fillauer KD. Evolution and development of the silicone suction socket (3S) for below-knee prostheses. *J Prosthet Orthot.* 1989;1(2):92–103.

80. Roberts RA. Suction socket suspension for below-knee amputees. *Arch Phys Med Rehabil.* 1986;67:196–199.

81. Quigley MJ. Prosthetic management: Overview, methods, and materials. In: Bowker JH, Michael JW, eds. *Atlas of Limb Prosthetics: Surgical, Prosthetic, and Rehabilitation Principles.* 2nd ed. St. Louis: Mosby-Year Book; 1992:67–79.

82. Michael J. Energy storing feet: A clinical comparison. *Clin Prosthet Orthot.* 1987;11(3):154–168.

83. Barth DG, Schumacher L, Thomas SS. Gait analysis and energy cost of below-knee amputees wearing six different prosthetic feet. *J Prosthet Orthot.* 1992;4(2)63–75.

84. Esquenazi A, Torres MM. Prosthetic feet and ankle mechanisms. *Phys Med Rehabil Clin North Am.* 1991;2(2):299–310.

85. Campbell JW, Childs CW. The S.A.F.E. foot. *Orthot Prosthet.* 1980;34(3):3–16.

86. Wing DC, Hittenberger DA. Energy-storing prosthetic feet. *Arch Phys Med Rehabil.* 1989;70:330–335.

87. Gitter A, Czerniecki JM, DeGroot DM. Biomechanical analysis of the influence of prosthetic feet on below-knee amputee walking. *Am J Phys Med Rehabil.* 1991;70(3):142–148.

88. Hittenberger DA. The Seattle foot. *Orthot Prosthet.* 1986;40(3):17–23.

89. Burgess EM, Hittenberger DA, Forsgren SM, Lindh D. The Seattle prosthetic foot: A design for active sports: preliminary studies. *Orthot Prosthet.* 1983;37(1):25–31.

90. Arbogast R, Arbogast JC. The carbon copy II: From concept to application. *J Prosthet Orthot.* 1988;1(1):32–36.

91. Macfarlane PA, Nielsen DH, Shurr DG, Meier K. Perception of walking difficulty by below-knee amputees using a conventional foot versus the flex-foot. *J Prosthet Orthot.* 1991;3(31):114–119.

92. Merlette J. Springlite foot: A composite material engineer's approach to design. *J Assoc Childrens Prosthet Orthot Clin* 1989;24(4).

93. Fernie GR, Griggs G, Bartlett S, Lunau K. Shape sensing for computer aided below-knee prosthetic socket design. *Prosthet Orthot Int.* 1985;9:12–16.

94. Michael JW. Reflections on CAD/CAM in prosthetics and orthotics. *J Prosthet Orthot.* 1989;1(3):116–121.

95. Saunders CG, Foort J, Bannon M, Lean D, Panych L. Computer aided design of prosthetic sockets for below-knee amputees. *Prosthet Orthot Int.* 1985;9:17–22.

96. Topper AK, Fernie GR. An evaluation of computer aided design of below-knee prosthetic sockets. *Prosthet Orthot Int.* 1990;14:136–142.

97. Houston VL, Burgess EM, Childress DS, Lehneis HR, Mason CP, Garbarini MA, et al. Automated fabrication of mobility aids (AFMA): Below-knee CASD/CAM testing and evaluation program results. *J Rehabil Res Dev.* 1992;29(4):78–124.

98. Gregory RT, Gould RJ, Peclet M, et al. The mangled extremity syndrome (M.E.S.): A severity grading system for multisystem injury of the extremity. *J Trauma.* 1985;25(12):1147–1150.

99. Johansen K, Daines M, Howey T, Helfet D, Hansen ST. Objective criteria to accurately predict amputation following lower extremity trauma. *J Trauma.* 1990;30(5):568–573.

100. Robertson PA. Prediction of amputation after severe lower limb trauma. *J Bone Joint Surg Br.* 1991;73(5):816–818.

101. Pinzur MS, Smith DG, Daluga D, Osterman H. Selection of patients for through-knee amputation. *J Bone Joint Surg Am*. 1988;70(5):746–750.

102. Gottschalk F, Kourosh S, Stills M, McClennan B, Roberts J. Does socket configuration influence the position of the femur in above-knee amputation? *J Prosthet Orthot.* 1989;2:94–102.

103. Unruh T, Fisher DF, Unruh TR, Gottschalk F, et al. Hip disarticulation: An 11 year experience. *Arch Surg.* 1990;125:791–793.

104. Esquenazi A, Micheo W, Vachranukunkiet T. Design and construction of a bicycle attachment for conditioning of below-knee amputees and its clinical application. *Proceedings of the International Society for Prosthetics and Orthotics*. Copenhagen, Denmark; 1986:373.

105. Dise-Lewis J. *Psychological Adaptation to Limb Loss in Comprehensive Management of the Upper-Limb Amputee.* Atkins JD, Meier HR III, eds. New York: Springer-Verlag; 1989:165–172.

106. Esquenazi A, Vachranukunkiet T, Torres M. A method for continuing bipedal ambulation in below-knee amputees with residual limb complications. *Arch Phys Med Rehabil.* 1983;64:482.

107. Bresler B, Berry FR. Energy characteristics of normal and prosthetic ankle joints. University of California, Berkeley. Series II, Issue 12, 1950.

108. Wagner J, Sienko S, Supan T, Barth D. Motion analysis of SACH vs. flex-foot in moderately active below-knee amputees. *Clin Prosthet Orthot.* 1987;11(1):55–62.

109. Bonstra AM, Fidler V, Spits GMA, et al. Comparison of gait using a multiflex foot versus a quantum foot in knee disarticulation. *Prost Orth Int.* 1993;17(2):90–94.

110. New York University Medical School. Lower-limb prosthetics. *Prosthet Orthot.* 1980.

111. Long IA. Normal shape-normal alignment (NSNA) above-knee prosthesis. *Clin Prosthet Orthot.* 1985;9:9–14.

112. Pritham CH. Biomechanics and shape of the above-knee socket considered in light of the ischial containment concept. *Prosthet Orthot Int.* 1990;14:9–21.

113. Sabolich J. Contoured adducted trochanteric-controlled alignment method (CAT-CAM): Introduction and basic principles. *Clin Prosthet Orthot.* 1985;9:15–26.

114. Michael JW. Current concepts in above-knee socket design. *Instr Course Lect.* 1990;39:373–378.

115. Pritham CH. Workshop on teaching materials for above-knee socket variants. *J Prosthet Orthot.* 1988;1:50–76.

116. Such CM. Modern above-knee fitting practice. A report on the ISPO workshop on above-knee fitting and alignment techniques, May 15–19, 1987, Miami, USA. *Prosthet Orthot Int.* 1988;12:77–90.

117. Taft CB. Radiographic evaluation of stump socket fit. *Artificial Limbs.* 1969;13(2):36–40.

118. Gailey RS, Lawrence D, Burditt C, et al. The CAT-CAM socket and quadrilateral socket: A comparison of energy cost during ambulation. *Prosthet Ortho Int.* 1993;17(2):95–100.

119. Jendrzejczyk D. Flexible socket systems. *Clin Prosthet Orthot.* 1985;9:27–31.

120. Kristinsson O. Flexible above-knee socket made from low density polyethylene suspended by a weight-transmitting frame. *Orthot Prosthet.* 1983;37:25–27.

121. Pritham CH, Fillauer C, Fillauer K. Experience with the Scandinavian flexible socket. *Orthot Prosthet.* 1985;39:17–32.

122. Volkert R. Frame type socket for lower limb prostheses. *Prosthet Orthot Int.* 1982;6:88–92.

123. Koike K, Ishikura Y, Kakurai S, et al. The TC double socket above-knee prosthesis. *Prosthet Orthot Int.* 1981;5:129–134.

124. Moore WS. Below-knee amputation. In: Moore WS, Malone JM, eds. *Lower Extremity Amputation.* Philadelphia: WB Saunders; 1989:118–131.

125. Linge K, Boot DA, Keetarut S.Application of the transcutaneous oxygen monitor to amputees undergoing early fitting of below-knee patellar tendon bearing prostheses. *J Prosthet Orthot.* 1988;1:18–23.

126. Bowker JH, Keagy RD, Poonekar PD. Musculoskeletal complications in amputees: Their prevention and management. In: Bowker JH, Michael JW, eds. *Atlas of Limb Prosthetics: Surgical, Prosthetic, and Rehabilitation Principles.* 2nd ed. St. Louis: Mosby-Year Book; 1992:665–680.

127. Levy SW. Skin complications of amputees. In: Bowker JH, Michael JW, eds. *Atlas of Limb Prosthetics: Surgical, Prosthetic, and Rehabilitation Principles*. 2nd ed. St. Louis: Mosby-Year Book Inc; 1992.

128. Rubin G, Dixon M, Fischer E. Lower extremity amputation problems: Etiology, manisfestations, and prevention. *Orthot Prosthet.* 1985;39(1):47–61.

129. Singson RD, Feldman F, Staron R, Fechtner D, Gonzalez E, Stein J. MRI of postamputation neuromas. *Skeletal Radiol.* 1990;19:259–262.

130. Odderson IR, Czerniecki JM. Reflex sympathetic dystrophy in an amputee: Case study. *Arch Phys Med Rehabil.* 1990;71:161–163.

131. Rosenthal AK, Wortmann RL. Diagnosis, pathogenesis, and management of reflex sympathetic dystrophy syndrome. *Compr Ther.* 1991;17:46–50.

132. Kozin F. Reflex sympathetic dystrophy syndrome. *Bull Rheum Dis.* 1986;36(3):1–8.

133. Kozin F, Ryan LM, Carerra GF, Soin JS, Wortmann RL. The reflex sympathetic dystrophy syndrome (RSDS): Scintigraphic studies, further evidence for the therapeutic efficacy of systemic corticosteroids, and proposed diagnostic criteria. *Am J Med.* 1981;78(Part 3):23–29.

134. Demangeat JL, Constantinesco A, Brunot B, Foucher G, Farcot JM. Three-phase bone scanning in reflex sympathetic dystrophy of the hand. *J Nucl Med.* 1988;29:26–32.

135. Brody MC, Andary MT. Treatment approaches to reflex sympathetic dystrophy. *Phys Med Rehabil Clin North Am.* 1993;4:165–174.

136. Lipton DE, Nagendran T. A rare cause of stump pain-herniated lumbar disc: A case report. *Alabama Med.* 1989;58:39–40.

137. Bulder ER, Smelt WLH. Onset of amputation stump pain associated with epidural anesthesia. *Anesth Analg.* 1991;72:394–396.

138. Jensen TS, Rasmussen P. Phantom pain and related phenomena after amputation. In: Wall PD, Melzack R, eds. *Textbook of Pain.* 2nd ed. New York: Churchill Livingston; 1990:508–521.

139. Jensen TS, Krebs B, Nielsen J, Rasmussen P. Phantom limb, phantom pain and stump pain in amputees during the first 6 months following limb amputation. *Pain.* 1983;17:243–256.

140. Loeser JD. Pain after amputation: Phantom limb and stump pain. In: Bonica JJ, Loeser JD, Chapman RC, Fordyce WE, eds. *The Management of Pain.* Philadelphia: Lea and Febiger; 1990:244–256.

141. Sherman RA, Sherman CJ, Parker L. Chronic phantom and stump pain among American veterans: Results of a survey. *Pain.* 1984;18:8–95.

142. Sherman RA, Sherman CJ. Prevalence and characteristics of chronic phantom limb pain among American veterans. *Am J Phys Med.* 1983;62:227–238.

143. Jensen TS, Krebs B, Nielsen J, Rasmussen P. Immediate and long-term phantom limb pain in amputees: Incidence, clinical characteristics and relationship to pre-amputation limb pain. *Pain.* 1985;21:267–278.

144. Katz J, Melzack R. Pain memories in phantom limbs: Review and clinical observations. *Pain.* 1990;43:319–336.

145. Sherman RA, Sherman CJ. A comparison of phantom sensations among amputees whose amputations were of civilian and military origins. *Pain.* 1985;21:91–97.

146. Sherman RA, Sherman CJ, Bruno GM. Psychological factors influencing chronic phantom limb pain: An analysis of the literature. *Pain.* 1987;28:285–295.

147. Sherman RA. Stump and phantom limb pain. *Neurol Clin.* 1989;7(2):249–264.

148. Vaida G, Friedmann LW. Postamputation phantoms: A review. *Phys Med Rehabil Clin North Am.* 1991;2:325–353.

149. Spiegel K, Kalb R, Pasternak GW. Analgesic activity of tricyclic antidepressants. *Ann Neurol.* 1983;13:462–465.

150. Jaeger H, Maier C. Calcitonin in phantom limb pain: A double-blind study. *Pain.* 1992;48:21–27.

151. Saris SC, Iacono RP, Nashold BS. Dorsal root entry zone lesions for post-amputation pain. *J Neurosurg.* 1985;62:72–76.

152. Pasnau RO, Pfefferbaum B. Psychologic aspects of post-amputation pain. *Nursing Clin North Am.* 1976;11(4):679–685.

153. Monga TN, Jaksic T. Acupuncture in phantom limb pain. *Arch Phys Med Rehabil.* 1981;62:229–231.

154. Bach S, Noreng MF, Tjellden NU. Phantom limb pain in amputees during the first 12 months following limb amputation, after preoperative lumbar epidural blockade. *Pain.* 1988;33:297–301.

155. Fisher A, Meller Y. Continuous postoperative regional analgesia by nerve sheath block for amputation surgery: Pilot study. *Anesth Analg.* 1991;72:300–303.

156. Brown PW. Rehabilitation of bilataral lower extremity amputees. *J Bone Joint Surg Am.* 1970;52(4):687–700.

157. Dunlap SW. Bilateral amputation: Above-knee and hip disarticulation. *Phys Ther.* 1969:49(5):500–502.

158. Canty TJ. Amputee rehabilitation. *Arch Phys Med.* 1951;32:219–221.

159. McCollough NC. The bilateral lower extremity amputee. *Orthop Clin North Am.* 1972;3(2):373–382.

160. Milling AWF. Multiple traumatic limb amputations. *Injury.* 1984;16(6):6.

161. Volpicelli LJ, Chambers RB, Wagner FW. Ambulation levels of bilateral lower extremity amputees. *J Bone Joint Surg Am.* 1983;65(5):599–605.

162. Hunter GA, Holliday P. Review of function in bilateral lower limb amputees. *Can J Surg.* 1978;21(2):176–178.

163. MacInnes MSA. Bilateral amputation of the legs: Patient care and rehabilitation. *Nursing Times.* July 1977;73(27):1033–1034.

164. Kerstein MD, Zimmer H, Dugdale FE, Levner E. Associated diagnoses which complicate rehabilitation of the patient with bilateral lower extremity amputations. *Surgery, Gynecology and Obstetrics.* June 1975;40:875–876.

165. Kerstein MD, Zimmer H, Dugdale FE, Levner E. Rehabilitation after bilateral lower extremity amputation. *Arch Phys Med Rehabil.* 1975;56:309–311.

166. Hamilton EA, Nichols PJR. Rehabilitation of the elderly lower-limb amputee. *Br Med J.* 1972;2:95–22.

167. Kitowski VJ, Pelosof HV, Leavitte LA. Comprehensive rehabilitation of a quadruple amputee. *South Med J.* July 1973;66(7):833–835.

168. Wynn Parry CB. The management of injuries to the brachial plexus. *Proc Roy Soc Med.* 1974;67:488–490.

169. Eldar R, Ohry A. Preparation of a hospital rehabilitation system for war and other disasters. *Medicine and War.* 1990;6(7):105–111.

170. Steinbach T. Total rehabilitation for amputees in special conditions. *Prosthet Orthot Int.* 1977;1(2):125–126.

171. Curley MD, Walsh MJ, Triplett RG. Some adjustment indices of oral-maxillofacial war casualties, limb amputees, and non-injured veterans. *Military Medicine.* 1982;147;572–574.

172. Ryan TC, Jones DT, White WL, Orr TE, Walters W. Management and rehabilitation of naval battle casualty amputees. *Ann Surg.* 1946;124(6):1047–1056.

173. Khoury SA. Evaluation of amputees. *Int J Rehabil Res.* 1983;6(4):449–454.

174. Kejlaa GH. The social and economic outcome after upper limb amputation. *Prosthet Orthot Int.* 1992;16:25–31.

175. Sheikh K. Return to work following limb injuries. *J Soc Occup Med.* 1985;35:114–117.

176. Helm P, Engel T, Holm A, Kristiansen VB, Rosendahl S. Function after lower limb amputation. *Acta Orthop Scand.* 1986;57:154–157.

177. Schafter R. Occupational therapy for lower extremity problems. *Am J Occup Ther.* 1973;27(3):132–137.

178. Goldberg RT. New trends in the rehabilitation of lower extremity amputees. *Rehabilitation Literature.* 1984; 45(1–2):2–11.

179. Brown LKW. Traumatic amputation: Mechanism of injury, treatment and rehabilitation. *AAOHN Journal.* 1990;38(10):483–486.

180. Shepherd WG, Caine D. Vocational end results following rehabilitation of upper-extremity amputees. *Med J Australia.* 1968;2(4):167–169.

181. Davis DJ, Paasuke L. Vocational evaluation and rehabilitation. In: Delisa JA, Currie DM, Gans BM, Gatens PF Jr, Leonard JA Jr, McPhee MC, eds. *Rehabilitation Medicine: Principles and Practice.* Philadelphia: JB Lippincott; 1988.

Chapter 5

SPINAL CORD INJURY REHABILITATION

JAMES W. LITTLE, M.D., PH.D.[*]; BARRY GOLDSTEIN, M.D., PH.D.[†]; AND MARGARET C. HAMMOND, M.D.[‡]

[*]*Assistant Chief, Spinal Cord Injury Service, V.A. Puget Sound Health Care System; Associate Professor, Department of Rehabilitation Medicine, University of Washington, Seattle, Washington 98108.*

[†]*Staff Physician, Spinal Cord Injury Service, V.A. Puget Sound Health Care System; Assistant Professor, Department of Rehabilitation Medicine, University of Washington, Seattle, Washington 98108.*

[‡]*Chief, Spinal Cord Injury Service, V.A. Puget Sound Health Care System; Associate Professor, Department of Rehabilitation Medicine, University of Washington, Seattle, Washington 98108.*

INTRODUCTION

Spinal cord injury (SCI) among the civilian population of the United States occurs at the rate of 7,500 to 10,000 new injuries a year. Young men are the most common victims (80% of the total injured are males) and the peak age of injury is 19 years.[1] Impacts at high speeds are most often the cause: automobile accidents are responsible for 48%, falls for 21%, acts of violence for 15%, sports for 14%, and others for 2%.[1] The most common site of the injury is the cervical cord (54% of cases), then the thoracic (36%), and the lumbar cord (10%).[1] About 250,000 people (roughly 0.1%) of the U.S. population are spinal cord injured.

The demographic profile of the war injured is less well-documented. In Vietnam, 0.9% of those admitted to U.S. Army hospitals had incurred SCIs; 3.8% of those patients died during the initial hospitalization.[2] In various Israeli wars, the percentage of injuries with gunshot wounds to the spinal cord varied between 0.2% during the War of Independence in 1948–1949 and 1.1% in the Sinai Campaign of 1956.[3]

Up until World War II, death following SCIs commonly ensued from uncontrollable urinary tract infections (UTIs) and pressure sores. During the Balkan Wars of 1912–1913, 95% of the soldiers with SCIs died within a few weeks. During World War I, 80% of American troops with SCIs died before they could be shipped home. Prior to this time, the attitude remained essentially what it was when the first recorded description of SCI was made on Egyptian papyrus: "an ailment not to be treated,"[4] a condition for which there was no hope.

During World War II, the attitude of fatalism toward the spinal cord injured began to change. Advances in anesthesia, surgical techniques, blood transfusions, and antibiotics all contributed to increased survival. Teams of physicians, nurses, and therapists in Great Britain established SCI units and set up procedures for meticulous care and rehabilitation.[5] These protocols allowed the injured not only to survive, but in many cases to return to their communities.

At the conclusion of World War II, the United States government established the first comprehensive SCI unit at Hines Veterans Administration Hospital in suburban Chicago, using the British model. Compared with the dismal mortality in World War I, over 75% of the paraplegics from World War II were alive 20 years later. Of the 2,500 American paraplegics from that war, over 50% returned to the job force. In 1976, regional centers for treatment of the spinal cord injured were made available to civilians through the Spinal Cord Injury Model Systems Program.

With improved care, particularly of urologic and skin complications, mortality has fallen; late mortality (ie, after initial rehabilitation) was 1.7 times higher for those injured in the 1940s than those injured in the 1960s.[6] In the 1970s and 1980s survival has continued to improve; 7-year survival was 87% during this period.[7]

As mortality has declined, the focus for SCI care has gradually shifted. Initially, the target was defining and adopting procedures and practices to control the often fatal sequelae. Now the focus is directed more toward retraining individuals for independence and return to community.

EMERGENCY TREATMENT IN THE FIELD

Medic (Minutes Postinjury)

Field medics should suspect spine or SCI if the patient suffers loss of consciousness, major trauma, or any complaint referable to the spine (pain, tenderness) or spinal cord (sensory changes, weakness, priapism).[8] In these instances, the following measures then apply: (*a*) do not move the victim until the medic team arrives, unless he is in a life-threatening situation; (*b*) provide cardiopulmonary resuscitation (CPR) but use a jaw-thrust maneuver, rather than a head-tilt or chin-lift maneuver that could further damage the cervical spine; (*c*) immobilize the spine, using manual traction, spine board, and extrication collar or straps so that displaced bony fragments will not further compromise the cord; (*d*) insert a large gauge intravenous line; and (*e*) monitor for impaired breathing and signs of shock.[9] In conditions of war, it may not be possible to observe these protocols because of danger to the evacuation team. In Vietnam, for example, 11%[2] of war wounds were sustained by those caring for the wounded.

First and Second Echelon MTF (Min/H Postinjury)

In treating the patient, as soon as possible, the

treatment team at the first and second echelon medical treatment facility (MTF) should

- maintain systolic blood pressure ≥ 90 mm Hg with pressor agents (eg, dopamine, phenylephrine) and intravenous fluids to replace losses, but avoid over-hydration, which can lead to neurogenic pulmonary edema[10];
- maintain oxygenation with O_2 per nasal prongs or face mask and endotracheal or nasotracheal intubation (avoiding neck movements in those with possible cervical spine involvement) and Ambu bag ventilation[11];
- maintain spinal alignment;
- perform a quick motor examination of elbow flexion-extension, finger abduction, knee flexion-extension, ankle plantar and dorsiflexion, and a limited sensory examination of pin-prick and position sense in the hands and feet to monitor for neurologic deterioration or improvement;
- begin methylprednisolone intravenously as a 30 mg/kg bolus over 15 minutes followed by continuous infusion at 5.4 mg/kg/hour for 24 hours[12,13]; and,
- insert a Foley catheter to ensure bladder drainage and for close monitoring of urine output.

Third Echelon Treatment Facility (Hours Postinjury, No Neurosurgeon Available)

Once the patient has arrived at a treatment center, the staff should

- continue to monitor airway, breathing, and circulation by checking vital signs, arterial blood gas, vital capacity, and inspiratory effort;
- continue to maintain systolic blood pressure > 90 mm Hg with pressor agents (eg, dopamine, phenylephrine) and limited fluid replacement, avoiding pulmonary edema; atropine may be needed for bradycardia < 50 beats per minute;
- continue to monitor urine output via Foley catheter;
- insert nasogastric tube to decompress the gastrointestinal (GI) tract because of paralytic ileus;
- obtain a detailed history including the mechanism of injury, weakness, sensory changes or loss of consciousness at any time after the injury;
- perform a detailed neurologic examination to further define the level and severity of the SCI and to identify associated neurologic injuries (eg, peripheral nerve, plexus or head injury);
- consider a head computed tomography (CT) scan if head injury is suspected;
- rule out associated injuries of the chest with chest radiographs, of the abdomen with physical examination and diagnostic peritoneal dialysis, and of the long bones with physical examination and radiographs; and
- evaluate the spine with physical examination, looking for tenderness or step-off (ie, misalignment), and with imaging studies (Exhibit 5-1), obtaining lateral radiographs of the entire spine, and anterior-posterior and oblique views of the injured spine segments.

Adequate imaging of vertebral level C-7 to T-1 may require manual traction downward on the arms, swimmer's position of the arms, midline tomogram, or a CT scan.

ACUTE NEUROSURGICAL MANAGEMENT AT MILITARY HOSPITAL (HOURS/DAYS POSTINJURY)

Restoring and maintaining spine alignment and assuring decompression of the spinal cord are primary objectives in the military hospital. Spine instability and cord compression are identified via further imaging studies (see Exhibit 5-1). Misalignment is reduced either with skull tongs and traction or surgically, and then alignment is maintained with external or internal means. External stabilization consists of tongs and traction, halo-vest, hard cervical collar (Philadelphia or Minerva), or body jacket. Internal fixation uses wires, rods, plates and screws, and bone grafts to achieve bony fusion across unstable spinal segments.

Early indications for spinal surgery are (*a*) failure to realign the spine nonoperatively; (*b*) decompression of the spinal cord by removal of bone or soft tissue from the spinal canal; (*c*) a penetrating wound that requires debridement; and (*d*) deteriorating neurologic function, though uncommon.[9,14,15] A late indication for surgery is pseudoarthrosis with progressive deformity. A spine is considered stable when there is no progressive neurologic deficit, no progressive spinal deformity, and no spinal pain. Achieving stability requires that bony and ligamentous structures heal. Bony fusion is usually achieved within 2 to 6 months with cervical injuries, gener-

EXHIBIT 5-1

IMAGING STUDIES IN ACUTE SPINAL CORD INJURY

Cervical Spine, Lateral View

1. Include from base of skull to T-1.
2. Identify misalignment of vertebrae or spinous processes.
3. Note prevertebral soft tissue swelling; width < 7 mm at C-3.
4. Note widening of spaces between spinous processes.
5. Vertebral bodies and disk spaces should be full height.

Cervical Spine, Flexion-Extension Views

1. Perform only if NO neurologic deficit, NO spine tenderness, and NO abnormality show on static roentgenograms. These flexion-extension views are to detect subtle instability.
2. Patient must be cooperative; sitting, he slowly flexes then extends the neck, stopping if he feels pain or neurologic change. A lateral c-spine radiograph is taken both flexed and extended.
3. Greater than 3.5 mm displacement or > 11° angulation indicates instability.

Emergent Myelogram CT Scan or Magnetic Resonance Imaging (MRI)

1. Indications: (*a*) assure cord decompression if neurologically incomplete SCI, (*b*) discrepancy between spine fracture and neurologic level, (*c*) deterioration in neurologic status.
2. Nonferromagnetic cervical collar, tongs, or halo for MRI.
3. Hematoma on MRI shows increased T-2, decreased T-1 density subacutely (ie, > 24 hours, due to extracellular methemoglobin); cord hematoma suggests poor prognosis for neurologic recovery.
4. CT scan is more sensitive than MRI for spine fractures; MRI detects some ligamentous injuries but sensitivity is unknown.

ally requiring 2 to 3 months, and thoracolumbar injuries needing 4 to 6 months. Ligamentous injuries are less predictable and require immobilization of 8 weeks. They may necessitate late surgical stabilization.

Wartime SCIs are often gunshot wounds. These military missile injuries differ from civilian bullet wounds because of the missiles' high velocity, large caliber, or both. In destroying more tissue, these missiles increase the likelihood of soft tissue and bone infection. Under these circumstances, debridement, wound exploration, bullet fragment removal, and 2 weeks of broad spectrum antibiotics have been advocated, particularly if the alimentary canal has been perforated. [16–19]

Studies of civilian gunshot wounds reveal that bullets or shrapnel fragments damage the spinal cord directly by passing through or lodging in the spinal canal, or indirectly by transmitting shock waves when bullets pass through adjacent tissue. Indirect SCI by bullet shock waves was noted in 27% of civilian cases.[20] Neurologically complete SCI is associated with larger caliber bullets and thoracic gunshot wounds[20]; cervical or lumbar gunshot wounds often yield an incomplete SCI. Cybulski and colleagues found motor recovery in 56% of those with lower lumbar level gunshot wounds, in 29% of those with thoracolumbar level injuries, and in less than 3% of those with gunshot injuries at or above vertebral level T-10.[21] Reviews of military missile injuries have also demonstrated a better prognosis for motor recovery in those with cauda equina level injuries.[22–24]

Bullet fragment removal has been advocated to prevent cerebrospinal fluid (CSF) leak, infection (meningitis or osteomyelitis), pain, lead toxicity, and neurologic decline from bullet migration or chronic inflammatory response. A few have advocated it to promote neurologic recovery. For civilian missile injuries, bullet removal does appear to promote motor recovery for injuries at the T-12 to L-4 vertebral level but not at higher thoracic or cervical levels.[19] Bullet removal does not prevent complications of CSF leak, pain, or meningitis.[19,25] Fourteen days of

antibiotic administration appears to reduce the incidence of spinal infection when a missile passes through the colon prior to striking the spine.[19]

In addition to spine stability and neurologic recovery, another major focus of early treatment is the prevention of SCI complications. A partial list of the early complications includes acute respiratory failure, atelectasis, pneumonia, bradyarrythmia, hypotension, autonomic hyperreflexia, deep venous thrombosis (DVT) and pulmonary emboli, ileus, gastritis and ulcers, fecal impaction, UTI, detrusor-sphincter dyssynergia, kidney and bladder stones, hypercalcemia, heterotopic ossification (HO), myotendinous contracture and capsule tightness, musculoskeletal and neuropathic pain, spasticity and other hypertonus, posttraumatic syringomyelia, and depression. The prevention and treatment of these complications is addressed in the later section entitled Preventing and Managing SCI Complications.

PATHOPHYSIOLOGY OF SPINAL CORD INJURY

The two types of SCIs are penetrating and nonpenetrating. Penetrating injuries are less common and typically result from a knife blade or bullet lacerating the cord directly.

Nonpenetrating injuries commonly result from bone or herniated disk material compressing the spinal cord or nerve roots. Those with narrow spinal canals, either congenital or because of degenerative changes, are at greater risk for this type of cord injury (Figure 5-1). Nonpenetrating SCI may also result from traction, hemorrhage, or ischemia. The severity of cord damage is proportional to the force and duration of the mechanical trauma; it is also dependent upon the ability of the vertebral column to dissipate those forces.[26]

Either penetrating or nonpenetrating injuries may compromise gray matter, white matter, or nerve roots or any combination. Gray matter contains the interneurons, which receive descending motor and segmental reflex input, and the motoneurons, which provide output to muscles. Being more vascular, gray matter is thought to be more vulnerable to mechanical trauma. Gray matter damage typically extends one or two segments rostral and caudal to the cord injury, but may be more extensive if the cord blood supply has been disrupted; all segments involved are known as the zone of injury. White matter is composed of ascending and descending fibers at the periphery of the cord. Pathologic studies of cord trauma show greater gray

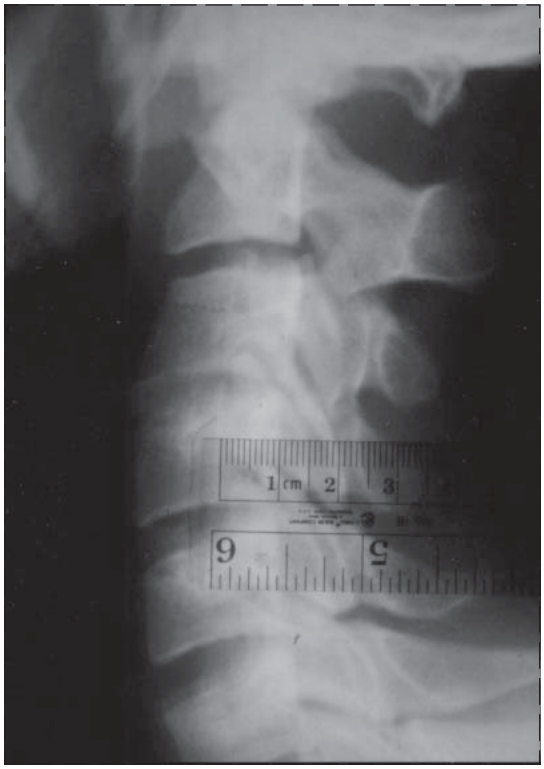


Fig. 5-1. Congenital cervical stenosis. This 33-year-old male football player and All-Service safety became immediately quadriplegic after tackling a ball-carrier. Acute respiratory failure required emergent intubation and artificial ventilation. Note the congenitally narrow cervical canal with an anterior–posterior (AP) diameter of 12 mm. The Pavlov ratio, the ratio of the canal AP diameter to the vertebral body AP diameter, is 0.5; the lower limit of normal is 0.8. Roentgenograms also demonstrated anterior subluxation of C-3 on C-4, for which he underwent anterior diskectomy and fusion. Over several weeks, the patient recovered sufficient diaphragm movement to wean from the ventilator. Over six months, he regained sufficient lower extremity movement to be independent in ambulation with a single point cane. He also experienced good recovery in his left upper extremity such that he became independent in self-care skills; his right upper extremity remained nonfunctional. Voluntary bowel and bladder control returned.

than white matter involvement.[27–29] Damage to gray matter causes segmental changes with denervation muscle atrophy and impaired reflexes. Damage to white matter is more disabling because it results in loss of motor control and sensory input not only at but also below the site of injury; hypertonia and hyperreflexia accompany weakness and sensory loss in such white matter involvement. Nerve root injury often results in an asymmetric level of injury.

Spinal cord damage can arise indirectly from vascular disruption (Figure 5-2). Thus, laceration of the aorta, artery of Adamkiewicz, or a vertebral artery can result in cord impairment.[30–32] Another type of ischemic cord injury is decompression sickness, where hyperbaric exposure (as in underwater diving) followed by sudden decompression, results in gas bubble formation and bubble emboli, which occlude the arterial supply to the spinal cord.[33,34]

While the force of the injury is the most important initial factor, there are several secondary processes that may contribute to cord damage. Studies show that even when a cord injury is clinically complete, the spinal cord is not usually transected. Some argue that much damage results from secondary neurologic injury, a consequence of ischemia, edema, hematomyelia, demyelination, persisting mechanical pressure, lactic acidosis, intracellular influx of calcium, increase of lipid peroxidation, and

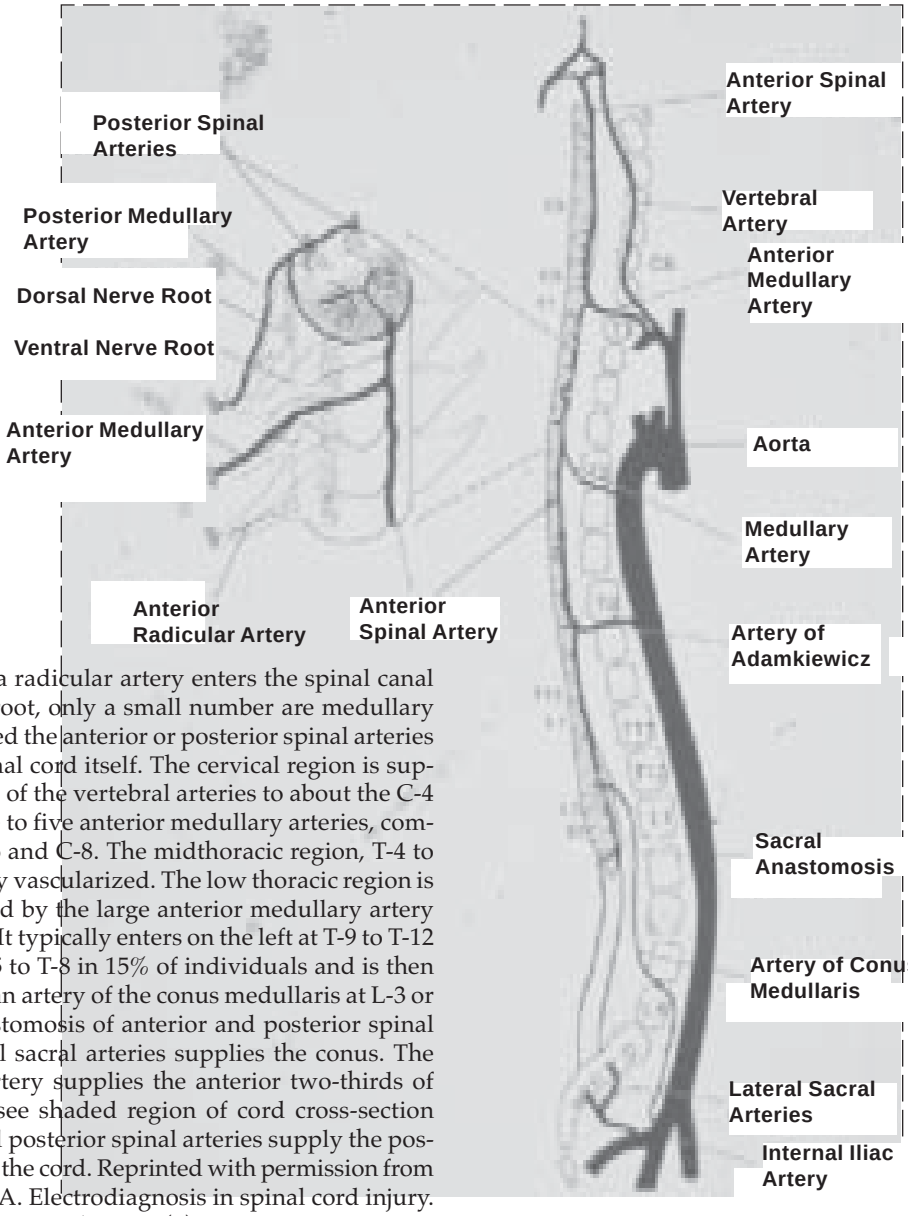


Fig. 5-2. Though a radicular artery enters the spinal canal with each nerve root, only a small number are medullary arteries, which feed the anterior or posterior spinal arteries and thus, the spinal cord itself. The cervical region is supplied by branches of the vertebral arteries to about the C-4 level and by three to five anterior medullary arteries, commonly at C-3, C-6 and C-8. The midthoracic region, T-4 to T-8, is often poorly vascularized. The low thoracic region is primarily supplied by the large anterior medullary artery of Adamkiewicz. It typically enters on the left at T-9 to T-12 but it arises at T-5 to T-8 in 15% of individuals and is then accompanied by an artery of the conus medullaris at L-3 or L-4. A sacral anastomosis of anterior and posterior spinal arteries and small sacral arteries supplies the conus. The anterior spinal artery supplies the anterior two-thirds of the spinal cord (see shaded region of cord cross-section above); the paired posterior spinal arteries supply the posterior one-third of the cord. Reprinted with permission from Little JW, Stiens SA. Electrodiagnosis in spinal cord injury. *New Dev Electrodiagn Med*. 1995;5(3):581.

free radical formation.[35–38] Various early treatments such as hyperbaric oxygen, cord cooling, naloxone, thyrotropin releasing hormone, osmotic diuretics, and others have been proposed to minimize this secondary neurologic injury.[38] However as yet, only high-dose methylprednisolone[12,13] and late anterior decompression[14,39] have been shown to promote neurologic recovery after SCI in humans. Methylprednisolone is effective if given within the first 8 hours postinjury, but even then only modest gains in sensory and motor recovery have been demonstrated.[12,13,40] Because all motor and sensory improvements were grouped together and no measure of functional outcome was used in these studies, the functional significance of methylprednisolone intervention is not known. Another intervention to promote recovery is late anterior decompression (ie, 1–12 mo post-SCI). This procedure reportedly allowed functional recovery in those with incomplete SCI, and residual cord or root compression for those whose recovery had plateaued for 4 weeks or more.[14] Another small study suggests that intravenous gangliosides given in the first month may also improve motor recovery; this recovery is thought to be mediated by white matter long tracts.[41]

In contrast, animal models of incomplete SCI demonstrate that much of the recovery is mediated by spared white matter axons that substitute for those pathways that have degenerated, rather than by resolution of conduction block in white matter axons due to resolution of ischemia, edema, and demyelination. The mechanisms that allow for this substitution of function by spared pathways likely include rapid-acting denervation supersensitivity and slower-acting synaptogenesis. When spared descending white matter pathways and spinal reflex pathways undergo this trauma-induced reactive synaptogenesis, the result is both motor recovery and spinal hyperreflexia. The slow pace of this motor recovery and the gradual onset of this spasticity may be explained by the slowness of synaptogenesis.

One way to enhance this process may be to increase the activity in the spared neural pathways since neural activity seems to be a necessary condition for such recovery mediated by spared pathways.[42] In animal models of stroke, administration of central nervous system stimulants (eg, amphetamine) with exercise has enhanced recovery. These observations suggest that remobilization of the patient and active exercise are essential factors in optimizing recovery of function after SCI.

Two other methods of regulating activity in the spared neural pathways of the cord, medications and growth factors, are as yet unexplored, and a third issue, the optimal treatment for minimizing developing spasticity during this period of recovery, is unresolved. With stroke patients, some advocate use of reflex facilitation during strengthening exercises to enhance motor recovery; however, this may promote the development of more spasticity. Alternatively, aggressive early treatment to suppress spasticity with medication and physical modalities may suppress the development of spasticity but it may not optimize recovery of motor function. Resolving these issues will allow more effective rehabilitation of acute, incomplete SCI in the future.

In contrast to white matter or long tract recovery, zone of injury recovery involves recovery of cord neurons, (gray matter recovery) and root or roots at the site of injury, as is well-documented by Ditunno and colleagues.[43,44] Mechanisms to explain zone of injury recovery include resolution of conduction block or reactive synaptogenesis by descending pathways in the spinal cord, resolution of conduction block or motor axon sprouting by lower motoneurons, or muscle fiber hypertrophy. These recovery mechanisms mitigate two types of weakness—upper motoneuron weakness and lower motoneuron weakness, both of which can be identified electrophysiologically.[45] The optimal rehabilitation interventions for these two types of weakness are not yet known.

NEUROLOGICAL EVALUATION AND PROGNOSIS

The degree of impairment depends on the level and extent of the injury. During development, the spinal cord grows less than does the spine; thus, both for the cervical (Figure 5-3) and the lumbosacral cord (Figure 5-4), the vertebral bodies and dorsal root ganglia are displaced caudal to their corresponding cord segments.

The following are standards in completing the initial evaluation and determining prognosis. The level of a complete SCI is defined as the last level with normal sensation (Figure 5-5, Table 5-1) and antigravity motor strength (ie, grade 3 or better; Table 5-2). If there is a difference from side-to-side, then the level of injury on each side should be described (eg, right C-5, left C-6). If there is a difference between the sensory and motor levels, then each should be described separately (eg, sensory C-6, motor C-8).[46–50] The American Spinal Injury As-

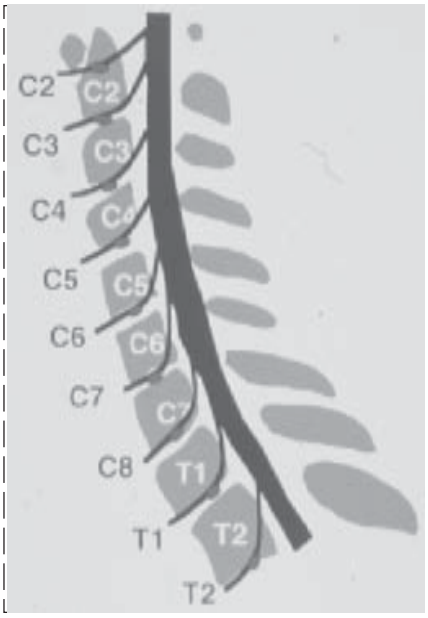


Fig. 5-3. Cervical spine, spinal cord, nerve roots, dorsal root ganglia (shaded ovals), and spinal nerves are shown. Note that there are seven cervical vertebrae but eight cervical cord segments and nerve roots. The C-1 to C-7 spinal nerves exit above their respective bony vertebrae but T-1 exits below. The dorsal root ganglia lie in the intervertebral foramina.

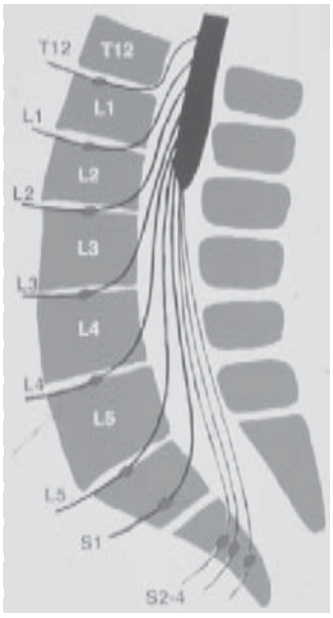


Fig. 5-4. Lumbosacral spine, spinal cord, conus medullaris, and cauda equina relationships are shown. Note that during development, the spine grows much longer than does the spinal cord; thus, lumbosacral cord segments are rostral to their respective spine segments. Observe the considerable length of the nerve roots of the cauda equina, that individual roots exit the canal below their respective spine segment, and that the dorsal root ganglia (shown as shaded ovals) lie in the intervertebral foramina. The spinal cord typically ends at the Ll–2 spinal level in a tapered conus medullaris that contains the S2–4 cord segments, but may end as high as T-12 or as low as L-2, as in this drawing.

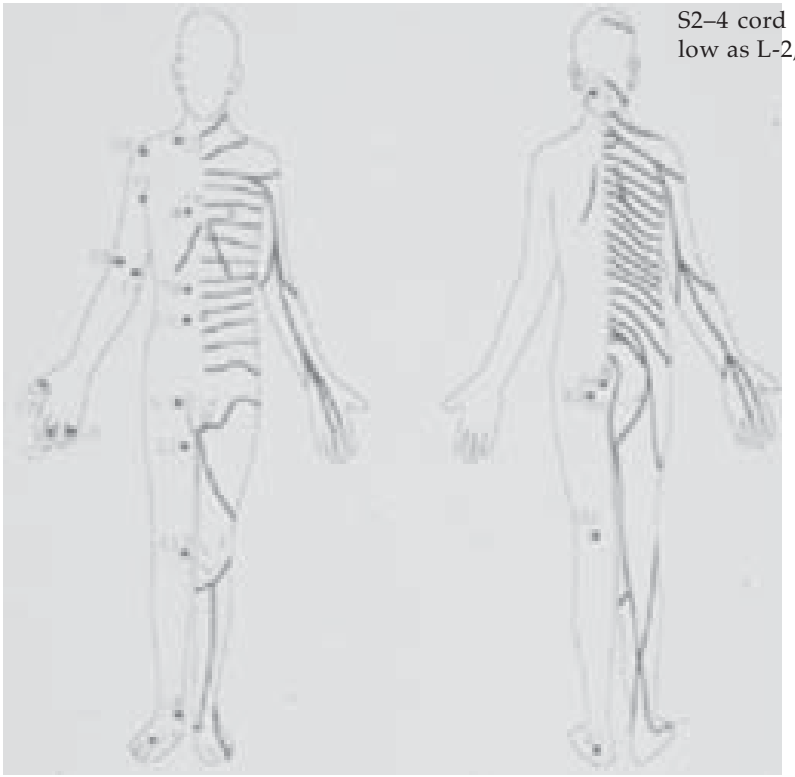


Fig. 5-5. Dematomes and midpoints (squares) commonly used for clinical testing are shown.

TABLE 5-1

NEUROLOGIC EVALUATION: SENSORY LEVELS

Level	Area of Cutaneous Sensation
Cranial Nerve V	Face (pain, temperature)*
C-2	Parietoccipital scalp
C-3	Lateral neck
C-4	Acromioclavicular
C-5	Lateral antecubital fossa
C-6	Digit 1
C-7	Digit 3
C-8	Digit 5
T-1	Medial antecubital fossa
T-2	Apex of axilla
T-4	Nipple line
T-10	Umbilicus
T-12	Inguinal ligament
L-2	Midanterior thigh
L-3	Medial femoral condyle
L-4	Medial malleolus
L-5	Dorsum of foot, 2nd metacarpophalangeal joint
S-1	Lateral heel
S-2	Midline popliteal fossa
S-3	Ischial tuberosity
S-4,-5	Perianal area

*The nucleus of the spinal trigeminal tract relaying pain and temperature sensation extends caudally into the upper cervical spinal cord and is somatotopically organized. Perioral afferents descend the least and synapse in the medulla; afferents from the lateral face descend most caudally into the upper cervical cord. This concentric representation around the mouth is known as the "onion skin pattern of Dejerine." Lesions of the rostral cervical spinal cord result in sparing of pain and temperature periorally but absence, over the lateral, upper, and lower face.

TABLE 5-2

NEUROLOGIC EVALUATION: MYOTOMAL LEVELS

Level	Area of sensation
C-1,-2,-3	Trapezius, SCM, upper cervical paraspinals, prevertebral neck muscles
C-4	Diaphragm
C-5	*Biceps brachii*, brachialis
C-6	*Extensor carpi radialis longus*
C-7	*Triceps brachii*
C-8	*Fexor digitorum profundus* (3rd digit)
T-1	*Abductor digiti minimi*, 1st dorsal interosseus
T-6,-7,-8,-9,-10	Bevor's sign*
L-2	*Iliopsoas*, hip adductors
L-3	*Quadriceps femoris*
L-4	*Tbialis anterior*
L-5	*Extensor hallucis longus*, hip abduction
S-1	*Gastrocnemius, soleus*
S-2,-3,-4	Anal sphincter

SCM: sternocleidomastoid
*Bevor's sign represents upward movement of the umbilicus when the patient attempts a sit-up from supine-lying.
The italicized muscles are the standard muscles used for classification of the level of injury by the American Spinal Injury Association.[1] These muscles are innervated by more than one root level, but reduction to a representative level is useful for injury classification. (1) American Spinal Injury Association. *Standards for Neurological and Functional Classification of Spinal Cord Injury.* 1992.

sociation (ASIA) has stated that partial sparing of sensation and voluntary movement (ie, less than grade 3 strength) may be present up to three segmental levels below the level of a complete cord injury; this is referred to as the zone of partial preservation. Any sparing four or more segments below this level has been classified as an incomplete SCI. Recently, ASIA has adopted a new classification of complete vs incomplete SCI based on the preservation of perianal sensation and voluntary anal sphincter or toe flexor contractions.[24]

Incomplete SCI is more variable. The level of injury is established similar to complete SCI; the sensory level is the most caudal segment with normal sensation and the motor level is the most caudal segment with antigravity strength. Incomplete SCI has been classified in three ways: (1) by the preservation of sensation and motor strength for the sacral segments,[24] (2) by the Frankel scale (Tables 5-3 and 5-4),[47] and (3) by the pattern of clinical deficits (eg, central, anterior or Brown-Séquard's syndrome) suggesting the transverse extent of the cord injury (Table 5-5).[51–53]

The likelihood of a complete vs incomplete SCI relates to the level of injury. If the injury is at the cervical level (C-5 being most common), 40% to 60% will be motor complete. If it is at the thoracic level, (T-12 being most common) 70% to 80% will be complete except at T-12 where the rate is 44%. At the lumbosacral level, only 10% to 25% are complete.[1]

TABLE 5-3

FRANKEL GRADING OF INCOMPLETE SPINAL CORD INJURY

Grade	Neurological Deficits*
A	Sensory and motor loss complete
B	Sensory incomplete, motor loss complete
C	Motor loss incomplete, nonfunctional
D	Motor loss incomplete, functional
E	Normal function

*Lower extremity sensation, motor strength, and function are assessed.

TABLE 5-4

FUNCTIONAL RECOVERY VERSUS INITIAL FRANKEL GRADE

Grade on Admission*	D or E† (%)	Regressing (%)
A	2	NA
B	20	4
C	50	1
D	98	2

*Frankel grade on admission for initial rehabilitation
†D and E: functional or normal recovery
NA: not applicable
Data source: Stover SL, et al. *Spinal Cord Injury: The Facts and Figures.* Birmingham: University of Alabama; 1986.

The neurologic findings at 72 hours postinjury are better predictors of functional lower extremity return than is an examination performed within hours of the cord injury; a wait of this length allows mild conduction block in the spinal cord to resolve and associated injuries (eg, mild head injury) and alcohol or drug effects to dissipate.[49,54] The most favorable prognostic sign for recovery is the return of voluntary movement below the injury; even a trace of voluntary toe movement at 72 hours is highly favorable for some functional motor recovery (see Table 5-4).[55] Another positive indicator is preserved pain and temperature sensations below the level of injury, often perianally (ie, sacral sparing); 66% to 88% of those with spared pin-prick sensation ambulated, whereas only 11% to 14% of those with just touch sensation spared went on to ambulate.[54,56] In the absence of any voluntary movement,

TABLE 5-5

SYNDROMES OF INCOMPLETE SPINAL CORD INJURY

	Clinical Features	Prognosis
Central cord	Common; lower > upper limb recovery; intact pain and temperature sensation	Often recover bladder and bowel function/control and ambulation
Brown-Séquard	Common; unilateral impaired pain and temperature sensation contralateral to paretic lower limb; unilateral impaired vibration and position sense ipsilateral to paretic limb	Usually recover bladder and bowel function/control and ambulation
Anterior cord	Common; bilateral absent pain and temperature sensation; marked paresis	Functional recovery uncommon
Posterior cord	Rare; absent position and vibratory sensation; intact pain and temperature sensation	Usually recover bladder and bowel function/control and ambulation
Cauda equina	About 10% of all cord injuries; often motor incomplete and with asymmetric deficits	Commonly recover bladder and bowel function/control and ambulation
Conus medullaris	Often with cauda equina injury; symmetric paresis and sensory loss	May recover ambulation; rarely recover bladder and bowel function/control

TABLE 5-6

NEUROLOGIC EVALUATION: SEGMENTAL REFLEXES

	Level	Stimulus	Response	Reflex Type
Biceps jerk	C-5,-6	Tap biceps tendon	Elbow flexion	Stretch
Brachioradial jerk	C-5,-6	Tap radius	Elbow flexion	Stretch
Triceps jerk	C-7	Tap triceps tendon	Elbow extension	Stretch
Finger jerk	C-8,T-1	Tap FDP, FDS tendon	Finger flexion	Stretch
Hoffmann's	C-8,T-1	Flick distal finger	Thumb flexion	Stretch
Deep abdominal	T-5 to T-12	Tap abdominal wall	Abdomen contraction	Stretch
Superficial abdominal	T-5 to T-12	Firmly stroke abdomen	Abdomen contraction	Cutaneous
Cremasteric	L-1,-2	Stroke inner thigh	Testicle elevation	Cutaneous
Adductor jerk	L-2,-3	Tap medial thigh	Knee adduction	Stretch
Knee jerk	L-3,-4	Tap patellar tendon	Knee extension	Stretch
Medial hamstring jerk	L-5	Tap medial hamstring tendon	Knee flexion	Stretch
Ankle jerk	S-1	Tap Achilles tendon	Ankle plantarflexion	Stretch
Extensor plantar	L-5,S-1	Scrape plantar foot	Great toe down (up=Babinski), pathological	Cutaneous
Bulbocavernosus	S-2,-3,-4	Pinch/tug glans	Anal sphincter contraction	Cutaneous
Anal reflex	S-2,-3,-4	Prick perianally	BC sphincter contraction	Cutaneous

BC: bulbocavernosus FDP: flexor digitorum profundus FDS: flexor digitorum superficialis

presence of a bulbocavernosus reflex or other reflex below the level of SCI (Table 5-6), is an unfavorable prognostic sign. It indicates the presence of segmental spinal cord excitability without any descending voluntary activation. From the civilian SCI model system, 31% of all injuries are motor incomplete and recover some lower extremity motor function.[1]

Various incomplete SCI syndromes have been described (see Table 5-5). Several relate to the presumed transverse extent of the cord injury; they are Brown-Séquard and central and anterior cord syndromes. Brown-Séquard syndrome is attributed to a cord hemisection, and is often the result of a penetrating injury, such as a knife or gunshot wound. The prognosis for return of functional ambulation and voluntary bladder control is good.[52,57] Central cord syndrome is typically due to a cervical cord injury and is associated with greater lower than upper limb recovery. This pattern of greater upper than lower limb deficit may reflect greater gray than white matter damage or greater damage to medial descending motor pathways in lateral funiculi of the white matter.[58] Central cord syndrome often occurs in individuals with a narrow cervical canal, either congenital or from osteophytes or other degenerative changes when the neck is hyperextended as in "whiplash," or when they suffer a fall. Bony fractures or instability are often absent. Anterior cord syndrome typically results from a cervical burst fracture or disk herniation, impinging on the anterior spinal artery, anterior cord, or both. It is not known whether direct mechanical pressure or disruption of arterial blood flow is the major factor. The outlook for functional recovery of the lower limbs is poor in anterior cord syndrome.

Cauda equina syndrome results from lumbar fractures at L-2 and below, with damage to the anterior and posterior roots. Central lumbar disk herniation or major trauma, as with gunshot wounds or seat belt injuries, are common causes of cauda

TABLE 5-7

ASHWORTH SCALE FOR GRADING MUSCLE TONE (RESISTANCE TO PASSIVE JOINT MOVEMENT)

Grade	Muscle Tone
0	Normal tone
1	Slight hypertonus
2	Mild hypertonus, joint moves easily
3	Moderate hypertonus, joint moves with difficulty
4	Rigid

Source: Ashworth B. Preliminary trial of carisoprodol in multiple sclerosis. *Practitioner*. 1964;192:540–542.

equina injury. Prognosis for functional recovery is good because the roots are less vulnerable to mechanical trauma than is the cord itself, and the motor axons have some capacity for regeneration to proximal muscles and to the bladder.[59] Often in conjunction with cauda equina syndrome, conus medullaris syndrome involves damage to the sacral segments of the cord, resulting in bowel, bladder, and sexual dysfunction. Fractures at T-12 to L-2 level or damage to the arterial supply of the lumbosacral cord are the most common causes of conus medullaris syndrome.

"Spinal shock" represents depressed spinal reflexes and weakness caudal to an SCI. This is likely caused by loss of normal tonic descending facilitation, since a block of conduction in suprasegmental pathways results in hyperpolarization of cord neurons.[60] Spinal reflexes then gradually return over days, to weeks, to months and become hyperactive as spasticity, flexor and extensor spasms, and hypertonia.[61,62] Some reflex activity may return as early as 24 hours after complete SCI, such as the bulbocavernosus reflex or the tibial H-reflex, even though tendon reflexes usually return weeks or months later. In those with incomplete SCI, tendon reflexes and spasticity may appear within days of the injury (see Tables 5-6 and 5-7).[63] No spinal shock is noted if myelopathy develops gradually as with a cord tumor, cervical stenosis, or syringomyelia; presumably hyperactive reflexes, such as a Babinski sign and ankle clonus, develop before overt weakness because neuroplasticity mechanisms, such as sprouting by reflex afferents and by spared descending pathways, mediate hyperreflexia and spared voluntary movement.

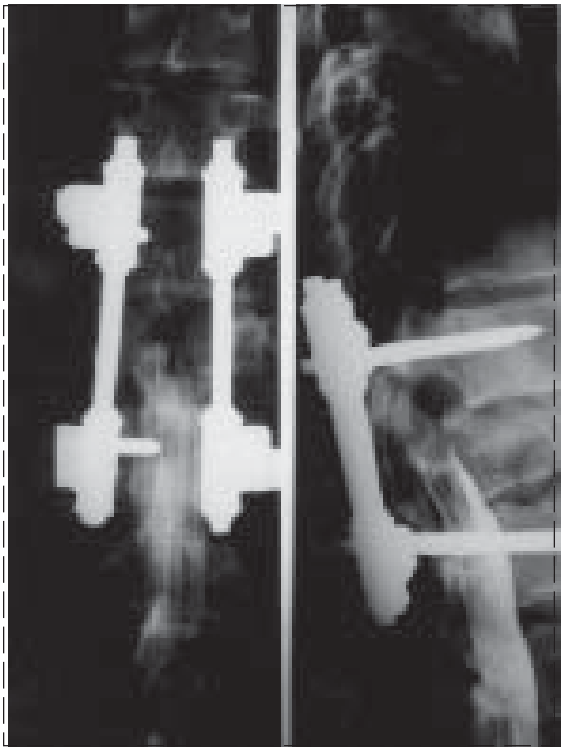


Fig. 5-6. Persisting bony impingement of the cauda equina. This 36-year-old male fell about 100 feet, landing on his buttocks, when his parachute partially collapsed. He was immediately paraplegic and radiographs revealed an L-1 burst fracture. An L-1 laminotomy, decompression of a retropulsed bony fragment and posterior Synthes instrumentation were performed. By two months some strength had returned in the lower extremities and ambulation training began in physical therapy; however, serial Cybex testing revealed worsening of his hamstring strength and repeat peroneal F-wave latencies, and motor evoked potential (MEP) latencies became more prolonged. The above lumbar myelogram was obtained and revealed a persisting complete block to dye flow at L-1 and inadequate bony decompression of the cauda equina. The patient was referred to orthopedic surgery for an anterior decompression; he subsequently regained functional strength in his lower extremities, becoming ambulatory without aids except for bilateral ankle-foot orthoses.

Motor and sensory recovery at the zone of injury is common, even in those with complete SCI. For example, 100% of C-5 and 25% of C-4 quadriplegic subjects regained functional wrist extension.[64] Fifty percent of Frankel A & B subjects and 100% of Frankel C & D subjects regained one segment of cord function by 2 months.[65] Seventy-five percent of those with grade 1 strength and 90% with grade 2 strength at 1 week regained functional use in up-

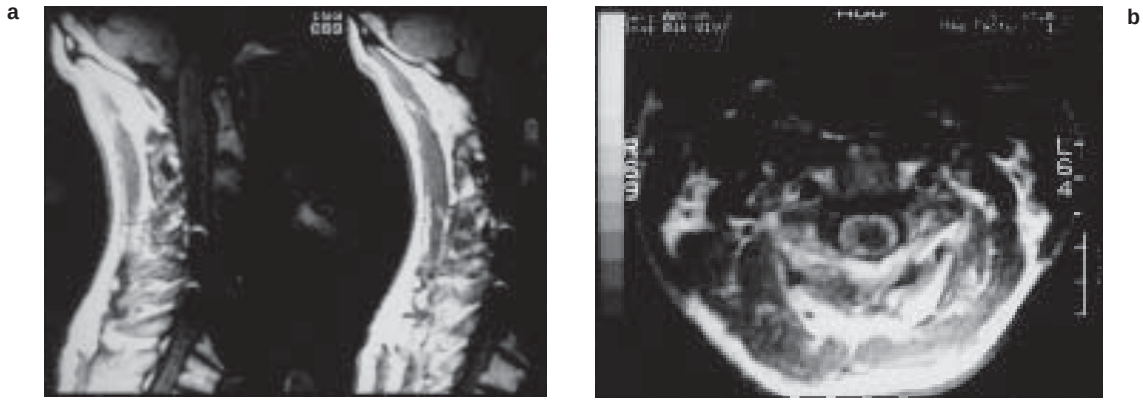


Fig. 5-7. Acute posttraumatic syrinx. This 31-year-old male developed complete C-6 quadriplegia after diving into shallow water; a C-6 burst fracture was evident on radiographs. Initially, wrist extensor strength improved but then at 5 months began to deteriorate, worse on the left than the right. Motor evoked potential latencies to the left biceps and extensor carpi radialis longus became more prolonged. Magnetic resonance imaging revealed a large posttraumatic syrinx extending rostrally to the C-1 level (**a**) and more evident on the left (**b**). Shunting of the syrinx resulted in marked improvement of wrist extension strength and lessening of the patient's neck and arm pain.

per limb muscles by 6 months.[43] Different types of upper limb weakness can be identified electrophysiologically[45] but the optimal rehabilitation interventions for these different types of weaknesses have not been clearly established.

Electrophysiologic studies can be helpful in evaluating SCI. [59,66–69,] They aid in understanding the pathophysiology of the neurologic deficits. For example, they can answer questions such as: Is this primarily upper motoneuron or lower motoneuron-type weakness? Is axonal conduction block or axonal degeneration present? Such knowledge about the pathophysiology can improve prognoses and thus rehabilitation planning. Serial long tract or segmental conduction studies (eg, somatosensory evoked potentials, motor evoked potentials, F-waves or H-reflexes) can aid in determining if residual cord or root compression is present or if neurologic deterioration has occurred (Figures 5-6 and 5-7).

SPINE EVALUATION AND MANAGEMENT

To minimize pain, optimize recovery of the spinal cord, and prevent progressive deformity, it is essential to reduce malalignment and immobilize the spine so bony and ligamentous injuries can heal. The following issues apply to specific vertebral levels of spinal injury.

Atlanto-Occipital Dislocations

These dislocations are commonly missed because they are typically ligamentous injuries; plain radiographs may reveal distraction between the occiput and C-1, often with anterior, vertical, and posterior displacement or any combination of these.[70] Occiput-C-1 injury should also be suspected if (*a*) prevertebral soft tissue swelling is present; (*b*) basion (anterior margin of the foramen magnum)-to-dens is more than 5 mm; (*c*) the clivus is not pointing to the tip of the dens; (*d*) the mastoid does not overlie the dens; or (*e*) the Powers ratio is > 1.0 (Powers ratio is the distance from the basion-to-posterior arch of C-1 divided by the distance from the opisthion [posterior margin of foramen magnum-to-the anterior arch of C-1]). Computed axial tomography with sagittal and coronal reconstructions is usually needed to fully define the instability. Definitive treatment is occipit-to-C-2 fusion with postoperative halo-vest immobilization (Exhibit 5-2). Because the stabilizing ligaments are potentially damaged, axial traction can cause brainstem injury and must be used with extreme caution.

C-1 Ring Fracture

The C-1 Jefferson fracture is a burst fracture of the ring of the atlas, the result of axial loading. If open mouth radiographs reveal lateral displacement of the lateral masses of C-1 on C-2 (sum total overhang on both sides ≥ 7 mm), then the transverse ligament is likely ruptured and the fracture is un-

EXHIBIT 5-2

TYPES OF CERVICAL SPINE ORTHOSES

Soft Cervical Collar: Comfort only, no limitation of spine movement

Philadelphia, Miami J Collar: Limits flexion, not rotation; not effective for upper cervical segments

SOMI* Cervicothoracic Orthosis: Limits flexion, not as good for extension; has forehead strap to allow removal of mandibular pad for eating

Minerva Cervicothoracic Orthosis: Limits flexion, extension, and rotation; not as restrictive as halo-vest

Halo-Vest Cervicothoracic Orthosis: Most restrictive cervical immobilization, but some "snaking" between vertebrae; minimal distraction, may lose alignment mobilizing in halo-vest from traction

*Sternal-Occipital-Mandibular

Source: Benzel EC, Hadden TA, Saulsbery CM. A comparison of the Minerva and halo jackets for stabilization of the cervical spine. *J Neurosurg*. 1989;70:411–414.

stable. The fracture usually decompresses the canal because bony fragments move laterally, often sparing the cord. Treatment is halo-vest immobilization for 3 months.[71]

C-2 Odontoid Fracture

C-2 odontoid or dens fractures are common; they are classified according to the level of dens injury. Type I is a fracture through the upper dens. Type II is at the junction of the dens and the body of the axis. Type III extends into the cancellous bone of the C-2 vertebral body. Type I is rare but usually heals in a Minerva cervicothoracic brace. Type II is associated with poor healing and is managed with a halo-vest or C-1 to C-2 fusion; odontoid displacement ≥ 6 mm is associated with nonunion, so operative fusion is recommended.[72,73] Type III usually heals with halo-vest immobilization.

C-1 to C-2 Atlanto-Axial Instability

C-1 to C-2 instability is due to transverse ligament disruption and is diagnosed on lateral radiograph as increased atlanto-dens separation (ie, > 3 mm for adults, > 5 mm for children). It is usually managed with posterior C-1 to C-2 fusion and postoperative halo-vest immobilization.[74]

C-2 Ring or Hangman's Fracture

The Hangman's fracture is a misnomer; the hanged man, not the hangman, suffers this fracture. Neurologic function may be spared. Type I is minimally displaced (ie, < 3 mm) and is managed with a Minerva cervicothoracic brace for 8 to 12 weeks. Type II is displaced ≥ 3 mm and is managed with a halo-vest for 12 weeks. Type III is associated with C2-3 facet dislocation and usually requires C2-3 fusion with postoperative halo-vest immobilization.[75]

Lower Cervical Spine Injuries

Unilateral facet fracture or dislocation or both is associated with about 25% displacement of adjacent vertebral bodies; bilateral facet involvement is associated with 50% or more displacement. Most can be reduced with cervical traction, although posterior fusion is often used to control persisting instability. Anterior injuries such as a vertebral body burst fracture or acute cervical disk herniation are treated via anterior vertebrectomy or diskectomy followed by a bone graft and often a metal plate to hold the graft in place; postoperatively, a Minerva cervicothoracic brace or a Philadelphia cervical collar may be used to help assure spinal alignment until bony fusion is achieved. Posterior arch injuries, as evidenced by widened spinous processes or facet/laminae fractures, are managed through posterior bony fusion supplemented by wiring between spinous processes or posterior metal plates with pedicle screws. Again, an external cervical brace is usually recommended to assure bony fusion.[76]

Thoracolumbar Fractures

Fractures are most common at the thoracolumbar junction (T-11 to L-2), the junction between the stiff thoracic spine and the mobile lumbar spine. Compression fractures represent wedging of the vertebral body anteriorly with preserved height posteriorly (the anterior column of Denis)[77]; compression fracture of a single thoracic level can be followed with serial radiographs and managed with bed rest, analgesics, and a lumbosacral corset or thoracolumbar Taylor brace for comfort. Lumbar or multilevel thoracic compression fractures are often

managed with TLSO (thoracolumbosacral orthosis, a custom-molded, body jacket); posterior fusion may be required if angulation is greater than 60°. Burst fractures are common axial loading injuries with failure of both the anterior and posterior vertebral body (ie, the anterior and middle columns of Denis) with variable compression of the cord by retropulsed bony fragments.[78] Characteristic imaging studies include loss of height with cortical fracture of the posterior vertebral body on lateral radiograph, increased interpedicular distance on AP radiograph, and retropulsed vertebral bone fragment into the canal on CT scan. Burst fractures may be managed with bed rest in extension for 7 weeks then mobilized in a TLSO for 7 to 11 weeks if there is less than 15° kyphosis, less than 50% canal narrowing, and less than 40% vertebral body wedging. More severe burst fractures require operative treatment to decompress the cord and to prevent progressive deformity. For those neurologically intact, posterior Edwards rods and bony fusion at T-10 to L-2 and posterior plate/pedicle screw constructs at L-3 to L-5 have been recommended.[79] For those neurologically impaired, anterior decompression and Kaneda instrumentation (anterior rods with screws into the vertebral body above and below the fracture) at T-11 to L-3 and posterior decompression with posterior plates/pedicle screws at L4-5 have been recommended.[79] Common flexion-distraction thoracolumbar fractures include seat-belt type injuries and fracture dislocations. Seat-belt type injuries cause damage to the middle and posterior columns of Denis[77]; they do not typically cause neurologic injury. Fracture-dislocation injuries damage all three columns of Denis and result in subluxation or dislocation of adjacent vertebrae. In general, if the middle column of Denis is disrupted then the spine is unstable; exceptions include fractures above T-8 if the ribs and sternum are intact, fractures below L-4 if the posterior elements are intact, and Chance fractures (single-level seat-belt type injuries of the middle and posterior columns with the latter involvement through bone).[77,80] Unstable flexion-distraction thoracolumbar injuries, such as seat-belt type injuries and fracture dislocations, are managed using posterior stabilization with posterior rods (Harrington, Luque, Edwards, or Cotrel-Dubousset rods) or a plate/pedicle screw system. All of the above anterior or posterior operative stabilization procedures are followed by immobilization in a TLSO for 4 to 6 months to assure solid bony fusion.[80,81]

INITIAL REHABILITATION (WEEKS/MONTHS POSTINJURY)

Functional Outcome

Rehabilitation goals vary according to the level of injury and the extent of damage to the cord.[82] When the injury is complete, the functional outcome depends to a large degree on the level of injury. The lower the cord level, the more voluntary movement is preserved and the greater the expectations for independence.

Mobility

Essential to a resumption of life outside the hospital is mobility. In teaching mobility, self-care, and other functional tasks, the following general principles apply: (*a*) start with the simple and move toward the more complex; (*b*) break down tasks into components and begin with discrete units that can be learned separately and then combined into completed units; and (*c*) teach the patient to substitute with head motions, momentum, and preserved muscles for weakened or absent muscles. The expected optimal outcome for a given patient, based on the level and the completeness of the injury, dictates how and which muscles should be trained (Table 5-8).[82]

Mobility encompasses a spectrum of movement from bed mobility (turning from side-to-side, moving from supine-to-sitting), to sitting balance transfers involving wheelchair (from wheelchair-to-bed, wheelchair-to-car, wheelchair-to-floor), to standing balance and ambulation (wheelchair or walking). Each task is mastered, as the patient is able, in physical therapy.

Wheelchair Use

One of the first objectives is to get the patient sitting upright, a task usually attended to by physical therapists (PTs) and nurses. During this procedure, the patient is monitored closely for orthostatic hypotension (OH), a common condition brought on by prolonged bed rest and the deficient vasoconstriction that accompanies disruption of the sympathetic nervous system (see section titled Orthostatic Hypotension later is this chapter). Two aids in achieving upright sitting are the tilt table and the reclining wheelchair. Progressive increase of the verticality of the tilt table challenges the cardiovascular system; over time, this improves systolic blood pressure and cerebral blood flow in the

TABLE 5-8

EXPECTED FUNCTIONAL OUTCOME FOR COMPLETE SPINAL C0RD INJURY

Cord Level	Preserved Muscle	Eating	Dressing	Transfers	Mobility		Writing
					Indoor	Outdoor	
C-1,-2	Trapezius	D	D	D	I	I	I
	Sternocleidomastoid			lift or pivot	power wc sip and puff		computer sip and puff
C-3,-4	Diaphragm	D	D	D	I	I	I
	Neck flexor/extensor			lift or pivot	chin control		mouthstick
C-5	Deltoid	I	D	D	I	I	I
	Biceps	cuff		lift or pivot	power wc		cuff
C-6	Latissimus	I	I?	I?	I	I?	I
	Serratus anterior	cuff		loops	manual wc		wrist-driven
	Extensor carpi radialis			sliding board			splint
C-7	Triceps	I cuff	I reachers adapted clothes	I	I manual wc	I	I wrist-driven
C-8 to T-1	Hand	I	I	I	I	I	I
	Intrinsics				manual wc		
T-2 to T-6	Intercostals	I	I	I	I	I	I
	Paraspinals				manual wc		
T-7 to T-12	Abdominals	I	I	I	I manual wc	I	I
L-2	Iliopsoas	I	I	I	I crutches	I? KAFOs	I
L-3	Quadriceps	I	I	I	I crutches	I AFOs	I
L-4,-5	Tibialis anterior	I	I	I	I	I	I
	Extensor hallucis longus				crutches	AFOs	
L-5 to S-1	Hamstrings	I	I	I	I	I	I
	Gluteus medius				AFOs	AFOs	
S-1,-2	Gluteus maximus	I	I	I	I	I	I
	Gastrocnemius-soleus						
S-3,-4	Anal/urethral sphincters	I	I	I	I	I	I

AFO: ankle-foot orthosis
D: dependent
I: independent
I?: possibly independent
KAFO: knee-ankle-foot orthosis
lift: hydraulic lift transfer
pivot: quad pivot transfer
wc: wheelchair

upright sitting and standing positions. For expected wheelchair users, the same can be achieved at the bedside in a reclining wheelchair with elevating leg rests; the staff can immediately recline the chair back if the patient develops symptomatic hypotension.

As the patient regains mobility, spinal alignment and neurologic status may be affected. If this is suspected, frequent radiographs and neurologic examinations are required to be certain that gravity, postural changes, and muscular forces do not compromise alignment. Once stable sitting is achieved, the patient is fitted for a wheelchair.

The wheelchair is the patient's key to mobility. Helping the patient select the correct chair is a complex task. The chair should be custom fit according to leg length, thigh length, pelvis width, trunk height, and forearm length. It should be efficient, appropriately stable, maneuverable, and matched to the strength and coordination of the user; a wheelchair that enables one patient to negotiate a curb with a "wheelie" may put another at risk for a dangerous fall. Extras to consider include antitip devices, lap trays, arm rests, and foot and leg supports—items that further tailor the chair to the physical condition and needs of its user. Aesthetics and cost must be considered as well.

Individuals with quadriplegia as high as the C-5 level may be able to propel a manual wheelchair. The benefits are exercise, backup in the event that the power wheelchair malfunctions, and the option of traveling in a passenger car. Propulsion for these higher level cervical injuries is possible by the use of the biceps brachii, brachialis, and anterior deltoid muscles in a closed kinetic chain to convert the regular elbow / shoulder flexion to elbow extension when the hand is fixed on the wheelchair handrim by friction. Many of these same individuals will also require a power chair for outdoor mobility involving distances, rough terrain, or grades.

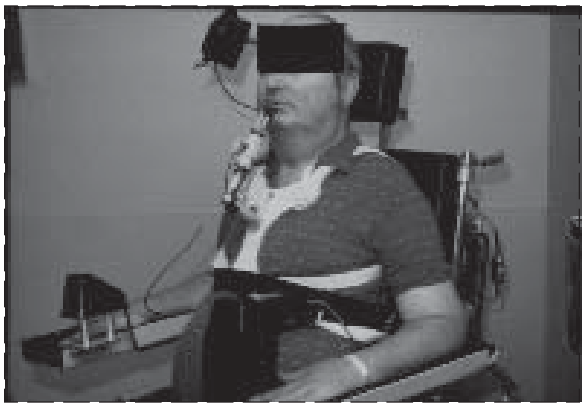


Fig. 5-8. Chin control. This individual with C-4 quadriplegia uses chin control to steer and to recline his power wheelchair and to operate an environmental control unit (ECU) with an infrared interface to a speaker phone, television and light controls.

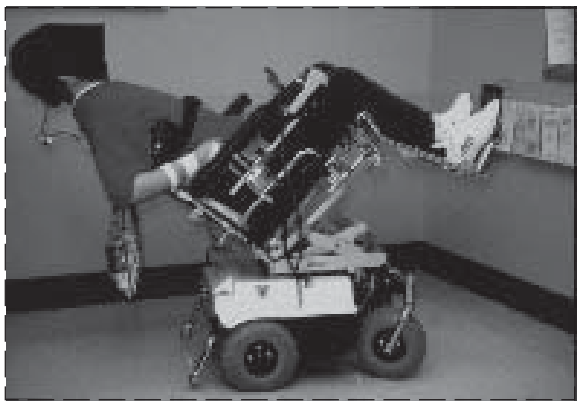


Fig. 5-9. Joystick control and tilt-in-space recline. This individual with C-5 quadriplegia has had sufficient recovery of C-5 innervated shoulder external rotators (supraspinatus, infraspinatus), shoulder abductors (supraspinatus, deltoid), and shoulder flexors and extensors (anterior and posterior deltoid) to operate a joystick hand control for his power wheelchair and a forearm lever for his tilt-in-space recline. This latter type of recline eliminates the hip extension stimulus, which can elicit extensor spasms.

Power wheelchairs are the only feasible means of mobility for some. With the power chair, in addition to the above considerations, the method of control must be determined: manual joystick, chin, sip and puff straw, mouth stick, or tongue. (Figure 5-8). For these patients, reclining mechanisms to minimize the risk of skin breakdown are essential. Two such mechanisms are the zero-shear recline or the tilt-in-space recline; the latter is less likely to elicit extensor spasms (Figure 5-9).

Walking

From sitting, some patients will move to walking. Ambulation goals differ depending on the level of the SCI and whether the cord injury is complete or incomplete. For complete SCI, the goals are exercise, short distance, and level mobility. Those with complete injuries at the T-6 level or below with no other major medical complications are the usual candidates for this training. Contraindicators are limited lumbar spine extension or limited hip, knee, and ankle joint motion. Initially, patients are taught to stand in parallel bars and maintain balance with-

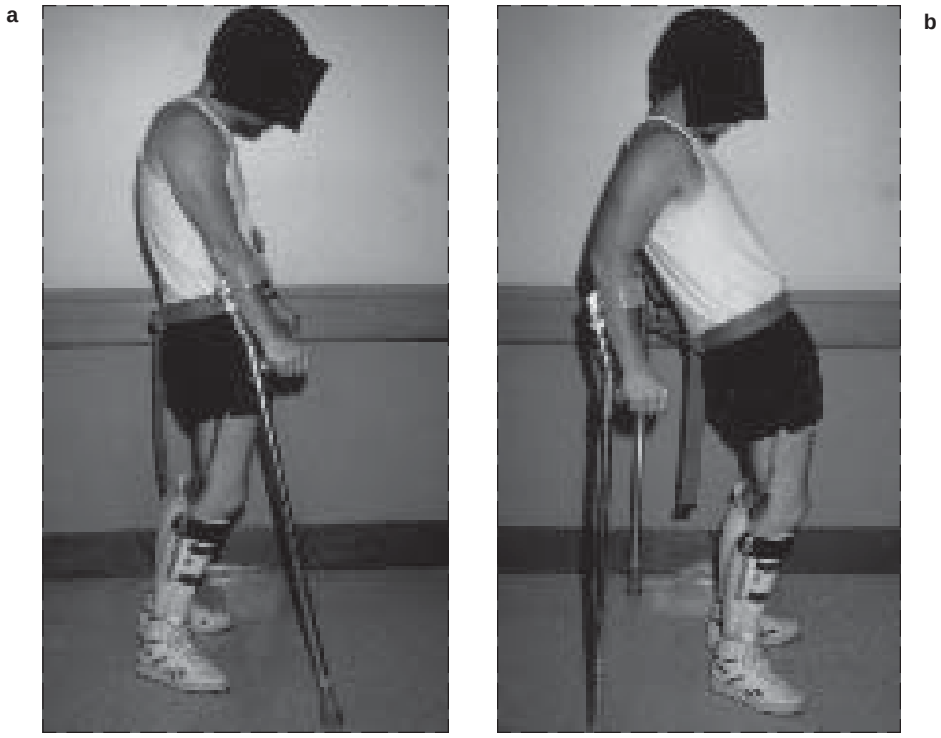


Fig. 5-10. Swing-through gait. This T-6 paraplegic individual ambulates up to one block at high energy cost, using bilateral knee-ankle-foot orthoses, locked at the knee and ankle. He maintains hip extension by positioning the center of gravity of his upper body behind the axis of rotation of his hip joints, both (**a**) at the beginning and (**b**) at the end of each swing-through stride.

out arm support, using temporary knee-ankle-foot orthoses. They then learn to walk with a swing-to gait, using parallel bars for support; with this gait, upper body center of gravity must be behind the axis of rotation of the hip joint to maintain hip extension (Figure 5-10). The energy expended for this type of walking can be as much as 800% of able-bodied walking and manual wheelchair propulsion.[83] This type of walking also requires considerable motor coordination. Because of the high energy and coordination demands, many patients trained in this skill do not continue after discharge. Accordingly, only those who demonstrate good progress and sustained motivation with temporary bracing should be fitted with custom knee-ankle-foot orthoses and proceed to learning a swing-through gait. This requires use of forearm crutches while the patients plant their feet in front of their shoulders for faster walking.

For incomplete SCI, the goals vary widely from assisted standing-pivot transfers to long distance ambulation, depending on the extent of sparing of motor and proprioceptive function and the degree of spasticity. For those with considerable sparing and rapid recovery, therapy is primarily the reconditioning of muscles that have atrophied from disuse. Those with little sparing and slow recovery require months of spontaneous healing, strengthening, and functional training. For the latter, recovery is less complete and spasticity often interferes with movement, although extensor spasms can aid standing.

Driver's Training and Community Mobility

The ultimate community mobility is the automobile. Some patients with injuries as high as C-6 can learn to drive with hand controls, although at the C-6 level, a specialized van with sensitized steering and braking is often required. Those at C-7 or lower can use a passenger car with hand controls. Vision, including visual scanning (the absence of which can sometimes be compensated for with panoramic and side mirrors), reaction time, absence of seizures, adequate spasm control, cognitive awareness, hand function, transfer skills, and a financial source for the necessary equipment and insurance are additional factors determining whether the patient is a candidate for driver's training. If the patient passes this screening, then a vehicle with ap-

propriate adaptations should be acquired. Adaptations include hand controls for brakes and gas, steering wheel attachments, power seats, and vans with hydraulic lifts.

Self-Care

Along with increased mobility, independence in self-care is a major step toward independence for those with SCI. Self-care includes feeding, bathing, dressing, grooming, and toileting. Those with injuries at the C-7 level or below can usually achieve independence in all of these activities. Occupational therapists (OTs) and rehabilitation nurses work with patients to master new techniques for accomplishing these tasks. Alternate strategies such as using a tenodesis grip for holding eating utensils or writing implements must be learned and mastered. A tenodesis grip involves using wrist extension to passively achieve some finger flexion for grip. A wrist-driven flexor hinge splint improves the strength of this tenodesis grip. Other special equipment also aids independence; for example, reachers, universal cuffs, built-up handles on utensils, and a tub bench. Tendon transfers[84] or an implantable functional electrical stimulator[85] may achieve a more functional pinch, although costs and training time are considerable.

With C-6 level or higher injury, partial independence may be achieved. For those tasks that require assistance, instruction takes place on two levels: the patient is taught how to teach others; and a caregiver, often a member of the family, is instructed in how to render assistance or complete the activity. Independence in self feeding with set-up is an example. The assistance needed may include fitting the universal cuff and utensil on the patient, positioning food, opening containers, and cutting meat. With similar assistance, some grooming and hygiene tasks such as combing hair, brushing teeth, shaving, and washing face and upper body may be mastered.

Living Skills

Living skills (meal preparation, shopping, check writing, housekeeping, and so forth) are necessary tasks of every day life and must be relearned and adaptation made. These skills are often reacquired with the help of OTs. Again, reachers and other specialized equipment are used as aids. Spared muscles substitute for paretic muscles, a motor learning task that requires practice. These tasks can usually be managed by patients with injuries sparing C-7 or more. Those with higher injuries will need assistance that may include environmental control systems and computers. These can be accessed via mouth stick, sip and puff, tongue touch pads, eye movements, or voice. Environmental control systems allow the use of telephones, appliances, sound equipment, intercoms, televisions, lights, and door openers—all items that can be powered by electricity through remote control. The complexity of the system will vary according to the extent of the incapacitation and financial resources. Computers allow word processing and written communication, spreadsheet use and money management, graphics and computer-aided design, access to books through CD-ROM discs, on-line bibliographic services and databases, and recreation. The use of a computer, accessed through the keyboard using hand or mouth sticks or through scanning and Morse code, allows a degree of control and a range of vocational and recreational options heretofore impossible.

Vocational and Avocational Pursuits

Computers may be one significant component of an individual's return to an abundant life that includes work and recreation. The patient's significant stake in the outcome makes it essential that he or she take the initiative in exploring what opportunities are available. Tools to aid the person in this search include psychological testing, vocational counseling, assessment of physical capacities, vocational remediation and training (where appropriate), identification of sources of financial assistance to support the training and education, peer counseling, and instruction in job seeking tactics. The rehabilitation staff can also communicate with potential employers. Hiring an individual with a disability can raise a host of unsettling issues that may center on physical accommodations for a disabled person and the psychology of responding to someone who is permanently disabled. Providing technical assistance for work space modifications and the dispelling of fears and stereotypes about the disabled can also aid patients in their employment pursuits.

How the patient will want to utilize time that is not controlled by the demands of living is his or her domain. SCI, at least initially, opens enormous amounts of time for personal pursuits. The patient should be helped to use this opportunity. Eventually, many will want to return to ordered forms of recreation and public activities. Community outings such as attending sports and cultural events, eating at restaurants, shopping, and so forth may serve this need; some will want to return to a form of

physical exercise or sport. Archery, boating, football, camping, hunting, flying, golf, scuba diving, swimming, sailing, basketball, table tennis, weight lifting, tennis, horseback riding, wheelchair racing, wheelchair dancing, and sit skiing have all been successfully pursued by people with SCI. Learning that even with a disability, satisfaction and camaraderie can be found in play with others is essential for SCI patients to realize the positive potential of their future life.

Patient and Family Education

A critical element in a patient's adjustment to disability is the patient's family. Initially, the concerns of the patient and family center on prognosis. Response to these concerns should respectfully acknowledge the uncertainty and difficulty the patient may have in accepting new limitations. Hope, even if based on improbable outcome, helps patients cope with the initial grief. Acceptance of the new reality generally comes with time. Patients and their families can be coached into accepting the necessity of learning alternate methods of mobility and self care even though they long for recovery to the preinjury state.

During rehabilitation the focus is to educate the patient, family, and caregivers on the nature of SCI, how to maximize health, and how to access community resources. Educational topics include the following:

- level and completeness of the SCI, which determine the anatomic and physiological correlates;
- prognosis for motor recovery and spasticity;
- current research on spinal cord regeneration and new technology;
- care of neurogenic bladder and bowel;
- prevention of skin breakdown;
- management of autonomic hyperreflexia;
- psychological adjustment to disability;
- attendant care management;
- sexual adjustment and fertility;
- vocational and educational options;
- avocational outlets such as wheelchair sports;
- finances; and
- housing.

Various educational formats can be used: a patient manual for self instruction and later reference, weekly class discussions, peer interaction, and one-on-one instruction provided by each member of the rehabilitation team. Intake and discharge panels with the rehabilitation team and the patient and his or her family further complement this instruction. The intake panel defines specific goals for the hospitalization and addresses specific questions related to prognosis and course of treatment. The discharge panel discusses plans for follow-up, equipment needs, and accessing community resources. For those who will be discharged partially dependent, family members and other caregivers need to be instructed in their roles. An on-site apartment for independent living allows patients and their caregivers to rehearse typical days that include shopping, meal preparation, transfers, toileting, and bathing with staff backup available as needed. Day and weekend passes are also scheduled once the patient is medically stable and sufficiently trained in self care and mobility. Community outings with the recreation therapist that include experience with public transportation, shopping malls, movies, grocery shopping, banking, and eating in restaurants allow patients to practice their recently acquired mobility and self care skills and to begin experiencing community reintegration.

Housing, Finances, and Community Reintegration

Social work can assist patients in understanding social security disability, veterans affairs (VA) benefits, separation from the military, Medicare/Medicaid benefits, accessible public housing, nursing homes, housing modifications, advocacy groups for the disabled, legal resources, and protections. In addition, occupational therapy can assist patients in understanding accessibility, including ramp inclines, door widths, and so forth.

HEALTH MAINTENANCE (YEARS POSTINJURY)

Lifetime annual evaluations are standard for discharged SCI patients, although initially the patient may need more frequent visits. Because of the risk of medical complications and functional decline, the annual assessment should be comprehensive. In addition to a general history and physical examination, assessments of urologic and neurologic, skin functional status, and equipment needs should be included. The physical health of the patient is only one aspect under review during this examination; the patient's psychosocial and vocational adjustments should also be assessed with additional ex-

pertise sought from other members of the rehabilitation team when appropriate.

Late mortality and morbidity due to SCI are higher in those patients with quadriplegia than paraplegia (1.4-fold higher) and higher in those with complete rather than incomplete SCI (1.5-fold higher).[6] At age 20, the late mortality rate of those with SCI is 8-fold that of the age-matched general population (7.2 vs 0.9 per 1,000); at age 70, the mortality rate on those with SCI is 1.5-fold that of others (75.1 vs 50.2 per 1,000).[6] Mortality is due to urologic causes in 24%, cardiovascular in 23% and pulmonary in 14%; however, mortality rate due to urologic causes has fallen dramatically from 43% of the deaths occurring in the 1940s and 1950s to 10% of the deaths occurring in the 1980s and 1990s.[6]

Preventing and Managing SCI Complications

During acute rehabilitation and thereafter, the SCI team must watch for medical complications associated with SCI. Many need early diagnosis and treatment to minimize long-term sequelae. In one clinical study, pressure sores and UTIs had annual incidences of 23% and 20%, respectively, and were the most common complications.[6] Some complications develop most often in the first several years postinjury (eg, hydronephrosis, spasticity, contractures, and suicides). Other complications demonstrate greater prevalence with increasing time since the SCI (eg, musculoskeletal problems). Still other complications are most closely associated with the patient's age (eg, cardiovascular complications, pneumonia, and renal stones). Common SCI complications are discussed below.

Pulmonary Complications

The inspiratory phase of breathing requires active muscle contraction and depends primarily on the diaphragm innervated at C-3, C-4, and C-5 levels. Resting expiration is passive and depends on the viscoelastic properties of the lung and chest wall; forced expiration is active, as in a cough for clearing secretions from the lungs, an action requiring rapid contraction by the abdominal muscles innervated at the T-6 to L-1 levels; the clavicular portion of the pectoralis major contributes to active expiration in quadriplegic individuals.[86] Impairment of these two pulmonary actions (inspiratory phase of breathing and forced expiration) cause most SCI pulmonary complications.[87]

Acute Respiratory Failure. The earliest pulmonary complication is acute respiratory failure. This condition develops most commonly in those with cervical cord injury at the C-5 level or higher and may appear immediately after the injury or develop over hours to several days as respiratory muscles fatigue. Close monitoring is needed to detect the muscle fatigue *before* a respiratory arrest, and emergent intubation is required; the fatigue manifests itself as tachypnea, decreased tidal volume, decreased inspiratory pressure, hypoxemia, and hypercapnia. A partial pressure of CO_2 above 40 mm Hg or vital capacity below 1,000 mL suggests the need for intubation and mechanical ventilation. Careful attention must be directed to aggressive pulmonary toilet and early detection and treatment of pneumonia.[88] The majority of these patients eventually wean from the ventilator over a period of days, to weeks, to months. Forced vital capacities (FVCs) typically improve over a period of months, presumably a combination of spontaneous neurologic recovery and strengthening of the respiratory muscles. After 1 to 2 weeks, if weaning is not imminent, a tracheostomy is performed; prolonged endotracheal intubation has been associated with airway complications such as subglottic stenosis. A tracheostomy tube also allows direct tracheal suctioning and bronchoscopy and communication through leak speech.

For intubated individuals, communication is a major issue. Initially, tongue clicking or exaggerated eye blinks can be used for yes or no signaling, but head nodding may not be possible because of spine instability or neck muscle paralysis with C-1 or brainstem level injuries. Communication can also be pursued through lip reading or a communication board. Eventually, as pulmonary status stabilizes, patients can use leak speech, where the tracheostomy cuff is deflated and large inspiratory volumes are delivered, enabling air to leak around the tracheostomy tube and upward through the larynx, allowing speech. Fenestrated tracheostomy tubes, with built-in windows to provide continuous air leak for speech, and talking tracheostomy tubes, which supply alternative sources of air, are available at more expense. Ventilator-dependent patients must always have with them a suction device for clearing secretions and an Ambu bag in the event of ventilator failure.

Other factors affecting inspiration are abdominal distention and posture.[89] The quadriplegic patient begins at a considerable disadvantage because the chest wall retracts during inspiration when the descent of the diaphragm creates a negative intrathoracic pressure. This intercostal retraction, which is normally prevented by intercostal muscles,

cancels a portion of the inspiratory effort. Further exacerbating the problem is the tendency of the atrophied abdominal wall muscles and abdominal organs to displace anteriorly, pulling the diaphragm downward when the patient is sitting. Diaphragm muscle contractions then begin in a shortened position and generate less inspiration. Ileus, acute gastritis, and fecal impaction can all cause abdominal distention to further compromise diaphragm descent. An elastic abdominal binder can help counteract this displacement when patients are sitting upright; it may also increase the loudness of speech.[90] Obstructive sleep apnea, more prevalent in SCI individuals than in the general population, may lead to disrupted sleep, daytime drowsiness, personality changes, and even cor pulmonale; quadriplegia, obesity, sleeping in the supine position, and sedative ingestion may be predisposing factors.[91,92]

Improved pulmonary function has been reported following breathing exercise programs, arm ergometry, and functional electrical stimulation.[86,93,94] Prevention programs (eg, smoking cessation, flu and pneumococcal vaccinations) should be established for all SCI patients, with a particular emphasis on quadriplegic individuals. Periodic pulmonary function tests are useful as part of a health maintenance program.

Atelectasis, Pneumonia, Aspiration, Impaired Cough. Pneumonia is the leading cause of death during the first several months following SCI.[57,95] Almost all SCI patients, with the exception of those with lesions at a low thoracic level or below, have impaired cough because of the loss of cough-elicited abdominal muscle contraction. Those with cervical cord injury have impaired inspiratory effort as well. Impaired cough and impaired inspiration predispose to atelectasis. Mucous hypersecretion and hyperviscosity, along with impaired cough, contribute to mucous plugging. All of these factors predispose to pneumonia. Those most vulnerable are patients with high cervical cord injuries. The site of pneumonia is most often the left lower lobe, where the sharper angulation of the left mainstem bronchus makes suctioning more difficult.

The following precautions promote clearing and reduce the risk of pneumonia: (*a*) turn the patient at least every 2 hours to promote gravity-assisted postural drainage, (*b*) offer incentive spirometry, (*c*) clap the chest wall and assist cough by manually compressing the abdomen in synchronization with the patient's cough (ie, quad cough), (*d*) institute intermittent positive pressure breathing with bronchodilator followed by assisted coughing, and (*e*) use a rotating bed (eg, Roto-Rest Traumabed, Kinetic Concepts, San Antonio, Texas).[70,96] Training in glossopharyngeal breathing (GPB) can improve assisted coughs and may allow time off a ventilator for some patients with rostral cervical cord injuries.[97]

Fever, increased purulent sputum, altered auscultation of the lungs, and chest radiographic changes suggest pneumonia. Treatment for pneumonia involves more frequent and more aggressive pulmonary hygiene and parenteral antibiotics. Thick secretions and consequent mucous plugs may require bronchoscopy.

An additional pulmonary problem is aspiration. Those with SCI at T-10 or above are less able to cough effectively and clear the airway if they aspirate. Precautions to minimize aspiration risk include (*a*) no oral intake and gastric decompression by nasogastric tube for those with gastroparesis or ileus, (*b*) upright or side-lying for eating, and (*c*) no assisted coughing by manual abdominal compression immediately after meals. Patients with suspected aspiration should have a swallowing evaluation that compares the effects of various consistencies, amounts, and techniques and develops optimal feeding strategies. Hyperextension of the neck in a cervical brace is a predisposing factor to aspiration, which should be considered. As a long-term precaution, family members and caregivers should be trained to perform the Heimlich maneuver for someone who is lying down or sitting.

Cardiovascular Complications

A variety of cardiac complications can compromise health for the spinal cord injured. Several etiologic factors contribute. With cervical or upper thoracic cord lesions, sympathetic outflow to the heart and blood vessels is disrupted; the heart is then influenced by unbalanced parasympathetic activity. Manifestations of deficient sympathetic outflow include bradyarrhythmia, asystole, other rhythm disturbances, postural hypotension, and autonomic hyperreflexia. Loss of tonic arteriolar vasoconstriction results in vascular pooling and a lower baseline blood pressure; expected blood pressure for a person with quadriplegia is 90/60 mm Hg. In addition, decreased lower extremity muscle activity leads to venous stasis and the compromise of venous return. A low level of high density lipoprotein (HDL) as a result of reduced physical exertion is a long-term cardiovascular risk factor.

Bradyarrhythmia, Sinus Arrest. Bradyarrhythmia leading to cardiac arrest is a serious, early complication, its incidence peaking at day 4 postinjury

in those with cervical cord injuries.[98] These injuries impair cardiac sympathetic outflow arising at T-1 to T-4, which accelerate the heart. The remaining innervation is parasympathetic; vagal input slows the heart rate. If deceleration is severe, cardiac arrest may result. Tracheal suctioning or hypoxemia can trigger such a bradyarrhythmia, presumably via a vagovagal reflex. Preventive measures include preoxygenation and atropine prior to suctioning to inhibit the cholinergic receptors of the vagal efferents to the heart. An atropine dose of 0.6 mg to 1.0 mg given intravenously 15 minutes prior to suctioning may prevent these vagal-mediated bradycardic episodes. Oral ephedrine sulfate or continuous intravenous infusion of very low-dose isoproterenol have also been suggested; quadriplegic subjects are hypersensitive to these adrenergic agents.[98] Occasionally patients may require transvenous cardiac pacing.

Succinylcholine, a depolarizing paralytic medication, is often given as an adjunct to general anesthesia, but in SCI patients this can result in profound hyperkalemia with cardiac arrest because the neuromuscular junction is hypersensitive to cholinergic agents.[47] Succinylcholine is absolutely contraindicated in SCI patients.

Deep Venous Thrombosis, Pulmonary Embolus. Another common and potentially fatal complication of acute SCI is DVT with consequent pulmonary emboli.[88,99] Deep venous thrombosis usually develops within the first 3 months postinjury, with a peak at 10 to 14 days. It is uncommon after 3 months. Known predisposing factors are venous stasis, a hypercoagulable state following trauma, and venous intimal damage. All of these factors are commonly present in SCI patients. Preventive measures include performing a baseline venous Doppler examination to exclude early DVT, prescribing subcutaneous heparin (5,000 units used twice daily), and applying venous sequential compression pumps to the lower extremities.[100,101] Twice daily thigh and calf circumference measurements aid early detection of DVT. A difference of as little as 1 cm is suggestive; care for accuracy and consistency of measuring points is essential. If a sudden increase or asymmetry in lower extremity circumference develops or if the patient experiences unexplained low-grade fever or sudden onset dyspnea or chest pain, then DVT must be suspected and prompt action taken.[102] Clinicians must always have a high index of suspicion. Terpie found that fewer than 50% of patients with documented venous thrombosis had the characteristic signs of fever, redness, and swelling.[103] Diagnosis can be made with duplex scanning alone or with a simple Doppler examination and confirmatory contrast venogram. Surveillance via Doppler, duplex, or impedance plethysmography scanning may need to be done at least twice weekly for the first 2 weeks. The differential diagnosis includes lower extremity fracture, lower extremity hemorrhage, HO, dependent edema, cellulitis, and postphlebitic edema. If confirmed, DVT is treated with continuous intravenous heparin, venous compression stockings and Ace wraps, and bed rest without lower extremity range-of-motion exercises for 1 week. Heparin is discontinued after oral Coumadin becomes therapeutic; Coumadin is then continued for 3 months. If anticoagulation therapy is not possible in a given patient or if pulmonary emboli occur despite anticoagulation, then an inferior vena cava filter should be considered. Quad coughing should not be performed in patients with vena caval filters.

Autonomic Hyperreflexia. Autonomic hyperreflexia, also often called autonomic dysreflexia, is a delayed sequela of high thoracic or cervical cord injury.[104] This unique manifestation of SCI above the T-7 level presents after skeletal muscle hyperreflexia returns. Loss of supraspinal control results in unmodulated norepinephrine release and exaggerated vasoconstriction of arterioles receiving sympathetic innervation from below the level of the cord injury. Resultant blood pressure elevation may be moderate (140–160/90–100 mm Hg) or severe (> 180/110 mm Hg). The trigger is bladder or bowel overdistension or some other noxious stimulation. Symptoms are pounding headache, flushing, and diaphoresis. These symptoms result from high and potentially life-threatening systolic hypertension, sometimes reaching 250 mm Hg and requiring the following emergency measures: (*a*) elevate the head of the bed to promote dependent pooling of blood and to lower the risk of intracerebral bleed; (*b*) eliminate viscus distension or noxious stimulus quickly by catheterizing the bladder or untwisting a condom catheter; an indwelling catheter may need to be replaced; (*c*) if hypertension persists, give sublingual nifidepine 10 mg and repeat every ten minutes as needed to a total of 30 mg.[104,105] Other antihypertensive medications are also used including phenoxybenzamine, prazosin, mecamylamine, nitrates, and clonidine.[105] Nitropaste may be useful for hyperreflexia that lasts hours or days. Once the blood pressure has been lowered, then an attempt must be made to identify and eliminate or minimize other noxious stimuli contributing to autonomic hyperreflexia. Bowel impaction should be removed with digital disimpaction, using premedication with

nifidepine and xylocaine jelly as a rectal anesthetic to avoid further aggravating the condition. Peptic ulcer, cholecystitis, appendicitis, bowel obstruction, rectal fissure, ureteral stone, UTI, infected ingrown toe nail, fracture, or labor and delivery are other common stimuli that should be considered.[104] In SCI patients with renal disease, autonomic hyperreflexia must be distinguished from renal vascular hypertension.

Severity varies among patients; in some, blood pressure elevations cause excessive diaphoresis while in others, hypertension can lead to retinal or intracerebral hemorrhage, seizures, or death.[106,107] Patients are encouraged to carry an Autonomic Hyperreflexia Treatment Card to facilitate prompt and appropriate treatment by health professionals less familiar with complications of SCI.

Orthostatic Hypotension. An early and occasionally chronic problem that is less threatening but nonetheless disabling, is symptomatic orthostatic hypotension (OH). With OH, patients complain of dizziness, lightheadedness, and fainting. In general, OH is most severe in patients with higher lesions (typically cervical and high thoracic SCI) and in patients with longer bed rest. Several factors may contribute: impaired vasoconstriction and consequent pooling of blood in the lower limbs and splanchnic bed, decreased sympathetic drive to the heart, and relative volume depletion. Most at risk are quadriplegic patients; they typically run blood pressures of 90/60 mm Hg. These patients often become orthostatic when first sitting in the morning. To minimize these episodes, such patients can be fitted with an abdominal binder, venous compression stockings, and Ace wraps; liberal salt and fluid intake and elevating leg rests may also help avert orthostasis. The patients may require ephedrine sulfate (25–50 mg one-half hour before sitting) or mineralocorticoid as well. In acute SCI, OH improves with repeated sitting trials, which promote fluid retention; quadriplegics are known to run high renin and aldosterone levels, a presumed mechanism for compensating for orthostasis. Late worsening of OH could suggest development of posttraumatic syringomyelia or a silent myocardial infarction.

Silent Myocardial Infarction and Coronary Artery Disease. As individuals with SCI live longer, cardiovascular disease has become a major source of morbidity and mortality, and is likely related to increased incidence of obesity, diabetes, and lowered HDL levels after SCI.[108,109] Another cardiac complication for the cervical cord injured is a silent heart attack. Cardiac ischemia and infarction in this population may be painless because the cardiac afferents course along cardiac sympathetic nerves and enter the spinal cord at T-1 to T-4. In such cases, the clinical manifestations may be subtle and nonspecific, perhaps increased orthostasis, dyspnea on exertion, orthopnea, or increased pedal edema.

Gastrointestinal Complications

Altered sympathetic outflow due to thoracic or cervical SCI may also alter GI function and cause delayed gastric emptying, increased gastric acid secretion, and disturbed colonic myoelectric activity.[110] SCI can alter motility of the descending and sigmoid colon and anal sphincter by impairing parasympathetic outflow from sacral segments. Acute abdomen is often diagnosed late with a resulting high morbidity and mortality in patients with SCI above T-10 having impaired somatic sensation; such patients may report poorly localized pain at a later stage of acute abdomen.[111,112] The physician caring for the SCI patient must maintain a high index of suspicion for acute abdomen and be alert to its minimal manifestations in SCI, which may be limited to tachycardia, increased spasticity, and autonomic hyperreflexia.

Ileus. Ileus or loss of GI motility is noted in 63% of SCI patients for several days and occasionally for weeks after acute SCI.[113] To minimize the possibility of vomiting, aspiration, and compromised respiration, acutely injured SCI patients often should have a nasogastric tube placed for decompression. The patient may report nausea and loss of appetite. Abdominal or gastric distention and absent bowel sounds may require managing the patient NPO (nothing by mouth) with intravenous fluids and a nasogastric tube for suctioning. Metoclopramide is reportedly of benefit for prolonged gastroparesis or ileus. [114]

Stress Gastritis or Ulcer. Stress gastritis or ulcer is another early complication, reported in 5% to 20% of acute SCI patients.[115,116] The most vulnerable are the aged and those with injuries above T-5, where spinal shock leaves the parasympathetic stimulation to the stomach unopposed, resulting in a consequent increased in acid secretions. Additional sources of irritation to the stomach lining are corticosteroids, which decrease mucosal resistance; gastric distention; hypotension; and sepsis. Additional risk factors are mechanical ventilation and heparinization. Prevention involves use of an H_2 blocker and antacids to maintain a pH greater than 4, continuous suction via nasogastric tube to prevent gastric overdistension, and monitoring gastric secre-

tions for occult blood. Should prophylaxis prove inadequate, upper GI endoscopy or angiography (if the cervical spine is unstable) will confirm GI bleeding. If active bleeding is detected, then blood pressure must be maintained with fluids and transfusions and coagulation defects corrected. Laser coagulation and intraarterial vasopressin may be of benefit.

Obstipation, Fecal Impaction, Pseudo-Obstruction, Megacolon. Decreased colonic motility, increased colonic compliance, and perhaps anal sphincter spasticity in those with chronic SCI may result in obstipation and fecal impaction.[117] Key elements of a bowel program include (*a*) adequate fluid intake (1,500–2,000 mL/day), (*b*) high fiber diet (40–60 gm/day), (*c*) bulk cathartics such as psyllium hydrophilic muciloid, (*d*) regular mealtimes, and (*e*) timed bowel programs with evacuations scheduled for every day or every other day, using rectal suppositories (glycerin or bisacodyl) and digital stimulation as necessary. Evacuation intervals of more than 3 days increase the risk of impaction and incontinence. The goal of a good program is continence with focus on techniques that minimize mechanical damage and irritation to the colon and promote long term health.[118]

Patients reporting poor results with a bowel program may manifest nausea, vomiting, abdominal distension, early satiety, and shortness of breath from compromised diaphragm descent. Reviewing the elements of the bowel program and ensuring that patients are not on medications that slow gut motility (eg, anticholinergics, narcotics, tricyclics, clonidine, etc.) are the first steps to remediation. In addition, the patient may require periodic hyperosmotic laxatives (milk of magnesia, lactulose, sorbitol). If this is unsuccessful, saline or phosphosoda enema or whole gut irrigation can be tried. Because enemas tend to stretch the bowel causing a loss of muscle tone and perhaps autonomic hyperreflexia, they should be used judiciously. In severe cases, nasogastric decompression, with or without a rectal tube, may be required. Rarely, colonoscopy or surgical decompression must be considered if cecal diameter is greater than 12 cm. For those with recurrent bowel obstruction or markedly prolonged bowel programs, refractory to conservative measures, colostomy may be an optimal solution.[119] The mean time spent on bowel care in 20 SCI patients with chronic bowel problems, dropped from 99 minutes to 18 minutes per day after a colostomy.

Cholelithiasis and Colon Carcinoma. The prevalence of gallstones and colon carcinoma is elevated in spinal cord injured patients.[118] The increase is 3-fold in cholelithiasis and 4-fold with colon carcinoma. In quadriplegic or high thoracic SCI, cholelithiasis can be associated with few symptoms, delayed diagnosis, and autonomic hyperreflexia. Treatment with chenodeoxycholic acid or cholecystectomy should be considered. Slow colon motility could contribute to the increased incidence of colon carcinoma. Delay in diagnosis of colorectal disease is common, sometimes leading to life-threatening abdominal emergencies.[120] Regular screening with fecal occult blood tests, flexible proctosigmoidoscopy, barium enema (beware of barium retention), and colonoscopy should be considered.

Endrocrinologic Complications

Two endocrinologic complications that are brought on or exacerbated by SCI are hypercalcemia and insulin-dependent diabetes.

Hypercalcemia. Hypercalcemia is uncommon but occurs most often in male adolescent quadriplegics in the first months after injury. In addition to gender and youth, risk factors include complete neurologic injury, high cervical injury, and prolonged immobilization. Symptoms and signs are subtle and include malaise, anorexia, nausea, vomiting, constipation, polydipsia, and polyuria. Left untreated, it can progress to lethargy and coma. Laboratory studies reveal hypercalcemia, hypercalciuria, and low creatinine clearance. Treatment includes intravenous saline with furosemide and remobilization. Additional measures may be needed to inhibit osteo-clast-mediated bone resorption: bisphosphonates (etidronate, pamidronate), calcitonin, mithramycin, glucocorticoids, and gallium nitrate.[121,122]

Insulin-Dependent Diabetes Mellitus. Spinal cord injury has profound metabolic consequences resulting in disorders of carbohydrate and lipid metabolism.[108] Abnormal glucose tolerance and hyperinsulinemia are common following SCI. SCI complicates the management of diabetes mellitus in two ways: (1) increased insulin resistance caused by the decreased muscle mass and obesity of the SCI patient, and (2) absence of adrenergic hypoglycemic symptoms (diaphoresis and tachycardia) in quadriplegics. For the second complication, cultivating an increased awareness of the neuroglycopenic symptoms (drowsiness, impaired mental status, seizure) is vital. Treatment for hypoglycemia with subcutaneous or intramuscular glucagon or intravenous dextrose should be anticipated. Treatment for hyperglycemia is standard: caloric restriction, exercise, oral hypoglycemic agents, and insulin therapy.

Skin Issues

Pressure Sores. Pressure sores are a major bane of SCI care. Incidence has been estimated as high as 42% to 85% in some centers.[123] Prevention is vital to controlling SCI health care costs and optimizing patient functioning. The cost of treating pressure sores in the United States is estimated to be $3 to $5 billion per year.[124,125] Causes are unrelieved direct pressure, skin shearing, and prolonged exposure to moisture. Activities and conditions that predispose to the development of pressure sores are prolonged sitting, severe spasticity, contractures, edema, anemia, poor nutrition, bruises and skin damage resulting from falls or scrapes, worn or inadequate cushions, urinary or fecal incontinence, and smoking.[126,127] Avoiding these conditions is a matter of education, awareness, behavioral change, and commitment—factors heavily dependent on the psychological well-being of the individual and the supportiveness and competency of the medical staff and caregivers. Biochemical changes have also been noted in skin following SCI and can contribute to increased susceptibility to pressure ulcers.[128–130]

Standard preventive measures for the acutely injured SCI patient include avoiding pressure by using a foam mattress and turning every 2 hours. Some conditions that put acute SCI patients at risk are anesthetic skin; immobility from paralysis; hypotension; spine immobilization measures; poor nutrition and subsequent weight loss; fever; urinary and fecal incontinence; flexor spasm; and sometimes altered mental status such as coma, depression, or chronic alcohol use. From 20% to 40% of patients develop pressure sores during the first month following SCI.[131]

Alternatives to standard measures are specialized beds such as the Roto-Rest bed and Stryker or Foster turning frames. These are expensive but of particular use when patients are in cervical traction. Low-air-loss beds (eg, Clinitronbed, Support Systems International, Inc., Charleston, South Carolina, or Kinnairbed, Kinetic Concepts, San Antonio, Texas) are also expensive but are useful for patients with multiple active pressure sores that result in insufficient lying surfaces and for those who are recovering from myocutaneous flap surgery. Less frequent turning is usually possible with use of these special beds.

No matter the predisposing factors, the cause of pressure sores is prolonged pressure or shear or both, sufficient to cause underlying skin and muscle necrosis from ischemia. Pressures as small as 20 cm H_2O occlude capillary blood flow. This low level of tolerance means it is impossible to distribute the weight of the torso over buttocks and posterior thighs without blocking capillary flow. Thus, for all but children (with less body weight), it is theoretically impossible to design a cushion that will eliminate ischemia. However, optimal distribution of weight over sitting or lying surfaces can prevent the development of necrosis as the inevitable capillary ischemia is regularly relieved by turning or by pressure releases. When lying down, patients require turning every 2 to 4 hours. When sitting, a pressure release for 15 seconds every 15 minutes is usually recommended. The greater frequency of pressure releases in sitting derives from the distribution of body weight over a smaller surface.

Pressures are highest over bony prominences; consequently, pressure sores arise most commonly over the ischial tuberosities and calcanei while sitting, and the greater trochanters, occiput, sacrum, scapulae, malleoli, and calcanei while lying. Other secondary factors that contribute to pressure sores are impaired circulation resulting from peripheral vascular disease as in diabetes mellitus, poor nutrition with hypoproteinemia and anemia, and obesity.

Relief of pressure while sitting in a wheelchair is achieved with cushions that distribute weight optimally. A well-fitted wheelchair, with a solid rather than sling seat, a high-density foam cushion, and pressure releases for 15 seconds every 15 minutes are the standard precautions. Specialized cushions are available for those at high risk: the Jay cushion (Jay Medical Ltd., Boulder, Colorado), made of contoured foam with an overlying gel pad that redistributes weight from the ischial tuberosities to the posterior thighs; and the Roho cushion, constructed of multiple inflated bulbs that distribute weight over a wide area by varying the position of the upper body.[132]

In addition, patients or caregivers must be taught to check the skin twice daily for any nonfading redness or skin breakdown. If noted, patients must alter their positions and routines to keep the affected areas free of pressure until the redness disappears or the skin heals. Patients are taught to avoid burns by avoiding cigarette ash falling on anesthetic skin, to avoid hot water bottle use or placing hot plates or mugs on their thighs, to sit away from fires or radiators, and, to check bath water with a thermometer (less than 98°F or 36.5°C). They also learn to avoid applying condom catheters or leg straps too tightly (Figure 5-11), to keep nails trimmed, to check shoes for nails or other sharp objects before donning, and to avoid tight shoes. Males must routinely

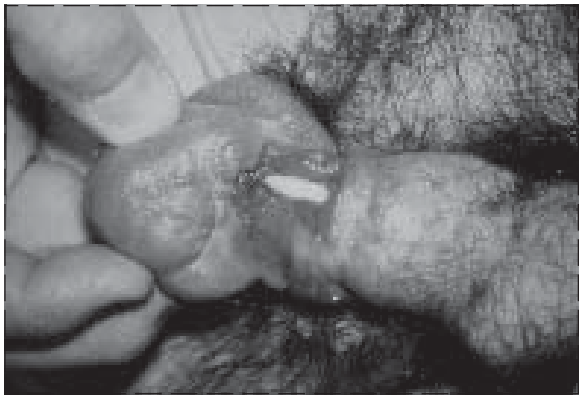


Fig. 5-11. Penile and urethral sore from tight condom catheter. This individual applied his condom catheter too tightly using elastic tape, resulting in a deep penile sore, which extended into the urethra. Such complications can be avoided only through patient or attendant education.

pull the scrotum forward after transferring to sitting positions, to avoid sitting on insensate testicles.

Pressure sores usually develop in 30% of acute SCI patients because adequate preventive measures have not been instituted and followed. In defining and classifying these sores, the following scale has evolved:

- Grade 1: nonfading redness, an incipient sore.
- Grade 2: partial thickness skin loss, involving epidermis or dermis, but not exposing subcutaneous tissue.
- Grade 3: full thickness skin loss exposing or involving subcutaneous tissue but not involving muscle.
- Grade 4: exposed muscle or fascia, no bone observed.
- Grade 5: bone exposed.
- Grade 6: joint space involved.

Superficial pressure sores can heal with conservative treatment; deeper wounds may require surgical intervention. The choice to use surgery (generally with ulcers of grades 4-6) is made when the sore is too large to heal with conservative measures over a period of 3 to 4 months, when a deep infection must be drained, or when a bony deformity must be corrected. For superficial wounds without bacterial infection, special hydrocolloid dressings are available.[133] Bedside debridement with scalpel, enzymatic ointments, or wet-to-dry dressings are used to clean the wound of necrotic tissue. Deep or undermined sores are packed with wet-to-dry sterile gauze to clean the wound thoroughly. When clean, granulated tissue is noted, wet-to-wet gauze or hydrocolloid dressings can be used to maintain a moist wound environment, which encourages epithelization.[120] Minimizing anemia and promoting nutrition are other measures that enhance healing. Recent research suggests that growth factors (eg, platelet-derived growth factors) increase the rate of healing. High voltage pulsed current has also been evaluated.[5] These interventions, along with a detailed analysis and correction or mitigation of the factors contributing to the development of the sores, are essential.

If the choice is surgery, myocutaneous flaps are the standard treatment. A myocutaneous flap frees up skin and underlying muscle while preserving the blood supply, and repositions it over the sore. The most common examples are gluteus maximus rotation flaps to cover sacral or ischial sores; hamstring V-Y advancement flaps to cover ischial sores; and tensor fascia lata, vastus lateralis, or rectus abdominus flaps to cover trochanteric sores. Skin grafts can be used over granulated tissue. The possible need for future grafts should be considered when tissue sites are chosen. Postoperative care requires rigorous attention to ensure success. Following surgery, patients are best served by management on a Clinitron bed (Support Systems International, Inc., Charleston, South Carolina) for a period of 3 weeks to minimize pressure on the flap. During this time, avoiding any tension on flap incisions is essential. For patients with spasms, this may mean increased spasmolytic medication and positioning to minimize hip flexor spasms. After 3 weeks, if the flap is healing well, patients progress to a regular hospital bed with a foam mattress, and lower extremity range-of-motion with direct observation of the flap suture lines. At a later date, patients are gradually remobilized in a fitted wheelchair with appropriate cushion and reinforcement of the need to perform regular pressure releases and twice daily skin checks.

Cellulitis, Osteomyelitis. Most cellulitis and osteomyelitis connected with SCI originate in pressure sores through the direct spread of infection.[134] With cellulitis (a spreading local infection in the skin), the common presenting symptoms are swelling, redness, local warmth, and fever. In osteomyelitis, the symptoms are fever, shaking chills, and purulent drainage. Laboratory findings in both include leukocytosis, elevated erythrocyte sedimentation rate, and positive blood cultures. A radiograph may show periosteal reaction or lytic lesions (Figure 5-12). Bone scans are of limited use because

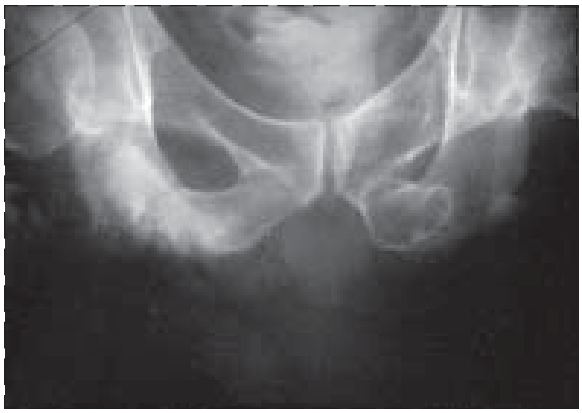


Fig. 5-12. Osteomyelitis underlying an ischial decubitus. This paraplegic individual with a history of paranoid schizophrenia developed recurrent ischial decubiti despite patient education to perform twice daily skin checks and pressure releases every 15 minutes and despite being fitted with a contoured foam/gel cushion. Note the irregular, sclerotic ischial bone indicating osteomyelitis below the decubitus.

they are usually positive below a pressure sore, whether the underlying bone is infected or not. Definitive diagnosis of osteomyelitis is possible through bone biopsy and histologic confirmation, with more reliable results obtained through a needle biopsy, which avoids the bacteria in the decubitus tract.

Cellulitis is treated with oral or parenteral antibiotics usually for 10 to 14 days. Osteomyelitis requires 6 weeks or more of antibiotic administration and may also require local bone debridement and a myocutaneous flap to cover an associated pressure sore.

Musculoskeletal Issues

The musculoskeletal effects of SCI are many and include HO, muscle contracture, spinal deformity, osteoporosis predisposing to fractures, and tendon or muscle rupture related to use of upper limbs for mobility.

Heterotopic Ossification. HO, also known as myositis ossificans and paraosteoarthropathy, is abnormal bone formation in the soft tissue around large joints below the level of the cord injury, most commonly the hips.[135] The reported incidence is 16% to 53% after SCI.[136,137] The cellular basis for HO is unknown, but presumably it represents a metaplastic transformation of mesenchymal or endothelial cells to osteoblasts. The onset of HO is usually 1 to 4 months following SCI, but it may develop late after surgery or local trauma. Its presence alone is often not significant; it becomes significant in 10% to 20% of the SCI population if it compromises joint range-of-motion, thus impairing function and predisposing to pressure sores; slows venous return leading to increased peripheral edema and DVT; or leads to local peripheral nerve damage.[103,138] Heterotopic ossification is reportedly more common in males, younger patients (< 30 years old), more complete injuries, and those with spasticity.[139,140]

Symptoms are swelling, warmth, redness, and fever. Without this inflammatory phase, a noted reduction in joint motion may be the first warning. Early confirmation is made in the presence of high level serum alkaline phosphatase and with a positive technetium 99 bone scan. Ultimately, a radiograph will reveal fluffy densities in the soft tissues around the joint (Figure 5-13). Cellulitis, hematoma, tumor, and DVT should be excluded.

Initial treatment, which ideally begins early when the ossification process is most amenable to reversal, usually involves maintenance of joint range-of-motion and oral etidronate disodium. Indomethacin prevents HO formation after total hip replacement and may be useful after SCI as well,

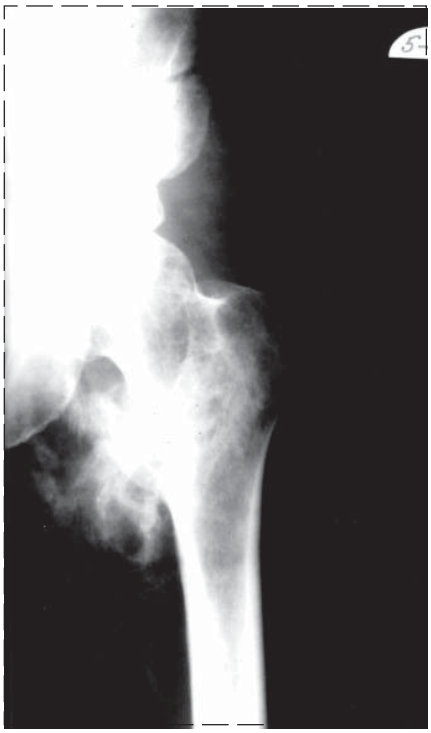


Fig. 5-13. Heterotopic ossification. Fluffy, amorphous radiograph densities around the hip indicate heterotopic ossification.

although this needs to be evaluated. Coumadin may also reduce the incidence of HO.[141]

For established HO, forceful manipulation or surgical resection can be considered once a bone scan confirms the HO is mature. These procedures often reinitiate the HO formation.[119,135] Associated local radiation therapy and continued treatment with disodium etidronate or indomethacin may minimize this recurrence.[137]

Myotendinous Contracture, Joint Capsule Tightness. In addition to HO, myotendinous contracture and joint capsule tightness can limit joint motion. Finger flexion deformities compromise skin care; ankle plantar flexion contractures compromise transfers, standing, and walking; and hip flexion contractures can prevent prone lying. Hip and knee extension contractures interfere with wheelchair sitting. Even when mild, hip and knee flexion contractures increase the energy cost of standing and walking, and contractures of finger flexors commonly impair grasp and pinch. Prevention requires daily joint range-of-motion exercises, provided by a caregiver if the patient is unable to do them without help. Early contracture or capsule tightness may be reversed with ultrasound diathermy and static stretch; ultrasound must be used cautiously in those with impaired or absent sensation. Established severe contracture or capsule tightness requires surgical release.

Osteoporosis, Fractures. The paralysis of SCI results in loss of bone density, for which there is no known treatment. This osteoporosis predisposes to fracture, particularly of the femur (Figure 5-14). In non-weight-bearing limbs, such fractures are usually managed with soft, well-padded splints that can be removed twice daily to ensure intactness of the skin.

Spine Instability, Charcot's Spine, Spine Deformity. A late consequence of spinal fracture may be progressive spinal deformity, arising from unrecognized ligamentous injury or other spinal instability.[142] This instability and associated excessive spinal movements can result in severe degenerative changes of the vertebral body, which must be differentiated from a malignant or infectious process. These degenerative changes are referred to as neurogenic spinal arthropathy or Charcot's spine. A needle or open biopsy of the degenerated vertebral body may be required to rule out malignancy or osteomyelitis. This condition can lead to pain and a progressive gibbus of the thoracic spine with a pressure sore affecting overlying skin, which can in turn become infected and lead to vertebral osteomyelitis and psoas abscess. Progressive spinal deformity may also lead to ascending neurologic loss, particularly when it affects the cervical spine. Typically, surgical stabilization is required, although external bracing may be most appropriate if age or medical complications preclude surgery.

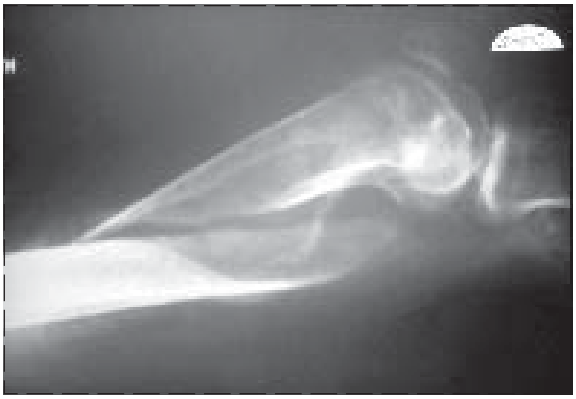


Fig. 5-14. Femur fracture in osteoporotic bone. This male with long-standing paraplegia flexed his trunk forward with his knees extended in long-sitting while tying his shoes; this resulted in a distal femur fracture. The osteoporosis that develops in long bones of paralyzed individuals due to the absence of normal compressive and tensile forces of weight bearing and muscle contraction increases the risk of fracture with minor falls or moderate stresses.

Weight-Bearing Shoulder. The assault to the musculoskeletal system is not limited to muscle, bone, and joint below the level of the injury, it also impacts the musculoskeletal components above the SCI level, which must compensate for the paralysis. Prolonged use of the upper extremities for weight-bearing, such as in push-up transfers, predisposes to degenerative changes in the shoulder.[83,143–145] Impingement syndrome, rotator cuff tears, and muscle rupture may all be evident.[146] Prevention and treatment involve controlling obesity, minimizing weight-bearing as in sliding board or hydraulic lift transfers, avoiding use of an overhead trapeze for transfers, substitution of forward or side-lean pressure releases for push-up pressure releases, nonsteroidal antiinflammatory medications, strengthening exercises, power wheelchairs, and surgery such as acromioplasty with rotator cuff tendon repair.[144]

Neurologic Issues

A variety of secondary neurologic complications can develop following an SCI, including spasticity, pain, posttraumatic syringomyelia, and peripheral nerve entrapment.

Spasticity and Other Hypertonus. Spasticity develops in those with upper motoneuron damage and spared reflex pathways; it is one type of hypertonus, an increased resistance to passive joint movement. Spasticity appears when segmental and descending inhibition is lost and new synaptic connections by the spared reflex pathways are acquired. Other forms of hypertonus associated with SCI include hyperactive cutaneomuscular reflexes, tonic stretch reflexes, segmental myoclonus, and dystonia.

Spasticity results from hyperactive phasic stretch reflexes mediated by 1A afferents from the muscle spindle. The 1A afferents respond to rapid stretch as in a tendon tap or rapid passive movement of a joint. True spasticity then is triggered by fast movements or muscle vibration, stimuli that elicit discharge from 1A afferents. Clinically, spasticity manifests as hyperactive tendon reflexes, clonus, velocity-dependent hypertonus, and extensor spasms. The latter are commonly elicited by the passive stretch of hip flexors, for example, as the patient moves from sitting to supine-lying position (Figure 5-15) or as the patient performs a push-up pressure release.[147] These extensor spasms often interfere during transfers, although some patients use them to aid standing.

Hyperactive cutaneomuscular reflexes are another form of hypertonus, which manifests as flexor spasms and a Babinski response. These are common indicators of ongoing noxious stimulation as with a chronic decubitus ulcer (Figure 5-16), or recurrent hip subluxation (Figure 5-17).[148] Such flexor spasms can interfere with prone or supine positioning in bed, with bed mobility, transfers, and walking; they can also interfere with sleep and contribute to decubiti.

Fig. 5-16. Flexor spasm associated with a trochanteric decubitus. This male with spastic paraparesis demonstrates hip and knee flexor spasms in association with a large, grade 4 trochanteric decubitus. The decubitus is a noxious stimulus that increases flexor tone, and in turn, the flexor hypertonus limits bed rest to left or right side lying, aggravating the trochanteric pressure sore; supine and prone lying are difficult to achieve with such severe hip and knee flexor spasms.

Less common than either of the above, but more disabling are hyperactive tonic stretch reflexes. These appear to result from length-dependent discharge of Group II stretch reflex afferents of the muscle spindle. They can be distinguished from

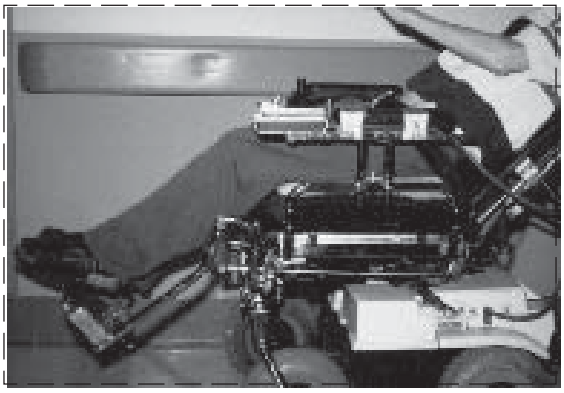


Fig. 5-15. Extensor spasms. These are a common manifestation of spasticity after SCI. This quadriplegic male demonstrates an extensor spasm, including hip and knee extension; hip adduction; ankle plantarflexion; abdominal muscle contraction; and elbow, wrist, and finger extension, elicited by the hip flexor stretch during rapid recline in his power wheelchair.

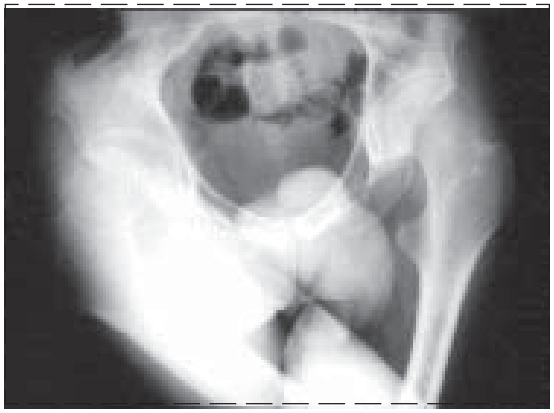


Fig. 5-17. Hip flexor/adductor spasms and hip subluxation. Severe hip flexor and adductor spasms in this paraparetic male have resulted in hip subluxation. In turn, the hip subluxation acts as a noxious stimulus, which further aggravates hip flexor and adductor spasms.

myotendinous contracture or joint capsule tightness by simultaneously recording electromyographic activity from the muscle during passive stretch or by performing an anesthetic nerve block.

Spasticity and other hypertonus require treatment depending on the degree to which they interfere with function, hinder the caregivers' tasks, or contribute to medical complications as with skin break down, falls, interrupted sleep, or pain.[149–151] Treatment benefits must always be weighed against the possible beneficial effects of spasticity; for example, positioning extremities or aiding standing transfers and walking (Figure 5-18).

Once spasticity is determined to be more detrimental than beneficial and any nociceptive sources such as UTI, renal stone, or fecal impaction have been ruled out, then various treatments may be undertaken. The least invasive is daily passive stretching of hypertonic muscles, which reduces tone and spasms for several hours and maintains joint range-of-motion. Often this is not enough and oral medications—baclofen, diazepam, dantrolene, or clonidine—must be tried alone or in combination. If these prove insufficient, as they often do with moderate or severe spasms, and the patient has some preserved voluntary movement, the physician should consider a reversible treatment such as intrathecal baclofen. Severe spasms can sometimes be suppressed only by irreversible treatments; among them are phenol motor-point or nerve blocks, tenotomy or tendon transfers, lumbar cord myelotomy, or complete or selective posterior rhizotomy.

Fig. 5-18. Functional extensor spasms. This male with T-4 paraparesis can elicit lower extremity extensor spasms by positioning his upper body behind the axis of rotation of his hips. This stretch of his hip flexors elicits and maintains extensor spasms, which he uses functionally for standing transfers. His only voluntary movement in his lower extremities is trace movement in his toes.

Pain. Severe, disabling pain is present in 5% to 49% of chronic SCI patients.[25,152] Surveys of SCI outpatients have revealed that chronic pain, rather than paralysis, often prevents employment. Commonly this pain originates centrally, within the spinal cord. This central pain presents with any combination of a burning, tingling, or lancinating quality that is unaffected by neck movements or posture and is thought to represent spontaneous discharge of neurons in the ascending pain pathways. Generally it is not present acutely, but develops weeks to months following SCI. The pain associated with incomplete cervical cord injury tends to be continuous and burning.

A patient with cauda equina injury experiences a series of stabbing pains that radiate down one or both lower limbs every few seconds to minutes; these shocklike pains may be continuously present or reappear intermittently. Another type of pain presents as a segmental hyperesthesia over one or two dermatomes at the level of injury. This hypersensitivity to light touch may only be a minor inconvenience and often does not require treatment. Occasionally it responds to desensitization or transcutaneous electrical nerve stimulation. Other treatments for these three types of central pain include nonnarcotic analgesics (acetaminophen, acetylsalicylic acid, ibuprofen); anticonvulsants (phenytoin, carbamazepine, clonazepam); and tricyclic antidepressants (amitriptyline, doxepin). Anticonvulsants may inhibit spontaneous discharge of central neurons that are mediating the pain sensations. Tricyclic antidepressants may reduce monoamine reuptake and thereby reduce pain transmission. Side effects of these medications can be significant. Carbamazepine has resulted in aplastic anemia, agranulocytosis, and thrombocytopenia. Other adverse effects of anticonvulsants include confusion, ataxia, nystagmus, and hepatic abnormalities. Tricyclic antidepressant side effects include sedation, urine retention, other anticholinergic effects, and OH.

Chronic narcotic use (eg, methadone, oxycodone, etc.) should be administered only in a

highly structured setting in which the patient's compliance and psychological status can be carefully monitored.[95] Nerve blocks, surgically sectioning the ascending pain pathways, and even cordotomy or cordectomy do not permanently relieve these types of pain.[153] Recently, epidural electrical stimulation and focal lesions of the dorsal root entry zone (DREZ) have been advocated.[154,155] The indications and long term success of these procedures is not known.

It is essential to distinguish these kinds of central dysesthetic SCI pains from pain arising due to mechanical causes such as persisting disk herniation impinging a root or the cord, an enlarging posttraumatic syrinx, or degenerative arthritis. These latter types of pain are generally aggravated by spinal movements or postural changes and thus may be clinically distinguishable from central pain. Treatment involves addressing the underlying cause of mechanical pain, if possible.

Posttraumatic Syringomyelia. Posttraumatic syringomyelia is an uncommon but clinically subtle and potentially severely disabling complication.[64,156] The prevalence of clinically significant posttraumatic syringomyelia has been estimated at 4.5% for quadriplegic subjects and 1.7% for paraplegics. Posttraumatic syringomyelia results from an enlarging fluid-filled cyst (syrinx) at the level of the cord injury, which leads to progressive spinal cord damage and is also referred to as ascending cystic myelopathy. One proposed pathophysiological explanation of posttraumatic syringomyelia is that the initial SCI leads to hemorrhagic cysts at the site of injury, which liquefy into fluid-filled cysts. The cysts enlarge with Valsalva's maneuver or repeated spine movements, which traction the cord and force CSF from the subarachnoid space by way of perivascular spaces (Virchow-Robin spaces) into the cysts. Such enlarging cysts may develop within months after an SCI but more typically develop over many years. They may extend rostrally or caudally from the cord injury and can extend the length of the spinal cord up to the brainstem.

The earliest symptoms and signs may be subtle (Figure 5-19). They include pain, often aggravated by postural change; altered spasticity; sweating; worsening OH; ascending loss of pain and temperature sensation; and ascending loss of segmental reflexes. Ascending weakness is a late manifestation. Its appearance often indicates that the condition is advanced and may not be reversible, although the pain may be remediated. Magnetic resonance imaging (MRI) is definitive for diagnosing posttraumatic syringomyelia and has revealed that 60% of all SCI patients have small cysts. Serial clinical examinations, repeat MRI, and serial electrodiagnostic studies (specifically, F-wave latencies and central motor conduction times by motor evoked potentials) are the available methods for determining if the cysts are progressive.[69,157]

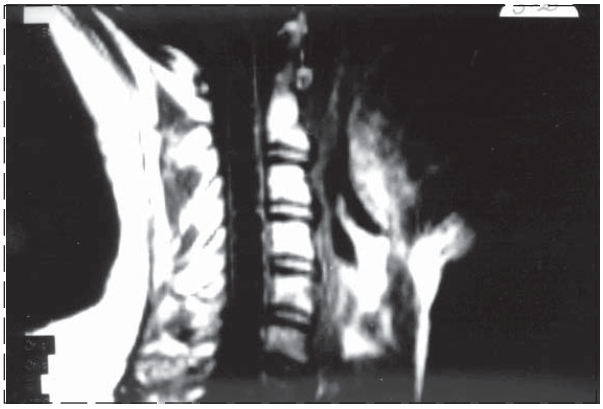


Fig. 5-19. Posttraumatic syringomyelia. This male with T-6 paraplegia at eight years postSCI noted loss of reflex bladder emptying and intermittent numbness in his fingers. Physical exam revealed pin prick sensory loss at T-4 on the left and at T-6 on the right and asymmetric tendon reflexes in the upper extremity. An MRI revealed a large syrinx extending rostrally and caudally from the level of the midthoracic cord injury; rostrally the syrinx extended to the foramen magnum. Despite this large syrinx, he denied any weakness or functional loss. This case illustrates the often subtle manifestations of posttraumatic syringomyelia as it initially presents.

Treatment may include minimizing Valsalva maneuvers and spine movements and surgical decompression with syringosubarachnoid, syringopleural, or syringoperitoneal shunts to remove the CSF and prevent its reaccumulation. For those with complete cord injury, cordectomy is an alternative.

Peripheral Nerve Entrapment. Peripheral nerve entrapment results from the high demands on the upper extremities of quadriplegics and paraplegics and is common after SCI.[158] Predisposing factors include (*a*) increased use of the upper extremities for transfers and wheelchair propulsion, resulting in an increased incidence of carpal tunnel syndrome; (*b*) absent pain sensation, particularly in posttraumatic syringomyelia, resulting in ulnar nerve entrapment; and (*c*) proximal compression of motor neurons and motor axons, resulting in double crush syndrome. Radial nerve entrapment, as in Saturday night palsy and thoracic outlet syndrome, also occurs with increased frequency. Preventive

measures include use of wheelchair gloves and avoidance of direct pressure to the ulnar groove. New sensory loss, weakness, or loss of function should prompt electrodiagnostic studies to rule out nerve entrapment. Wrist splints, elbow pads, and surgical decompression are common treatments.

Genitourinary Complications

Urosepsis and renal failure cause significant morbidity and mortality for those with SCI, although this has markedly improved since the 1940s. Standard care must provide annual urologic evaluations to eliminate conditions predisposing to urosepsis and renal failure. Understanding how the level of the cord lesion relates to bladder function is useful.[159,160] Upper motor neuron dysfunction is generally associated with spinal fractures at T-12 and above (ie, cervical and thoracic fractures). For patients with these conditions, voluntary control from the brain is disrupted, yet reflex activity recovers after spinal shock dissipates. Lower motoneuron dysfunction generally occurs when the bony injury is at L-1 and below. These individuals do not regain reflex contraction; the bladder remains flaccid and overfills. Mixed dysfunctions occur with partial injuries to the lumbar spine such as combined conus-cauda equina injuries, with combinations of upper and lower motor neuron damage. Whatever the type of dysfunction, the goals of treatment are to imitate as closely as possible normal lower urinary tract functions with low-pressure storage, continence with adequate emptying when voiding, infection control, and protection of the ureters and kidneys.[138,161]

SCI patients are at increased risk for renal compromise.[162] Acutely, rhabdomyolysis and shock can lead to acute renal failure. Sepsis can lead to ischemic renal injury and aminoglycoside antibiotics can cause toxic acute tubular necrosis. Radiocontrast dye for intravenous urography can cause acute renal insufficiency. Various antibiotics, including penicillin derivatives, methicillin, cephalosporins and sulfas can cause acute interstitial nephritis, while nonsteroidal antiinflammatory medications can impair renal hemodynamics and yield azotemia. Factors predisposing to chronic renal failure in SCI patients include high pressure voiding with reflux nephropathy, nephrolithiasis from urea-splitting bacteria and hypercalciuria, chronic pyelonephritis, and amyloidosis from chronic decubiti and osteomyelitis. Renal function should be regularly followed and impairment detected early and treated.

Urinary Retention, Incontinence. During the initial period of spinal shock, before reflex emptying develops, bladder drainage is managed with an indwelling Foley catheter until fluid intake can be regulated to less than 2 L per day. Patients are then managed with sterile intermittent catheterization (IC) every 4 to 6 hours to keep maximal bladder distension to less than 500 mL.

After several months, reflex detrusor contractions develop; this can be anticipated in those with preserved bulbocavernosus and anal reflexes. When spontaneous reflex bladder emptying develops, anticholinergics may be needed and IC may need to be continued; or in males, the frequency of IC is decreased, postvoid residuals monitored, and urinary drainage collected in a condom catheter and leg bag.[163] Recently, some[164] have advocated intentional bladder overdistension for women in conjunction with IC to avoid the need for anticholinergics. Postvoid residuals should be less than 100 mL to minimize concentrations of bacteria in the urine. The less common lower motoneuron bladder (where the S-2, S-3, S-4 reflex arc is interrupted) can be managed with IC and fluid restriction or condom catheter drainage and Valsalva's maneuver. High bladder pressures can be associated with Credé over the bladder and can lead to vesicoureteral reflux and hydronephrosis.[165]

Detrusor-Sphincter Dyssynergia, Vesicoureteral Reflux, Hydronephrosis. Spinal cord injury patients with upper motoneuron lesions rostral to and sparing the conus (S-2, -3, -4) usually develop reflex bladder emptying several months following SCI. Many of these patients will also develop detrusor-sphincter dyssynergia, in which the reflex detrusor contraction is opposed by a simultaneous external urethral sphincter contraction.[166–168] The result is high bladder pressures that lead to detrusor muscle hypertrophy and a further increase in bladder pressure. This increased pressure may, in turn, result in vesicoureteral reflux, hydroureter, hydronephrosis, and renal failure. This process is often clinically silent.[169] Urodynamics (ie, cystometrogram with simultaneous external urethral sphincter electromyography and fluoroscopy) is used to detect the presence of high pressure bladder emptying or vesicoureteral reflux. Yearly upper tract imaging (intravenous pyelogram, or renal ultrasound, or nucleide renal scan) and creatinine clearance should be obtained to further monitor renal status.

Treatment of high pressure voiding may include one of the following approaches: anticholinergic medication (oxybutynin, propantheline) and every-

6-hour catheterization, indwelling Foley catheter, or transurethral sphincterotomy.[170,171] Detrusor-sphincter dyssynergia is not the only cause of high pressure voiding. Outflow obstruction and increased bladder pressure can also be caused by internal sphincter (bladder neck) spasms, prostate enlargement, urethral stricture, or a too tightly applied condom catheter. Internal sphincter spasm may improve with α-adrenergic antagonist medication (phenoxybenzamine, prazosin) or bladder neck incision. A promising new treatment for external sphincter spasm or prostate enlargement is transurethral placement of a permanent stent, a small mesh coil that holds the urethra open.

Urinary Tract Infection

Bacteriuria ($> 10^5$ organisms per mL) is a nearly inevitable consequence of neurogenic bladder.[161,172] If asymptomatic, this bacteriuria is not treated. Symptoms of UTI prompting antibiotic treatment may include fever, shaking chills, leukocytosis, hematuria, pyuria (white blood cell count > 10 per high power field), and unexplained increase in spasticity. Preventing UTI may involve ensuring adequate bladder drainage with a low postvoid residual, perineal hygiene, cleaning drainage tube and bag, clean catheterization (washing hands and catheter with soap and water for those on IC), and eliminating urinary tract stones.[173] Those with recurrent UTI may benefit from bacterial suppression with methenamine mandelate or hippurate, trimethoprim-sulfamethoxazole, or nitrofurantoin, though resistant bacteriuria invariably develops.[161] High fevers and shaking chills suggest urosepsis, which requires broad antibiotic coverage (including pseudomonas coverage), placement of an indwelling catheter to assure bladder drainage, and a renal ultrasound to rule out upper tract obstruction. Failure of response to antibiotic within 48 to 72 hours or septic shock may prompt an abdominal CT scan to rule out perinephric abscess.

Bladder and Renal Calculi. Bladder stones may cause bladder spasms and are often removed by transurethral cystolithectomy. Renal stones are most commonly infectious struvite stones caused by vesicoureteral reflux and urea-splitting organisms. Another risk factor for stone formation is hypercalciuria in response to paralysis and urinary stasis. If stones are large enough to obstruct the ureter, then SCI patients must be monitored closely since upper tract obstruction may be relatively silent until urosepsis develops. Such upper tract stones may be removed by extracorporeal shock wave lithotripsy (ESWL) or percutaneous procedures.

Bladder Carcinoma. Spinal cord injury patients with long term indwelling catheters have an increased risk of bladder carcinoma.[174] One approach to early diagnosis and treatment of such bladder cancers is to perform yearly cystoscopy and biopsy of the bladder for those who have had indwelling catheters for more than 10 years.

Sexual Function and Fertility. Sexuality is the disclosure of maleness or femaleness in an individual. SCI may alter but never negates that expression.[175] The potential exists to make the expression richer and more thoughtful and ingenious. The recently injured may need to be reassured of this. In particular, young males may find their definition too narrowly focused on genitals. To broaden this, they need to be helped in making a compassionate and honest reevaluation of their physical selves and provided with, if they are willing to explore alternatives, detailed information about aids and options. They should also be helped to realize that in sexual as well as in other human relationships, qualities such as kindness, humor, and intelligence are powerful and even erotic. A thorny issue for many is their increased physical dependency and how in this altered situation they retain a sense of power, control, and responsibility to those around them.

If such patients are willing to explore the topic, this is some of the specific information they should have. They should know that sexual desires for both males and females are psychological and hormonal and not affected by nerve damage. In addition, although the level of sensation below the site of injury may be diminished or absent, males, particularly those with neurologic lesions above S-2, -3, -4 or incomplete lower lesions, are sometimes able to achieve and maintain erections (the overall rate is 50% for all categories of injury). External appliances, intracavernosal injection (prostaglandin E, papaverine), or an implanted penile prosthesis provide additional options.[176]

Although many men with SCI are able to achieve erections, most are unable to ejaculate. Vibration-induced ejaculation or electroejaculation can yield sperm for artificial insemination in some. Autonomic hyperreflexia is a small risk with these procedures in men with injuries at or above T-6.

Fertility in men diminishes to less than 5% with clinically complete injuries. Semen quality declines rapidly after SCI, possibly from recurrent UTIs and

increased temperature of the testicles. Nonetheless, artificial insemination with semen obtained by electroejaculation has resulted in some pregnancies and some healthy livebirths.[177]

For females, loss of sensation and lack of vaginal lubrication are the primary physical changes. Pleasurable sexual experience for both sexes may be realized through focusing on skin, lips, and other areas where sensation is intact. Use of a vibrator and lubricants can also be helpful. The fear of bladder incontinence is often a concern, but provision of towels, some humor and acceptance, bladder emptying by catheterization, and decreased fluid intake prior to intercourse should resolve this. Information about manual stimulation, oral–genital sex, positioning, and reducing or using spasticity can be discussed if the patient is willing to explore these topics.

Female fertility returns to preinjury level once the body has recovered from the initial trauma and menses return. Oral contraceptives and intrauterine devices carry extra risks for these patients, making foam and condoms better birth control choices. Undetected labor pains leading to an unsupervised birth, a prolonged labor because of compromised ability to push, and autonomic hyperreflexia in those with SCI above T-7 are additional risks of pregnancy and childbirth in this population.

Because of the complex issues and the extraordinary demands on the patient for reworking many aspects of his or her life after SCI, sexual and marital counseling are standard components of rehabilitation. Respect for individual values and sensitivity are essential in handling this emotionally loaded, highly personal topic.

Psychological Issues

Assessment and support are the two major psychological services in SCI. Assessment is an evaluation of the individual's potential to learn, to think, and to interact with his environment and other human beings. It includes a description of impairments that may be remediated and those that are unlikely to be altered. Some of the tools of assessment are standardized tests that measure psychosocial function and intelligence. Those most commonly used include the Minnesota Multiphasic Personality Inventory,[178] the Strong-Campbell Interest Inventory[178] for vocational interest testing, and intelligence tests like the Wechsler Adult Intelligence Scale[178] and the Wide Range Achievement Test.[178] Other tests can be used to assess neuropsychological functions in patients where head injury is a complicating factor, such as the Halstead-Reitan Neuropsychological Battery[178] or the Luria-Nebraska Neuropsychological Battery.[178] What should emerge from these assessments is a clear picture of the individual's potential for rehabilitation and appropriate guidelines for educational and vocational pursuits.

Support, the other major psychological service to SCI patients, should extend to both patients and family members. The support should be grounded in knowledge of the patient's history, disposition, values, limitations, and potential. SCI psychologists assist individuals in making new lives when part of the old has been shattered. Other members of the rehabilitation team also contribute to the patient's psychosocial adjustment to SCI, particularly the social worker, vocational counselor, and recreation therapist. Most important, no matter what the level or completeness of injury, is the patient's willingness to engage with life. In a population that is mostly male, young, and action oriented, this is a Herculean remaking that requires the patient to find necessary energy reserves to make a new contract with life that will include a radical shift in self-image and paradigm. What makes this possible is hope—hope for satisfaction in meaningful relationships and genuine contributions. Helping the patient to find this hope is the rehabilitation team's most crucial assignment. Quality of life gradually improves in most SCI patients so that by 4 years postinjury, it is 87% of normal on a visual analogue scale.[179] Quality of life is slightly worse for those with quadriplegia as compared with paraplegia and slightly worse for those with complete as compared to incomplete cord injuries.[180]

Denial or inability to accept the SCI is common and can interfere with rehabilitation, although expectations for recovery may be a source of hope and motivation. Patients are counseled to focus on current rehabilitation issues without destroying their hopes for recovery. Confronting patients on their denial can destroy the physician–patient relationship. Eventually, most patients come to the physician and ask for information on prognosis.

Associated Head Injury. A significant percentage of SCI patients incur closed head injury.[181–183] Resulting agitation, impulsiveness, impaired judgment, and impaired new learning may all impact rehabilitation.

Premorbid Personality. Premorbid factors affect rehabilitation outcomes. Risk-taking behavior, and

drug and alcohol abuse are associated with some cases of SCI and affect adjustment to disability after SCI. More preinjury education is associated with greater likelihood of employment after SCI; thus, among those with less than 12 years of education only 38% have worked at some time after SCI, compared to 93% of those with 16 or more years of education.[62] These premorbid factors should be considered during acute rehabilitation.

Reactive Depression and Suicide. A depressed mood is common following SCI. When depression is extreme or prolonged, it can interfere with rehabilitation and precipitate suicide. Suicide was the known cause of death in 6.3% of 9,135 SCI patients, 4.9 times higher than a matched general population.[184] Risk factors for suicide include chronic pain and alcohol or drug abuse.

Marital Adjustment and Discord. Divorce is common after SCI. Factors that may contribute to divorce are altered family roles, physical dependence on spouse as a caregiver with subsequent burnout, and impaired sexual function.

ORGANIZATION OF A SPINAL CORD INJURY UNIT

Acute trauma facilities, such as those in the U.S. military, do not usually have comprehensive SCI units, but rather, they have orthopedists, neurosurgeons, physiatrists, PTs and OTs, psychologists, social workers, and skilled nurses who can decompress the spinal cord, establish spine stability, prevent complications, and begin the initial process of rehabilitation. As soon as they are medically stable, patients are transferred to a specialized SCI rehabilitation facility; the U.S. military has a longstanding agreement with the U.S. Department of Veterans Affairs to transfer patients to one of the 23 VA SCI centers.

Physician support for a comprehensive SCI facility includes physiatrists, urologists, orthopedic surgeons, neurosurgeons, internists, and plastic surgeons. Other essential health professionals are rehabilitation nurses, PTs and OTs, psychologists, social workers, vocational counselors, orthotists, recreation therapists, respiratory therapists, and dietitians.

Specialized facilities for an SCI unit are uro-dynamics with videofluoroscopy, myelography with CT, MRI, a physical and occupational therapy gym, and a wheelchair accessible pool. All patient care space must be wheelchair and gurney accessible. An independent living apartment, where SCI patients can live on their own with spouse or attendant support, is particularly useful when the patient's ability to return to the community is uncertain.

A specialized SCI unit can anticipate and minimize complications and enhance functional outcomes compared to a medical, surgical, or general rehabilitation ward, which results in shorter hospitalizations and less economic cost. The features of an SCI unit that allow these gains are specialized knowledge, unique team relationships, and specialized equipment and facilities. Such specialized SCI centers allow clinical and applied basic research to further improve care for SCI individuals.

CONCLUSION

Spinal cord injury was fatal prior to World War II. Most SCI patients now survive, return home completely or partially independent, and live near-normal life spans with satisfactory quality of life. For most SCI patients, these favorable outcomes can be expected if the emergency, acute, and rehabilitation interventions during the first 6 months postinjury are appropriate. Spinal cord injury leads to multiorgan dysfunction. Strength, sensation, blood pressure control, bladder and bowel emptying, and sexual function are often impaired. Life threatening or disabling conditions affecting different organ systems must be prevented or diagnosed and treated early, such as pressure sores, deep venous thrombosis with pulmonary embolism, autonomic hyperreflexia, orthostatic hypotension, heterotopic ossification, posttraumatic syringomyelia, depression, and suicide. In addition, SCI patients must resume function to the degree possible, given the level and completeness of SCI. Training and adaptive equipment is required for independent mobility and independent self-care. Guidance and instruction is required for psychological, social, financial, vocational, and avocational adjustment to the disability.

An SCI rehabilitation unit includes an interdisciplinary clinical staff and specialized equipment and protocols to prevent and treat SCI complications and to train and instruct SCI patients functionally. These specialized facilities achieve greater functional outcome with fewer complications. U.S. military facilities refer spinal cord injured soldiers to the 23 SCI Services of the Department of Veterans Affairs for comprehensive rehabilitation.

REFERENCES

1. Stover SL, et al. *Spinal Cord Injury: The Facts and Figures.* Birmingham: University of Alabama; 1986.

2. Hardaway RM. Viet Nam wound analysis. *J Trauma.* 1978;18:635–643.

3. Ohry A, Rozin R. Acute spinal cord injuries in the Lebanon war, 1982. *Israel J Med Sci.* 1984;20:345–349.

4. Yashon, D. *Spinal Injury.* New York: Appleton-Century-Crofts; 1978.

5. Griffin JW, Tooms RE, et al. Efficacy of high voltage pulsed current for healing of pressure ulcers in patients with spinal cord injury. *Phys Ther.* 1991;71:433–444.

6. Whiteneck GG, Charlifue SW, Frankel HL, et al. Mortality, morbidity, and psychosocial outcomes of persons spinal cord injured more than 20 years ago. *Paraplegia.* 1992;30:617–630.

7. DeVivo MJ, Kartus PL, Stover SL, Rutt RD, Fine PR. Seven-year survival following spinal cord injury. *Arch Neurol.* 1987;44:872–875.

8. Davne SH. Emergency care of acute spinal cord injury. *J Am Para Soc.* 1983;6:42–46.

9. Tator CH, Duncan EG, Edmonds VE, Lapezak LI, Andrews DF. Comparison of surgical and conservative management in 208 patients with acute spinal cord injury. *Can J Neurol Sci.* 1987;14:60–69.

10. Longo WE, Ballantyne GH, Modlin IM. Colorectal disease in spinal cord patients: An occult diagnosis. *Dis Colon Rectum.* 1990;33:131–134.

11. Rhee KJ, Green W, Holcroft JW, Mangili JA. Oral intubation in the multiple injured patient: The risk of exacerbating spinal cord damage. *Ann Emerg Med.* 1990;19:511–514.

12. Bracken MB, Shepard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal cord injury. *N Engl J Med.* 1990;322:1405–1411.

13. Bracken MB, Shepard MJI, Collins WFI, et al. Methylprednisolone or naloxone treatment after acute spinal cord injury: 1-year follow-up data. *J Neurosurg.* 1992;76:23–31.

14. Anderson PA, Bohlman HH. Anterior decompression and arthrodesis of the cervical spine: Long-term motor improvement: Improvement in complete traumatic quadriplegia. *J Bone Joint Surg Am.* 1992;74 (pt.2):683–691.

15. Wilmot CB, Hall KM. Evaluation of the acute management of tetraplegia: Conservative versus surgical treatment. *Paraplegia.* 1986;24:148–153.

16. Roffi RP, Waters RL, Adkins RH. Gunshot wounds to the spine associated with a perforated viscus. *Spine.* 1989;14:808–811.

17. Romanick PC, Smith TK, Kopanky DR, Oldfield D. Infection about the spine associated with low velocity missile injury to the abdomen. *J Bone Joint Surg Am.* 1985;67:1195.

18. Schaefer SD, Bucholz RW, Jones RE, Carder HM. The management of transpharyngeal gunshot wounds to the cervical spine. *Surg Gynecol Obstet.* 1981;152:27–39.

19. Waters RL, Adkins RH. The effects of removal of bullet fragments retained in the spinal canal: A collaborative study by the National Spinal Cord Injury Model Systems. *Spine.* 1991;16:934–939.

20. Waters RL, Adkins RH, Yakura J, Sie I. Profiles of spinal cord injury and recovery after gunshot injury. *Clin Orthop.* 1991;267:14–21.

21. Cybulski GR, Stone JL, Kant R. Outcome of laminectomy for civilian gunshot injuries of the terminal spinal cord and cauda equina: Review of 88 cases. *Neurosurgery.* 1989;24:392–397.

22. Clark RA Jr. Analysis of wounds involving the lumbosacral canal in the Korean War. In: Mierowsky A, ed. *Neurological Surgery of Trauma.* Washington DC: US Army, Office of The Surgeon General; 1965:337–344.

23. Pool JL. Gunshot wounds of the spine: Observations from an evacuation hospital. *Surg Gynecol Obstet.* 1945;81:617–622.

24. Wannaker GT. Spinal cord injuries: A review of the early treatment in 300 consecutive cases during the Korean conflict. *J Neurosurg.* 1954;11:517–524.

25. Richards JS, Stover SL, Jaworski T. Effect of bullet removal on subsequent pain in persons with spinal cord injury secondary to gunshot wounds. *J Neurosurg.* 1990;73:401–404.

26. Albin MS. Acute spinal cord trauma. In: Shoemaker WC, Thompson WL, Holbrook PR, eds. *Textbook of Critical Care.* Philadelphia: WB Saunders; 1984:928–936.

27. Jellinger K. Neuropathology of spinal cord injuries. *Handbook Clin Neurol.* 1976;25:43–121.

28. Kakulas BA. The clinical neuropathology of spinal cord injury: A guide to the future. *Paraplegia.* 1987;25:212–216.

29. Schneider H. Acute and chronic pathomorphological reactions to cord injury. In: Schramm J, Jones S, eds. *Spinal Cord Monitoring.* Berlin: Springer-Verlag; 1985:103–120.

30. Eltorai IM, Juler G. Ischemic myelopathy. *Angiology*. 1979;30:81–94.

31. Foo D, Rossier AB. Anterior spinal artery syndrome and its natural history. *Paraplegia.* 1983;21:1–10.

32. Sliwa JA, Maclean IC. Ischemic myelopathy: A review of spinal vasculature and related clinical syndromes. *Arch Phys Med Rehabil.* 1992;73:365–372.

33. Francis TJR, Pearson RR, Robertson AG, Hodgson M, Dutka AJ, Flynn ET. Central nervous system decompression sickness: Latency of 1,070 human cases. *Undersea Biomed Res.* 1989;15:403–417.

34. Melamed Y, Shupak A, Bitterman H. Medical problems associated with underwater diving. *N Engl J Med.* 1992;326:30–35.

35. Braughler JM, Hall JD. Central nervous system trauma and stroke. *Free Radic Biol Med.* 1989;6:289–301.

36. Papadopoulos SM. Spinal cord injury. *Curr Opin Neurol Neurosurg.* 1992;5:554–557.

37. Waxman SG. Demyelination in spinal cord injury and multiple sclerosis: What can we do to enhance functional recovery. *J Neurotrauma.* 1991;9:S105–117.

38. Young W. Pharmacologic therapy of acute spinal cord injury. In: Errico TJ, Bauer RD, Waugh T, eds. *Spinal Trauma.* Philadelphia: JB Lippincott; 1991:415–433.

39. Bohlman HH, Anderson PA. Anterior decompression and arthrodesis of the cervical spine: Long-term motor improvement. *J Bone Joint Surg Am.* 1992;74:671–682.

40. Bracken MB. Treatment of acute spinal cord injury with methylprednisolone: Results of a multicenter, randomized clinical trial. *J Neurotrauma.* 1991;8:S47–52.

41. Garland DE, Orwin J. Resection of heterotopic ossification in patients with spinal cord injuries. *Clin Orthop Rel Res.* 1989;242:169–176.

42. Little JW, Harris RM, Lerner SJ. Immobilization impairs recovery after spinal cord injury. *Arch Phys Med Rehabil.* 1991;72:408–412.

43. Mange KC, Marino RJ, Gregory PC, Herbison GJ, Ditunno JF. Course of motor recovery in the zone of partial preservation in spinal cord injury. *Arch Phys Med Rehabil.* 1992;73:437–441.

44. Wu L, Marino RJ, Herbison GJ, Ditunno JF. Recovery of zero-grade muscles in the zone of partial preservation in motor complete quadriplegia. *Arch Phys Med Rehabil.* 1992;73:40–43.

45. Little JW, Powers RK, Micklesen P, Moore D. Electrodiagnosis of upper extremity weakness in acute quadriplegia. 1992.

46. American Spinal Injury Association. *Standards for Neurological and Functional Classification of Spinal Cord Injury.* Atlanta, Ga: ASIA; 1992.

47. Frankel HL, Hancock DO, Hyslop G, et al. The value of postural reduction in the initial management of closed injuries of the spine with paraplegia and tetraplegia. *Paraplegia.* 1969;7:179–192.

48. Harris P, Karmi MZ, McClemont E, Matlhoko D, Paul KS. The prognosis of patients sustaining severe cervical spine injury. *Paraplegia.* 1980;18:324–330.

49. Maynard FM, Reynolds GC, Fountain S, Wilmot C, Hamilton R. Neurological prognosis after traumatic quadriplegia. *J Neurosurg.* 1979;50:611–616.

50. Young JS, Dexter WR. Neurological recovery distal to the zone of injury in 172 cases of closed, traumatic spinal cord injury. *Paraplegia.* 1979;16:39–49.

51. Bosch A, Stauffer ES, Nickel VL. Incomplete traumatic quadriplegia. *JAMA.* 1971;216:473–478.

52. Little JW, Halar EM. Temporal course of motor recovery after Brown-Sequard spinal cord injuries. *Paraplegia.* 1985;23:39–46.

53. Schneider RC, Crosby EC, Harris-Russo R, Gosch HH. Traumatic spinal cord syndromes and their management. *Clin Neurosurg.* 1973;20:424.

54. Marino RJ, Crozier KS. Neurologic examination and functional assessment after spinal cord injury. *Phys Med Rehabil Clin N Am.* 1992;3:829–852.

55. Holdsworth F. Fractures, dislocations and fracture-dislocations of the spine. *J Bone Joint Surg Am.* 1970;52: 1534–1545.

56. Foo D, Subrahmanyan TS, Rossier AB. Post-traumatic acute anterior spinal cord syndrome. *Paraplegia.* 1981;19:201–205.

57. Roth EJ, Park T, Pang T, Yarkony GM, Lee MY. Traumatic cervical Brown-Sequard and Brown-Sequard-plus syndromes: The spectrum of presentations and outcomes. *Paraplegia.* 1991;29:582–589.

58. Quencer RM, Bunge RP, Egnor M, Green BA, Puckett W, Naidich TP, Post MJD, Norenberg M. Acute traumatic central cord syndrome: MRI-pathological correlations. *Neuroradiology.* 1992;34:85–94.

59. Little JW, DeLisa JA. Cauda equina injury: Late motor recovery. *Arch Phys Med Rehabil.* 1986;67:45–47.

60. Barnes CD, Joynt RJ, Schottelius BA. Motorneuron resting potentials in spinal shock. *Am J Physiol.* 1961;203: 1113–1116.

61. Burke D. Spasticity as an adaptation to pyramidal tract injury. In: Waxman SG, ed. *Advances in Neurology: Functional Recovery in Neurological Disease.* New York: Raven Press; 1988;47.

62. Krause JS. Employment after spinal cord injury. *Arch Phys Med Rehabil.* 1992;3:163–169.

63. Ashworth B. Preliminary trial of carisoprodol in multiple sclerosis. *Practitioner.* 1964;192:540–542.

64. Williams B. Post-traumatic syringomyelia: An update. *Paraplegia.* 1990;28:296–313.

65. Mange KC, Ditunno JF, Herbison GJ, Jaweed MM. Recovery of strength at the zone of injury in motor complete and incomplete cervical spinal cord injured patients. *Arch Phys Med Rehabil.* 1990;71:562–565.

66. DeLisa JA, Little JW. Electrodiagnosis and recovery of function. *Am J Phys Med Rehabil.* 1988;67:44–49.

67. Donovan WH, Bedbrook GM. Sensory and motor activity in the posterior primary rami following complete spinal cord injury. *Arch Phys Med Rehabil.* 1980;61:133–138.

68. Katz RT, Toleikis RJ, Knuth AE. Somatosensory-evoked and dermatomal-evoked potentials are not clinically useful in the prognostication of acute spinal cord injury. *Spine.* 1991;16:730–735.

69. Little JW, Robinson LR. AAEM case report #24: Electrodiagnosis in post-traumatic syringomyelia. *Muscle Nerve.* 1991;15:755–760.

70. Alvarez SE, Peterson M, Lunsford BR. Respiratory treatment of the adult patient with spinal cord injury. *Phys Ther.* 1981;61:1737–1745.

71. Callahan RA, Schlicke LH. A rational approach to burst fractures of the atlas. *Clin Orthop.* 1981;154:18–21.

72. Clark CR, White AA. Fractures of the dens: A multicenter study. *J Bone Joint Surg Am.* 1985;67:1340–1348.

73. Greenberg MS. *Handbook of Neurosurgery.* 2nd ed. Lakeland: Greenberg Graphics; 1991.

74. Lipson SJ. Fractures of the atlas associated with fracture of the odontoid and transverse ligament ruptures. *J Bone Joint Surg Am*. 1977;59:940–942.

75. Levine AM, Edwards CC. The management of traumatic spondylolisthesis of the axis. *J Bone Joint Surg Am.* 1985;67:217–226.

76. Murphy KP, Opitz JL, Cabanela ME, Ebersold MJ. Cervical fractures and spinal cord injury: Outcome of surgical and nonsurgical management. *Mayo Clin Proc.* 1990;65:949–953.

77. Derrickson J, Ciesla N, Simpson N, Imle PC. A comparison of two breathing exercise programs for patients with quadriplegia. *Phys Ther.* 1992;72:763–769.

78. Denis F. The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. *Spine.* 1983;8:817–831.

79. Hanley EN, Simpkins A, Phillips ED. Fractures of the thoracic, thoracolumbar, and lumbar spine: Classification, basis for treatment, and timing of surgery. *Semin Spine Surg.* 1990;2:2–7.

80. Denis F, Davis S, Comfort T. Sacral fractures: An important problem: Retrospective analysis of 236 cases. *Clin Ortho Rel Res.* 1988;227:67–81.

81. Whitesides TE. Traumatic kyphosis of the thoracolumbar spine. *Clin Orthop Rel Res.* 1977;128:78–92.

82. Staas W, et al. Spinal Cord Injury Rehabilitation. In: DeLisa JA, et al, eds. *Principles and Practice of Rehabilitation Medicine.* 1st ed. Philadelphia: JB Lippincott; 1988.

83. Waring WP, Maynard FM. Shoulder pain in acute traumatic quadriplegia. *Paraplegia.* 1991;29:37–42.

84. Johnstone BR, Jordan CJ, Buntine JA. A review of surgical rehabilitation of the upper limb in quadriplegia. *Paraplegia.* 1988;26:317–339.

85. Peckham PH, Keith MW, Freehafer AA. Restoration of functional control by electrical stimulation in the upper extremity of the quadriplegic patient. *J Bone Joint Surg Am.* 1988;70:144–148.

86. De Troyer A, Estenne M, Heilporn A. Mechanism of active expiration in tetraplegic subjects. *N Engl J Med.* 1986;314:740–744.

87. Carter RE. Respiratory aspects of spinal cord injury management. *Paraplegia.* 1987;25:262–266.

88. Schmitt J, Midha M, McKenzie N. Medical complications of spinal cord disease. *Neurol Clin.* 1991;9:779–795.

89. Maeda CJ, Baydur A, Waters RL, Adkins RH. The effect of the halovest and body position on pulmonary function in quadriplegia. *J Spinal Disord.* 1990;3:47–51.

90. Hoit JD, Banzett RB, Brown R, et al. Speech breathing in individuals with cervical spinal cord injury. *J Speech Hear Res.* 1990;33:798–807.

91. Bonekat HW, Andersen G, Squires J. Obstructive disordered breathing during sleep in patients with spinal cord injury. *Paraplegia.* 1990;28:392–398.

92. Short DJ, Stradling JR, Williams SJ. Prevalence of sleep apnea in patients over 40 years of age with spinal cord lesions. *Paraplegia.* 1992;55:1032–1036.

93. Arnold PM, McVey PP, Farrell WJ, Deurloo TM, Grasso AR. Functional electrical stimulation: Its efficacy and safety in improving pulmonary function and musculoskeletal fitness. *Arch Phys Med Rehabil.* 1992;3:665–668.

94. Walker J, Cooney M, Norton S. Improved pulmonary function in chronic quadriplegics after pulmonary therapy and arm ergometry. *Paraplegia.* 1989;27:278–283.

95. Farkash A, Portenoy RK. The pharmacological management of chronic pain in the paraplegic patient. *J Am Para Soc.* 1986;9:41–50.

96. Reines HD, Harris RC. Pulmonary complications of acute spinal cord injuries. *Neurosurgery.* 1987;21:193–196.

97. Clough P. Glossopharyngeal breathing: Its application with a traumatic quadriplegic patient. *Arch Phys Med Rehabil.* 1983;64:384–385.

98. Lehmann KG, Lane JG, Piepmeier JM, Batsford WP. Cardiovascular abnormalities accompanying acute spinal cord injury in humans: Incidence, time course and severity. *J Am Coll Cardiol.* 1987;10:46–52.

99. Waring WP, Karunas RS. Acute spinal cord injuries and the incidence of clinically occurring thromboembolic disease. *Paraplegia.* 1991;29:8–16.

100. Green D, Lee MY, Lim AC, et al. Prevention of thromboembolism after spinal cord injury using low molecular weight heparin. *Ann Intern Med.* 1990;113:571–574.

101. Kulkarni JR, Burt AA, Tromans AT, et al. Prophylactic low dose heparin anticoagulant therapy in patients with spinal cord injuries: Retrospective study. *Paraplegia.* 1992;30:169–172.

102. Weingarden SI, Weingarden DS, Belen J. Fever and Thromboembolic Disease in Acute SCI. *Paraplegia.* 1988;26:35–42.

103. Terpie AGG. Thrombosis prevention and treatment in spinal cord injured patients. In: Bloch RF, Basbaum M, eds. *Management of Spinal Cord Injuries.* Baltimore: Williams & Wilkins; 1986:212–220.

104. Colachis SC. Autonomic hyperreflexia with spinal cord injury. *J Am Para Soc.* 1992;15:171–186.

105. Braddom RL, Rocco JF. Autonomic dysreflexia: A survey of current treatment. *Am J Phys Med.* 1991;70:234–241.

106. Eltorai I, Kim R, Vulpe M, et al. Fatal cerebral hemorrhage due to autonomic dysreflexia in a tetraplegic patient: Case report and review. *Paraplegia.* 1992;30:355–360.

107. Kursh ED, Freehafer A, Persky L. Complications of autonomic dysreflexia. *J Urol.* 1977;118:70–72.

108. Bauman WA, Spungen AM, Raza M. Coronary artery disease: Metabolic risk factors and latent disease in individuals with paraplegia. *Mt Sinai J Med.* 1992;59:163–168.

109. Yekutiel M, Brooks ME, Ohry A, et al. The prevalence of hypertension, ischemic heart disease, and diabetes in traumatic spinal cord injured patients and amputees. *Paraplegia.* 1989;27:58–62.

110. Cosman BC, Stone JM, Perkash I. Gastrointestinal complications of chronic spinal cord injury. *J Am Para Soc.* 1991;14:175–180.

111. Bond W. Acute abdomen in spinal cord injured patients. In: Lee BY, Ostrander LE, Cochran GVB, Shaw WW, eds. *The Spinal Cord Injured Patient: Comprehensive Management.* Philadelphia: WB Saunders; 1991:18–23.

112. Juler GL, Eltorai IM. The acute abdomen in spinal cord injury patients. *Paraplegia.* 1985;23:118–123.

113. Berlly MH, Wilmot CB. Acute abdominal emergencies during the first four weeks after spinal cord injury. *Arch Phys Med Rehabil.* 1984;65:687–690.

114. Miller F, Fenzi TC. Prolonged ileus with acute spinal cord injury responding to metaclopramide. *Paraplegia.* 1981;19:43–45.

115. Albert TJ, Levine MJ, Balderston RA, Cotler JM. Gastrointestinal complications in spinal cord injury. *Spine.* 1991;16(10S):522–525.

116. Kewalramani LS. Neurogenic gastroduodenal ulceration and bleeding associated with spinal cord injuries. *J Trauma.* 1979;19:259–265.

117. Glick ME, Meshkinpour H, Haldeman S, et al. Colonic dysfunction in patients with thoracic spinal cord injury. *Gastroenterology.* 1984;86:287–294.

118. Stickler DJ, Chawla JC. An appraisal of antibiotic policies for urinary tract infections in patients with spinal cord injuries undergoing long-term intermittent catheterization. *Paraplegia.* 1988;26:215–225.

119. Stone JM, Wolfe VA, Nino-Murcia M, Perkash I. Colostomy as treatment for complications of spinal cord injury. *Arch Phys Med Rehabil.* 1990;71:514–518.

120. Longe RL. Current concepts in clinical therapeutics: Pressure sores. *Clin Pharm.* 1986;5:669–681.

121. Carey DE, Raisz LG. Calcitonin therapy in prolonged immobilization hypercalcemia. *Arch Phys Med Rehabil.* 1985;66:640–644.

122. Maynard FM. Immobilization hypercalcemia following spinal cord injury. *Arch Phys Med Rehabil.* 1986;67:41–44.

123. Richards JS. Pressure ulcers in spinal cord injury: Psychosocial correlates. *SCI Digest.* 1981;3:11–18.

124. Edberg EL, Cerny K, Stauffer ES. Prevention and treatment of pressure sores. *Phys Ther.* 1973;53:246–252.

125. Knight AL. Medical management of pressure sores. *J Fam Pract.* 1988;27:95–100.

126. Lamid S, El-Ghatit AD. Smoking, spasticity and pressure sores in spinal cord injured patients. *Am J Phys Med.* 1983;62:300–306.

127. Thiyagarajan C, Silver JR. Aetiology of pressure sores in patients with spinal cord injury. *Brit Med J.* 1984;289:1487–1490.

128. Rodriguez GP, Claus-Walker J. Biomechanical changes in skin composition in spinal cord injury: A possible contribution to decubitus ulcers. *Paraplegia.* 1988;26:302–309.

129. Rodriguez GP, Claus-Walker J, Kent J, et al. Collagen metabolite excretion as a predictor of bone and skin-related complications of spinal cord injury. *Arch Phys Med Rehabil.* 1989;70:442–444.

130. Rodriguez GP, Claus-Walker J, Kent MC, et al. Adrenergic receptors in insensitive skin of spinal cord injured patients. *Arch Phys Med Rehabil.* 1986;67:177–180.

131. Woolsey RM. Rehabilitation outcome following spinal cord injury. *Arch Neurol.* 1985;42:116–121.

132. Garber SL. Wheelchair cushions for spinal cord injured individuals. *Am J Occup Ther.* 1985;39:722–725.

133. Shannon ML, Miller B. Evaluation of hydrocolloid dressings on healing of pressure ulcers in spinal cord injured patients. *Decubitus.* 1988;1:42–46.

134. Sugarman B. Osteomyelitis in spinal cord injured people. *J Am Para Soc.* 1984;7:73–75.

135. Garland DE. A clinical perspective on common forms of acquired heterotopic ossification. *Clin Orthop Rel Res.* 1991;263:13–29.

136. Rossier AB, Bussat P, Infante F, et al. Current facts on para-osteoarthropathy. *Paraplegia.* 1973;11:36–41.

137. Stover SL. Heterotopic ossification after spinal cord injury. In: Bloch RF, Basbaum M, eds. *Management of Spinal Cord Injuries.* Baltimore: Williams & Wilkins; 1986:284–302.

138. Gardner BP, Parsons KF, Machin DG, Galloway A, Krishnan KR. The urological management of spinal cord damaged patients: A clinical algorithm. *Paraplegia.* 1986;24:138–147.

139. Lal S, Hamilton BB, Heinemann A, et al. Risk factors for heterotopic ossification in spinal cord injury. *Arch Phys Med Rehabil.* 1989;70:387–390.

140. Wittenberg RH, Peschke U, Botel U. Heterotopic ossification after spinal cord injury: Epidemiology of risk factors. *J Bone Joint Surg Br.* 1992;74:215–218.

141. Buschbacher R, McKinley W, Buschbacher L, et al. Warfarin in the prevention of heterotopic ossification. *Am J Phys Med Rehabil.* 1992;71:86–91.

142. Glennon TP, Madewell JE, Donovan WH, Bontke CF, Spjut HJ. Neuropathic spinal arthropathy after spinal cord injury. *Spine.* 1992;17:964–971.

143. Pentland WE, Twomey LT. The weight-bearing upper extremity in women with long term paraplegia. *Paraplegia.* 1991;29:521–530.

144. Silfverskiold J, Waters RL. Shoulder pain and functional disability in spinal cord injury patients. *Clin Orthop.* 1991;272:141–145.

145. Wylie EJ, Chakera TMH. Degenerative joint abnormalities in patients with paraplegia of duration greater than 20 years. *Paraplegia.* 1988;26:101–106.

146. Bayley JC, Cochran TP, Sledge CB. The weight-bearing shoulder. The impingement syndrome in paraplegics. *J Bone Joint Surg Am.* 1987;69:676–678.

147. Kuhn R. Functional capacity of the isolated human spinal cord. *Brain.* 1950;73:1–51.

148. Davis R. Spasticity following spinal cord injury. *Clin Orthop.* 1975;112:66–75.

149. Little JW, Merritt JL. Spasticity and associated abnormalities of muscle tone. In: DeLisa JA, et al, eds. *Principles and Practice of Rehabilitation Medicine.* 1st ed. Philadelphia: JB Lippincott; 1988:430–447.

150. Little JW, Micklesen P, Umlauf R, Britell C. Lower extremity manifestations of spasticity in chronic spinal cord injury. *Am J Phys Med Rehabil.* 1989;68:32–36.

151. Whyte J, Robinson KM. Pharmacologic management. In: Glenn MB, Whyte J, eds. *The Practical Management of Spasticity in Children and Adults.* Philadelphia: Lea & Febiger; 1990.

152. Mariano AJ. Chronic pain and spinal cord injury. *Clin J Pain.* 1991;8:87–92.

153. Jefferson A. Cordectomy for intractable pain in paraplegia. In: Lipton S, Miles J, eds. *Persistent Pain: Modern Methods of Treatment.* London: Grune and Stratton; 1983;4:115–132.

154. Nashold BS, Vieira J, El-Naggar AO. Pain and spinal cysts in paraplegia: Treatment with drainage and DREZ operation. *Brit J Neurosurg.* 1990;4:327–336.

155. Young RF. Clinical experience with radiofrequency and laser DREZ lesions. *J Neurosurg.* 1990;72:715–720.

156. Rossier AB, Foo D, Shillito J, Dyro FM. Posttraumatic cervical syringomyelia: Incidence, clinical presentation, electrophysiological studies, syrinx protein and results of conservative and operative treatment. *Brain.* 1985;108:439–461.

157. Little JW, Robinson LR, Goldstein B, Stewart D, Micklesen P. Electrophysiologic findings in post-traumatic syringomyelia: Implications for clinical management. *J Am Para Soc.* 1992;15:44–52.

158. Gellman H, Sie I, Waters RL. Late complications of the weight-bearing upper extremity in the paraplegic patient. *Clin Orthop Rel Res.* 1988;233:132–135.

159. Fam B, Yalla SV. Vesicourethral dysfunction in spinal cord injury and its management. *Semin Neurol.* 1988;8:150–155.

160. Kaplan SA, Chancellor MB, Blaivas JG. Bladder and sphincter behavior in patients with spinal cord lesions. *J Urol.* 1991;146:113–117.

161. Lloyd LK. New trends in urologic management of spinal cord injured patients. *Cent Nerv Syst Trauma.* 1986;3:3–12.

162. Vaziri ND. Renal insufficiency in patients with spinal cord injury. In: Lee BY, Ostrander LE, Cochran GVB, Shaw WW, eds. *The Spinal Cord Injured Patient: Comprehensive Management.* 1st ed. Philadelphia: WB Saunders; 1991:134–157.

163. Cardenas DD, Kelly E, Krieger JN, et al. Residual urine volumes in patients with spinal cord injury: Measurement with a portable ultrasound instrument. *Arch Phys Med Rehabil.* 1988;69:514–516.

164. Yanagita T, Iwatsubo E, Haraoka M, Osada Y. *Japanese J Urol.* 1993;84:1954–1960.

165. Tempkin A, Sullivan G, Paldi J, Perkash I. Radioisotope renography in spinal cord injury. *J Urol.* 1985;133:228–230.

166. Perkash I. Pressure response during cystomanometry in spinal cord injury patients complicated with detrusor-sphincter dyssynergia. *J Urol.* 1979;121:778–782.

167. Wyndaele JJ. Urethral sphincter dyssynergia in spinal cord injury patients. *Paraplegia.* 1987;25:10–15.

168. Yalla SV, Blunt KJ, Fam BA, Constantinople NL, Gittes RF. Detrusor-urethral sphincter dyssynergia. *J Urol.* 1977;118:1026–1029.

169. Lamid S. Long-term follow-up of spinal cord injury patients with vesico-ureteral reflux. *Paraplegia.* 1988;26:27–34.

170. Madersbacher H. The various types of neurogenic bladder dysfunction: An update of current therapeutic concepts. *Paraplegia.* 1990;28:217–229.

171. Wyndaele JJ. Pharmacotherapy for urinary bladder dysfunction in spinal cord injury patients. *Paraplegia.* 1990;28:146–150.

172. Stover SL, Lloyd LK, Waites KB, Jackson AB. Neurogenic urinary tract infection. *Neurol Clin.* 1991;9:741–755.

173. Sanderson PJ, Rawal P. Contamination of the environment of spinal cord injured patients by organisms causing urinary tract infection. *J Hosp Infect.* 1987;10:173–178.

174. Bickel A, Culkin DJ, Wheeler JS. Bladder cancer in spinal cord injury patients. *J Urol.* 1991;146:1240–1242.

175. White MJ, Rintala DH, Hart KA, Young ME, Fuhrer MJ. Sexual activities, concerns and interests of men with spinal cord injury. *Am J Phys Med Rehabil.* 1992;71:225–231.

176. Lloyd LK, Richards JS. Intracavernous pharmacotherapy for management of erectile dysfunction in spinal cord injury. *Paraplegia.* 1989;27:457–464.

177. Rawicki H, Lording DW. Assisted fertility in complete paraplegia: Case report. *Paraplegia.* 1988;26:401–404.

178. Trieschmann RB. *Spinal Cord Injuries: Psycholgical, Social, and Vocational Rehabilitation.* 2nd ed. New York: Demos; 1988.

179. Luce JM. Medical management of spinal cord injury. *Crit Care Med.* 1985;13:126–131.

180. Lundqvist C, Siosteen A, Blomstrand C, Lind B, Sullivan M. Spinal cord injuries: Clinical, functional and emotional status. *Spine.* 1991;16:78–83.

181. Davidoff GN, Roth EJ, Richards JS. Cognitive deficits in spinal cord injury: Epidemiology and outcome. *Arch Phys Med Rehabil.* 1992;73:275–284.

182. Richards JS, Brown L, Hagglund K, et al. Spinal cord injury and concomitant traumatic brain injury. *Am J Phys Med Rehabil.* 1988;67:211–216.

183. Roth E, Davidoff G, Thomas P, et al. A controlled study of neuropsychological deficits in acute spinal cord injury patients. *Paraplegia.* 1989;27:480–489.

184. DeVivo MJ, Black KJ, Richards JS. Suicide following spinal cord injury. *Paraplegia.* 1991;29:620–627.

Chapter 6

TRAUMATIC BRAIN INJURY

NATHAN D. ZASLER, M.D., F.A.A.P.M.&R., F.A.A.D.E.P., C.I.M.E.* AND CATHERINE DEVANY SERIO, PH.D†

*Medical Director, Concussion Care Centre of Virginia, Ltd.; Chief Executive Officer and Medical Director, Tree of Life, L.L.C., 10120 West Broad Street, Suite G, Glen Allen, VA 23060

†Cinical Neuropsychologist, Private Practice, 1161 West River Street, Suite 390, Boise, ID 83702

INTRODUCTION

The area of traumatic brain injury (TBI) rehabilitation is fast becoming one of the most highly technical, medically complex areas of physiatry. Physiatrists are physicians specializing in Physical Medicine and Rehabilitation (PMR). Given the broad scope and complexity of the field of brain injury rehabilitation, this chapter will provide only an overview of clinically germane topics in TBI rehabilitation rather than a review of all topics pertinent to military personnel following TBI. Important philosophical, clinical, and programmatic aspects have been emphasized regarding the clinical care of this special patient population.

Brain injury rehabilitation, unlike any other subspecialty area in PMR, has undergone a rapid maturation over the last 10 to 15 years. Additionally, the neuromedical multidisciplinary expertise required to care for this special patient population is not matched by any other area of rehabilitation. with which these authors are familiar. Much of the literature pertinent to the clinical care of individuals with TBI is scattered among various medical disciplines, including neurosurgery, physiatry, general surgery, neurology, psychiatry, ophthalmology, otolaryngology, orthopedic surgery, and gastroenterology. There is, however, a growing literature base in areas outside of medicine that is pertinent to rehabilitative care of military personnel with TBI. This literature encompasses areas such as neuropsychology, neurophysiology, neuroanatomy, physical therapy, occupational therapy, speech language pathology, therapeutic recreation, rehabilitation nursing, and rehabilitation social work.

Rehabilitationists have the opportunity to not only support ongoing neurological recovery, but to also affect facilitation of this recovery and allow for better functional adaptation to residual neurologic impairments, as well as physical disabilities. This chapter will consolidate important information regarding rehabilitative care for military personnel with TBI; care which will maximize not only the neurologic and functional outcomes, but also the rehabilitation cost efficiency from "coma to community."

NOMENCLATURE

Although there is general consensus regarding the importance of a common nomenclature within this field of brain injury rehabilitation,[1] one limiting factor has been the lack of consistency in neuromedical and "technical" rehabilitative terminology. Without a standardized nomenclature, clinicians cannot know if they are comparing "apples to apples" or "apples to oranges." One of the most frequently misused or misunderstood areas of nomenclature is that pertaining to low level neurologic patients following brain injury. Terminology such as coma, vegetative state, persistent vegetative state, akinetic mutism, and locked-in syndrome are frequently used interchangeably when, in fact, the neurobehavioral status is variable in each of these diagnoses, which affects the care as well as prognosis.[2] Another area of frequent miscommunication deals with the term "head injury" as opposed to "brain injury." Although cranial trauma (head injury), can be seen concomitantly with TBI, often the two diagnoses are not synonymous. Many of the sequelae that result from presumptive TBI may be due to other diagnostic entities such as cranial trauma, cranial adnexal trauma, or cervical injury secondary to hyperextension-hyperflexion injury ("whiplash"). Labeling an individual as having a head injury when he actually has a traumatic brain injury not only exacerbates patient denial regarding the diagnosis, but is pathophysiologically and diagnostically inaccurate.[3]

Another area of nomenclature misuse is description of muscle tone. Many times clinicians incorrectly interchange the terms "spasticity" and "hypertonicity." Spasticity is an increase in tonic stretch reflexes that are velocity dependent and associated with increased tendon reflexes; this is one component of upper motor neuron syndrome. Hypertonicity, on the other hand, describes an increase in tone of a muscle during movement when the subject attempts to relax.[4] Both of these terms should be differentiated from rigidity, which is not associated with hyperreflexia, has no clonus or clasp-knife response, and is not velocity dependent.

Clinicians should also accurately define brain injury etiology; specifically, open or penetrating TBI should be differentiated from closed or nonpenetrating TBI. Unlike civilian populations, military personnel may incur much higher percentages of penetrating injuries. Most occur as a result of missile wounds such as bullets or assaults with blunt objects. An open injury always occurs when the dura, a membrane immediately covering the brain,

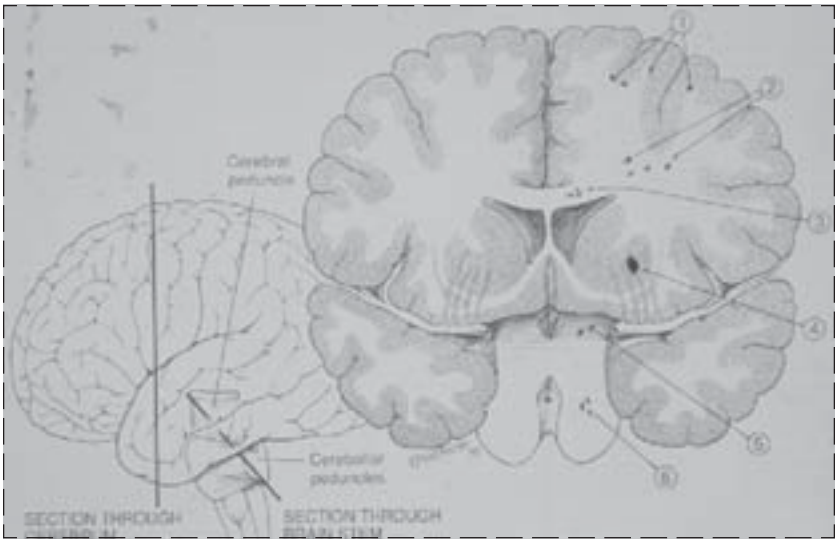


Fig. 6-1. Diagrammatic representation of neuropathologic changes associated with diffuse axonal shear injury relative to areas of higher predilection for this type of insult. (1) axonal shear at gray/white matter junction; (2) axonal shear injury in corona radiata; (3) axonal shear injury in corpus callosum; (4) axonal shear injury of lenticul-striate vessels leading to basal ganglia infarction/hemorrhage; (5) axonal shear injury of cerebral peduncles; (6) axonal shear injury of cerebellar peduncles. Reprinted with permission. Copyright Robert Shepherd. Shepherd Visual Communications: Richmond, VA;1993.

is penetrated. Nonpenetrating (closed) TBI more commonly results from acceleration-deceleration type injuries and falls. Confusion continues regarding descriptive terms that delineate the neuropathology of TBI. Often, punctate hemorrhages are documented as focal contusions even though the two are not similar in appearance or pathoanatomical origin. Classically, punctate hemorrhages are a result of diffuse axonal shear injury, which may occur, to varying degrees, in all brain injuries and affects not only axonal integrity, but also neurovascular structures (Figure 6-1). Contusions, on the other hand, occur as the result of direct impact to the involved area, resulting in either bland (nonhemorrhagic) or hemorrhagic lesions (Figure 6-2). Diffuse axonal injury and focal cortical contusion, comprise the "primary" etiologies of brain injury. Proper use of such neuroanatomic and neuropathologic nomenclature has significant implications for diagnosis, treatment, and prognosis.[5,6]

Although the above examples comprise only a fraction of the commonly misunderstood or misused nomenclature, they represent a global clinical issue that must be addressed through appropriate education and professional demands for nomenclature consistency.

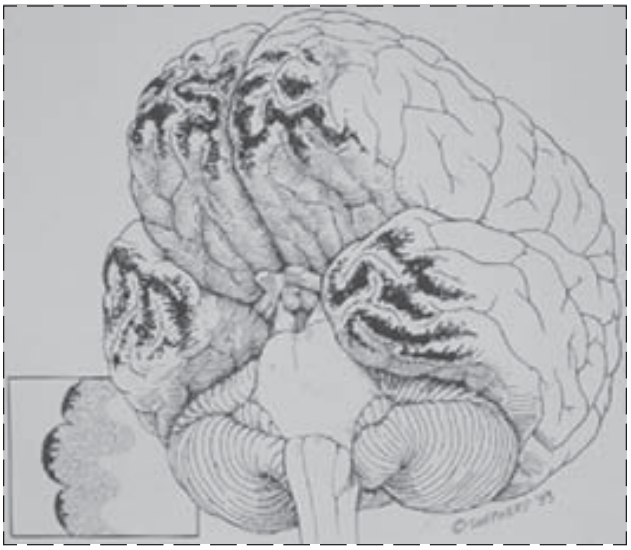


Fig. 6-2. Diagrammatic representation of neuropathologic changes and areas of predilection for focal cortical contusion involving the medial frontal lobes and anterolateral temporal lobes. Reprinted with permission. Copyright Robert Shepherd. Shepherd Visual Communications: Richmond, VA;1993.

EPIDEMIOLOGY

Discussion of TBI epidemiology, either civilian or military, is often complicated by the lack of consistency regarding (*a*) how brain injury is defined (nonpentrating vs penetrating brain injury), (*b*) the differences in screening methodologies for TBI (TBI plus multitrauma vs TBI alone), and (*c*) the differences in populations studied (inclusion or exclusion of subpopulations, such as patients with a prior history of substance abuse or TBI, and so forth). It might seem surprising that during the last decade, more deaths have resulted from nonwartime TBI than all the U.S. wars fought since the founding of the Republic. In the civilian population, accidental death is the fourth most frequent cause of death in

the United States, exceeded only by coronary artery disease, cancer, and cerebral vascular disease. The primary cause of death to both sexes ages 15 to 24 years is accidental injury.[7] Conservative civilian statistics suggest that more than two million individuals per year incur traumatic brain injury and approximately one quarter of these require hospitalization.[8] Most deaths associated with TBI occur at the time of injury or within the first several hours thereafter. Of those individuals who survive, 50,000 to 70,000 will be left with permanent neurologic impairments that will prevent their return to a preinjury lifestyle.

In civilian populations, motor vehicle accidents account for about 50% of all TBIs. Falls are the second most common cause of injury at approximately a 20% incidence; violence, including gunshot wounds and assaults, account for 12%; followed by recreational injuries at about 10%.[9] In civilian populations, research demonstrates that males typically are injured two to three times more frequently than females. Males between the ages of 15 to 24 years have the highest rate of injury. The frequency distribution of TBI relative to age is trimodal, with the young adult group being of highest frequency. The two other peaks occur during infancy and in the geriatric population.

Some studies[10] have attempted to examine racial correlates of TBI in civilian populations and have found that suburban whites seem to have about a 50% lower incidence of TBI than suburban and inner city blacks. Most TBI in inner city blacks apparently correlates with assault, whereas the single most common cause in both suburban blacks and whites is automobile accidents.[10] Unfortunately, child abuse contributes to over 50% of infant TBI. Falls account for most brain injuries in the geriatric population.

Military studies examining incidence and type of TBI include the extensive data collected and published by the Vietnam Head Injury Study group. Interested readers can find a discussion of this detailed follow-up study at the end of this chapter, in a section entitled The Vietnam Head Injury Study: Overview of Results to Date. The data suggest that penetrating TBI accounts for approximately 50% of all military-related injuries.[11] Penetrating injuries, high velocity bullets, and fragments from explosive devices seem to predominate as the mechanisms of injury.[12] During nonwartime, U.S. Army epidemiological studies have shown a fairly equal incidence of concussion across sexes; although, in general, males predominated with regard to intracranial injury and TBI.[13] McCarroll and Gunderson[14] published data from a 5-year survey that indicated that skull fractures and intracranial diagnoses were found almost exclusively in males; whites had a higher incidence of injury than blacks, and only 10% of the injuries were alcohol related (97% of which occurred in males).

PROGNOSTICATION ISSUES

Rehabilitation physicians are often asked to make prognostic statements about patients who have suffered TBI. The main factors that can aid the postinjury prognostication of this patient population include preinjury, injury, and postinjury parameters. A significant amount of literature has been published dealing with acute injury and the prognostic implications of clinical and laboratory parameters.[15] Few sound data are available that do justice to preinjury and postinjury factors relative to their role in prognosis of either neurologic or functional outcome.

Preinjury Parameters

The preinjury parameters that must be considered when analyzing a patient's prognosis include (*a*) psychosocial background; (*b*) history of learning disability; (*c*) prior psychological and psychiatric problems, or both; (*d*) prior history of substance abuse; (*e*) prior brain injury (regardless of etiology); and (*f*) developmental history.[16,17] Available data support the conclusion that people with higher preinjury levels of function have a better functional outcome after TBI than individuals with suboptimal preinjury psychosocial or intellectual status, or both. Although it is poorly quantified, factors such as the amount of neural reserve, play at least some part in determining how well an individual will recover from TBI. Individuals with any significant prior injury may find an otherwise trivial neurologic insult much more devastating than if they not had such a history. Research literature also supports the general contention that younger patients typically have a better outcome than older patients, from both neurological and functional standpoints regardless of injury severity, although this relationship of age does not appear to be linear.[18]

Injury Parameters

In the acute care setting, a significant number of clinical parameters can be analyzed separately or

together to assess both neurologic and functional outcome prognosis. Probably the best known of these is the Glasgow Coma Scale (GCS)[19] score. The scale provides clinicians with a brief, standardized neurologic assessment tool for use in the acute care setting. The GCS score has been shown to be highly correlated with acute morbidity and mortality, as well as, although not as strongly as, long term functional outcome. The three clinical parameters that make up the total GCS score (which ranges from 3 to 15) are (1) best motor response, (2)verbal response, and (3) visual response. The scores are differentially weighted with eye opening ranges from 1 to 4, best verbal response from 1 to 5, and best motor response from 1 to 6. GCS scores between 3 and 8 define severe neurologic insult; scores between 9 and 12, moderate; and scores between 13 and 15, mild. Although each of the three variables on the GCS holds prognostic utility in and of itself, the motor score has proved to be the most sensitive relative to long-term outcome. The predictive validity, specificity, and sensitivity increase significantly when all three parameters are conjointly assessed.

Variations of the GCS scores such as the Glasgow-Liege Scale allow for more sensitive prediction of outcome by taking into consideration clinical evidence of brain stem dysfunction.[20] Clinical findings that are useful in early prognostication include oculocephalic and oculovestibular reflex abnormalities, which may indicate significant brain stem damage. Longer durations of coma and posttraumatic amnesia have also been associated with poorer neurologic and functional outcomes. Advances in neuroimaging have enabled correlations to be made between early static imaging, as well as functional imaging and outcome. Electrophysiologic assessment by means of multimodal evoked potentials (including visual evoked responses, auditory brain stem responses, and somatosensory evoked responses) and a variety of electroencephalographic (EEG) modalities, such as compressed spectral analysis and quantitative EEG, have also been correlated either singularly or in multifactorial analysis of outcome prognosis. A variety of cerebrospinal fluid markers, including creatine kinase and lactate dehydrogenase, have also been studied relative to their correlation with the severity of central neurologic damage. Cerebrospinal fluid neurotransmitter and neurotransmitter metabolite levels have also been studied relative to their relation with acute neurologic morbidity and mortality; levels tend to have a direct correlation with a higher degree of neurologic morbidity. Acute medical variables that have been associated with a worse outcome include mass lesions on imaging, protracted elevated intracranial pressure beyond 40 mm Hg, and cardiopulmonary complications.[21]

Concurrent hypoxic ischemic injury, whether internal or external, must also be considered as a comorbidity that is associated with poorer short- and long-term neurological and functional prognosis. Hypoxic ischemic insult may be focal or diffuse. The focal variety is generally the result of infarction of a vascular territory quite commonly in the distribution of the posterior cerebral artery as a result of transtentorial temporal lobe herniation. Diffuse hypoxic insult, similar to diffuse axonal injury as a result of trauma, has a predilection for damaging certain areas more than others, including the medial temporal lobe structures (hippocampi), basal ganglia, and purkinje fibers of the cerebellum. When severe, hypoxic insult may result in diffuse cortical neuronal loss. Ischemic insult may also result in so-called "watershed" infarctions in parenchymal areas between major arterial vascular territories.[22]

Postinjury Parameters

Numerous studies have been conducted in an attempt to provide clinicians with information to aid them in prognosticating outcome and morbidity in the postinjury phase. Typically, once a patient has reached rehabilitation, many clinicians are not as attuned as they should be to the prognosticatory significance of specific clinical findings or functional impediments, or both. The longer the duration of the vegetative state, particularly when over 3 months, the greater the likelihood that the individual will remain vegetative, all other things being equal. In minimally responsive patients, some researchers have correlated the presence of communicating hydrocephalus and central dysautonomia with poorer neurologic and functional outcomes.[23] Agitation during the recovery phase has been theorized to correlate with better than worse functional outcomes in comparison to withdrawn, psychomotorically retarded behavior. Anosognosia, or denial of deficit, can be a troublesome neurobehavioral sequela of TBI due to safety implications and the inability to appreciate the need for further rehabilitative interventions. Lower extremity flexion synergy patterns are typically considered to be a poor prognostic sign for functional ambulation. Significant behavioral problems tend to indicate a poorer prognosis for successful independent community reentry. Further research is obviously necessary to clarify which specific impairments are poor prognostic indicators for specific functional goals and abilities.

MECHANISMS OF NEURAL RECOVERY

Multiple mechanisms have been proposed in an attempt to explain recovery of function following central nervous system (CNS) insult after TBI. However, there is little empirical support for a causal link between the theorized phenomena and the resultant functional recovery. Resolution of transient neurophysiologic phenomena, including elevated intracranial pressure, edema, and hypoxia, may be responsible for some of the reversible impairments noted in the earlier stages postinjury. Modification of synaptic function has been suggested as a possible explanation for the phenomenon of diaschisis (reversible depression of parenchymal function associated with focal insult to adjacent areas of brain tissue). Alterations in neural connections through axonal regeneration (not necessarily functional) and collateral sprouting have also been suggested as neural mechanisms mediating recovery of function.

Other theories posited include functional substitution, vicarious functioning, and redundancy. Functional substitution entails the overt or covert use of alternative strategies to achieve the desired functional outcome. In other words, following CNS insult, the organism learns compensatory strategies to cope with its functional disabilities. Vicarious functioning implies that neural structures alter their function in some indeterminate manner to allow for subserving the direction of new functional tasks. Redundancy, on the other hand, implies that following neural insult, there are "dormant" neural circuits that have the capability of directing particular functions, but only do so "when called upon."[24]

The concepts of neuronal sparing and neuronal reorganization broadly define the two major putative neural mechanisms involved in enhancing the potential for functional reorganization and recovery of function following brain injury. Multiple experimental treatment regimens for inhibiting or blocking the "neurotoxic cascade" following TBI are presently being researched and are expected to have clinical applicability in the near future. Research utilizing agents that interfere with cholinergic and glutaminergic tertiary nerve cell death, and that impede calcium induced cellular damage and vasoconstriction, remains furtive. Free radical research continues at a very active pace. Experimental studies[25] have demonstrated that oxygen free radicals may be important mediators of brain injury and brain edema. Researchers have found a multitude of sources for oxygen radicals after neuronal injury, including xanthine oxidase, peroxidases, catecholamines, and amine oxidases. Agents being examined to impede oxygen-free-radical damage include superoxide dismutase, catalase, vitamin E, and dimethyl sulfoxide on lipid peroxidation inhibitors such as the 21-aminosteroids ("Lazaroids" being one example of this class of drugs).[25]

The release of excessive excitatory amino acids, including glutamate and aspartate, were initially hypothesized by Olney[26] in 1969 to be associated with neuronal death due either to acute osmotic lysis or delayed excess intracellular free calcium. Current thought among bench neural science researchers is that excitotoxic phenomena may render neurons dysfunctional without necessarily killing them. It is well known that TBI results in the widespread depolarization and nonspecific release of a multitude of neurotransmitters and neuromodulators, both excitatory (glutatmate, aspartate, and acetylcholine) and inhibitory (γ-amino butyric acid [GABA] and opioids). The resultant "sublethal" toxicity is theorized to be mediated by elevations in intracellular calcium levels.[25] The glutatmate receptor has three subtypes, named according to their selective agonists: (1) *N*-methyl D-aspartate (NMDA),(2) quisqualate, and (3) kainate. Laboratory studies[27] have demonstrated that NMDA receptor antagonists may protect against brain injury secondary to cerebral ischemia and trauma. Issues of dosing and toxicity need further investigation prior to undertaking human trials. Recent work[28] suggests a role for anticholinergic and GABAergic agents in suppressing some of the adverse side effects from this class of presumptive neuroprotective agents.

A variety of other neurotransmitter systems are presently being studied relative to their contribution to acute brain injury. Cholinergic systems seem to play variable roles in mediation of brain injury and neural recovery relative to the time postinjury. Evidence[29] suggests that acute anticholinergic drug administration after TBI tends to decrease the period of unconsciousness either through decreasing the extent of active inhibition of systems responsible for regulation of consciousness, or lessening the extent of neural injury. Researchers[25] have hypothesized that early anticholinergic therapy benefits on long term motor deficits may be more related to its effect at blocking release of excitotoxins.

Catecholamines, particularly norepinephrines, are actively being investigated relative to their role in recovery from TBI related behavioral deficits.

Early work by Hovda and Feeney[30] demonstrated that α-noradrenergic agonists and perhaps dopaminergic agents actually accelerated motor recovery following sensorimotor cortex injury; whereas their antagonists retarded recovery. The exact location at which noradrenergic fibers emanating from the locus ceruleus need to be "stimulated" to mediate accelerated motor recovery is a much debated area of current research. Boyeson[25] believes that the critical area is not related to diaschisis-like effects in the sensorimotor cortex itself, but rather to alterations in noradrenergic function in the cerebellum contralateral to the site of sensorimotor cortex injury. The present evidence[31] is strong for an acute role for noradrenergic treatment if given at appropriate times and under specific conditions. Conversely, in certain circumstances, noradrenergic antagonists may actually be detrimental relative to their potential to reinstate motor behavior deficits following acute brain injury.

Stimulation of GABAergic systems have been associated with ischemic events following both experimental and clinical brain injury. Studies have demonstrated that early administration of GABAergic agents (such as diazepam) may be deleterious relative to slowing of neural recovery and potential reinstitution of neural deficits. Evidence also suggests that increased central GABA levels may enhance glutamate neurotoxicity by mechanisms not yet clearly defined. Such evidence would suggest a more conservative approach to the use of GABAergic agonists, including benzodiazepines and valproic acid, in the very acute setting after TBI.[25]

One area of active clinical research that may turn out to be the "magic bullet" is that of hypothermia. Researchers are presently investigating the role of acute hypothermia in animal and human subjects to determine the effects on a variety of neurochemical cascades, as well as the morbidity and mortality associated with acute brain injury. Clifton and associates[32] have evaluated the effects of moderate hypothermia (30°C to 36°C) on mortality after experimental fluid percussion TBI in a rat model and found significant protection from hypothermia at the lower range of this temperature scale. Theoretically, hypothermia, among other explanatory mechanisms, may actually slow down the neurotoxicity associated with the multitude of events occurring immediately following TBI.

No neurotransmitter system acts in a totally isolated manner. Therefore, treatment with a specific neurotransmitter agonist or antagonist may directly or indirectly modify functioning in other neurotransmitter systems within the CNS. Clinically, "therapeutic cocktails" may be necessary, either with or without hypothermia treatment. The focus of these polypharmaceutical "potions" will become better as our understanding of neurotransmitter system interrelationships improves. Once the science of acute neurochemical alterations following TBI is better understood, the potential exists for acute treatment in the field by emergency medical technician personnel or in hospital emergency rooms with specific drug or hypothermia protocols, or both. As we "go where no man has gone before," the hope for more successful treatments for acute brain injury grows brighter by the day.[25,27,32]

Denervation supersensitivity and reactive synaptogenesis (also called axon collateral sprouting) have also been theorized[33] to play a potential role in neural reorganization; however, it remains unclear whether such reorganization is always adaptive as opposed to maladaptive.

Some clinicians[24] believe that functional recovery is a consequence of an inherent, albeit poorly defined and understood, ability on the part of the CNS to adapt to injury. It is critical to understand the realities of age-related, genetically driven, central processes that drive functional recovery. Specifically, neuronal sparing mechanisms in early development are distinct in comparison with those processes occurring in more mature organisms. In the real world, most of the evolutionary consequences of brain injury for nonhumans are functionally and physiologically maladaptive because they result in the organism being more prone to predation in the wild. Parallels can be drawn to what happens to humans after severe brain injury. In the best of all worlds, based on sound scientific rationale, rehabilitationists can intervene in the recovery process to beneficially (or even negatively) affect rehabilitation outcome.

Research[34] suggests that the rate of neurologic recovery is more amenable to interventional manipulation than the ultimate level of neurologic recovery. It should also be realized that faster is not necessarily better. There are inherent risks associated with rushing recovery. Specifically, maladaptive pathoanatomic mechanisms and functional behaviors may be triggered or reinforced, or both. The development of a more comprehensive understanding at a basic-science level of what really happens as patients recover following brain injury is critical if rehabilitationists are to intercede optimally into this complex process and maximize neurologic and functional recovery.[33]

ADVANCES IN CLINICAL TREATMENT: POSTACUTE PHARMACOTHERAPY

The basic tenant of positively affecting neurological outcome and functional status after brain injury through the use of pharmacologic agents is by no means new.[35,36] Nonetheless, most rehabilitation professionals have relied almost exclusively on nonpharmacologic modalities to address sequelae following traumatic and nontraumatic brain injury. Physiatrists, as of late, have become more comfortable at managing both the pharmacologic and the more traditional nonpharmacologic rehabilitative aspects of care of individuals following brain injury.

Until recently, there was little if any evidence that medications could make a difference in either the rate or plateau of neurologic and functional recovery following brain injury. Now, good evidence indicates that many acute, subacute, and chronic neurologic and functional sequelae resulting from brain injury can be lessened and potentially abated through the thoughtful and appropriate use of pharmacologic agents.[37] Many pharmacologic agents may have potential utility in altering function following brain injury. Much of what is known about pharmacologic TBI rehabilitation is based on theories derived from work done at the basic-science level with animal models, or from individual clinical experience. The peer reviewed scientific literature, as it presently stands, does not provide much well controlled, methodologically sound, prospective research data regarding this topic. Nonetheless, clinicians should be aware of the major pharmocologic agents in each neurotransmitter class in order to better grasp how they *may* have an affect, positive or otherwise, on neurological recovery and functional capabilities following brain injury. In addition, rehabilitation professionals should be familiar with the major side effects of these drugs. Although drug interactions, precautions, and contraindications must also be considered, these topics are beyond the scope of this chapter. The reader is referred to other sources for this information, but should remember that these reference texts provide guidelines and information only on Food and Drug Administration (FDA) approved drug uses and dosage ranges. It should be noted that many of the medications prescribed for post-TBI sequelae are not FDA approved for the particular application in which they are being utilized. Physicians should be aware of potential medicolegal issues inherent in utilizing non-FDA approved medications, medications in applications that are not FDA approved, or medications in higher doses than FDA approved. The following paragraphs will outline the major drug groups presently in use by physiatrists versed at neuropsychopharmacology and TBI rehabilitation.

The major catecholaminergic drugs are levodopa (L-dopa), amantadine, bromocriptine, pergolide, lisuride, and some of the more classic stimulant drugs, such as dextroamphetamine, methylphenidate, and pemoline. The classic dopamine agonist has historically been L-dopa. A combination formulation of L-dopa and Carbidopa is also available. The use of the combination drug minimizes peripheral (non-CNS) side effects and increases the amount available for CNS incorporation. L-dopa has its action presynaptically and is agonistic at both the D1 and D2 receptor sites.[38] Side effects are numerous, but the more frequent ones include dyskinesias, various bradykinetic episodes (ie, "on–off" phenomena), psychiatric disturbances, gastrointestinal disturbances (nausea, vomiting, anorexia, and slowing of gastric motility), as well as orthostatic hypotension. Carbidopa-L-dopa is available in ratios of 1:10 (100 mg L-dopa to 10 mg carbidopa) and 1:4 (100 mg L-dopa to 25 mg carbidopa). Most patients with clear clinical evidence of dopaminergic deficiency will respond to a 1:10 ratio provided the daily dosage of carbidopa is 70 mg or more. When the 1:4 ratio is used, the usual starting dose is 1 tablet three times a day, increasing by one tablet every 2 days up to a maximum dosage of 6 tablets daily. If the 1:10 ratio is used, the usual starting dose is 1 tablet three to four times a day, increasing by one tablet every 2 days, to a maximum of 8 tablets daily.[39] In addition to carbidopa, the enzyme inhibitors benserazide and L-deprenyl (a monoamine oxidase Type B inhibitor) have been used in conjunction with L-dopa in an attempt to increase therapeutic efficacy.

Amantadine hydrochloride has been utilized clinically as an antiviral agent, as well as an anti-Parkinsonian agent. Its exact mechanism of action is still not fully understood; however, it has been theorized to have a presynaptic action, as well as a possible postsynaptic action.[40] Some researchers have speculated that amantadine may also increase central cholinergic and GABAergic activity.[41] Therapy can be initiated at between 50 to 100 mg/d and increased to a maximum of 400 mg daily. Since the drug is not metabolized and is excreted unchanged in the urine, dosage adjustments must be made when there is concurrent decreased renal func-

tion, such as in the elderly or in patients with renal disease. Peripheral side effects include, but are not limited to, peripheral edema, lightheadedness, orthostatic hypotension, hot and dry skin, rash, and livedo reticularis. Livedo reticularis is a discoloration of the skin that occurs in a reddish-blue to purple blotchy pattern. The reaction tends to occur after at least 1 month of treatment and it may occur more commonly at higher doses. Livedo reticularis is totally benign and the medication does not need to be discontinued unless the cosmetic aspects outweigh the therapeutic benefits.[39] Central side effects, which are more commonly seen in the geriatric population, include confusion and hallucinations.

Due to the L-dopa "indirect" mechanism of action, researchers have pursued and developed several direct dopamine-receptor stimulating agents, all of which fall in the ergotalkaloid class. These direct agents include bromocriptine, lisuride, and pergo-lide. Both bromocriptine and lisuride are antagonistic at the D1 receptor and agonistic at the D2 receptor. Pergolide, on the other hand, is agonistic at both the D1 and D2 receptor sites. Bromocriptine mesylate tends to produce fewer problems with dyskinesias, but more problems with mental side effects, orthostasis, and nausea than L-dopa.[38] Clinical results have demonstrated a triphasic response to bromocriptine with dopamine agonism, occurring only in the midrange doses. Dosing should start with a test dose of 1.25 mg and, if tolerated, the patient can then receive 2.5 mg daily, increasing fairly quickly to a three to four times a day dose. Once at 10 mg/d, the dose can be increased every 4 days by 2.5 mg. Typically, clinical experience has dictated that doses higher than 60 mg/d are unnecessary in patients with acquired brain injury. The manufacturer has not established safety limits for dosages greater than 100 mg daily.

Pergolide and lisuride are relatively new agents in this country and there is little if any literature on their utility in the pharmacologic rehabilitation of individuals with brain injury. It should be noted that pergolide is an extremely potent dopamine agonist and only very small doses are required. In this author's limited experience with pergolide, most patients with brain injury are unable to tolerate the drug secondary to sedation. Lisuride is also extremely potent and therapeutic effects are typically seen with daily doses ranging from 4 to 10 mg daily.[38] Most of the ergot alkaloids also have concomitant central serotonergic receptor agonism, which might explain the high incidence of changes in mental status with this class of dopamine agonists.

The classic "psychostimulant" drugs include dextroamphetamine, methylphenidate, pemoline, and to a lesser extent, activating tricyclic antidepressants. These agents have typically been theorized to have mixed dopaminergic and noradrenergic agonist activity. Dextroamphetamine has been theorized to produce noradrenergic agonism by blocking the reuptake mechanism for norepinephrine. In higher doses, it is also dopaminergic by a similar mechanism of dopamine reuptake blockade.[42] Dosing of dextroamphetamine should be initiated at 5 mg once to twice daily. The maximum recommended dose of dextroamphetamine is 60 mg/d; however, there are little if any data addressing dosing limits in individuals following brain injury. To avoid problems with insomnia, the last dose of medication should be given at least 6 hours before the patient's bedtime. There is evidence that "pulsed" dosing of noradrenergic agonists by standard formulations rather than extended release dosing may be preferential with regard to the resultant psychostimulant effects. Generally, adults are fairly sensitive to psychostimulant therapy, particularly after brain injury. Relative or absolute "toxicity" may be manifested by anxiety; dysphoria; increased irritability; cardiovascular symptoms; headache; palilalia (pathological use of words and phrases); stereotypical thoughts; cognitive impairment; hallucinations; insomnia; and motor disorders including dyskinesias, tics, and worsening of spasticity.[43]

Methylphenidate hydrochloride is a mixed dopaminergic, noradrenergic agonist whose pharmacologic action is similar to amphetamines. The main sites of action appear to be the cerebral cortex and subcortical structures such as the thalamus. Dosing typically should be initiated at 5 mg twice a day and titrated up to a maximum dose of 60 mg daily. An extended release formulation is also available. The adverse effects of this drug are analogous to those of dextroamphetamine.

Pemoline is an oxazolidinone derivative stimulant with pharmacologic actions qualitatively similar to dextroamphetamine and methylphenidate. Evidence suggests that pemoline may have its stimulatory effect through dopaminergic mechanisms. The drug is typically dosed initially at 37.5 mg daily as a morning dose with increases of 18.75 mg made weekly, as appropriate. The effective dose typically ranges from 56.25 to 75 mg daily. The most frequently encountered adverse effects include insomnia and anorexia, both being dose related.

The major serotonergic drugs are trazodone hydrochloride, fluoxetine, buspirone, sertraline, par-

oxetine, and L-tryptophan. Trazodone hydrochloride is a triazolopyridine derivative that selectively inhibits serotonin uptake. Initial dosing should begin at low doses (50–150 mg), typically at bedtime with food. The dose should be on the lower end of the dosing range in geriatric patients secondary to more common side effects such as sedation and orthostatic hypotension. The dose may be increased by 50 mg/d every 3 to 4 days to a maximum of 400 mg daily. If closely monitored, as in an inpatient setting, the maximum dose may be as high as 5 mg/kg daily. Fluoxetine is also a serotonin reuptake inhibitor, but it tends to be more activating than other serotonergic drugs like trazodone. Initial dosing should be 20 mg/d as a morning dose. Doses above 20 mg/d should be given on a twice a day schedule with a maximum daily dose of no more than 80 mg. The major reported side effects include headache, nausea, nervousness, and insomnia.[44] Buspirone is a novel benzodiazepine anxiolytic which is theorized to work through its serotonergic agonist activity at the 5-HT1 receptor. It should also be noted that this medication is presynaptically antagonistic at the D2 dopaminergic receptor.[45] The medication should be initiated at a dose of 10 to 15 mg twice a day and increased over 4 to 6 weeks to a maximum of 60 mg/d, based on patient response and tolerance.[46] The main side effects with buspirone are dizziness, headache, nervousness, and lightheadedness.

Newer selective serotonin reuptake inhibitors (SSRIs), such as paroxetine and sertraline, also show promise in the treatment of affective disorders. Generally, paroxetine is dosed from 10 mg to 50 mg/d and may be taken as a single dose. The most common side effects are nausea, headache, dry mouth, and an altered sleep–wake cycle, as well as—albeit less commonly—ejaculatory dysfunction.[47] Sertraline, also an SSRI, is dosed from 50 mg to 200 mg daily. Commonly reported side effects are gastrointestinal (nausea and diarrhea), headache, tremor, dizziness, dry mouth, altered sleep–wake cycle, and male sexual dysfunction.[48] L-tryptophan is a serotonergic precursor that has recently received quite a bit of attention secondary to the incidence of eosinophilia-myalgia syndrome. This syndrome has been purportedly traced to a bad batch of this pharmacologic agent produced in Japan.[49] For now, its use remains barred by the FDA. Newer serotonergic uptake inhibiting drugs that are still under clinical investigation and may prove useful from a neuropharmacologic rehabilitative standpoint include citalopram and fluvoxamine.

The two most commonly used opioid antagonists are naloxone and naltrexone, the latter being preferred secondary to its oral route of administration and prolonged mode of action. Dosing typically starts low, with 12.5 mg to 25 mg daily with titration up to 150 mg/d with an average daily dose of 50 mg. Exact dosing schedules and upper limits for TBI have not been established. The major side effects relate to gastrointestinal complaints and hepatocellular injury.

Agents with GABAergic activity are commonly used in the general rehabilitation setting. It should be noted, however, that only a few of these can be recommended for use in a patient with concomitant brain injury, particularly in the early phase of neural recovery. Classic antispasticity agents such as valium and baclofen are GABAergic agents, GABA A and GABA B, respectively. Many of the presently available anticonvulsant agents are also GABAergic, specifically, valproate, barbiturates, and benzodiazepines. Other commonly utilized anti-convulsants, such as phenytoin and carbamazepine, are felt to mediate anticonvulsant effect through other neurochemical systems.[50] From a clinical standpoint, many GABAergic agents tend to be overly sedative with concomitant suppression of cognitive processes. The use of these agents in the subacute and chronic phases following brain injury should be examined carefully given their potential side effects.[37]

Valproic acid is typically dosed at 15 mg/kg/d. Dosages may be increased by 5 mg/kg/d to 10 mg/kg/d at weekly intervals until clinical efficacy is achieved or adverse side effects prevent further increases. Due to potential adverse gastrointestinal side effects, it is recommended that the drug be administered in two or more divided dosages. The maximum daily recommended dose is 60 mg/kg. Side effects are generally dose dependent.

Although various agents fall under the category of cholinergic substances, most of them have fairly limited utility secondary to their lack of CNS specificity, poor ability to penetrate the CNS, short half-life, and side-effect profile. Various drugs, including direct agonists, acetylcholine precursors, and acetylcholinesterase inhibitors have been utilized in an attempt to provide "cholinergic stimulation" following brain injury. Newer drugs such as tetrahydro-9-aminoacridine (THA), also commercially known as Tacrine, may hold better promise than more standard drugs, such as physostigmine.

The following discussion reviews some of the potential pharmacologic approaches to dealing with

posttraumatic impairments. The specific residua have been listed alphabetically.

Appetite Dysregulation

Alterations in appetite are common in patients with brain injury. The hyperphagic patient, or "bulimic type," must be contrasted with the hypophagic, or "anorectic type." Presumptive central neurochemical and neurophysiologic mechanisms responsible for alterations in appetite regulation form the basis of drug treatment for these functional sequelae.[51] The present consensus, based on animal as well as human studies,[52,53] suggests that serotonergic agonists (sertraline, fluoxetine, and fenfluramine), opioid antagonists (naltrexone), and possibly corticotropin releasing hormone may all inhibit feeding behavior. Interestingly, there is recent evidence that questions whether the anorexigenic effects of sertraline and fluoxetine are mediated by 5-hydroxytrytophan.[54] Central serotonergic antagonists such as cyproheptadine can be utilized when there are problems with anorexia or hypophagia.[52]

Ataxia

Various forms of brain injury can result in cerebellar ataxia, including trauma, stroke, tumor, degenerative conditions, and inherited ataxias such as Friedreich's ataxia. Several authorities have reported that the serotonergic precursor L-tryptophan can significantly improve cerebellar ataxia due to a variety of primary etiologies.[55–57] Oral thyrotropin-releasing hormone also appears to be a promising agent.[38,39] Other agents that have been utilized with some success include propranolol, gamma-vinyl GABA, acetazolamide, and phthalazinol.[57] Peterson and associates have reported good success with amantadine for Friedreich's ataxia, presumably through either a dopaminergic or more likely a GABAergic mechanism.[41]

Autonomic Dysregulation

One of the most challenging clinical conditions to treat following severe CNS injury is that of autonomic dysregulation with associated symptoms of hyperthermia,[58] diaphoresis, tachycardia, and tachypnea. Numerous neurochemical systems have been theorized to be involved with central control of temperature regulation, but relatively speaking, hypothalamic dopaminergic systems seem to play a very significant role.[59,60] Hyperpyrexia following brain injury has been successfully treated at a central level with dopaminergic agonists,[61] morphine, β-blockers, and neuroleptics. Dantrolene sodium has also been utilized to help decrease peripheral systemic effects such as rigidity commonly associated with this condition.

Cognitive Behavioral Dysfunction

Before any pharmacologic agent is administered in an attempt to improve cognitive function, one must first establish whether the individual's internal and external environments have been maximized and stabilized.

The examination of the internal environment should be composed of assessing the individual's present neuromedical condition. Neuromedical issues that could present as alterations in cognitive function include (*a*) basic metabolic aberrations; (*b*) nutritional depletion; (*c*) occult infection; (*d*) neuroendocrine dysfunction; (*e*) suboptimal cerebral blood flow or oxygenation, or both; (*f*) posttraumatic hydrocephalus; (*g*) late extra-axial collections; and (*h*) unrecognized posttraumatic seizure disorders. Other critical internal factors include the neuronatomic correlates of injury made evident by either static or dynamic imaging technologies; postinjury medical history (ie, significant hypoxic-ischemic injury, elevated intracranial pressures, etc.); and preinjury factors, such as prior brain injury, substance abuse, learning disability, or psychiatric illness.

The examination of the external environment must take into consideration the extent of stimulation and the cognitive-behavioral status of the individual at that particular time. It must be recognized that, structured or not, the extent and complexity of environmental stimulation must be gauged by the individual's cognitive-behavioral profile. A patient who is highly volatile, easily irritated, or hyperaroused will do better with less stimulation than with more. On the other hand, the individual who tends to be at the lower end of the functional scale, or who becomes confused in unfamiliar surroundings, will perform better, cognitively speaking, when provided with more structured stimulation in a familiar environment. Many times, individuals with brain injuries who have severe physical disabilities are assumed to be also disabled from a cognitive standpoint; this is not always the case. In these individuals, cognitive performance may actually suffer secondary to inadequate environmental stimulation, sometimes nearing the point of environmental sensory deprivation.

It is also critical to consider issues of aging (including the inherent decline in learning ability and retention of new information that is known to occur with aging) on the potential response of an individual to medication following TBI.[62] Additionally, certain situational variables appear to influence the performance of geriatric subjects; specifically, older individuals perform more poorly when task difficulty is high, or when complex encoding strategies or mnemonics are required.

A variety of neurochemicals have been hypothesized as being involved with the mediation of cognitive processes. It is rather a limited viewpoint that attempts to explain cognitive function by Occam's razor, that is, hypothesizing the simplest explanation possible to rationalize a particular process. Cognition is most likely a set of processes mediated through the interaction of a variety of neurochemical systems. Some of the neurochemical substrates that have been proposed to be involved in mediation of cognitive processes (both facilitory and inhibitory) include cholinergic, catecholaminergic, neuropeptidergic (vasopressin, thyrotropin releasing hormone [TRH], endogenous opioids, neuropeptide γ, and adrenocorticotropic hormones [ACTH]), GABAergic, and hormonal systems. Other substances (vitamin cofactors and trace metals) have also been theorized to play important roles in allowing normal neurophysiologic reactions to proceed unabated.

Many drugs have been advocated to improve memory, learning, and general cognitive function. Disappointingly, there is as yet no magic bullet, possibly as a result of the reflection of the nature of the basic neurophysiological and neurochemical processes in question rather than a lack of adequate understanding on the part of researchers regarding cognitive processes. Of the agents that have been studied, the response rates have been quite variable, or the sample populations or experimental methodologies (or both) have been suboptimal. Ultimately, research may find a "cognitive enhancement cocktail" that combines various agents in an attempt to normalize and even maximize the neurochemical environment deemed to be most conducive to enhancement of cognitive processes. At this time, there does not appear to be one single cognitive enhancing drug (CED) that works *all* the time for *every* individual who suffers from posttraumatic cognitive deficits.

In recent years, there has been a fairly extensive body of literature examining the potential influence of hypothalamic and pituitary neuropeptides on learning and memory.[63] Vasopressin analogues and ACTH have been reported to improve memory and learning in numerous test situations in humans and several species of animals. One hypothesis is that ACTH/MSH (melanocyte stimulating hormone) affects attentional and motivational processes, whereas vasopressin is more directly involved in memory processes. On the other hand, opioids specifically, beta-endorphin and met-enkephalin) seem to have amnestic qualities that can be reversed through administration of opioid antagonists, such as naloxone or naltrexone.[64] The only published double-blinded, placebo controlled studies that specifically examined the utility of vasopressin in persons with TBI found no clinically significant benefit.[65,66] Regardless of ultimate efficacy, the electrolytic effects of vasopressin on sodium homeostasis may be the limiting factor in clinical application of this CED. More recent research on the utility of TRH as well as vasopressin, has been conducted at the University of Washington in the Department of Rehabilitation Medicine as part of their Rehabilitation Research and Training Center in Traumatic Brain Injury.[67] Preliminary data seem encouraging regarding a potential role of these agents in memory enhancement mediated by cholinergic systems.

Of all neurotransmitter systems proposed to play a role in memory function, the cholinergic system has without question received the most attention. Most of the work in this area emanates from research in senile dementia, Alzheimer's type. Although the scientific literature is mixed regarding the role of cholinergic pathways in memory function, an increasing number of drug studies[67] in humans and animals suggest that pretreatment with anticholinergic drugs disrupts memory storage, whereas cholinergic agonists may actually produce dose-dependent facilitation or disruption. Some research[67] also suggests that a neurochemical dissociation of cholinergic memory systems exists, such that cholinergic neurotransmission is required for declarative, but not procedural, memory. Interestingly, there may actually be a "therapeutic window" for cholinergic agents so that beneficial effects are present only at middle range doses and are absent at low range doses; whereas high doses lead to impaired cognitive function.[68]

The approach to treatment of cognitive deficits referable to cholinergic system augmentation may take one of three main routes: (1) precursor agents such as choline or lecithin, (2) anticholinesterases such as physostigmine or THA, or (3) direct cholinergic agonists such as bethanecol or oxotremorine.[69] Only a few scattered studies, with rather mixed results, have specifically addressed the utility of

cholinergic agents in individuals with TBI.[70–72] Tetrahydro-9-aminoacridine, a potent anticholinesterase, may be a cholinergic "drug of the future" secondary to the fact that it can be administered orally, has a relatively long half-life, and has a reasonable side effect profile.[73] Most recently, it has achieved attention relative to a potential role in the treatment of cognitive deficits associated with senile dementia. The utility of this specific drug is yet to be clarified in individuals with TBI. CDP-choline (cytidene 5′-diphosphocholine), an essential precursor in the synthesis of brain glycerophospholipids, has been studied in patients with TBI to enhance neurorecovery, including cognitive performance. Results to date, including several placebo controlled or double blind studies, or both, look promising.[74,75] For now, the pharmacological side effects and suboptimal modes of administration of many of the cholinergic agents limit their clinical usefulness.

A large body of evidence indicates that catecholamines may be involved in the modulation of learning and memory. A number of drug studies have suggested that drugs that disrupt catecholamine systems disrupt memory storage, while catecholamine agonists produce dose-dependent facilitation or disruption. As an example, amphetamine has been shown to have no effect at low doses, improvement at restricted dosage ranges, and impairment at higher doses.[76] The major catecholaminergic neurotransmitters are norepinephrine and dopamine. It is possible to affect the net balance of neurotransmitter effects, as well as turnover, through the administration of agents that ultimately affect the net activity at the postsynaptic receptor site. Drugs may exert their effect by increasing release from presynaptic stores (methylphenidate), increasing production and release from the presynaptic vesicles (L-dopa/carbidopa), decreasing reuptake into presynaptic vesicles (nortriptyline, desipramine), or acting directly at the postsynaptic receptor site (bromocriptine). Some agents, such as amantadine, may effectuate their ultimate agonistic effect at both the presynaptic and postsynaptic receptors through a variety of mechanisms.[77] The use of psychostimulants, such as amphetamine or Ritalin, typically results in improved concentration and performance, a suppression of fatigue, and an elevated mood. These noradrenergic drugs may also produce the adverse side effects of anorexia, hypertension, tachycardia, and aberrant behavioral changes (ie, euphoria or dysphoria). Although multiple studies have utilized catecholaminergic agonists after brain injury, very few have specifically assessed their utility for remediation of cognitive dysfunction.[78,79] One case study[80] involving the assessment of clonidine (a central α-2 noradrenergic agonist) found no benefit to this particular pharmacologic intervention.

Nootropics are a relatively new class of CNS-active drugs that have a direct functional impact on the higher integrative mechanisms of the brain. A few of the nootropic-like drugs that have been advocated to improve cognitive function include piracetam, etiracetam, aniracetam, pramiracetam, vincamine, dihydro-ergotamine, and centrophenoxine. Their chemical structures are quite different and their specific mechanisms of action are still unknown. Some of the proposed mechanisms of nootropic action include facilitation of dopamine release, increase of acetylcholine turnover, and inhibition of α-adrenoreceptors. One study[81] in TBI did demonstrate some beneficial effects of pramiracetam. Unfortunately, most of the more promising nootropic agents are still unavailable for clinical use in this country. The beneficial effects of Hydergine, a dihydrogenated ergot alkaloid, were recently reviewed by McDonald,[82] and he concluded that it produced some global improvement in memory. However, a recent well controlled study[83] using ergoloid mesylates for Alzheimer's disease failed to show any significant memory benefit. No studies have specifically assessed the utility of this drug for treatment of cognitive dysfunction in individuals after TBI.

Numerous drugs aside from Hydergine have been utilized to improve cognitive function secondary to their presumed beneficial effects on cerebrovascular blood flow. These drugs include papaverine hydrochloride, cyclanedelate, naftidrofuryl, and pentoxifylline. Although some literature suggests a beneficial effect of these agents in geriatric populations with concomitant "dementia," there has been no substantial exploration of the benefits of these agents in persons with cognitive dysfunction following TBI.

Given the neurochemical complexity of cognitive processes, it should not be surprising that pharmacologic agents may have the potential to actually impair cognitive processes in both noninjured and injured brains. It is critical to remain aware of the *relative* risks of certain pharmacologic agents in terms of their *potential* to impede cognitive processing.

Three main classes of drugs are felt to have the potential to interfere with cognitive functioning by way of their basic neurochemical mechanisms of action: (1) catecholaminergic antagonists, (2) GABAergic agonists, and (3) cholinergic antagonists. Agents that block catecholaminergic receptor

sites have been linked with deficits in attention, concentration, and memory. The main drug categories in this group are neuroleptics and antihypertensives. Neuroleptics such as haldol, thiothexene, and mellaril are primarily used for treatment of behavioral disturbances and act by dopaminergic blockade. Ideally, they should only be used for treatment of acute agitation and are rarely needed for long term behavioral management. Antihypertensives such as methyldopa, propranolol, and prazosin act by noradrenergic blockade and, therefore, may impair cognitve function in the individual with brain injury. All possible attempts should be made to avoid these agents given the availability of many other antihypertensives that act peripherally and are just as clinically effective (ie, ACE [angiotensin converting enzyme] inhibitors, calcium channel blockers, etc.).[84]

The multiple potential clinical uses of GABAergic agonists include seizure management, spasticity treatment, control of aggression, and as sedative hypnotics. The adverse cognitive effects that have been reported with this class of drugs include state-dependent learning, paradoxical agitation, and transient global amnesia. One must, therefore, realize that medications such as valium, baclofen, clonazepam, lorazepam, and temazepam, are not innocuous agents with regard to their potential cognitive side effects.

The association of anticholinergic use and cognitive impairment is by no means foreign to most practicing physicians. The fact that antidepressants are so commonly prescribed following TBI bespeaks judicial use of this class of medications, including an awareness of the relative anticholinergic potencies of specific agents.[84] Given the fact that newer and less anticholinergic agents are now available (eg, fluoxetine, trazodone, sertraline, and paroxetine), it would seem reasonable to assess the efficacy of these agents in individuals with TBI to ascertain the true potential of these agents in the treatment of organic affective disorders.[85]

Hemiinattention and Neglect

Ascending dopaminergic pathways have been experimentally implicated in mediation of attentional processes, including hemispatia neglect. Two small studies have demonstrated a potential utility of dopamine agonists, specifically bromocriptine, in the treatment of neglect secondary to cerebrovascular accident[86] and TBI.[87] Both studies utilized an A–B–A paradigm and demonstrated significant differences in testing performance, as well as functional capabilities, while patients were receiving dopamine agonist pharmacotherapy. Further studies are obviously warranted based on the encouraging results of these two.

Movement Disorders

A variety of movement disorders have been treated with some success following brain injury. These include dystonia, tremors, Parkinsonism, tics, akathisia, myoclonus, and dyskinesias (such as chorea, ballismus, and athetosis).

Dystonia, whether focal, segmental, or generalized, has been treated with a variety of agents, but with mixed results. Dopaminergic agonists and antagonists, anticholinergics, baclofen, benzodiazepines, and carbamazepine have all been utilized in the treatment of this class of movement disorders.[88,89]

Following TBI, tremors are typically of the postural or kinetic type (or both), whereas resting tremors are typically seen with nontraumatic degenerative cerebral disorders, which result in dopaminergic deficiency. Pharmacologic treatment tends to work better for nontraumatically induced tremor than for tremor resulting from trauma. A variety of drugs have been utilized, including β–adrenergic blocking agents, benzodiazepines, dopaminergic agents, valproic acid, and anticholinergics.[90–92] Drug induced tremor must always be considered a result of iatrogenic prescription or patient use of nicotine, or both.[93]

Parkinsonism, when a result of trauma, can be treated fairly well with pharmacologic intervention. Following diffuse brain injury, numerous authorities have reported patients with Parkinsonian-like symptoms, such as bradykinesia, dysarthria, decreased facial expression, and rigidity.[79,94] Drugs that have been shown to be effective for " posttraumatic Parkinsonism include dopaminergic agonists and, to a lesser extent, anticholinergics.

Tics are a rare consequence of acquired brain injury.[88,89] The drugs used to treat tics include GABAergic agonists, dopamine antagonists, and to a lesser extent, noradrenergic drugs, such as clonidine.[47]

Akathisia has been reported following brain injury and in animal models is thought to be associated with a relative dopaminergic deficiency in the prefrontal area. Successful treatment of akathisia using bromocriptine has been reported.[95] Other drugs that have been utilized, but with fairly limited success, include benzodiazepines and β-adrenergic blockers.[96] Myoclonus is a common sequela of severe hypoxic ischemic brain injury, but can also

be seen after nonhypoxemic brain injury. Cortical myoclonus must be differentiated from epilepsy partialis continua.[97] A variety of drugs, including benzodiazepines (clonazepam), serotonergic agonists (such as trazodone and L-tryptophan), valproic acid, primidone, and piracetam, have all been reported effective.[98,99]

Dyskinesias can occur in a variety of conditions and be manifested as ballismus, chorea, or athetosis. As a result of trauma, these types of movement disorders can result from thalamic or striatal injury, or both. Typically, the drugs that have shown some utility for TBI associated dyskinesias include dopaminergic antagonists and a variety of anticonvulsants, including carbamazepine, phenobarbital, valproic acid, and phenytoin.[88,100,101] It should be noted that certain dyskinesias may actually be atypical presentations of posttraumatic epilepsy.

Neurogenic Heterotopic Ossification

The only pharmacologic therapies presently available to minimize the extent of morbidity associated with neurogenic heterotopic ossification following brain injury involves the use of etidronate disodium[102] and nonsteroidal antiinflammatory agents (NSAIDs). Didronel presumably works by interfering with biological calcification; specifically, impairing the calcification of osteoid. When there is still an acute phase to the condition, NSAIDs have been advocated to decrease the suspected inflammatory component of this pathologic process.

Didronel therapy is typically initiated at 20 mg/kg/d and the dose is subsequently lowered after several weeks to months to 10 mg/kg/d. There are no well controlled, reliable trials examining the use of this agent in homogeneous brain injury populations; therefore, many, if not all, of the recommendations are based on spinal cord injury literature. The main side effect of the medication involves gastrointestinal complaints in the form of diarrhea and nausea.

Posttraumatic Seizures

At the present time, most neurosurgeons in this country use either phenytoin or phenobarbital for early management of seizures or seizure prophylaxis (or both) due to the fact that these medications can be administered parenterally (by intravenous route in the acute care setting). It is still unclear as to the exact utility of anticonvulsant agents in the prevention versus suppression of posttraumatic seizures.[103–105]

Recent literature by Temkin and associates[103] is highly supportive of the conclusion that phenytoin treatment for prophylaxis (treating with anticonvulsant medication to suppress potential seizures even though none have occurred) is efficacious only during the first week postinjury. Ongoing research also reveals that prophylaxis with other agents (such as carbamazepine) is also ineffective. Studies are presently underway to examine the prophylactic use of other agents, such as valproic acid. Except in very high risk cases, such as a penetrating brain injury, the common practice in most progressive rehabilitation centers is to discontinue anticonvulsant treatment if it has been prescribed prophylactically. Even in high risk cases it may be prudent to wean patients off medications and see how they do, treating only if they declare themselves. Whether or not to treat after one late (after the first week) seizure or to wait for the second late seizure remains controversial.

A current trend within the field of brain injury rehabilitation is to advocate the utilization of specific anticonvulsants in the postacute setting following brain injury (traumatic or nontraumatic), specifically, carbamazepine and valproic acid.[105,106] In general, carbamazepine should be a first line agent for treatment of partial seizures, whether simple or complex. On the other hand, valproic acid should be the agent of choice for multifocal epilepsy and generalized tonic-clonic seizures. This is not to say that select patients may not achieve better seizure control, potentially with fewer side effects, on agents such as phenytoin and phenobarbital. Clinicians should also be aware that valproic acid has been associated with encephalopathy and alterations in consciousness, most likely secondary to hyperammonemia.[107] On the other hand, recent experimental evidence suggests that valproate may be the most efficient agent relative to suppression of kindling phenomena. Another advantage that psychotropic anticonvulsants have over other seizure medications are their potentially positive effects on behavior. This is not to say that some patients may not do better on other agents, such as phenytoin for seizure control; however, the clinician must examine all aspects of a particular agent prior to instituting treatment. Obviously, drug interactions, side effects, cost, and compliance issues of the various anticonvulsant medications must be taken into consideration on a case-by-case basis.

Various studies[108–110] have demonstrated significant negative effects on cognitive function secondary to phenytoin and phenobarbital. A recent study[111] questioned these general findings, bringing

to light the need for further research in this area. A variety of newer agents, such as oxcarbazepine, felbamate, vigabatrin, flunarizine, lamotrigine, and others, are presently being studied in an attempt to develop more effective drugs with fewer cognitive and systemic side effects.[112]

Sexual Dysfunction

Following brain injury, it is common for individuals to have problems in the area of sexuality. One of the most regular complaints is alteration in libido.[113] Hyposexuality can be treated with a number of different pharmacologic agents, including activating antidepressants, yohimbine (a noradrenergic agonist), dopamine agonists, and hormonal supplementation.[114] Hypersexuality, on the other hand, is a relatively rare clinical condition that is more difficult to broach from a pharmacotherapeutic standpoint. Hormonal agents, specifically, medroxyprogesterone acetate, have been utilized to "chemically castrate" individuals with severe hypersexuality problems.[115] For patients who have bitemporal involvement and associated hypersexuality as seen in Kluver-Bucy syndrome, carbamazepine is generally considered the treatment of choice.[116] Other agents that may hold potential utility for treatment of the hypersexual patient following brain injury include serotonergic GABAergic and opioid agonists.[115] There are obviously significant ethical and medicolegal ramifications in the utilization of agents affecting sexual drive in this population.

Speech and Language Disorders

A number of different medications have successfully been used for a variety of speech and language disorders in patients with brain dysfunction. Bromocriptine has been reported to improve speech dysfunction in patients with diffuse TBI with dosages ranging from 20 to 40 mg daily.[117] Another series of studies demonstrated the efficacy of bromocriptine in the treatment of dysphasia, specifically, the transcortical motor variant.[118,119] Animal studies have yielded some support for the role of dopaminergic pathways in both spontaneous and reflex swallowing,[120,121] leading to human studies that support the potential efficacy of dopamine agonist therapy for dysphagia following brain injury utilizing L-dopa/carbidopa.[122] Parkinsonian hypokinetic dysarthria has been treated with low dose clonazepam (0.25–0.5 mg/d); the probable mechanism for its efficacy being striatal GABAergic agonism.[123] Lastly, a case of posttraumatic adult onset stuttering responsive to anticonvulsant treatment has been reported, suggesting that ictal speech disorders should always be considered in this patient population.[124]

NEUROMEDICAL ISSUES

There are numerous neuromedical issues that the physiatrist must take into consideration when treating the survivor of TBI. Early identification of cranial nerve injuries not only has potential prognostic significance, but is also critical relative to institution of appropriate treatment regimens.[125] The clinician must be familiar with the diagnosis and management of central hyperthermia and autonomic dysregulation syndrome,[61,126] neurogenic heterotopic ossification,[127] hypertonicity,[128] fractures,[129] nutritional issues,[130] pulmonary and tracheostomy problems,[131] peripheral nerve injuries including neuropathies and plexopathies,[132] and concurrent spinal cord injury.[133] It is also critical for the physiatrist to have specific knowledge of subpopulations of TBI patients, including pediatric and geriatric.[134–136] A number of neurologic conditions may not manifest themselves until sometime after the initial neurologic insult. Posttraumatic communicating hydrocephalus may occur in up to 8% of survivors of severe TBI and typically present with evidence of neurologic deterioration or plateau in the face of ongoing ventriculomegaly without associated sulcal enlargement.[137] Sophisticated procedures, such as cerebrospinal fluid infusion studies, may assist the clinician in making a more definitive diagnosis and proceeding with shunting, the definitive treatment. Subdural hematomas and hygromas may develop and progress in the postacute period, thereby providing some rationale for the practice of follow-up imaging.[138] Posttraumatic seizures and epilepsy are relatively common clinical conditions, particularly after severe TBI, and physiatrists should have a thorough understanding of the potential clinical presentations, workup, and treatment of this class of posttraumatic disorders.[139] Although rare, other late neurological disorders include posttraumatic movement disorders, neuropsychiatric complications, functional deterioration associated with aging, and certain neuroendocrine disturbances.

Cognitive and behavioral problems are frequent following all severities of TBI. Cognitive dysfunction may take many forms. Clinicians must ad-

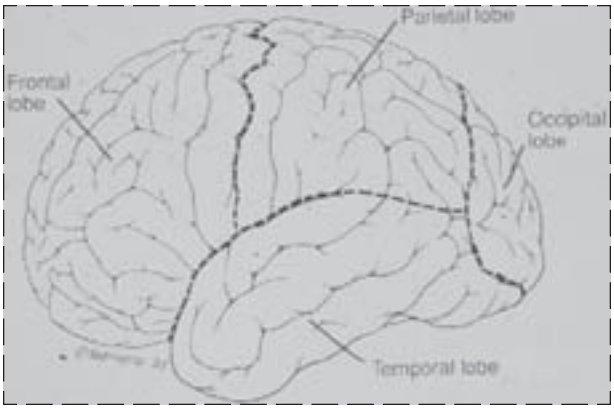


Fig. 6-3. Basic surface anatomy of the brain demonstrating the relative demarcations for the four main lobes: frontal, parietal, temporal, and occipital. Reprinted with permission. Copyright Robert Shepherd. Shepherd Visual Communications: Richmond, VA; 1993.

equately delineate the type of dysfunction present to determine if it is at all remediable. Many concurrent issues may exacerbate cognitive problems, including inappropriate medication prescription, vestibular problems, sleep–wake cycle dysfunction, chronic pain due to posttraumatic headache or myofascial dysfunction, and visuovestibular dysfunction. Although, as previously mentioned, there are no magic bullets for cognitive dysfunction, the professional may want to consider more traditional cognitive remediation interventions, including computer based cognitive retraining. There is generally a trend toward functionally based remediation of cognitive dysfunction, preferably in the environment in which the behavior occurs, whether at home or work.[140] As previously discussed, pharmacotherapeutic interventions may also augment the potential benefit gained from more traditional physiatric interventions.[141] Patients with other factors exacerbating their cognitive dysfunction must have these factors addressed concurrent with other interventions.

Behavioral issues are relatively commonplace following TBI. Comprehensive assessment should include elicitation of an adequate history, use of symptom rating scales and behavioral assessment measures, evaluation of brain structure and function by way of imaging and electrophysiological assessment, and neuropsychological evaluation. Several behavioral assessment tools are available, including the Structured Clinical Interview,[142] Neurobehavioral Rating Scale,[143] Hamilton Rating Scale for Depression,[144] and the Overt Aggression Scale.[145] A variety of neurobehavioral alterations may be seen following brain insult. Personality changes, intellectual deterioration, and affective disorders may manifest themselves as a direct result of TBI. Other conditions, not directly related to TBI, must also be taken into consideration, including posttraumatic stress disorder, reactive depression, and sleep–wake cycle disturbances related to pain from myofascial dysfunction or orthopedic injuries, or both. Organically based affective disorders may present as depression, mania, delirium, and psychosis. Anxiety, aggression, irritability, mood lability, and sleep–wake cycle disturbances are also quite common in this patient population.[146] Clinicians should be aware of both behavioral and pharmacological approaches to the management of behavioral issues.[147] Some of the other issues that may aggravate behavioral problems following TBI include chronic pain, vestibular dysfunction, organically based fatigue, medication side effects, substance abuse, preinjury psychologic and psychiatric dysfunction, organic affective disorders, recurrent head injury, learned maladaptive behaviors, and situational specific conflicts.[146] Clinicians should also be familiar with the array of neurobehavioral conditions that may be seen with brain injury, both focal and diffuse. Knowledge of regional brain anatomy (Figure 6-3) and neurobehavioral correlates is critical for the practicing clinician who works with TBI survivors (Table 6-1).

TABLE 6-1

NEUROBEHAVIORAL PROBLEMS ASSOCIATED WITH DAMAGE TO SPECIFIC LOBES OF THE BRAIN

Lobe	Problems
Frontal	Orbitofrontal and dorsolateral neurobehavioral syndromes
Parietal	
Dominant	Gerstmann's syndrome (finger agnosia, dysgraphia, dyscalculia, and left-right disorientation)
Nondominant	Topographical disorientation, dysprosodia, and neglect
Temporal	Episodic dysphoria, alterations in libido, episodic dyscontrol, temperolimbic epilepsy (TLE)
Occipital	Anton's syndrome, cortical blindness

ADVANCES IN FUNCTIONAL ASSESSMENT

As brain injury rehabilitation has matured, clinicians have realized the critical need for valid and reliable assessment and outcome evaluation measures. The development of such measures has resulted from a lack of applicability of overall health and functional measures from other areas of medical rehabilitation, and general and mental health. Physiatrists working with TBI survivors must delineate the cornucopia of impairments that each patient presents, and must also identify how these specific impairments result in disability and handicap.

Clinicians have tried to develop outcome and functional assessment measures that correlate with real world function. These measures may also be used to track functional progress, response to a specific treatment or nontreatment, and to assess the efficacy of specific interventions or programs, by utilizing review and case management for possible outcome prognostication. Prior to utilizing any measure for any purpose, clinicians need to be fully apprised of the specificity and sensitivity of these scales, as well as their validity and reliability.[148]

Many general status measures have been developed for TBI,[149] including the Disability Rating Scale, Rancho Los Amigos Scale, Glasgow Outcome Score, and Glasgow Assessment Scale. Other measures have been designed specifically to use with patients at low levels of neurological function, including the Coma Recovery Scale, Coma/Near Coma Scale, Sensory Stimulation Assessment Measure, and Western Neurosensory Stimulation Profile. Several scales address multidimensional functional status evaluation, including the Functional Inventory Measure, Functional Assessment Measure and Patient Evaluation Conference System, and others.

There are a number of more focused clinical measures for delineation of communication, cognitive, behavioral and psychosocial function. Measures addressing community and vocational integration, and life satisfaction and stress have been less well developed for this specific patient population. Refer to Exhibit 6-1 for a review of specific measures for individuals with TBI.

It is critical for clinicians to realize that there are inherent limitations in functional assessment and outcome measures. For example, measures, per se, do not give evidence of intervention effectiveness. Use of functional measures to "prove" effectiveness is at best an inference based either on research design or preexisting causal knowledge garnered from controlled research.

There are also a number of domains of function that are not adequately addressed in TBI functional assessment and outcome measures. Some of these parameters that are particularly critical include psychosocial functioning, avocational activities, neurobehavioral status, higher level physical and cognitive deficits, limitations secondary to lack of insight and judgement, and job maintenance capability.

Given the aforementioned, it is extremely important for clinicians to understand why they are utilizing a specific measure, and the strengths and limitations of each instrument. When applied appropriately, functional outcome and assessment measures can serve the clinician, third party payers, patient, and family in a positive manner. When used indiscriminately, these measures can do more harm than good.

EXHIBIT 6-1

BRIEF REVIEW OF SOME OF THE MORE FREQUENTLY UTILIZED FUNCTIONAL ASSESSMENT MEASURES IN TBI REHABILITATION

General Scales

- Disability Rating Scale
- Glasgow Outcome Scale
- Modified Barthel Index
- Functional Independence Measure (FIM)
- Functional Assessment Measure (FAM)
- Rancho Los Amigos Scale
- Adapted PULSES
- Glasgow Assessment Schedule

Scales for Persons with Severe Alterations in Consciousness

- Coma Recovery Scale
- Western Neurosensory Stimulation Profile
- Sensory Stimulation Assessment Measure
- Coma/Near Coma Scale

MODEL SYSTEMS CONTINUUM OF CARE FOR TRAUMATIC BRAIN INJURY

The need for a comprehensive continuum of neuromedical and rehabilitative care for individuals with TBI, regardless of severity, cannot be overemphasized.[150] Coordination of services, across medical disciplines and among healthcare specialists will increase the ultimate quality of patient care. Timely and appropriate intervention also decreases both short- and long-term morbidity and mortality associated with TBI.[151] The VA medical system has recently designated a select number of its hospitals to serve as brain injury treatment centers and provide comprehensive services to soldiers with TBI. In the military there are frequently no physiatrists assigned to evacuation or corps-level hospitals.

Leadership Issues

It has become apparent from the Model Systems[152] experience that the success of the interdisciplinary team, and the system itself, is dependent on a strong team leader. For a team to fully integrate across interdisciplinary as well as multidisciplinary "barriers," there must be firm guidance and strong leadership from a senior clinician. The central leadership role is, in essence, the glue that holds the system together. In the field of brain injury rehabilitation, many challenges face physicians relative to both clinical and nonclinical training. These include brain injury experience during residency, specialization controversies, continued medical education, and an ability to serve in administrative capacities as leader of an interdisciplinary or transdisciplinary team.[152,153]

General TBI Education

Ignorance of TBI sequelae is quite common in the community at large and among health-care professionals. Regional TBI education by neuromedical and rehabilitative professionals who specialize in TBI care, both acute and chronic, should be included in a comprehensive rehab-ilitation program. This would serve not only to increase awareness of the problems faced by individuals with TBI, but would improve the quality of care given to such individuals.[152]

Emergency Medical Services

Appropriate emergency medical services are an essential feature of comprehensive TBI neuromedical care continuum. The ultimate goal of a well trained and efficient casualty evacuation service is rapid, early intervention; patient stabilization; and subsequent transport to a *predesignated* medical facility that can provide the equivalent of level 1 trauma center care for patients with severe head injuries (as defined by a GCS score of 3 to 8). Designated military medical facilities treating moderate and severe brain injury should have neuroimaging facilities available; specifically, computed tomography (CT) scanning and magnetic resonance imaging (MRI). Based on ongoing research, other imaging modalities, including single photon emission computed tomography (SPECT) may soon become standard. Appropriate ground and air transportation services should be available to transport more severely injured patients to level 1 trauma centers or their functional equivalent. Expeditious evacuation from the accident or battle scene to acute neuromedical facilities will allow for more accurate neurosurgical diagnosis and treatment, thereby minimizing secondary brain injury and its associated morbidity and mortality. Any patient seen in emergency room facilities who is subsequently discharged to duty should have appropriate supervision and adequate monitoring, as well as referral for follow-up if needed. If appropriate supervision is unavailable, the patient should be admitted to the hospital for observation.[152]

Professionals evaluating more subtle head injuries and TBI should be aware of criteria that justify hospital admission, such as GSC scores of less than 15, focal neurologic deficits, altered mental status, abnormal CT scans, and so forth.[3] Mild brain injured patients evaluated in emergency room facilities should be given general information sheets and provided with appropriate information regarding postconcussional disorders. Line commanders should be educated regarding postconcussive symptoms and the specifics of monitoring behavioral and performance abilities of their soldiers. If the soldier exhibits any lasting postconcussive problems, more definitive assessment is warranted through referral to a larger military hospital.

Acute Neurosurgical Care

Patients admitted for treatment or observation, or both, regardless of severity of injury, should be screened, preferably by a rehabilitation medicine

consultation service. Proper communication across disciplines, that is, neurosurgery and rehabilitation medicine, has proven to be a critical factor in the development of a continuum of quality care for TBI patients. Additionally, it is not uncommon that problems that lead to potential morbidity issues and higher level cognitive linguistic deficits are sometimes overlooked by acute care physicians, thereby necessitating the involvement of a physiatric consultant. If possible, interdepartmental rounds should occur to review patient condition, individual team recommendations, and disposition issues. A timely and efficient hierarchy of communication should exist in order to inform consulting rehabilitative services of patient admissions to the neurosurgical service. Routine interservice communications ultimately allow for smoother transitions of care if and when the patient is transferred to the care of the rehabilitation medicine physician.

Neuromedical and rehabilitative issues that can be addressed by the physiatrist for patients with mild TBI include diagnosis and treatment of postconcussive symptoms, such as posttraumatic headaches, audiovestibular disorders, balance disorders, visual changes, olfactory and gustatory dysfunction, and cognitive behavioral deficits.[3]

For more severely injured patients, the major issues of neuromedical management to be addressed by the physiatrist include skin care, bowel and bladder management, behavioral management, tone control, maximization of nutritional status, maintenance of joint range-of-motion, and optimization of the patient's potential for maximum neurologic and functional recovery through both pharmacologic and nonpharmacologic modalities. During the acute period of treatment, particularly in cases of severe injury, it is essential to have a multidisciplinary neuromedical staff available.[152] The staff must be familiar with complications associated with TBI including, but not limited to, neurogenic heterotopic ossification, traumatic myositis ossificans, posttraumatic seizures, spasticity management, neuroendocrine disorders, neuroophthalmologic problems, olfactory dysfunction, audiovestibular deficits, orthopedic injuries, dysphagia, tracheostomy management, and posttraumatic psychologic, as well as psychiatric disturbances.[154]

Services that should be readily available for consultative purposes as part of the acute continuum of TBI care include neuroophthalmology, ophthalmology, gastroenterology, general and orthopedic surgery, neurology, dental medicine, oral surgery, plastic surgery, urology, substance abuse, dermatology, and ear, nose and throat.

In both acute neurosurgical and acute brain injury rehabilitation care, prevention of morbidity is a critical contribution of the physiatrist and rehabilitation team. Specifically, aggressive efforts should be made to counteract the adverse effects of immobility. Early care that can decrease the complications associated with protracted immobilization are (*a*) passive and passive assisted range-of-motion exercises to decrease muscle atrophy, (*b*) mobilization efforts (ie, getting the soldier out of bed once intracranial pressures [ICPs] are controlled), (*c*) contracture prevention through positioning, (*d*) ranging and splinting, (*e*) turning protocols to prevent skin breakdown, and (*f*) deep vein thrombosis prophylaxis. Spasticity treatment, including potential use of neurolytic agents for motor point or nerve blocks (or both), should also be a focus of early rehabilitative care.

Acute Brain Injury Rehabilitation

Once patients are medically stable, and at the discretion of the consulting physiatrist, certain patients with brain injury may meet criteria for admission to acute inpatient brain injury rehabilitation programs. Ideally, these units should be dedicated to the severe TBI patient population, in both the space allocated for the unit and the treatment team. In the modern military, this may mean certain designated continental medical centers or specialized VA hospital facilities. Use of a transdisciplinary team approach, and training the staff to be sensitive to medical and psychosocial issues commonly encountered after TBI helps to maximize treatment efficacy. Specific admission criteria should be developed for all units.

As appropriate, some programs should attempt to designate separate units or a small portion of bed space to "early recovery management programs"[2] for patients who exhibit slow neurologic recovery. Ideally, patients should be admitted within the first 3 months postinjury; however, patients who are further along than 3 months postinjury should also be considered as candidates. It should be understood that their potential for recovery is much smaller than the potential of those patients who are within the first 3 months postinjury.

Slow to recover patients should generally be given a 2 to 3 month trial of inpatient care with the goal set at maximization of their recovery potential and minimization of neurologic and functional morbidity. If there are no significant improvements, other dispositions, such as long-term placement, need to be considered. Long-term care facilities,

typically based in skilled nursing homes, should be staffed by healthcare professionals adept at dealing with the multitude of functional and neuromedical issues relevant to TBI survivors. Some opportunity for rehabilitative follow-up is critical in order to assess neurologic or functional change, whether positive or negative, and the need for appropriate modification of existing treatment plans.[154]

Ideally, a transdisciplinary team approach should be implemented when working with survivors of TBI. The team works with the patient and the family to maximize recovery from both a neurologic and functional standpoint. The team consists of a variety of disciplines beyond the medical ones. These disciplines include rehabilitation nursing, physical therapy, occupational therapy, speech-language pathology, cognitive therapy, neuropsychology, rehabilitation nursing, rehabilitation social work, dietetics, pharmacy, and spirituality/religion. Team rounds and conferences should be held regularly, and, when possible, all treating team members should be in attendance. This process increases the team's ability to assess and treat the variety of issues that may be new or ongoing with regard to the individual's recovery and rehabilitation. Preferentially, didactic lectures, journal club, and weekly administrative meetings should be held to promote the cohesiveness of the team.[153]

All patients admitted to an inpatient brain injury rehabilitation unit should receive a complete neuromedical workup to fully assess any factors that may be compromising their neurological or functional recovery process. The standard neuromedical workup should include a thorough history and physical; including full neurologic and functional assessment; EEG; static brain imaging; neuroendocrine assessment as indicated; as well as full nutritional and metabolic evaluation, and comprehensive evaluation to rule out concurrent infection. To rule out inappropriate or excessive medications, a thorough review and assessment of medications must be conducted, preferably in conjunction with a pharmacist. As indicated, patients should be seen by a psychiatric consultant for evaluation and treatment of postinjury neurobehavioral sequelae. Preinjury substance abuse issues, which may effect short- and long-term potential, should be addressed as early as possible with the assistance of substance abuse consultants.[6,155]

A critical part of any brain injury rehabilitation program, regardless of the neurologic or functional level of the patient, should involve family training. Early and ongoing family involvement as active members of the rehabilitation process, including the time after discharge, improves patient outcome and should be encouraged. Institutional as well as community resources should be developed to allow families to cope with the changes that have incurred in their own lives, as well as the life of their loved one. Such resources include rehabilitation service support groups and community support groups through such organizations as local and state chapters of the National Head Injury Foundation (NHIF). (The main office is in Washington, DC and can be contacted at 202-296-6443 or 1-800-444-6443.)

Outpatient Clinical Services

Once patients are discharged from an inpatient brain injury rehabilitation unit they will require ongoing therapeutic services. Patients who are referred to the outpatient clinic, regardless of the severity of injury, should be evaluated by a transdisciplinary outpatient team. If such a team is not available, there should be an assessment performed by the releasing rehabilitation medicine physician in conjunction with the new unit's available therapy staff. All relevant areas should be addressed, including issues relating to mobility status, activities of daily living (ADLs), communication, bowel and bladder status, cognitive and behavioral status, sexuality, and vocational and avocational status. Both patient and family should be included in these discussions.

Appropriate professional resources should be available to work with patients in the outpatient clinic setting. These professional resources include psychologic, neuropsychologic, rehabilitation nursing, and rehabilitation social work. Outpatient clinic staff, including the physician, should be familiar with resources within the community and the surrounding regions so they can optimize the quality of long term care services for the patient and his family. Such services include driver evaluation designed to assess driving skills after TBI, vocational services, behavioral management programs, transitional and independent living programs, and long term care facilities. Appropriate coordination of outpatient rehabilitation services, including day rehabilitation and outpatient therapies, is critical to providing a smooth transition from inpatient status. Families should be referred to a variety of community resources, including their local chapter of the Brain Injury Association (BIA). Community mental health services and recreational programs should be recommended as needed. Literature on TBI from various sources, including BIA, should be distributed to patients and families to increase their understanding and awareness of TBI deficits.

Special diagnostic and therapeutic programs should be implemented in all facilities caring for TBI patients, and for those individuals who have suffered so-called mild traumatic brain injury with associated postconcussive symptoms or cognitive-behavioral dysfunction, or both. Implementation of such programs requires that the treating physician have a thorough knowledge of the etiology, diagnosis, and management of these problems. Additionally, adequate resources, either within the institution of origin or in the surrounding community, are necessary for accurate diagnosis and management of specific postconcussive disorders, including audiovestibular dysfunction, cognitive and behavioral disturbance, and posttraumatic headache. An integral part of such a program should include compensatory strategies for attention deficits, memory problems, and impaired mental flexibility; rapid processing is imperative. For the patient with mild TBI, the neuropsychological staff must have specialized training and expertise in TBI evaluation, interpretation, and treatment. Counseling services for patients and families should be available to help their adjustment to associated sequelae. Higher level cognitive and behavioral changes, which may interfere with vocational reintegration, need to be addressed by a qualified vocational specialist.[156]

Neurobehavioral Programs

Neurobehavioral programming is one of the least met needs of survivors of TBI relative to the continuum of clinical care. Typically, neurobehavioral programs are rendered in skilled nursing facilities or in community based environments. Only a few select programs exist in acute care hospital settings, and those are for the most severe patients who also might require aggressive and significant neuromedical workups. This problem stems from a lack of a generally agreed-on programmatic content for this aspect of treatment. Additionally, the level of expertise in nonpharmacologic and pharmacologic management varies greatly across programs. Given that psychosocial and behavioral issues make a major subset of posttraumatic sequelae and compromise individual survivor capacities for significant community reentry, it is surprising that so few resources have been allocated to this clinical service.[157]

Home Based Services

In recent years, one of the major advances in service provision has been the development of home based, neurologic rehabilitation care. This service has allowed patients to make quicker transitions from institutionalized care to their home environments. It also negates the significant concerns regarding the general applicability of information learned in environments foreign to the home setting (ie, instructions). Many community based rehabilitation programs go beyond administrative home care and extend their services to vocational and avocational activities. The successful programs to date have utilized a model of physician directed transdisciplinary care, with a sensitivity to the general preponderance of the individual's nonneuromedical needs. General healthcare trends suggest that such programs, from both a clinical and cost efficacy standpoint, may usurp many of the present modalities of providing rehabilitation care.[158]

Vocational Rehabilitation

An ultimate measure of how successful rehabilitative efforts have been is how well survivors of TBI are able to reintegrate into the work place. Numerous methodologies are being utilized to facilitate work reentry, including vocational retraining, supported employment, sheltered workshops, and work hardening.[159] The combination of early intervention and follow-up tends to maximize results of vocational reentry efforts; however, it does not guarantee success. Vocational reentry efforts must focus on the functional strengths of the person with TBI, and must also be keenly sensitive to areas of functional deficit. It is not yet known which methodology works best for patient population subtypes relative to preinjury job characteristics and education, postinjury sequelae, or sociocultural background, but to achieve a maximal level of survivor interdependency, both in and outside the work place, skills that are applicable in the "real world" must be taught.[160]

Trends in Service Provision: Case Management

Case management is probably one of the strongest movements in the field of brain injury rehabilitation. In the best of worlds, case management can provide TBI patients a safety net in several ways: (*a*) the case manager can assist in securing needed services while at the same time assuring that every dollar spent is used judiciously; (*b*) case management can provide a "common-thread" that spans the network of services each survivor must negotiate; and (*c*) when properly applied, timely institution of case management services, for patient and

family, produces better outcomes of neurologic and functional morbidity and lowers overall costs.[161]

Community Based Living Alternatives

For survivors of TBI who have significant cognitive and behavioral, or physical limitations, the options for community based living are typically quite limited. Just as a continuum exists for general service provision, so does one exist for community based living. The most restrictive settings are usually institutional and involve 24-hour supervised living environments and structured daily therapeutic intervention. As the environment becomes less restrictive, based on client ability to function more safely and independently, the level of supervision, as well as the intensity of that supervision, typically decreases. Additionally, clients tend to spend less time institutionalized and more time "in community" with concomitant increases in personal freedoms and free choices.[162] One area in the service continuum for community based living that needs more attention is that of accessible and affordable housing for cognitively and physically challenged TBI survivors.[163] As comprehension of how to maximize client interdependency and simultaneously protect civil rights and other constitutional freedoms improve, the continuum of community based services will ultimately develop and grow.

For a continuum of neuromedical and rehabilitation care to be truly effective, there must be a multifaceted approach that involves preventative education, as well as rehabilitation; and that develops institutional and community based TBI services, maximizes communication across medical disciplines, and promotes better integration of rehabilitation professionals in the long-term management of individuals with TBI. To broaden service access, there is also a need to network community providers who are inside and outside the immediate field of rehabilitation. Lastly, healthcare providers and clinicians must be willing and committed to examining the efficacy of rehabilitation interventions. This is the only way to fully maximize the neurologic and functional outcome for TBI patients, and to also optimize their reintegration into society.[164,165]

CURRENT ISSUES IN THE REHABILITATION OF THE SOLDIER WITH TRAUMATIC BRAIN INJURY

Role of the Physiatrist

A physiatrist is a practitioner of PMR, a recognized medical specialty established in 1947. It is concerned with (*a*) the optimal functional restoration of patients with disabilities; (*b*) physical treatment of neuromuscular impairments; and (*c*) the use of electrodiagnostic studies, including electromyography, and evoked potentials. The more familiar medical specialties, such as internal medicine, orthopedic surgery, and neurology, address the diagnosis and treatment of specific diseases or conditions that result in disability. By comparison, a physiatrist focuses on the diagnostic, therapeutic, and management procedures that will potentially enhance an individual's residual functional capabilities. As opposed to other medical specialties, the emphasis, both in terms of residency training and eventual clinical practice, is on the maximal physical functional capacity and psychosocial adjustment of the physically challenged individual.

Physiatrists approach the patient from a holistic view and address not only the rehabilitative and neuromedical issues, but also lead the interdisciplinary therapy team in the long-term care of the disabled individual. Physiatry, clinically rooted in basic sciences that include anatomy, kinesiology, exercise and muscle physiology, nerve physiology, and biomechanics, also has a clinical suprastructure that combines elements of internal medicine, neurology, neurosurgery, cardiology, rheumatology, orthopedics, pediatrics, geriatrics, and the behavioral sciences. This broad foundation of scientific and clinical knowledge particularly qualifies the physiatrist to evaluate and treat the complicated problems of individuals with disability, and to manage interdisciplinary and transdisciplinary teams.[153]

Mild Traumatic Brain Injury

Mild TBI, although quite prevalent, is still poorly understood by most clinicians, and thereby promulgates many of the fallacies and foibles common to this diagnostic label. This discussion is designed to educate professionals and acknowledge the need to diagnose and treat individuals with mild TBI appropriately from as early postinjury as possible.

Mild TBI accounts for approximately 80% of all traumatic brain injuries.[166,167] It typically results from motor vehicle accidents and involves young males 15 to 24 years of age.[166,168] The role of alcohol use in injuries resulting in mild TBI seems to be significant and that fact should not be ignored in the development of accident prevention programs.[168,169] Many individuals who incur mild TBI do not seek medical attention in acute hospital settings, if at all,

thereby causing an underestimation of the true incidence of this phenomenon.

Given the incidence of this condition, it is crucial for practitioners to be familiar with the diagnosis and treatment of problems that individuals with mild TBI may present. Postconcussive sequelae may have an adverse impact on an individual's ability to function well in a number of different spheres, including physical, emotional, social, marital, vocational, and avocational.[170] The professional healthcare provider must also be aware of resources within the immediate community that provide neuromedical and nonmedical services for this special population of patients. The BIA is an excellent source of information on TBI, mild and otherwise, for professionals, "survivors," and families alike. Typically, each state has its own affiliate association of the BIA.

Mild TBI is defined as a traumatically induced physiological disruption of cerebral function as manifested by at least one of the following: (*a*) loss of consciousness of no longer than 20 minutes; (*b*) any loss of memory, either retrograde (memory loss for events prior to concussive injury) or anterograde (memory loss occurring after the injury and reflecting a time between injury and the point at which continuous memory is regained); (*c*) any alteration in mental status at the time of the accident, even in the absence of loss of consciousness or amnesia; (*d*) physical symptoms that are potentially brain related (eg, nausea, headache, dizziness, tinnitus, visual aberrations, olfactory deficits, or extended periods of fatigue); and (*e*) development of posttraumatic cognitive deficits that cannot be completely accounted for by emotional factors. Given these factors, TBI severity must not exceed the following in order to qualify as mild: (*a*) GCS score of 13 to 15 without subsequent worsening; (*b*) posttraumatic amnesia of 24 hours or less; and (*c*) loss of consciousness lasting no longer than 30 minutes.[171] Recently, several investigators have questioned the inclusion of individuals with intracranial lesions under the mild diagnosis, even if they meet the diagnostic criteria. This call for reconsideration of the present classification is due to the apparent higher incidence of significant neurobehavioral sequelae and resultant functional disability that occur in this subpopulation.[172]

It is critical to recognize that loss of consciousness is not essential for a diagnosis of TBI. Concussive injuries can occur without loss of consciousness, a so-called mild concussion. Recent evidence[172] also suggests that the severity of associated neuropsychological deficits is independent of the neurological status immediately following the injury. It should be noted here that even seemingly sound scientific studies, which have attempted to address such issues, have weaknesses and faults that open them up to further criticism.[173]

Concussive injuries occur along a continuum from mild to classic, the latter involving loss of consciousness. Direct impact to the skull is not necessary to incur TBI. The pathophysiologic hallmark of concussive brain injuries is strain, which can occur secondary to acceleration forces on the head, a stretched cervical spine, and skull distortion due to pressure gradients. Strain disrupts axonal function along a range from physiological disruption, due to transient alterations in membrane function, to actual pathological changes secondary to direct axonal injury.[174] Although the magnitude of strain may vary relative to anatomic variations, the force vectors are normally directed centripetally from the brain's center of gravity, which is approximately in the area of the pineal gland. Due to the centripetal nature of the force vectors, the cerebral cortex and lower brain stem are affected first, followed by the upper brain stem. Many of the transient physiologic responses seen in experimental models of brain injury can help explain the clinical picture of transient coma, pupillary and corneal areflexia, and decerebration seen with upper brain stem dysfunction.[174]

Rehabilitative evaluation of TBI patients should include a thorough history and a physical examination. Historical information should include accident circumstances, alteration in consciousness (dazed vs true loss of consciousness), presence and duration of retrograde (preevent) and anterograde (postevent) amnesia, blood alcohol level and drug screen (if available), as well as initial GSC score. Other significant information pertaining to the initial evaluation includes neurological status and any diagnostic data such as brain CT or MRI, cervical spine or skull films, or both. Pertinent preinjury information should be elicited, including (*a*) prior psychologic or psychiatric problems, or both; (*b*) history of learning disability; (*c*) prior substance abuse; (*d*) criminal record; and (*e*) any history of prior loss of consciousness or TBI. All these factors may adversely effect neurologic and psychologic recovery.[175] Due to the array of injuries and symptoms that many postconcussive patients may have incurred, it is imperative to consider central neurologic dysfunction as well as the peripheral injuries related to cervical hyperextension-hyperflexion injury (whiplash) and cranial and cranial adnexal trauma. Common postconcussive disorder symptoms are

- visual dysfunction: blurry and double vision;
- audiologic dysfunction: tinnitus, high frequency hearing loss;
- vestibular dysfunction: dizziness secondary to peripheral vestibular dysfunction;
- olfactory dysfunction;
- balance dysfunction;
- cognitive-behavioral alterations;
- sleep–wake cycle dysfunction; and
- fatigue.

The physical examination should include a thorough neurological evaluation, including a test of higher level cognitive and linguistic function. It is critical for the physician to be familiar with the associated musculoskeletal and peripheral neurologic (both somatic and autonomic) sequelae of head trauma, as well as cervical spine flexion and extension injuries. Adequate and timely recognition of myofascial pain disorders related to traumatic injury of the neck or cranium is critical due to the array of symptoms (both somatic and autonomic) that can be related to referred pain from trigger points in the facial, cranial, cervical, and upper back musculature.[176]

For proper diagnosis and treatment of injury related problems, it is critical to differentiate head trauma sequelae from true brain injury sequelae. Many postconcussive symptoms that are purported to be secondary to brain injury may actually be head injury sequelae. Head injury sequelae that may be mistaken for TBI-related problems include certain visual disturbances, olfactory and gustatory deficits, audiovestibular deficits, headaches, and peripheral nerve dysfunction (both somatic and sympathetic).[175,177] Postconcussive symptoms due to brain injury may include various types of visual sequelae; audiovestibular, olfactory, and gustatory deficits; headaches; imbalance; excessive daytime somnolence and fatigue; and sexual dysfunction.[178] Maximization of functional potential and expeditious recovery necessitates the appropriate use of pharmacologic agents to treat postconcussive problems of organic affective disorders, sleep–wake cycle disturbances, fatigue, and decreased libido.[170]

As necessary, patients should also be referred for therapy services. Physical therapy referrals should mainly focus on myofascial concurrents of injury and balance retraining, as well as vestibular habituation training. Occupational therapy services are appropriate if the patient presents with issues germane to higher level organizational difficulties that affect ADL, driving problems, decreased performance at work or school, perceptual difficulties, and functional memory and problem solving difficulties. Speech therapy referrals are appropriate when the patient presents with reading problems, auditory difficulties, verbal and written expression impairments, and pragmatic language deficits. Therapy should preferably be administered in an interdisciplinary, function oriented fashion.[175]

Neuropsychological evaluation is a critical component in the diagnosis and ongoing treatment of persons with mild TBI. This evaluation provides objective evidence of higher level cognitive and behavioral, linguistic, and motor dysfunction, not typically evident on bedside evaluation, and it also provides a basis providing compensatory strategies to both patient and family.[179] Neuropsychological testing also provides potentially critical medicolegal information that can be followed to demonstrate initial and subsequent profiles consistent with those seen after similar injuries. Neurodiagnostic tests may provide useful clinical and medicolegal information in this patient population. These tests utilize static, as well as functional imaging (SPECT and positron emission tomography); electrographic monitoring by EEG (with or without compressed spectral analysis); evoked potentials including, but not limited to, BAER (brain stem auditory evoked response) and cognitive evoked potentials (so-called P-300s); quantitative EEG; electronystagmography with calorics; posturographic evaluation; polysomnography; and olfactory and gustatory evaluation, among others.[3]

Patients and family members should be given educational materials and the clinician should explain the condition, history, and prognosis. A frequent and tragic occurrence in clinical practice is the patient with true postconcussive deficits who has gone from doctor to doctor only to be told there is nothing wrong, to the point where the patient actually thinks he is "losing his mind." The patient and family *must* understand what has happened to him, why he feels and behaves the way he does, what the prognosis is, and what can be done about the condition.[3]

In the early period after mild TBI, the patient should follow the directions of the emergency room physician. For the first few days after a more significant injury, the patient should be told to rest, avoid alcohol and caffeinated beverages, keep a regular schedule, avoid recreational drugs, not overdo, avoid distractions, and return to a daily routine gradually. If symptoms continue for more than a week, the patient should consider consulta-

tion with a brain injury physician who is experienced with postconcussive patients.

Given the variety of postconcussive symptoms that may exist following mild TBI, it is unlikely that a true postconcussive syndrome exists. Given the potential complexity of such injuries, it is not uncommon for one set of symptoms to go totally undiagnosed or be misdiagnosed. The astute clinician will assess all the aforementioned potential diagnostic factors before developing an integrated holistic treatment plan. Some of the more frequently reported postconcussive symptoms include headache, dizziness, memory problems, weakness and fatigue, nausea, and tinnitus. Based on sound research and the experience of innumerable clinicians, most individuals who incur mild TBI do not have long-term sequelae. A definite subpopulation of patients with mild TBI, however poorly defined, do have persistent, and sometimes, disabling long term somatic and neuropsychological sequelae. It is critical to take into consideration both organic and nonorganic factors that might give cause to protracted periods of disability after otherwise innocuous insults to the brain.

Accessibilty to advanced neurodiagnostic facilities and to subspecialists, such as neurootologists, neuroophthalmologists, and neuropsychiatrists, may be critical in patient diagnosis, treatment, and medicolegal settlement. Preferably, the primary physician should have subspecialty knowledge and sufficient clinical experience in dealing with this patient population to truly optimize patient care. Ideally, a continuum of clinical services, both neuromedical and rehabilitative, should be available for this patient population, including full physical examinations (including a thorough cognitive-behavioral assessment), neurodiagnostic workups as indicated, and functional assessment. Sophisticated clinicians can assist the primary physician with further diagnoses (Table 6-2) and treatment interventions aimed at maximizing functional status and community reentry. Additionally, such tests can provide additional corroboration for relevant medicolegal purposes. Appropriate use of pharmacologic interventions can also contribute significantly in the treatment of this patient population (Table 6-3). Generally, the practice is to not make final neuromedical or functional prognoses

TABLE 6-2

NEURODIAGNOSTIC EVALUATION OF POSTCONCUSSIONAL DISORDERS

Posttraumatic Sequela	Neurodiagnostic Procedure
Vestibular dysfunction—peripheral and central	Electronystagmography with calorics
Eye movement disorders	Electrooculography
Olfactory and gustatory deficits	Chemosensory evaluation
Perilymphatic fistula	Posturography ?
Sensorineural and conductive hearing loss	Audiologic evaluation
Neuralgic scalp pain	Diagnostic block with lidocaine
Structural parenchymal abnormalities	Computed tomography and/or magnetic resonance imaging
Cerebral blood perfusion abnormalities	Single photon emission computed tomography*
Cerebral metabolic abnormalities	Positron emission tomography*
Electroencephalographic abnormalities	Sleep deprived electroencephalogram or variant thereof, BEAM (brain electrical activity mapping)* ?
Attentional deficits	Cognitive evoked potential (P-300)* ?
Balance dysfunction	Posturographic evaluation
Sleep disturbance	Polysomnography
Erectile dysfunction	Nocturnal penile tumescence (NPT) monitoring
Comprehension deficits	Central auditory processing

*Still in research phase
?Questionable efficacy

TABLE 6-3

PHARMACOLOGIC INTERVENTIONS FOR COMMON POSTTRAUMATIC SEQUELAE

COMMON POSTTRAUMATIC SEQUELA	PHARMACOLOGIC INTERVENTION
Anxiety	Serotonergic agonist Buspirone, sertraline, trazodone, and fluoxetine ?
Basilar artery migraine (BAM)	Antimigraine regimens Psychotropic anticonvulsants
Depression	Tricyclic antidepressants (TCAs) Newer generation serotonergics Monoamine oxidase inhibitors (MAOIs) Lithium carbonate Carbamazepine
Emotional lability and/or irritability	Serotonergic agonists Psychotropic anticonvulsants TCAs Lithium carbonate
Libidinal alteration	
Decreased	Noradrenergic agonists, hormone replacement if low to borderline low
Increased	Serotonergic agonist, hormonal treatment—cyproterone or medroxy-progesterone acetate
Myofascial pain/dysfunction	Nonsteroidal antiinflammatory agents (NSAIDs) TCAs and other antidepressant type medications Mild muscle relaxants
Neuralgic pain	Capsaicin TCAs and related compounds Carbamazepine and other anticonvulsants NSAIDs Local anesthetic blockade
Posttraumatic stress disorder	Antidepressant medications Psychotropic anticonvulsants Propranolol Clonidine MAOIs Lithium Benzodiazepines
Sleep initiation problems	Serotonergic agonists—trazodone
Sleep maintenance problems	Catecholaminergic agonists—nortriptyline
Tinnitus	Gingko biloba ? Tocainide ?
Vascular headache	Antimigraine regimens used in the following protocols Symptomatic Abortive Prophylactic
Fatigue	Catecholaminergic agonists Methylphenidate Caffeine Amantadine
Cognitive dysfunction	Nootropes Catecholaminergic agonists Cholinergic agonists and/or precursors Neuropeptides ? Vasoactive agents ?

?: Questionable efficacy

until at least 18 to 24 months postinjury, due to the anticipated neurologic recovery curve. During the recovery period from postconcussive disorders, judgments should be made by the treating physician and rehabilitation team to determine if the soldier should be placed in a supervised setting or one that does not require full work capacity skills. Work reentry should occur in a structured, monitored fashion to minimize risks of frustration and failure, and to optimize long-term reentry success (see the section on Vocational Reentry for further discussion).

Healthcare professionals should remain aware that secondary gain, malingering, chronic pain, and preinjury psychologic and psychiatric disorders may have an impact on subjective symptoms or the recovery course. If there is opportunity for financial or other conscious secondary gains, the issue of malingering should always be kept in mind.[180] The majority of symptomatic patients, however, are *not* malingerers and do have some sort of legitimate injury, although it may not necessarily relate to TBI. Prior to labeling anyone with a diagnosis that may have a negative impact on functional status and community reintegration, careful scrutiny must be made of all cases to determine any contributing factors, conscious or otherwise, as well as the methodical neuromedical procedures.

Early identification of patients with mild TBI is critical in assuring timely and adequate identification of treatable sequelae. The evaluation and treatment of this patient population must be holistic in nature, ideally utilizing an interdisciplinary rehabilitative model and, as appropriate, a multidisciplinary neuromedical model. Rehabilitative efforts should focus on neuropsychological assessment; physical therapy for vestibular habituation, balance retraining, and myofascial dysfunction; occupational therapy for provision of compensatory strategies for functional cognitive deficits; and speech language pathology for cognitive-linguistic and pragmatic deficits. Appropriate neuromedical diagnosis and treatment, reassurance, education, support, counseling, and regular monitoring are also essential components that will optimize the expediency and quality of overall functional recovery.[170,178]

Low Level Neurologic States: Terminology

One of the most confusing issues for families as well as many professionals is the language used to describe the condition of an individual after TBI. The word *coma* simply conveys that the patient is neither alert nor aware. The comatose patient remains unconscious and "asleep." Typically, there is absence of vegetative functions (such as sleep–wake cycles) during coma. From a neurologic standpoint, coma can be fleeting or prolonged, but generally lasts no longer than 3 to 4 weeks.[181] Most patients with very short durations of coma (ie, seconds) will generally not suffer any significant degree of long-term disability or impairment; however, a small percentage do have temporary and sometimes even permanent problems. Generally, the longer the period of coma the more extensive the associated brain damage. Typically, longer periods of coma are correlated with more extensive diffuse axonal injury. Once comatose, patients will take one of three possible courses: (1) they will regain some level of consciousness; (2) they will die; or (3) they will emerge into a vegetative state. More than 50% of individuals with severe brain injury will die, regardless of the quality of care rendered during the acute period. The majority of those who survive will eventually emerge to some level of consciousness. Patients who emerge from coma into vegetative state may remain vegetative for a very short period of time or may remain in this state permanently, the so-called "permanent vegetative state."[2]

The term *vegetative state* is a confusing one and commonly misunderstood. It is not meant to imply that the person has become a "vegetable," instead it refers to neurologic changes, such as a return of sleep–wake cycles and progression from a state of nonarousal to arousal. Very few patients with severe brain injury (approximately 1%–3%) will remain permanently in a vegetative state.[182] Permanent vegetative state is a prognostic term, not a diagnostic one, and should, therefore, only be used when it is quite clear that the patient will permanently remain in this state.[183] The length of time someone has been in a vegetative state should always be specifically qualified. When there has been significant hypoxic brain injury (a secondary form of brain injury), the prognosis may be more clear cut earlier on in the patient's course, but even in these cases the permanent vegetative state label should not be given until at least 3 months postinjury.[182,183]

Recent guideline development has resulted in further evolution of nomenclature and practice guidelines for management of persons with severe alterations in consciousness. One example of this has been the recommendation to dispense with the use of the phrase "persistent vegetative state" because it adds nothing to the diagnostic accuracy of the vegetative state. Additionally, the assignation, at 1 month postinjury, of the term "persistent" is totally arbitrary. The word "permanent" should be

assigned only after 12 months have passed in a vegetative state following trauma or 3 months following hypoxic ischemic brain injury. It must be realized nonetheless, that even in such cases, the statistical odds of emergence from vegetative state do not reach 100%, although they certainly are nearing this level. The Aspen Workgroup, which has met over the last few years, has also identified a further subgroup of patients who are at the very impaired end of the severe disability category, and although not vegetative, demonstrate intermittent signs of awareness. The suggested terminology for describing this previously not–well-studied or identified subgroup of patients is the "minimally conscious state."[182]

Social Concerns

Significant ethical and legal issues surround the care and potential withdrawal of care from patients in the persistent vegetative state.[184,185] Decisions regarding withdrawal of medical support, whether artificial breathing machines, nutrition, or medications, should probably not be made until at least 2 years postinjury. In actuality, if someone is truly vegetative at 1 year postinjury, that patient is *extremely* unlikely to regain consciousness, although such cases have been reported. Clinicians who deal with this patient population must familiarize themselves with the position papers that have been published on this topic.[182,183,186,187] Clinical care and ethical issues are distinctly different for individuals with profound and irreversible paralysis who have retained consciousness and cognition, such as in locked-in syndrome. Ethically, legally, and medically if these patients are medically judged to have the capacity to make such a decision, they have the inalienable right to forego life-sustaining therapy.[187]

Stimulation Programs

In this era of high technology and aggressive medical care, coma stimulation programs seem to have taken a foothold as an integral part of most continuums of rehabilitative care for patients with severe brain injury, regardless of the specific etiology. It is disconcerting, therefore, to learn that the content of coma situation programs is quite variable. Additionally, little, if any, methodologically sound literature supports the efficacy of such programs in terms of altering the course of neurologic recovery either with regard to the maximization of final neurologic outcome or an increased rate of recovery.[2]

There are major issues regarding how coma stimulation may have a beneficial effect on a brain that is "unaware" of its environment. Pathologic[181] studies of patients who have died while in a coma or vegetative state show that there is no consistent pathology associated with these conditions. In the early descriptions of the persistent vegetative state, it was theorized that significant brain stem involvement was a requisite neuropathologic finding; however, subsequent studies[188] have shown a variety of neuropathologic findings that vary from extremely diffuse gliosis to relatively grossly normal appearing brains. More importantly, experimental data on cerebral glucose metabolism in patients in the vegetative state might lead to questions of how, if at all, any external stimulation can effect any positive change in this patient population.[189,190] Care should be taken when applying these results to the TBI population at large because most of the patients studied had suffered significant hypoxic brain injury; however, data still *suggest* that patients who are truly vegetative from a neurobehavioral standpoint do *not* have the dynamic physiologic cerebral function to support *any* type of cognitive processes.

A most important issue to address in assessing the efficacy of coma stimulation is the standardization of terminology across all disciplines. The lack of a common neurologic and neurobehavioral terminology, such as coma, vegetative state, and persistent vegetative state, is a major obstacle presently facing all professionals who treat individuals with severe brain injury. Additionally, there is a great need for standardization of assessment tools for low-level patients, whether they are comatose, vegetative, or severely disabled. At the present time, the ongoing development of assessment tools by many institutions does not appear to be a constructive process because of the nonuniformity of data that are subsequently generated, and the inability to compare treatment design and efficacy data across centers.

Most coma stimulation is not *coma* stimulation at all, but rather vegetative or low-level stimulation. Therefore, it is probably appropriate, both from a standpoint of proper terminology and, ultimately, from a standpoint of reimbursement, to dispense with the phrase *coma stimulation*. Additionally, labeling a program as offering coma stimulation suggests that this is the sole or major component of such a program; such an implication is a disservice to the third party payers, the families, and most important, the patients. Appropriate management of the low-level patient, whether comatose or vegetative, should be comprehensive in nature with sen-

sory stimulation (rather than coma stimulation) merely being *one* component of such a program.

It is important to differentiate between environmental and structured sensory stimulation from both clinical and research perspectives. Environmental stimulation simply implies that the patient is subjected to ongoing environmental stimuli of various types, including sights, sounds, textures, and so forth. Due to the fact that these are environmental stimuli, they are not presented in any type of structured or systematic fashion, such as music in the room, a picture mobile, or the smells of food or perfume on a loved one. Structured sensory stimulation implies that stimuli are presented in a labor intensive, systematic fashion, whereby the patient is subjected to multisensory stimulation, which typically includes tactile, visual, vestibular, auditory, olfactory, and gustatory sensations. It may be surprising to some proponents of environmental and sensory stimulation programs that there are presently no well-controlled, prospective, statistically significant, population based studies to indicate that such programs actually alter the recovery course or final neurologic outcome of patients who are comatose or vegetative. Most of the studies that are used by proponents of stimulation programs are flawed by multiple factors, including small sample size, retrospective nature, lack of control groups, lack of correlation to functional status and recovery, poor descriptions of possible neuromedical factors (ie, hypoxic ischemic brain injury), lack of scientific peer review, or inadequate information regarding control group selection criteria, or all of the above.[2,191]

There are two recent experimental studies[192,193] that are much more methodologically sound, although they still lack a significant sample population size or adequate control groups, or both. Neither of these studies showed any significant beneficial effect from sensory stimulation. It should be noted that there are animal studies, particularly rodent models, that do indicate a beneficial effect of enriched environments relative to sensory deprivation on recovery processes after experimentally induced brain injury. However, none of the animal protocols utilized vegetative or comatose subjects. Additionally, the relationship of lower animal nervous system recovery to human recovery in persistent vegetative state requires a quantum leap in phylogenic physiologic assumptions.[194]

Given what is presently known about individuals in coma and vegetative states, from both a neuromedical and rehabilitative standpoint, it would seem that present knowledge and literature, when taken as a whole, do not strongly support the utility of structured sensory stimulation as a means of altering the course of neurologic recovery after severe brain injury. Nonetheless, various professionals have *theorized* that sensory stimulation, although not empirically validated, might help structure the patient's interaction with the environment, monitor the patient, and lastly, increase the input into the reticular activating system.[195]

Program descriptions, such as coma stimulation and sensory stimulation, should be abolished and more appropriate phraseology such as early recovery management programs (ERMPs) or "medically complex care." Appropriate interdisciplinary management of this patient population involves the issues of potential morbidity prevention and provides appropriate neuromedical and rehabilitative interventions to maximize neurologic and functional outcome. An ERMP program should focus on several main goals, including maintenance of range of motion, prevention of complications associated with immobility, prescription of appropriate orthotics for preventive and corrective positioning, prescription of seating systems for transport and mobilization, and treatment of neuromedical conditions that could potentially compromise ongoing neurologic recovery and are germane to low level neurologic states. For those who wish to read further on this topic, the management of this patient population has been delineated in several publications.[194–196]

Coma stimulation programs, per se, should not be the sole means by which patients undergo frequent and close reevaluation. A rhetorical argument could be that the rationale of coma stimulation, (ie, stimulating patients to "wake up" faster than they would without stimulatory intervention) may actually be detrimental to the organism. Specifically, what if coma and the vegetative state are normal evolutionary responses to severe brain injury? Would not the more severely injured brain need more time to recover than the less severely injured one? In this sense, trying to rush the process might actually be detrimental to the long-term viability and recovery of the injured organism. Given that there is presently little or no solid evidence that structured sensory stimulation is harmful, there is still great controversy and debate over whether it is even efficacious, or if this aspect of clinical service should be maintained in any ERMP. Theories aside, the current evidence strongly suggests that rehabilitation professionals need to more closely scrutinize the role of any stimulation program in the greater context of the rehabilitative care of these patients. If sensory stimulation is offered, it should

be done in a cost efficient, ethical, and responsible fashion and not as the major component of the total program. For those patients who remain vegetative beyond 1 year and become permanently vegetative, it is best to take a step back and ask the question, "when is enough, enough?" Based on present knowledge about recovery from prolonged vegetative state, it would seem reasonable to withdraw *intensive* rehabilitation efforts, including coma stimulation, after 1 year. A full neuromedical workup must be performed prior to labeling any patient as persistently vegetative. All patients should receive ongoing monitoring for *any* change in neurologic or functional status before *and* after the 1 year mark. If and when changes occur, decisions should be made regarding whether or not to proceed with more intensive rehabilitation efforts.

The controversies surrounding coma stimulation will continue until more definitive studies are available to answer the questions at hand. Until that time, rehabilitation professionals must strive to maintain better uniformity with regards to nomenclature, treatment strategies, and assessment tools. By doing so, clinicians will be better able to assess the efficacy of their interventions, and will be able to provide families with much needed information, which will ultimately lead to a higher quality of patient care, both ethically and qualitatively.

NEUROBEHAVIORAL OUTCOME, ASSESSMENT, AND INTERVENTION

Following TBI, individuals of all injury severity levels are often confronted with a range of neurobehavioral problems. Cognitive deficits, such as impaired memory and concentration abilities, are commonly noted. Interpersonal difficulties, depression, and diminished self-awareness are also typical after TBI. Comprehensive assessment of neurobehavioral functioning is essential to develop effective rehabilitation plans for military personnel after brain injury. The following section outlines a protocol for conducting neurobehavioral evaluations on military personnel with brain injury. An overview of the evaluation process addresses the development of referral questions, interviewing strategies, test selection and administration, and development of treatment recommendations. These guidelines are preceded by a review of the neurobehavioral outcome literature. Research on cognitive outcome is presented first. Next, an overview of psychosocial sequelae highlights emotional, behavioral, and substance abuse problems after TBI. Finally, a rationale for utilizing a neurobehavioral methodology is presented as a preface to the evaluation protocol and a section on intervention.

Cognitive Outcome Literature

An undisputed consequence of TBI is its effects on cognitive functioning. Residual deficits in memory, processing speed, and other cognitive factors often compromise a patient's ability to resume preinjury activities, including work and self-care.[197,198] Unfortunately, research that delineates the recovery of cognitive functioning after TBI is scarce.[199] Of the existing investigations, few have reported on recovery beyond the first year postinjury and even fewer have explored the course of recovery following an open head injury (eg, penetrating missile wound).

Most of the existing research on recovery comes from Glasgow, Scotland, where Jennett and colleagues studied cognitive outcomes after severe brain injuries.[200,201] The Glasgow researchers found that recovery occurs with, and is characterized by, rapid early improvement, which slows down and levels off in most patients by 6 months. Notably, these studies relied on a global outcome measure, the Glasgow Outcome Scale (GOS), to assess change over time. The GOS is an ordinal scale that assigns patients to one of five broad outcome categories: (1) dead, (2) persistent vegetative state, (3) severe disability, (4) moderate disability, and (5) good outcome.[202] Because the GOS is a global measure, it may not be sensitive to the more subtle cognitive changes beyond the first 6 months.

More recent studies[203] challenge the assumption that cognitive recovery halts at 6 months postinjury. Using a matched control design, Dikmen and colleagues administered an expanded version of the Halstead-Reitan Neuropsychological Test Battery to moderately and severely injured patients. Patients and control subjects were tested longitudinally at 1, 12, and 24 months postinjury. The investigators concluded that significant improvements occurred beyond the first year. However, recovery in the second year tended to be more specific, depending on the severity of the injury and type of function. In contrast, the first year of recovery was marked by improvements in all functional areas. Dikmen's group also noted that despite signs of recovery, "marked impairment" across a broad spectrum of neuropsychological functions was still present at 2 years postinjury.

An earlier investigation by this same research group[199] proposed a deficit-proportional model to account for differences in recovery curves. Using this statistical model, they concluded that the amount of cognitive recovery is proportional to the initial severity of the deficit. In general, brain injured patients are expected to recover a portion of their initial losses over time. Accordingly, individuals with substantial losses (eg, moderate to severe TBI) show greater amounts of improvement, but also have more residual deficits. In contrast, those with less initial impairment (eg, mild to moderate TBI) show smaller amounts of improvement and fewer residual deficits. According to these authors, the deficit-proportional model affords better prediction of cognitive outcome than do other models that assume a constant change over time, regardless of injury severity. In turn, the deficit-proportional model highlights the predictive power of initial severity indexes, such as duration of coma, in forecasting cognitive recovery.

Psychosocial Outcome Literature

Emotional and Behavioral Sequelae

Unfortunately, the effects of TBI are not typically confined to cognitive functioning.[204] Many patients, including those with minor injuries, undergo undesirable emotional and behavioral changes.[205,206] These changes may persist for many years after the injury and include such problems as increased anger, frustration, and depression.[204,207] Underscoring the significance of these problems is the testimony of relatives who report that personality and behavioral changes cause more problems than cognitive and physical deficits.[205,207,208] Moreover, research suggests that emotional problems often impede a brain injured person's ability to return to work.[97,209] The path of emotional and behavioral sequelae following TBI has been charted through a series of outcome studies typically focused on severe closed head injury.[204,205,207,208,210] One of the earliest investigations took place in Denmark, where Thomsen followed a group of 50 severely injured patients and their families for up to 5 years postinjury.[211] Patients were examined by a neurologist, a neuropsychologist, and a speech pathologist to assess physical and cognitive functioning. Relatives were interviewed in regards to a number of areas, including the patient's emotional and psychological status. During their interviews, 84% of the relatives complained of changes in the patient's character, including irritability, hot temper, loss of spontaneity, emotional lability, and emotional regression. Reportedly, these changes in personality created the greatest problems in daily living. In contrast, relatives reported that motor dysfunction and other physical problems were not problematic.

Ten years after the initial investigation, Thomsen published a follow-up study[207] on 40 patients from the original sample. The follow-up included an expanded version of the original relatives' questionnaire, which was administered up to 15 years after the patient's injury. Thomsen found that the majority of patients remained unchanged since her initial investigation 10 years prior; moreover, the relatives again reported that the most salient changes were psychosocial in nature. Summarizing her findings, Thomsen[207] wrote that "though physical impairment, dysarthria, and defects of memory remained severe in many cases, the psychosocial sequelae presented the most serious problems." Thomsen further noted that two thirds of the patients in her study underwent "permanent changes in personality and emotion." Unfortunately, most patients lacked awareness of the changes in their character, making it difficult for them to modify their behavior.

Thomsen's conclusions are corroborated by a number of other studies on psychosocial outcome after TBI.[204,205,208,210,212] In an early outcome study from Scotland, McKinlay and colleagues[212] assessed a group of severely brain injured patients at 3, 6, and 12 months postinjury. Using relatives' reports, the researchers charted the course of emotional, cognitive, and physical outcome during the first year after injury. At 3 months, the most commonly endorsed problem was related to cognitive functioning; specifically, 86% of relatives stated that the patient had processing speed deficits. In comparison, emotional problems, such as mood changes and social withdrawal, were infrequently endorsed. At 6 months, slow processing speed was again the most commonly endorsed problem (69%), followed by "bad temper" (56%). At 1 year postinjury, the most frequently endorsed problems were bad temper and cognitive slowness, both at 67%. In summary, problems related to cognitive and physical functioning tended to decrease, whereas emotional and behavioral problems tended to become more frequent during the first year after injury.

This same group of Scottish researchers continued to explore the course of psychosocial recovery from severe TBI in a series of outcome studies. In 1987, they reported on 134 patients who were within 7 years postinjury.[205] Patients and some close relative were questioned about an assortment of areas,

including physical, communication, emotional, behavioral, and cognitive problems. Patients were reported to have altered personalities, evidenced by increased irritability, anger, depression, and mood swings. A disturbing number of patients threatened violence (47%) or acted violently (26%) toward a relative. Other commonly reported problems included disturbances in memory and concentration. Patients and relatives agreed that physical complications caused relatively few problems.

In a comparable study,[204] a group of British researchers reported on psychosocial adjustment 7 years after severe traumatic brain injury. Their findings were remarkably consistent with those from the Danish and Scottish investigations. Relatives reported that behavioral, personality, and memory difficulties interfered the most with long-term adjustment after brain injury. Oddy's[204] group also found a pervasive pattern of social isolation among patients and their families. Only half of those surveyed had even "limited contact" with friends. Other studies[208,210] confirm that social isolation is a major barrier following TBI. Socialization difficulties are often attributed to negative changes in the patient's personality.[208]

A clear pattern emerges from these international outcome studies. First, research indicates that the course of recovery and adjustment following brain injury varies over time. In the period immediately following the injury, the patient's physical and cognitive status are of primary concern. Just as these problems begin to subside after the first year, personality and behavioral problems become more noticeable. Many patients reportedly exhibit aggressiveness, increased irritability, mood swings, and depression. Unfortunately, lack of awareness about their personality changes contributes to interpersonal difficulties, and ultimately, social isolation.

The issue of emotional adjustment following mild TBI has thus far been understudied. However, preliminary research[206] suggests that persons with mild brain injury report more emotional problems following their injury than do severely injured persons. Leininger and colleagues[206] administered the Minnesota Multiphasic Personality Inventory[213] (MMPI) as a measure of emotional and personality function to groups of mildly and severely injured outpatients. The authors found that the emotional profile of individuals with mild TBI was indicative of greater subjective distress than was the profile of severely injured persons. Specifically, individuals with mild TBI showed significantly higher elevations on clinical numbers scale 1, 2, 3, 7, and 8. Individuals with similar profiles were highly distressed and reported feelings of depression, social isolation, and somatic concerns (Table 6-4). Because these results were based on responses within the first year postinjury, more research is needed to delineate the long term emotional sequelae of persons with mild TBI.

TABLE 6-4

EMOTIONAL PROFILES OF PERSONS WITH MILD VS SEVERE TRAUMATIC BRAIN INJURY

Level of severity	Symptoms
Mild	Somatic concerns, clinical depression, anxiety, feelings of social inadequacy, interpersonal alienation, difficulty concentrating and/or confusion
Severe	Clinical depression, interpersonal alienation, difficulty concentrating, confusion

Substance Abuse

The widespread use and abuse of alcohol in American society is a well documented phenomenon. Analysis of national survey data indicates that more than two thirds of all Americans have at least an occasional drink.[214] Highest drinking rates are seen among young adult males, 19% of whom meet the criteria for classification as heavy drinkers. The rate of alcohol abuse among Americans is staggering. A report[215] published by the National Academy of Sciences revealed that 10 to 13 million American adults either abuse or are dependent on alcohol. Other investigations[216,217] have concluded that as many as 12% of American drinkers consume excessive and potentially lethal amounts of alcohol. While these statistics reflect the population at large, it is reasonable to assume that problems with alcohol abuse generalize to a significant portion of military personnel.

From the clinician's point of view, the many issues regarding the use and misuse of alcohol by individuals after TBI tend to be extremely problematic. Some of the alcohol-related issues that professionals must deal with include fears about potential lowering of seizure threshold, alcohol induced cognitive and behavioral changes, neuro-physical side effects, possible suppression of concomitant

neural recovery processes, and interactions with various medications. Probably the most important, yet not necessarily most obvious, factor that can be levied against alcohol consumption after TBI is the greater potential for recurrent brain injury. Of additional interest, although not well studied, is that after significant brain injury a person's CNS becomes more sensitive to the effects of ethanol. Many of these issues are poorly understood by most professionals dealing with this patient population, yet there are reasonable solutions, some possibly surprising, to most of these concerns.

Many clinicians advise their TBI patients not to drink even though they have no real rationale for doing so. It is known that ethanol has multiple effects on CNS neurotransmitter systems, the strongest of which appears to be GABAergic mediated. Recent literature[218] suggests that GABAergic agents may actually impede neurologic recovery following brain injury; this thereby, provides at least one piece of evidence in support of this practice. More research is obviously needed to clarify the potential detriment caused by ethanol during the early neural recovery processes.

Probably one of the most controversial alcohol-related issues facing clinicians is the concern about the lowering of the seizure threshold. It is commonly perceived that individuals with documented seizure disorders will experience problems with seizure control if they use alcohol; however, this is not confirmed by the few experimental studies that have tested this hypothesis. The rare situations where alcohol can act as a convulsant drug, and the mechanisms whereby long term neurotoxic effects of alcohol may lead to chronic epilepsy, have been poorly studied and need further clarification.[219]

The effects of acute alcohol intoxication on the CNS can have a significantly more profound effect on someone who already has preexisting neurologic dysfunction secondary to TBI. Although no neuropathologic changes, per se, have been associated with acute intoxication, alterations in the neuronal membranes, because of the incorporation of alcohol and central neurotransmitter aberrations, have been clearly demonstrated. The reason why individuals with TBI are seemingly more susceptible to the effects of alcohol may be due to posttraumatic alterations in CNS membrane permeability, neurochemical alterations, or some other, unidentified factor.[219]

Acute alcohol intoxication may worsen already existing postinjury behavioral sequelae, including akathisia, aggressiveness, irritability, and disinhibition. From a cognitive perspective, a variety of posttraumatic neuropsychological problems may be further compromised, including staying on task, mental processing speed and flexibility, learning, problem solving, attention, concentration, judgment, and reasoning. From a neurophysical standpoint, acute intoxication adversely affects all motor behaviors, from the simplest to the most complex, including ambulation, speech, eye movements, and postural control. Many TBI patients, who do not drink any alcohol at all can relate at least one incident where they have been stopped by police for suspicion of alcohol intoxication.[219]

Chronic effects of alcohol on the CNS have been well documented. Given the neuropathology associated with TBI (specifically, primary injury due to focal cortical contusion with greatest propensity for frontal and temporal parenchyma, or diffuse axonal injury, or both, as well as frequent cases with some degree of secondary brain injury), it would seem unwise for anyone with a brain injury to consume alcohol on a chronic basis. This word of caution carries more weight given the occurrence, in alcoholics, of significant neuropathologic changes with chronic alcohol consumption, including brain shrinkage, loss of frontal cerebral cortical neurons, and cerebellar degeneration.[219]

Alcohol ingestion may actually be contraindicated in conjunction with various medications, including antidepressants, anxiolytics, benzodiazepines, neuroleptics, anticonvulsants, lithium carbonate, and many others. Induction of hepatic enzymes with chronic alcohol ingestion can alter drug levels of agents hepatically metabolized. Acute alcohol intoxication may cause serum levels of certain drugs to rise secondary to competition for binding sites, and due to its diuretic effect, alcohol can potentially cause lithium toxicity. The added sedative effects of alcohol with many pharmacologic agents must also be taken into consideration because it can adversely affect safety in driving, operation of equipment, and other activities.

Lastly, it is well documented that much of the morbidity and mortality associated with TBI is linked to alcohol consumption. Given this fact, it becomes obvious that the individual who drinks after sustaining a TBI is at higher risk for recurrent injury than one who does not. Accidents with alcohol are typically related to vehicular mishaps, falls, or fights. The aforementioned facts should be sufficient justification for more conservative recommendations, rather than more liberal ones, regarding alcohol use following traumatic brain injury.[219]

Several published investigations[218,220] have revealed troublesome data regarding the relationship

between brain injury and alcohol use. The investigators examined admission blood alcohol levels among persons with TBI and found that many TBI patients had been drinking at the time of injury.[218,220] Galbraith and colleagues[221] completed a prospective study on 918 consecutive civilian brain injury admissions and found that 62% of male and 27% of female patients had positive blood alcohol levels on admission. Rimel and colleagues[218] investigated blood alcohol levels using 199 moderately injured patients (GCS = 9–12) and 538 patients with minor brain injury (GCS = 13–15) from the University of Virginia Hospitals. In screening blood alcohol levels, positive findings emerged for 78% of moderately injured patients and 53% of patients with minor injuries. Of the moderately injured group, 57% had blood alcohol levels greater than or equal to the 100 mg/dL, the maximum legal limit for driving in Virginia and many other states. Furthermore, 28% of those tested had blood alcohol levels in excess of twice the legal limit. Among minor-injury patients who tested positive, 43% exceeded the legal limit for intoxication while 23% had blood alcohol levels equal to or greater than twice the legal limit. These figures, in part, reflect the fact that more than a third of moderately injured and 10% of minor brain injury patients reportedly abused alcohol prior to their injury.

While alcohol clearly contributes to the incidence of traumatic injuries, its use also complicates the rehabilitation process. The use of alcohol can increase the expense of rehabilitation by interfering with physical recovery.[220] This situation is further complicated when alcohol is combined with prescription medications, causing adverse interactions.[222] The likelihood of alcohol interfering with the rehabilitation process is compounded when patients use intoxication as a coping mechanism. Research suggests that patients who suffer physical disabilities related to brain injury are likely to consume alcohol in their effort to subdue emotional distress.[223,224] In spite of these concerns, few investigations have examined patterns of preinjury and postinjury alcohol use among head injured persons.

The potential of alcohol to impede the rehabilitation of head injured patients is alarming and indisputable. Unfortunately, there is a dearth of information regarding alcohol use and disability, and policies for substance abuse among this population are simply absent. Rohe and DePomolo[225] surveyed 52 rehabilitation medicine departments, only a fraction of which screened patients for substance abuse as routine policy and, furthermore, staff members were found to be generally ambivalent regarding the issue. For example, the majority of respondents (90%) supported prohibition of alcohol consumption for inpatients; however, half the sample felt that alcohol should be prescribed to outpatients for desirable reasons, including appetite stimulation and relaxation. The survey further revealed that patient and staff education programs concerning substance abuse were notably deficient. While over half of the programs expressed support for substance abuse education, only 29% actually provided such programs to patients and even fewer were available to the clinical staff. In reviewing these findings, Rohe and DePompolo concluded that the issue of substance abuse has been virtually ignored or unrecognized by rehabilitation professionals in the brain injury field.

Theoretical Rationale for Neurobehavioral Assessment

Historically, researchers have incorporated two assessment methodologies to describe the neurobehavioral consequences of TBI: (1) self-report measures of emotional and behavioral functioning, and (2) neuropsychological assessment. Researchers[218,226] have used questionnaires and interviews to elicit family members' and patients' perceptions regarding the behavioral and emotional effects of injury. Partly because of concerns related to diminished self-awareness arising from injury, most investigators have relied primarily on family members' rather than patients' reports. More recently, rehabilitation professionals have begun to use traditional psychodiagnostic measures, such as the MMPI, to assess the patient's personality functioning.[227]

Neuropsychological assessment procedures have also been used extensively to describe patients' diverse cognitive, intellectual, sensory, motor, linguistic, and perceptual skills.[228] Neuropsychological testing serves a variety of functions and offers an objective description of diverse abilities. Postinjury performance on neuropsychological measures may be compared with estimates of preinjury ability to help determine the consequences of the injury. Comparisons can also be made with normative data available for uninjured persons. Additionally, follow-up evaluations related to recovery, aging, pharmacologic intervention, and cognitive rehabilitation, can help delineate changes over time.[229]

Each assessment methodology is subject to bias and uncertain validity. Psychological denial may cause relatives and patients to minimize difficulties on self-report measures. Patient perceptions of disability may be affected by memory deficits and

impairment of self-awareness that arises from pathophysiological changes. Psychological distress may contribute to exaggerated reports by relatives. Neuropsychological tests are clearly more objective than self-report measures and they also avoid the issue of biased reporting. Unfortunately, several factors contribute to uncertainties regarding the value of neuropsychological assessment. A clear relationship between neuropsychological test scores, ability to perform daily living activities, and patients' and relatives' perceptions of sequelae has not been demonstrated.[230] Furthermore, neuropsychological assessment procedures represent an unusual and highly structured situation. As such, test results may not provide a good representation of characteristic behavior evident in daily living activities.

Efforts to enhance the validity of conclusions regarding neurobehavioral outcome and the complexity of interrelationships among outcome variables have contributed to an increased reliance on a combination of assessment methodologies. Neurobehavioral assessment offers this comprehensive methodology by combining neuropsychological assessment techniques with measures of behavioral and emotional functioning. Consolidation of these methodologies assures that a comprehensive picture of the patient's emotional, behavioral, and cognitive problems is presented.

A Protocol for Neurobehavioral Assessment

This section presents an overview of issues pertaining to neurobehavioral assessment of military personnel following a TBI. The suggested protocol highlights four key elements of the assessment process: (1) developing the referral question, (2) interview and behavioral observations, (3) test selection and administration, and (4) interpretation and reporting. While the information presented herein is intended to benefit any interested reader, administering and interpreting the evaluation requires the supervision of a well-trained professional, typically a clinical neuropsychologist.

Developing the Referral Question

Military personnel with probable TBI will typically be referred for neurobehavioral evaluation by their physicians. Primary objectives of the evaluation include description of the patient's neuropsychological strengths and deficits (eg, intellectual, cognitive, sensory, and psychomotor skills), description of the patient's emotional and behavioral status, and development of a treatment plan. In addition to these broad objectives, explicit referral questions can enhance the value of the examination by guiding the assessment process. Consultation with referral sources is essential to ensure that the lengthy examination process meets the unique needs of each patient and answers the questions that the treating professionals deem most important. Typical referral questions among military personnel will include those that assess the extent of the patient's disability and the probability that he may return to active duty. Examples of other referral questions are presented in Exhibit 6-2.

Interview and Behavior Observations

An initial interview with the patient provides useful data regarding any neurobehavioral problems. The patient's reactions to the injury are of particular concern because depression and other forms of psychological distress often accompany TBI.[231] Clinicians should assess the patient's perception of emotional changes, present coping ability, and level of pessimism. Patients should also be screened for the presence of depressive symptoms, including suicidal ideation or intent. Symptoms of depression after TBI include

- difficulty enjoying activities;
- feelings of worthlessness;
- feelings of hopelessness;
- lack of initiative;
- lack of self-confidence;
- feelings of sadness, being "blue";
- diminished appetite;
- changes in sleep patterns;
- changes in weight; and
- suicidal ideation or intent.

A medical examination can help distinguish between pathophysiological changes that have arisen from injury, and psychological changes. An interview with family members provides another important perspective on the patient's emotional status.

During the initial interview and throughout the evaluation, qualitative information may be gained by observing the patient's behaviors and reactions to the testing process. Examiners may, for example, observe whether the patient is able to recognize errors and, furthermore, if he demonstrates initiative to correct these errors. If present, socially inappropriate behaviors should be noted in the report because such behaviors can negatively impact the

EXHIBIT 6-2

SAMPLE REFERRAL QUESTIONS FOR A NEUROBEHAVIORAL EVALUATION OF MILITARY PERSONNEL

- Describe the patient's cognitive, intellectual, and psychomotor abilities.
  Which areas would you consider relative strengths and which areas would you consider to be weaknesses?
- To what extent are the patient's deficits attributable to his injury?
  What preinjury factors (eg, premorbid educational history, prior brain injury, substance abuse) might be contributing to the patient's impairment?
- What is the patient's present emotional status?
  Is there evidence of depression, feelings of hopelessness, or diminished self-esteem?
  Is the patient a suicide risk?
- Will the patient be able to return to his previous position in the military?
  If so, when might a return to active duty be possible?
  If not, are there other jobs within the military that would be better suited to this individual's abilities?
- Does the patient's injury require him to assume permanent disability status with the military?
- What is the patient's competency to drive, manage finances, operate machinery, and take medications?
  Will the patient require supervision for his personal safety or the safety of others?
- Does the patient evidence any behavioral problems (eg, aggressiveness, socially inappropriate, low frustration tolerance)?
- To what extent will the patient benefit from rehabilitation services, including pharmaceutical, psychotherapeutic, and behavioral management strategies?
- When will the patient require additional evaluation? For what purpose?

vocational and community reintegration process. Information should also be obtained about executive skills, including planning and organization, as well as problem-solving strategies. Rate of information processing and mental flexibility can also be assessed formally and informally during the examination process. This information can be used by vocational rehabilitation professionals to develop structured work environments, and by therapists who provide direct feedback to the patient.

Test Selection and Administration

The neurobehavioral test battery should include a variety of neuropsychological tests that measure cognitive functioning (eg, attention, processing speed, verbal, and auditory memory) and psychological tests that assess emotional and behavioral status. Qualitative and quantitative criteria should be considered when selecting tests for the assessment battery. Quantitative criteria reflect a test's empirical integrity as indicated by indexes of reliability and validity. Qualitative standards refer to a test's ability to address the referral question. For example, if the patient is being evaluated for suspected mild TBI, the test battery should include measures that are sensitive to mild brain injury.[232,233]

When selecting neuropsychological measures for the test battery, it is important to include diverse tests that are indicative of a wide variety of abilities. There are many neuropsychological tests available and clinicians occasionally have difficulty selecting the most appropriate test. As noted earlier, tests must meet reasonable standards of reliability and validity.[234] Recently, the validity of neuropsychological tests has become a central issue among researchers and clinicians in the field. Professionals are particularly interested in the predictive validity of neuropsychological tests. Predictive validity refers to the ability of test scores to predict performance on some criteria behavior, such as return to work. Some neuropsychological tests (such as the Paced Auditory Serial Addition Test and the Wechsler Memory Scale - Logical Memory subtest) have

demonstrated their ability to predict return to work after TBI.[197] Such tests could assist the clinician in addressing questions related to vocational reentry.

Other criteria to consider in selecting neuropsychological tests include the availability of normative data for persons varying in age, education, race, and sex. Normative scores from an appropriate comparison group help to establish the patient's level of functioning relative to his peers. Preference should also be given to neuropsychological tests that have been used extensively with the brain injury population, as evident in the research literature. Clinical neuropsychologists are also encouraged to use tests that have the greatest relevance to real-life functioning. For this reason, standardized measures of spelling, reading accuracy, reading comprehension, and arithmetic reasoning are useful. As a guide for the reader, other sources are available that specify commonly used neuropsychological tests and the corresponding skill areas they assess.[228,235] Assessment of the patient's emotional and behavioral status includes formal and informal methods. Informal methods consist of observing the patient's behavior during the testing process and interviewing the family. Formal methods of assessment incorporate psychological measures of personality and emotional status. Such measures should be used with caution, because most tests have been standardized on psychiatric as opposed to brain injured populations. Nonetheless, some tests can yield useful insights when cautiously interpreted.

Information from psychological tests should be compared with the patient's self-report, report of family members, and clinician's knowledge about the common effects of injury. For example, the Beck Depression Inventory[236,237] can provide useful information regarding self-esteem, levels of pessimism, appetite disturbance, and libido. However, this instrument should be interpreted carefully as it is highly susceptible to a social desirability response style. The MMPI[213] provides some indication of test taking attitudes and social desirability through examination of this test's validity scales. Other valuable information about depressive symptoms, family disturbance, somatic discomforts, and social isolation can be obtained through use of this instrument.[238,239]

Interpretation and Reporting

Clinical neuropsychologists are typically asked to make four important judgments for each cognitive skill area tested. A checklist has been developed to summarize and cogently present the results of these judgments (Exhibit 6-3[240]).

Clinicians also have the responsibility of commenting on the patient's ability to engage in certain activities that may have associated risks. Patients and family members frequently have questions about the patient's ability to drive, operate mechanical and electrical equipment, manage finances, and take medication as prescribed. Interview information about the patient's current re-

EXHIBIT 6-3

NEUROPSYCHOLOGICAL PROFILE: PERFORMANCE AND IMPAIRMENT LEVELS

1. The patient's ability in a given domain is determined by comparing his performance to the normal population. This comparison allows the clinician to estimate how the patient performed relative to persons of similar age, education, and sex.
2. A judgment is made of how the patient's performance has been affected by the injury. Reported sequelae, review of academic and vocational records, estimation of preinjury level of ability, and knowledge of common consequences of injury can help the clinician determine which skills are impaired relative to preinjury status.
3. Clinical neuropsychologists should estimate whether a patient's functioning in each area has improved, remained the same, or declined relative to previous testing. Direct comparisons with prior test scores can help establish whether therapy, subsequent injury or disease, recovery, or medication has affected patient status over time.
4. The neuropsychologist should review a profile of the patient's test scores to determine areas that are relative strengths as well as identify those areas that are clearly weakest in terms of the individual's overall performance.

sponsibilities is integrated with test information to estimate levels of competence in these skill areas. When possible, the opinions of other professionals should be solicited before reaching final determinations. For example, based on neuropsychological testing that reveals visuoperceptual problems, the clinical neuropsychologist may suggest that the patient submit to a formal driving evaluation before resumption of driving.

As noted earlier, interpretation of psychological tests should be done with particular caution as the majority of these tests were developed for use with psychiatric populations. Clinicians using the MMPI should avoid standard clinical interpretation of scale elevations because certain scales are commonly elevated over a T-score of 70 for persons with brain injury (numbers scale 1, 2, 3, 7, 8).[206,241] In these cases, standard interpretive labels, such as *schizophrenia*, would be inappropriate for patients with thought disturbances attributable to a brain injury rather than psychopathology. Clinicians who use the MMPI may derive the most valid conclusions about the emotional and personality functioning of brain injured persons by examination of responses to individual test items, including those designated as "critical items".

Practical recommendations tailored to the individual patient's needs should be included in the neurobehavioral report. In response to the problem areas identified through assessment, clinical neuropsychologists should suggest compensatory strategies and general health guidelines. All patients should, for example, be encouraged to abstain from the use of alcohol or illicit substances. Specific deficits, such as memory impairment, can be managed through the use of compensatory strategies. Numerous TBI patients report memory problems, yet they learn to cope with this handicap by maintaining a personal journal in which they record any information they need to remember. Patients who are depressed or having other adjustment problems should be encouraged to seek counseling or be evaluated for pharmacological intervention, or both. Ultimately, the value of the neurobehavioral evaluation will be seen in the clinician's ability to consolidate test scores, behavioral observations, and interviews into a set of realistic recommendations that will promote the patient's successful reintegration into the community.

Intervention

As detailed earlier, TBI often causes a diversity of emotional, behavioral, and cognitive problems, even among patients with mild injuries. A comprehensive neurobehavioral evaluation is invaluable for the diagnosis of problematic areas, and subsequent development of a treatment agenda. A variety of treatment modalities are appropriate for working with TBI individuals. Selecting the treatment of choice will depend on the needs and goals of the patient. The following section provides a brief overview of several popular treatment modalities: cognitive remediation, psychotherapy, and substance abuse counseling. For a more comprehensive review of this topic, interested readers should consult other resources.[235,242,243]

Cognitive Remediation

Individuals who sustain TBI typically suffer from a number of cognitive deficits, including impaired memory and concentration skills. The negative repercussions of cognitive deficits can be seen in all areas of function, ranging from vocational ability to social skills. For example, many patients are unable to recall news stories minutes after hearing them on television. These patients are then unable to discuss these events with others, leading them to feel socially inadequate.

Over the past two decades, cognitive remediation programs have been developed to treat problems with cognition.[243] Cognitive rehabilitation involves teaching patients strategies that will allow them to compensate for their cognitive deficits. Compensatory strategies range from simple behavioral techniques, such as keeping a written or taped memory log, to the use of mnemonic devices or computers. The efficacy of cognitive rehabilitation programs depends on a number of essential characteristics. These include multimodal training, integration of cognitive and skills training, intervention rooted in theory, and adaptation of training experiences to the real-world setting.[243]

Psychotherapy

Depression, low self-esteem, poor interpersonal relationships, and other emotional disturbances are often seen among persons with brain injury. Interestingly, patients with mild injuries report higher levels of depression than do persons with severe TBI.[206] This phenomena is attributable to the mildly injured patient's potential for greater insight and awareness of their deficits. Thus, awareness of deficits and loss caused by a brain injury may place individuals at greater risk for psychological maladaptation.

Psychotherapy is a well-established treatment modality that is intended to reduce an individual's emotional distress. The application of psychotherapy to the TBI population has only recently been demonstrated scientifically. Patients with brain injury may use the psychotherapeutic process as an opportunity to grieve the loss of their former, uninjured selves. Therapy may also teach brain injured clients effective methods for coping with depression and low self-esteem. Clients may also receive interpersonal training and feedback from their therapists, which may focus on ways to become more effective in social situations. In the context of the therapeutic relationship, the therapist is a source of empathy and support, and provides the soldier with objective guidance, facilitating the adjustment process.

Substance Abuse Counseling

Given the high incidence of preinjury alcohol abuse and the compromising effects alcohol and illicit drugs have on the rehabilitation process, professionals working in the field should closely monitor potential substance abuse problems among individuals with TBI. Interviews with the patient and family may directly address questions regarding the patient's usage patterns pre- and postinjury. If a family history of drug or alcohol problems, or both, is discovered, the patient should automatically be considered at risk. Other at-risk factors to be considered include the patient's sex (male) and age (under 25), job performance (inadequate productivity or excessive absenteeism), and blood alcohol levels at the time of injury.

A variety of measures may be undertaken to increase the likelihood of patient abstinence. Patients and families should certainly be educated about the potential dangers of substance abuse. Physicians and other rehabilitation professionals should communicate to their patients consistent guidelines about abstinence. Family, friends, and clinicians should promote participation in social and recreational activities that do not involve the use of alcohol. Finally, contracts with patients can be used to establish clear expectations, guidelines, and behavioral contingencies. Reward systems may be built into such contracts to reinforce compliant patients.

Because there are few treatment programs specifically designed to treat drug and alcohol problems among disabled individuals, professionals are encouraged to network with existing substance abuse agencies in their community in order to provide services for those in need. Rehabilitation professionals can serve to educate these agencies about specific issues related to TBI. In turn, substance abuse counselors can instruct rehabilitation professionals about substance abuse and treatment while also meeting the intensive treatment needs of disabled patients. Given the dangers of alcohol and illicit drug use and their compromising effects on recovery, substance abuse treatment must be aggressively confronted among TBI patients to protect the integrity of rehabilitation.

VOCATIONAL REENTRY

Over the past decade, data on vocational outcome following TBI have begun to accumulate and the findings are not encouraging.[209,210,244] Admittedly, these outcome studies vary on a number of parameters, including (*a*) different levels of injury severity studied, (*b*) lack of a consistent definition of employment outcome (eg, return to work vs employability), (*c*) inconsistent verification of work status, and (*d*) lack of reliable follow-up over time.[209] Despite such differences, the data inevitably lead to the same conclusion—that TBI often compromises an individual's ability to return to preinjury employment levels. Vocational reentry following TBI is a crucial issue for the military. The following section provides an overview of this issue, beginning with a review of the employment outcome literature. Vocational rehabilitation models are then presented, with an emphasis on supported employment. Criteria for decision making regarding return to active duty are also considered, particularly with regard to mild brain injury.

Employment Outcome Literature

Vocational outcome research consistently indicates that unemployment is a common sequela of brain injury. In an exemplary study, Brooks and colleagues[205] followed 134 severely brain injured persons for up to 7 years and reported that fewer than 30% of the sample were employed at follow-up. When compared to an 86% preinjury employment rate, this finding indicates the prolonged adverse effects of brain injury. Another study[54] on severe TBI reported similarly disconcerting findings. Of 60 patients seen 3.5 years after their injuries, only 13% had returned to preinjury employment levels. The majority of the sample was either unemployed (52%) or employed in positions that were less de-

manding, such as sheltered workshops (35%).

Studies on work outcome for mixed severity groups are equally alarming. Lezak[228] studied persons with mild, moderate, and severe TBI at 3 years postinjury and found that only 15% were employed in situations consonant with their preinjury educational or vocational status. Similarly, McMordie and colleagues followed a mixed severity group for up to 27 years postinjury (mean = 7 years) and found that only 19% were competitively employed.[244] Even studies limited to mild brain injury (ie, no loss of consciousness) conclude that a return to previous levels of employment can be a difficult if not impossible task.[245]

In response to these staggering findings, researchers[205,244,246,247] have begun to identify the primary risk factors associated with employment difficulties after brain injury. Despite variability between studies (eg, differing levels of severity, variable time postinjury), a number of factors were consistently related to poor work outcome. These included the following: (*a*) cognitive difficulties, including memory and processing speed[205,244]; (*b*) longer duration of coma[244,247]; (*c*) age (ie, older patients had poorer outcomes)[244,246,247]; and (*d*) family attitudes.[246] Additionally, many investigators argued that personality and behavioral disturbances (such as impaired interpersonal skills, depression, and limited self-awareness) were the greatest impediments to successful return to work.[205,209]

In a 15-year follow-up, Dresser and associates[248] looked at prognostic factors related to work outcome among 864 U.S. veterans of the Korean War.[248] The sample of veterans had a mean age of 22.7 years at the time of injury. Three quarters of the sample sustained penetrating missile wounds, while the remainder suffered closed head injuries. Although Dresser's diagnostic criteria are unclear, the results indicate that the majority of the soldiers (70%) sustained mild to moderate injuries. In their conclusions, the researchers argued that the longer lengths of posttraumatic amnesia predicted poor vocational outcome. Other predictive factors included aphasia, seizures, motor impairment, and preinjury intellectual status.

Unfortunately, few studies chronicle the long-term effects of brain injury on return to work, making it difficult to validate Dresser's predictions. Nonetheless, the existing studies[205,207] confirm that employment difficulties persist for many years after brain injury. In one of the latest outcome studies, Thomsen[207] reported on 40 severely injured patients up to 15 years postinjury. While the vast majority of patients were employed prior to their injuries, fewer than 8% had resumed full-time employment despite many years of recovery.

In summary, the work outcome literature does not paint an encouraging picture. Findings suggest that regardless of injury severity, persons with brain injury are at a substantial risk for employment difficulties. A number of factors, including cognitive deficits and behavioral and personality disturbances, appear to increase the probability of unemployment. Unfortunately, these problems do not appear to wane with the passage of time, leaving patients and their families to face the financial and emotional burdens of chronic employment problems.

Vocational Rehabilitation: The Supported Employment Model

Military personnel who have sustained a brain injury must be directed down one of three vocational paths: (1) return to the position which they held prior to their injury, (2) placement and training for a different position that better matches their postinjury abilities, or (3) pursuit of permanent disability status. The last option is clearly the most expensive to the military and demotivating to the disabled soldier. The severest of cases, however, will require permanent disability in order to safeguard the injured person and others against potentially dangerous situations.

The ideal choice of returning the soldier to his prior position is appropriate when the injury has not compromised his ability to perform his duties safely and accurately. Making this decision requires a comprehensive assessment of the individual as well as the job. For example, an individual with a mild brain injury would have more difficulty returning to an executive position than to a manual job. Safety should also be a primary consideration as even a mild injury can reduce an individual's ability to make fast decisions or react quickly to a physical threat. Other considerations include the injured person's attitude; many people have difficulty accepting that it takes longer to do things than it did before their injury. Soldiers who do return to prior positions following a brain injury should be encouraged to work slowly and carefully until they again feel comfortable in their positions. This topic is further addressed in the section, Return to Active Duty Following Mild TBI: Decision-Making Criteria.

A third option for the soldier with brain injury is to place and train him in a new position that better matches his abilities and limitations. This approach

to vocational rehabilitation, known as supported employment, is designed specifically for individuals who need support to maintain employment.[249] Supported employment offers direct on-site job training and intervention to address problems associated with physically or mentally challenged employees. While supported employment was initially developed as a community intervention method, its basic components are applicable to the task of vocational reentry among military personnel. The following sections describe the components involved in implementing a supported employment approach, including client assessment, job assessment, job placement, job site training and enabling, and follow-along services.

Client Assessment

The function of client assessment is to identify the individual's interests as well as his strengths and weaknesses so that the most appropriate placement can be made. Individualized assessment may be best accomplished by synthesizing information from a variety of sources including the individuals themselves, family members, neuropsychologists, physiatrists, neurobehavioral assessments, and especially a functional assessment of the individual's skills. Neuropsychological tests can be used to identify specific cognitive deficits—not to limit employment options, but rather to specify areas that may require compensatory strategies. A thorough medical history, including history of substance abuse and treatment for psychiatric disorders, should also be reviewed. Interviewing family members can be particularly helpful since the families know how the individual performs in real-world settings. For instance, family members may have developed compensatory strategies for memory problems or found effective techniques for dealing with behavioral concerns. Other considerations include the presence of physical limitations or other medical conditions that might interfere with successful return to duty. The individual's work history and work interest should also be considered when developing a placement site.

In the supported employment model, a "job coach" is assigned to act as a liaison between the client and the placement site. Ideally, the job coach would have training in vocational rehabilitation, but an employee from the target placement site could also be trained for this position. Most importantly, the job coach should be the client's advocate. The development of rapport between the job coach and employment candidate is a primary factor in effectively implementing the supported employment model. If good rapport is developed in the initial stages of service delivery, it will improve the job coach's ability to solve employment problems that may develop. Failure to develop rapport may result in persistent resentment or power struggles within this all too critical partnership.

Job Assessment and Placement

Prior to job placement, the job assessment phase involves a formal analysis of the skills and responsibilities necessary to perform the task. Each job must be broken down according to functional areas. For example, the job of "combat soldier" would involve a number of functions ranging from "ability to operate an assault rifle" to "possess physical stamina necessary to survive under harsh physical conditions." A careful analysis of the job requirements is necessary to determine which position is most compatible with the individual's strengths and limitations.[250,251] At the point that an individual is determined to be a good candidate for a particular job, the job placement phase is initiated. This phase includes facilitating communication between the individual and his prospective coworkers, immediate supervisors and commander, as well as family members. Conditions of employment, including work schedule and job requirements, are clarified for all concerned so that there is less chance of confusion and errors in the initial employment period. If necessary, the job coach helps make transportation arrangements and provides transportation training to the individual.

For individuals with TBI, the component of job placement should include the broader concept of environment placement. For people who are easily distracted, the seemingly "perfect" job in a stressful or chaotic environment may result in job failure. Having job candidates observe a potential employment site allows them to observe the job first hand and evaluate if they like the job or if they feel comfortable trying the job. After being given skill training or physical adaptions, or both, the job coach and the employment candidate can decide together if the work is acceptable, or if it is more beneficial to consider a different position. The social aspects of dealing with coworkers, the particular employment supervisor, and the amount of socialization required in an employment setting are also key factors in making a successful placement with long-term retention.

Job Site Training and Enabling

Following the clarification of placement arrangements, the primary activity for supported employment is job-site training (enabling). Beginning the first day of employment, the job coach accompanies the new worker to the job site and provides one-to-one training to the degree of intensity needed for the individual. The degree of training intensity expands and contracts based on the individual's progress in assuming the work responsibilities. Training strategies include the use of behavioral and systematic instructional techniques, with heavy reliance on task analytic procedures, production training strategies, and data collection procedures as on-going processes. Other sources[249,252] detail the technology of job-site training within a supported employment approach.

For individuals recovering from TBI, persons with physical disabilities, or long-term mental illness, there are going to be other major activities in addition to training during the job-site enabling component.[251] Individuals with physical impairments in addition to cognitive deficits, will need to make adaptations or modifications to the work setting and equipment (or both) to enhance the individual's ability to perform the work activities. In some cases the job coach may have the expertise to make the needed adaptations. In other instances, the coach will need to act in the role of coordinator to access other resources that can provide the service. An example of this would be to arrange for an occupational therapist to visit the employment setting to confer on the physical positioning of the worker so that fatigue is avoided and maximum efficiency of movement can be achieved. Rehabilitation technologists or engineers are also resources that may need to be accessed to help the individual achieve the ultimate requirements of the job. Another example of service coordination would be accessing psychological counseling services to help the individual work on personal or interpersonal issues that may be interfering with his ability to work independently. Generally speaking, the job coach should provide direct support or access indirect support services as needed to help the individual succeed in the employment situation.

Finally, with regard to the job-site enabling component, the job coach plays a major role as advocate for the new worker with the employer, coworkers, and family members. The job coach will need to negotiate on behalf of the individual worker on any number of issues that may come up as a result of the new work situation. For example, the job coach may negotiate a trade with the individual's coworkers which would involve swapping job duties to match each individual's interests and skills. Advocacy is of major importance in the successful implementation of supported employment, and is an ongoing activity throughout the time an individual is employed.

Job-site training and enabling intervention need to be flexible, according to the level of independence demonstrated by the employee. At times, the level of independence may fluctuate due to disrupted sleep patterns, anxiety, certain medical changes, or cognitive deficits. Specific cognitive deficits can include problems with storage, retrieval, and processing of information. Feedback from the job coach to the employee regarding job concerns should be specific. Written instructions, daily journals, directive feedback, and role playing are strategies that can be individualized and used on job sites when appropriate.

As the soldier learns the work activities and begins to demonstrate competence in independently performing job duties, the job coach's level of involvement gradually diminishes. Ideally, the supported employment model is designed so that the job-site intervention can increase or decrease based on the needs of the individual worker. When the intervention requirements of the individual have stabilized at a low level (eg, less than 10% to 20% of the required work hours per week for a period of 30 to 60 days) the job coach will initiate an ongoing schedule of follow-along support. This model of supervised return to duty would be valuable for recuperating injured soldiers on a temporary disability retirement list. Although these soldiers are not on active duty, if they were to have a trial of active duty during this time, an objective assessment of fitness could be made. The "on-site" experience gained would then help make a determination of "fitness for duty" based on more objective data about duty performance capabilities.

Follow-Along Services

Follow-along services within a supported employment approach means that the job coach maintains regular contact with the employee, his supervisor, and family members throughout the duration of the employment situation. Contacts are scheduled as needed by the employee, including off-job site contacts with family members or the individual worker, or others involved with providing employ-

ment related support services. Follow-along intervention levels may need to increase in response to changes in the work environment, such as the introduction of new work equipment, which requires the employee to be retrained. Other occasions for increased follow-along services include the occurrence of interpersonal problems between the worker and his supervisor or another employee.

With TBI clients, follow-along needs to be as proactive as possible. Regular communication with the employee in a work setting can be an effective strategy for proactive troubleshooting. Compensatory strategies or physical adaptations may need to be modified or discontinued according to the employee's level of independence. When appropriate, supervisors, coworkers, and family members can provide the job coach with information regarding work-related issues. Most important, the job coach should be available to counsel the employee and to support his efforts to be a productive and satisfied member of the armed services.

Return to Active Duty Following Mild TBI: Decision-Making Criteria

Decisions regarding vocational reentry for military personnel are particularly complex when mild brain injury is involved. Soldiers who have sustained moderate to severe injuries will typically require job retraining in their previous position, placement in a new position, or disability status. In contrast, the subtler effects of mild brain injury often complicate the decision-making process regarding return to active duty. Making this decision requires a neurobehavioral assessment of the individual, including interviews with family members, peers, and supervisors. A number of critical questions should be answered, including (*a*) what the individual's emotional status is, (*b*) what his attitude about returning to his prior position is, and (*c*) to what extent might cognitive or behavioral deficits impair judgment of safety and decision-making skills. Safety should be a primary consideration as even a mild injury can reduce reaction time and impair an individual's ability to make quick and accurate decisions. The physical evaluation board (PEB) makes the official determination of fitness for duty.

In addition to assessment of the brain-injured soldier, the decision regarding return to active duty requires a comprehensive assessment of the target job. Identical to the procedure described in the context of supported employment, the task assessment phase involves analyzing the skills and responsibilities necessary to perform the job. Each job must be broken down into its functional elements, such as "operate a fork lift" and "supervise manufacturing employees for quality control." Descriptions should be as detailed as possible to help identify those skills that are necessary to perform the task. The job environment should also be assessed and rated for safety risks. Finally, coworkers should be interviewed regarding their expectations of the returning employee's duties, responsibilities, and limitations. This process is necessary to determine if coworkers are more likely to support or disrupt the reentry process.

Ultimately, the decision regarding return to active duty following mild brain injury hinges on three issues: (1) will reinstating active duty status compromise the individual's safety or that of his coworkers; (2) will emotional, behavioral, or cognitive deficits negate the individual's ability to successfully perform his job duties; and (3) will the individual have difficulty tolerating changes in his job performance that are caused by his brain injury (eg, decreased processing speed and frustration tolerance)? If these three questions can be answered with a "no," a decision to return the soldier to active duty would be warranted. Otherwise, the alternatives of retraining, placement in a different position (eg, the supported employment model), or disability should be considered.

FAMILY OUTCOME

There is little doubt that patients are not the only victims when TBI occurs. Many clinicians believe that immediate family members frequently suffer as much, and in some cases more, than patients themselves. The negative impact on family members is not surprising given the many common adverse behavioral, emotional, and intellectual sequelae, the dramatic shift in responsibilities and roles, and the difficulties inherent in finding rehabilitation services throughout the continuum of care. The research of Kozloff[253] and Jacobs[254] has indicated that extended family and friends are rarely available to provide long-term assistance. Consequently, the burden of care falls upon immediate family members.

Clearly, family members will play a central role in the rehabilitation of soldiers with brain injury. Because of their importance, family members should be included as an intrinsic part of the rehabilitation team. Important components of an effec-

tive rehabilitation program include the family's education regarding the effects of the injury, the treatment planning, and coping strategies. Clinicians have long been aware that families can make or break the rehabilitation process. To encourage relatives' positive involvement in the patient's recovery, professionals must appreciate the nature and course of family reactions to brain injury. The following section provides an overview of family reactions to TBI. A review of the literature is presented first, followed by guidelines for family assessment and intervention.

Family Outcome Literature

The long-term burden of caring for a person with brain injury is typically assumed by some member of the patient's family.[253,254] Family members frequently identify themselves as the primary providers of care and rehabilitative therapies (eg, physical therapy) for many years following the person's injury. Unfortunately, the vast majority of these individuals receive no formal training to help them assume this caregiving role.[254] Dependent on their own resources and abilities, families members must learn to cope with someone whose character and intellect have been permanently altered. Successful adaptation, although critical, is not common.[226] More often, family members report that the burden of caring for the patient forces them into social isolation[255] and depression.[256] These negative responses have far reaching consequences, such as effecting the duration and level of the patient's ultimate recovery.[212,257]

Family outcome following TBI, although a critical factor, has been an understudied topic.[258] Moreover, the existing literature is plagued by methodological shortcomings, such as inadequate description of samples and use of instruments with questionable reliability and validity.[259] Several exceptional studies do exist[205,212,226]; however, these studies typically focus on severe TBI or the acute stages of injury, or both, thereby limiting the scope of their findings.

Despite our limited knowledge of family outcome after brain injury, the existing literature has yielded a number of interesting findings. For example, families report that physical complications cause fewer problems than do cognitive and character changes,[211] which are more disruptive to family functioning.[260] Alterations in the patient's character result in numerous stresses for the family, including (*a*) feelings that the injured person is a "stranger"[256]; (*b*) changes in family roles[261]; and (*c*) difficulties managing the patient's behavior.[257] Family members often assume the brunt of the patient's frustration and are subjected to verbal abuse and threats of physical harm.[256,260] Lacking the formal training necessary to handle these stressors,[254] many relatives utilize maladaptive coping behaviors, such as taking tranquilizers and sleeping pills.[262]

Several investigators[256,257,263] have found that the burden of caring for someone with a brain injury places relatives at an increased risk for depression, anxiety, and physical illness. In a longitudinal study during the first year after severe TBI, Oddy and colleagues[257] reported on the responses of parents and spouses to the Wakefield Depression Scale[264] at 1, 6, and 12 months postinjury. Based on the Wakefield's clinical cutoff score, 39% of the relatives were considered depressed at 1 month postinjury in comparison to 25% at the 6 and 12 month intervals. The researchers concluded that the incidence of depression among relatives declines over the first year after injury. Oddy and associates[257] also investigated the prevalence of physical illness among relatives and found that approximately 25% had suffered at least one major illness since the injury.

In a comparable study,[263] researchers from Glasgow administered psychosocial measures to relatives of severely injured patients at 3, 6, and 12 months postinjury. Family members completed a number of surveys, including the Leeds anxiety and depression scales.[265] At 3 months, 34% of the relatives reported significant anxiety and 20% were depressed. These percentages rose at 6 months to 37% on the anxiety scale and 23% on the depression scale. At 12 months, the percentage of anxious relatives remained constant at 37% while depression increased to 26%. In contrast to Oddy's group,[257] the Glasgow researchers concluded that distress among relatives does not decline during the first year after injury.

Based on extensive clinical experience, Lezak[266] developed a stage model of family reactions following a relative's brain injury. Lezak proposed that families initially (ie, first 3 months following hospitalization) experience anxiety, confusion, and denial. During this period, the family believes that the patient will return to premorbid functioning within a year. As the family attempts to help the patient return to premorbid activities (eg, work and social events), the members begin to acknowledge the patient's cognitive and behavioral deficits. This marks the second stage which, according to Lezak's model, occurs 3 to 12 months following hospitalization. During this period, caregivers' initial bewilderment evolves into depression and anxiety as

their hopes for the patient's full recovery begin to fade. In the latter stages of Lezak's model (15 months and beyond), families must work through and accept the patient's permanent deficits. Consequently, the family structure must be reorganized to accommodate role changes. In some cases, this process requires emotional or physical detachment (or both) from the patient.

The existing literature suggests that family responses to TBI are moderated by a number of factors, including family, patient, and injury characteristics (Exhibit 6-4). Family characteristics, including demographic, dynamic, and economic factors, have been insufficiently studied and their impact on family outcome is ambiguous. The only exception is the characteristic of family relationships. Preliminary research suggests that spouses have more difficulty adjusting to the injured person than do parents.[207,211,256,262] Panting and Merry[262] were among the first researchers to compare the reactions of spouses and parents to a relative's brain injury. Family members of 30 British patients with severe TBI received questionnaires and structured interviews. Based on this information, Panting and Merry concluded that injury was a "great strain" on all family members. They also observed that spouses had more difficulty adjusting to the injury than did parents, although this conclusion was not supported by any data. Unfortunately, a number of other shortcomings, such as using instruments of questionable reliability and validity, limit the effectiveness of their findings.

In a later study, Mauss-Clum and Ryan[256] compared the responses from wives and mothers of neurologically impaired patients. Of the 30 patients included in the study, the majority had sustained TBI (57%), while the remainder had suffered vascular accidents (30%), or neurological degenerative disease. Overall, family members reported feeling frustrated, irritable, and annoyed with their situations. However, a greater proportion of wives were reportedly experiencing more negative emotional reactions and life changes than were mothers. Unfortunately, a number of factors confound this finding, including the patient's age and the type of injury incurred, making it uncertain if the differences are attributable solely to the variable of relationship.

Although methodological limitations dilute any conclusions, clinical evidence supports the legitimacy of a spouse–parent distinction. Experts in the field of brain injury rehabilitation identify familial role changes as an inevitable consequence of TBI. Typically, patients become more dependent on others and, in some cases, their behavior becomes more childlike. Accepting theses changes would presumably be less difficult for parents who consider dependency a natural part of a parent–child relationship,[207,211] while childish dependence is likely to be foreign to a marital relationship and, in turn, more stressful to the uninjured spouse. Panting and Merry[262] also suggest that marital relationships are especially burdened when children are involved. In such cases, uninjured spouses lose their partners' assistance in caring for their children and are essentially forced into single parenthood.

Certain characteristics of the patient have also been found to predict family outcome following brain injury. Current research[205,212] suggests that the severity of the patients' cognitive deficits is related to family outcome. However, this is based on two indirect lines of evidence and the findings should be considered preliminary. The first line of evidence comes from research on other disability groups, including rheumatoid arthritis, spinal cord injury, and multiple sclerosis. Bishop and Miller[267] conducted a discriminant validity study on a measure of family functioning, the McMaster Family Assessment Devise (FAD).[268] The FAD was designed to assess different dimensions of family functioning, including communication, problem solving, and affective responsiveness. Bishop and Miller[267] administered the FAD to family members of persons with physical disabilities (lupus erythematosus, rheumatoid arthritis, spinal cord injury, stroke, and multiple sclerosis) and to a "nonclinical" group (no family member with a chronic physical illness). They found that the "nonclinical" families were relatively high functioning. Furthermore, families from the lupus, arthritis, and spinal cord injury groups were functioning at levels comparable to the nonclinical fami-

EXHIBIT 6-4

FACTORS THAT MODERATE FAMILY REACTIONS TO TRAUMATIC BRAIN INJURY

- Relatives' relationship to the patient
- Patient's behavioral and emotional status
- Degree of patient's cognitive impairment
- Severity of injury
- Time postinjury

lies. In contrast, the stroke and multiple sclerosis (MS) groups were reporting significant disturbances in family functioning. Bishop and Miller[267] noted that the presence of cognitive disturbances among individuals with MS and stroke differentiated these patients from the other disability groups. In turn, Bishop and Miller concluded that "cognitive and mental disturbances have the most deleterious effect on family functioning."

The second line of evidence comes from the reports of relatives living with a person with brain injury. Researchers from Glasgow, Scotland, developed two rating scales for family members of persons with brain injury.[115,212] The first was a 7-point "subjective burden" scale on which the relative chose a number between 1 (I feel no strain as a result of the changes in my family member) and 7 (I feel severe strain as a result of the changes in my family member). On the second measure (objective burden), relatives rated the patient's functioning in different categories, including memory, emotion, language, physical, and disturbed behavior. A comparison of these rating scales revealed that relatives who reported greater memory impairment were likely to endorse higher levels of stress.

In addition to cognitive impairments, many survivors of TBI undergo emotional and behavioral changes. These changes include depression, apathy, emotional lability, pathological laughter, and irritability.[204,205,207,208,210] In a number of studies,[205,207,211] family members identified psychosocial changes as the greatest impediment to daily living. In a recent investigation, parents of adult brain injury survivors responded to a number of psychosocial outcome measures, including the Symptom Checklist-90 (SCL-90)[269] and the Hassles Scale.[270] Parents also rated their children on severity of psychosocial and physical impairment using the Sickness Impact Profile.[271] Tarter[272] reported that ratings of psychosocial dysfunction in the patients were correlated with severity of stress in the parents. Furthermore, parents who endorsed higher stress levels were more likely to be psychologically maladjusted. Tarter concluded that the patient's emotional and behavioral problems not only correlated with increased parental stress, but also with psychological distress among parents.

A final category of moderating variables pertains to characteristics of the injury, such as injury severity and time postinjury. Theories of coping with crisis suggest that coping responses are mediated by the severity of a stress as well as temporal factors.[273] The brain injury literature is consistent with this hypothesis. Many studies report that family reactions vary according to the severity of the patient's injury and the time postinjury.[212,257] Historically, studies relating injury severity to family outcome have been limited by methodological oversights, inconsistencies in classification, and restricted samples. In spite of these restrictions, research on injury severity and family outcome has yielded a number of noteworthy findings.

In several investigations, researchers found that marital instability is most common among severely injured persons and their spouses.[274,275] Using a battery of standardized measures, Peters and colleagues[274] compared all injury severity groups (ie, mild, moderate, severe) on dimensions of marital adjustment. Findings indicated that greater injury severity was associated with greater marital dysfunction, particularly in the areas of affectual expression and dual consensus. In an earlier study on long-term outcome, Walker[275] found that nearly 3 years postinjury, individuals with less severe injuries were more likely to be married than were severely injured individuals. This contrasts preinjury marital status, where the proportion of married individuals was equivalent across severity groups.

Preliminary findings indicate that injury severity interacts with time postinjury to affect the relative's perception of stress[205,212] and the psychological adaptation.[257] In an early outcome study, Livingston and colleagues[276] compared the relatives of individuals with mild and severe TBI at 3 months postinjury. Family members completed the Leeds depression and anxiety scale[265] along with a number of other measures. Relatives of severely injured patients were found to be significantly more anxious than were family members of individuals with mild TBI. Levels of depression were comparable for both groups of relatives.

In a longitudinal study described earlier, researchers in Glasgow[212] examined subjective burden among relatives of severely injured patients at 3, 6, and 12 months postinjury. Length of posttraumatic amnesia (PTA), which assesses the patient's orientation to person, place, and time, was used as an index of injury severity. Reportedly, longer durations of PTA were associated with higher levels of burden among relatives at 3 and 6 months postinjury. However, PTA and subjective burden were not significantly correlated at 12 months, suggesting that the relationship between injury severity and stress weakens over time.

In a long term outcome study, researchers[226] explored changes in familial stress levels after several years postinjury. Family members of 42 severely injured patients reported levels of subjective bur-

den at 1 and 5 years postinjury using a 7-point rating scale. Scores were collapsed into three categories of subjective burden: (1) low (1–2), (2) medium (3–4), and (3) high (5–7). At 1 year postinjury, 57% of relatives were reporting medium or high levels of subjective burden. In contrast, 89% of relatives reported medium or high burden at 5 years postinjury. Brooks and colleagues[226] concluded that relatives experience higher levels of stress as time postinjury increases.

Overall, these studies suggest an interesting relationship between injury characteristics and family outcome. Temporal factors appear to influence family reactions, as relatives report more stress over time.[226] This trend is likely related to the scarce availability of professional assistance beyond the acute stages of injury.[207,211] Initially, the patient and family receive a great deal of attention from medical professionals who are working to stabilize the patient. Once the patient is released from the hospital, the availability of resources declines and family members are left to deal with the patient on their own. Over time, the family must adjust to the patient's character and cognitive changes as well as increased financial pressures and social isolation. Relatives' reports that stress levels increase with time are, therefore, understandable.

The amount of time postinjury also appears to moderate the effects of injury severity on family outcome. Research suggests that injury severity is related to stress levels and anxiety among relatives within the first 6 months of injury.[212,276] However, initial severity indexes are not correlated with relative's depression or stress at 12 months.[212,257] These preliminary findings suggest that initial severity indexes are not predictive of relatives' psychosocial outcome beyond the acute stages of recovery. The absence of a correlation between injury severity and family outcome over time is reasonable because initial severity measures are only moderately related to patient outcome.[203,232,277] Individual differences account for wide variability in outcome among patients, even among the most severely injured.[203,277]

Assessment and Intervention

Any intervention strategy must be preceded by an assessment of the family's strengths, weaknesses, and needs. Because of their specialized training and experience, social workers and psychologists often play an important role in this evaluation process. Information gathered by them is often supplemented by other professionals in medicine and healthcare. Assessment of family systems can be accomplished through interview and clinical observation. Recently, several standardized assessment instruments have been developed to provide more objective means of analyzing family status. For example, the FAD[268] is a 60-item questionnaire divided into seven subscales. Descriptive information pertaining to communication, emotional involvement, roles, problem-solving skills, and rules for behavior can be derived through examination of scale score patterns. Scores can be used to establish goals for intervention and priorities can be developed by examining scale scores and responses to individual questionnaire items. Furthermore, global information regarding overall family "health" can be derived from the seventh subscale: the General Functioning Scale. Other well-developed measures of postinjury family functioning should also be considered, including the Family Adaptability and Cohesion Scales[278] and the Family Environment Scale.[279]

Clinicians may choose to use a measure developed specifically for use with families after brain injury, such as the Family Needs Questionnaire (FNQ).[280] Measures like the FNQ provide a wealth of information about how families react to and cope with the unique stresses associated with TBI. Using the FNQ, family members rate different needs on two dimensions: (1) perceived importance (not important, somewhat important, important, and very important); and (2) the extent to which each need is met (not met, partly met, met). Each dimension provides qualitatively different information about family needs after TBI. The importance of family needs is seen in its theoretical relationship to family coping and levels of stress. Presumably, families with fewer unmet needs adapt better and experience less stress than do families with more unmet needs. Additional information about quantitative family outcome measures can be found in the review by Bishop and Miller.[267]

The amount and type of intervention must complement the strengths, weaknesses, and needs of the family that were revealed in the evaluation process. Rosenthal and Young[281] identified six modes of family intervention that may be useful following TBI:

1. *Family education* provides family members with general information about traumatic brain injury and specific information about the patient.
2. *Marital and sexual counseling* directs couples toward restructuring marital roles and redeveloping a positive sexual relationship.

3. *Family counseling* helps members of the immediate (and extended) family cope with the changes in family roles, as well as issues of loss.
4. *Family support groups* provide emotional support through members sharing their experiences, problems, and information about community resources.
5. *Family networking* develops an extended family and friendship system to share the burden of care for clients and provide mutual support.
6. *Family advocacy* helps families learn to fully utilize existing community resources, enhance existing resources, and develop new resources.

Family members should be encouraged to develop realistic expectations for patients and help them to accomplish reasonable goals. Unreasonable or overly optimistic expectations enhance the likelihood of failure and ultimately contribute to depression, anxiety, and reduced self-esteem. Conversely, being overprotective and setting overly simplistic goals does not enable soldiers to fully use existing skills. Lifelong dependence accompanied by feelings of inadequacy and resentment can easily result from chronic underestimation the client's abilities. Treatment team members should carefully work with family members to develop appropriate expectations. Regular discussions of rehabilitation program goals and progress are helpful.

Although good interpersonal communication is hard to establish and maintain, frequent contact between rehabilitation professionals, clients, and families is an essential feature of effective rehabilitation systems. Professionals can develop positive relationships with family members through sharing information about community resources, focusing on positive client aspects, speaking in practical terms, and avoiding jargon. The use of jargon will not only create interpersonal distance between professionals and families, but may cause families to feel ignorant and overwhelmed.

Rehabilitation teams are encouraged to build family education systems that routinely provide information about the patient's problems, professional treatment strategies, and means by which the family may facilitate improvement. Ongoing education is necessary given the many different challenges faced at each point of the recovery process and significant changes in family members' abilities to accommodate information. Although a single team member may be designated to provide education, team members often share the responsibility. Information may be provided in the form of written materials, lectures, workshops, or individual meetings. Rehabilitation professionals are also encouraged to learn about community support and rehabilitation programs, and to routinely share their knowledge with clients and families.

Licensed professionals such as psychologists, counselors, and social workers are most qualified to provide marital counseling, family counseling, and family networking services. Credentialed mental health workers who are employed by the military would certainly be able to provide this treatment. However, mental health workers who have not been trained in TBI rehabilitation should collaborate with a physiatrist or clinical neuropsychologist. In the greater community, family support groups are usually available through such agencies as the local NHIF.

PREVENTION

One of the biggest issues in addressing the prevalent problem of TBI is the education of laypersons about ways to avoid it. Educational programs aimed at high school and college students, community service agencies, and the mass media have impacted greatly on compliance issues regarding seatbelts, drinking and driving, and helmet use with bicycles and motorcycles. No long term comprehensive TBI program hoping to have an impact on the devastation associated with this disability would be complete without such an educational component. TBI professionals and unit commanders should be involved in an organized military effort to address this "epidemic" through education of troops and officers.

CONCLUSION

There is no question that the field of brain injury rehabilitation is still in its infancy. We have learned quite a bit regarding what interventions can decrease neurologic and medical morbidity, increase functional capabilities, and potentially expedite both neurologic and functional recovery. We still require a better basic science foundation for our methodologies and clinical theories, although we have

come a long way in addressing this issue in the last decade. By encouraging continued examinations, the rationale behind our rehabilitative treatment and its overall efficacy we will only improve the quality of care being rendered to our patients and their families. In the interim, much of rehabilitation remains an integration of art and science; the key is to understand how the two interact.

REFERENCES

1. Dixon T. Systems of care for the head-injured. *Phys Med Rehabil*. 1989;3(1):169–181.
2. Zasler ND, Kreuzter JS, Taylor D. Coma stimulation and coma recovery: A critical review. *NeuroRehabilitation: An Interdisciplinary Journal*. 1991;1(3):33–40.
3. Zasler ND. Neuromedical diagnosis and management of post-concussive disorders. In: Horn LJ, Zasler ND eds. *Phys Med Rehabil*. 1992;6(1):33–67.
4. Katz RT. Mechanisms, measurement, and management of spastic hypertonia after head injury. *Phys Med Rehabil Clin North Am*. 1992;3(2):319–336.
5. Gennarelli TA. Mechanisms of brain injury. *J Emerg Med*. 1993;11(Suppl 1):5–11.
6. Kreutzer JS, Zasler ND, Devany CW. Neuromedical and psychosocial aspects of rehabilitation after traumatic brain injury. In: Fletcher G, Jann B, Wolf S, Banja J, eds. *Rehabilitation Medicine: State of the Art*. New York: Lea and Febiger; 1992: 63–102.
7. Chicago National Safety Council Statistics Department. *Accident Facts*. Chicago: Chicago National Safety Council; 1989.
8. Frankowski RF, Annegers JF, Whitman S. The descriptive epidemiology of head injury in the United States. In: Becker DP, Povlishock JT, eds. *Central Nervous System Trauma Research Status Report*. Bethesda, Md: National Institute of Neurological and Communicative Disorders and Stroke, NIH; 1985:33–44.
9. Annegers JF, Grabow MD, Kurland LT, Laws ER. The incidence, causes, and secular trends of head trauma in Olmsted County, Minnesota: 1935–1974. *Neurology*. 1980;30:912–919.
10. Whitman S, Coonley-Hoganson R, Desai BT. Comparative head trauma experiences in two socio-economically different Chicago-area communities: A population study. *Am J Epidemiol*. 1984;119:570–580.
11. Rautio J, Paavolainen P. Afghan war wounded: Experience with 200 cases. *J Trauma*. 1988;28(4):523–525.
12. Levi L, Borovich B, Guiburd JN, Grushkiewicz I, Lemberger A, Linn S, Schachter I, Zaaroor M, Braun J. Wartime neurosurgical experience in Lebanon, 1982–1985. I: Penetrating craniocerebral injuries. *Isr J Med Sci*. 1990;26(10):548–554.
13. McCarroll JE, Zych KA. Descriptive epidemiologic survey of head injury in the army: 1983 hospitalized cases. *Neuroepidemiology*. 1989;8:48–52.
14. McCarroll JE, Gunderson C. 5-year study of incidence rates of hospitalized cases of head injuries in the US army. *Neuroepidemiology*. 1990;9:296–305.
15. Jennett B, Teasdale G, Murray G, Murray L. Head injury. In: Evans RW, Baskin DS, Yatsu FM, eds. *Prognosis of Neurological Disorders*. New York: Oxford University Press; 1992:85–96.
16. Bond MR. Assessment of psychosocial outcome of severe head injury. *Acta Neurochir*. 1976;34:57–70.
17. Heiskanen O, Martilla I, et al. Prognosis of depressed skull fractures. *Acta Chir Scand*. 1973;139:605–608.
18. Choi SS, et al. Chart for outcome prediction in severe head injury. *J Neurosurg*. 1983;59:294–297.

19. Jennett B, Teasdale G. Assessment of coma and impaired consciousness: A practical scale. *Lancet.* 1974;2:74.

20. Born JD. The Glasgow-Liege Scale. Prognostic value and evolution of motor response and brainstem reflexes after severe head injury. *Acta Neurochir*. 1988;9:1–11.

21. Horn LJ, Cope DN. Traumatic brain injury. *Phys Med Rehabil.* 1989;3(1).

22. Katz DI. Neuropathology and neurobehavioral recovery from closed head injury. *J Head Trauma Rehabil*. 1992;7(2):1–15.

23. Groswasser Z, Sazbon L. Outcome in 134 patients with prolonged posttraumatic unawareness. *J Neurosurg*. 1990;72:81–84.

24. Whyte J. Mechanisms of recovery of function following CNS damage. In: Rosentahl M, Griffith ER, Bond MR, Miller JD, eds. *Rehabilitation of the Adult and Child with Traumatic Brain Injury*. Philadelphia, Pa; FA Davis. 1990:79–88.

25. Boyeson MG. Neurochemical alterations after brain injury: Clinical implications for pharmacologic rehabilitation. *NeuroRehabilitation: An Interdisciplinary Journal*. 1991;1(1):33–43.

26. Olney JW, Sharpe LG. Brain lesions in an infant rhesus monkey treated with monosodium glutamate. *Science.* 1969;166:386–388.

27. Lyeth BG, Jenkins LW, Hayes RL. The NMDA receptor: Theoretical and clinical implications in the acute treatment of traumatic brain injury. *NeuroRehabilitation: An Interdisciplinary Journal*. 1991;1(1):50–57.

28. McDonough JH Jr, Shih TM. A study of the N-methyl d-aspartate antagonistic properties of anticholinergic drugs. *Pharmacol Biochem Behav.* 1995;51(2–3):249–253.

29. Hayes RL, Pechura CM, Katayama Y, et al. Activation of pontine cholinergic sites implicated in unconsciousness following cerebral concussion in the cat. *Science.* 1984;233:301–303.

30. Hovda DA, Feeny DM. Amphetamine with experience promotes recovery of locomotor function after unilateral frontal cortex injury in the cat. *Brain Res.* 1984;298:358–361.

31. Boyeson MG, Feeny DM. The role of epinephrine in recovery from brain injury. *Soc Neurosci Abstr*. 1984;10:68.

32. Clifton GL, Jiang JY, Lyeth BG, Jenkins LW, Hamm RJ, Hayes RL. Marked protection by moderate hypothermia after experimental traumatic brain injury. *J Cereb Blood Flow Metab.* 1991;11(1):114–121.

33. Boyeson MG, Bach-y-Rita P. Determinants of brain plasticity. *J Neurol Rehab*. 1989;3:35–37.

34. Boyeson MG, Jones JL. In: Horn LJ, Zasler ND, eds. *Theoretical Mechanisms of Brain Plasticity in Therapeutic Implications in Medical Rehabilitation of Traumatic Brain Injury*. Philadelphia, Pa: Hanley & Belfus; 1996: 77–102.

35. Chavany JA. Rôle des causes occasionelles dans le déterminisme du ramolissement cérébral (réfléxions thérapeutiques à propos de). *Pratique medicale francaise*. 1928;7:285–295.

36. Sciclounoff F. L'acetylcholine dans le traitement de l'ictus hémiplegique. *Presse medicale*. 1934;42:1140.

37. Feeney DM, Sutton RL. Pharmacotherapy for recovery of function after brain injury. *CRC Crit Rev Neurobiology*. 1987;3:135–197.

38. Cedarbaum JM. Clinical pharmacokinetics of anti-Parkinsonian drugs. *Clin Pharmacokinetics*. 1987;13:141–178.

39. Berg MJ, Ebert B, Willis DK, et al. Parkinsonism: Drug treatment. *Drug Intelligence & Clinical Pharmacy*. 1987;21(1):10–21.

40. Gualtieri CT, Chandler M, Coons TB, et al. Amantadine: A new clinical profile for traumatic brain injury. *Clin Neuropharm*. 1989;12:258–270.

41. Peterson PL, Saad J, Nigro MA. The treatment of Friedreich's Ataxia with amantadine hydrochloride. *Neurology*. 1988;38:1478–1480.

42. Cooper JR, Bloom FE, Roth RH. *The Biochemical Basis of Neuropharmacology*. New York: Oxford University Press; 1986.

43. Gualtieri CT. Pharmacotherapy and the neurobehavioral sequelae of traumatic brain injury. *Brain Injury*. 1988;2:101–129.

44. Sommi RW, Crisomn ML, Bowden CL. Fluoxetine: A serotonin-specific, second generation antidepressant. *Pharmacotherapy*. 1987;7:1–15.

45. Kastenholz KV, Crismon ML. Buspirone, a novel non-benzodiazepine anxiolytic. *Clin Pharmacy*. 1984;3:600–607.

46. Eison AS, Temple DL. Buspirone: Review of its pharmacology and current perspectives on its mechanism of action. *Am J Med*. 1986;80:1–9.

47. Cardoni AA. Focus on paroxetine: A potent, selective serotonin uptake inhibitor for use in major depression. *Hosp Formul*. 1992;27:445–461.

48. Guthrie Sk. Sertraline: A new specific Serotonin reuptake blocker. *DICP*. 1991;25:952–961.

49. Slutsker L, Hoesly FC, Miller L, et al. Eosinophilia-myalgia syndrome associated with exposure to tryptophan from a single manufacturer. *JAMA*. 1990;264;213–217.

50. Bleck TP, Klawans HL. Covulsive disorders: Mechanisms of epilepsy and anticonvulsant action. *Clin Neuropharm*. 1990;13:121–128.

51. Rolls ET. Psychopharmacology and food. In: Sandler M, Silverstone, eds. *The Neurophysiology of Feeding*. New York, NY: British Association for Psychopharmacology Monograph. 1985:1–16.

52. Morley JE. An approach to the development of drugs for appetite disorders. *Neuropsychobiology*. 1989;21:22–30.

53. Childs A. Naltrexone in organic bulimia: A preliminary report. *Brain Injury*. 1987;1:49–55.

54. Garattini S, Bizzi A, Caccia S, Mennini T. Progress report on the anorectic effects of dexfenfluramine, fluoxetine, and sertraline. *Int J Obes.* 1992;16(3):43–50.

55. Sandyk R, Lacona RP. Post-traumatic cerebellar syndrome: Response to L-tryptophan. *Int J Neuroscience*. 1989;47:301–302.

56. Trouillas P, Brudon F, Adeleine P. Improvement of cerebellar ataxia with levorotatory form of 5-hydroxytryptophan: A double-blind study with quantified data processing. *Arch Neurology*. 1988;45:1217–1222.

57. Manyam BV. Recent advances in the treatment of cerebellar ataxia. *Clin Neuropharmacology*. 1986;9:508–516.

58. Rudy TA. Pathogenesis of fever associated with cerebral trauma and intracranial hemorrhage: Thermoregulatory mechanisms and their therapeutic implication. In: *4th International Symposium on the Pharmacology of Thermoregulation*. Oxford, UK: Karger; 1979: 75–81.

59. Lipton JM, Clark WG. Neurotransmitters in temperature control. *Ann Rev Phsyiology*. 1986;48:613–632.

60. Yamawaki S, Lai H, Horita A. Dopaminergic and serotonergic mechanisms of thermoregulation: Mediation of thermal effects of apomorphine and dopamine. *J Pharmacol Exp Ther*. 1983;227:383–388.

61. Rossitch E, Bullard DE. The autonomic dysfunction syndrome: Etiology and treatment. *Br J Neurosurg*. 1988;2:471–478.

62. Gordon M, Rreiksaitis HG. Drugs and the aging brain. *Geriatrics*. 1988;43:69–78.

63. Greidanus TJ, Van Wimersma B, Jolles J, De Wied D. Hypothalamic neuropeptides and memory. *Acta Neurochir*. 1985;75:99–105.

64. Serby M, Resnick R, Jordan B, Adler J, Corwin J, Rotrosen JP. Naltrexone and Alzheimer's disease. *Prog Neuropsychopharmacol Biol Psychiatry.* 1986;10:587–590.

65. Reichert WH, Blass JP. A placebo controlled trial shows no effect of vasopressin on recovery from closed head injury. *Ann Neurol.* 1982;12:390–392.

66. Bohnen NI, Twijnstra A, Jolles J. A controlled trial with vasopressin analogue (DGAVP) on cognitive recovery immediately after head trauma. *Neurology*. 1993;43:103–106.

67. Zasler ND. Pharmacologic Aspects of Cognitive Function Following Traumatic Brain Injury. In: Kreutzer JS, ed. *Cognitive Rehabilitation for Persons with Traumatic Brain Injury: A Functional Approach*. Baltimore, Md: Paul H Brooks; 1991: 87–93.

68. Davis KL, Mohs RC, Tinkleberg JR, Pfefferbaum A, Hollister LE, Kopell BS. Physostigmine: Improvement of long term memory processes in normal humans. *Science*. 1978;201:272–274.

69. Hock FJ. Drug influences on learning and memory in aged animals and humans. *Neuropsychobiology*. 1987;17:145–160.

70. Weinberg RM, Auerbach SH, Moore S. Pharmacologic treatment of cognitive deficits: A case study. *Brain Injury*. 1987;1(1):57–59.

71. Levin HS, Peters BH, Kalisky Z, High WM, Laufen AV, Disenberg HM, Morrison DP, Gary HE. Effects of oral physostigmine and lecithin on memory and attention in closed head injured patients. *Central Nervous System Trauma*. 1986;3(4):333–342.

72. Goldberg E, Gerstman JL, Mattis S, Hughes JE, Bilder TM, Sirio CA. Effects of cholinergic treatment on post-traumatic anterograde amnesia. *Neurology*. 1982;39:581.

73. Summers WK, Majovsky LV, Marsh GM, Packiki K, Kling A. Oral tetrahydroaminoacridine in long term treatment of senile dementia. *N Engl J Med.* 1986;315(20):1241–1287.

74. Levin HS. Treatment of post-concussional symptoms with CDP-choline. *J Neurol Sci.* 1991;103:39–42.

75. Maldonado VC, Perez JBC, Escario JA. Effects of CDP-choline on the recovery of patients with head injury. *J Neurol Sci.* 1991;103:15–18.

76. Zornetzer SR. Neurotransmitter modulation and memory: A new neuropharmacological phrenology? In: Lipton MA, DiMascio A, Killian KF, eds. *Psychopharmacology—A Generation of Progress*. New York: Raven Press; 1978: 637–649.

77. Gualtieri T, Chandler M, Coons TB, Brown LT. Amantadine: A new clinical profile for traumatic brain injury. *Clin Neuropharmacol*. 1989;12:258–270.

78. Gualtieri T, Evans R. Pharmacotherapy and the neurobehavioral sequelae of traumatic brain injury. *Brain Injury*. 1988;2(2):101–129.

79. Lal S, Merbitz C, Grip J. Modification of function in head-injured patients with Sinement. *Brain Injury*. 1988;2(3):225–234.

80. McIntyre FL, Gasquoine P. Effects of clonidine on post-traumatic memory deficits. *Brain Injury*. 1989;4(2):209–211.

81. McLean A, Stanton K, Cardenas D, Bergerud D. Memory training combined with the use of oral physostigmine. *Brain Injury*. 1987;1:145–159.

82. McDonald RJ. Hydergine. A review of 26 clinical studies. In: Guski J, ed. *Determining the Effects of Aging on the Central Nervous System*. Berlin: Schering; 1980; 121–138.

83. Jenike MA, Albert MS, Heller H, Locastro S, Gunther J. Combination therapy with lecithin and ergoloid mesylates for Alzheimer's disease. *J Clin Psychiatry*. 1986;47:249–251.

84. O'Shanick GJ, Zasler ND. Neuropsychopharmacological approaches to traumatic brain injury. In: Kreutzer JS, Wehman P, eds. *Community Integration Following Traumatic Brain Injury*. Baltimore, Md: Paul H Brooks; 1990: 15–27.

85. Glenn MB, Wroblewski BW. Update on pharmacology: The choice of antidepressants in depressed survivors of traumatic brain injury. *J Head Trauma Rehabil.* 1989;4(3):85–88.

86. Fleet WS, Valenstein E, Watson RT, et al. Dopamine agonist therapy for neglect in humans. *Neurology*. 1987;37:1765–1770.

87. Zasler ND, McNeny R. Neuropharmacologic rehabilitation following traumatic brain injury via dopamine agonists. *Arch Phys Med Rehabil.* 1989;70:12–13.

88. Koller WC, Wong GF, Lang A. Post-traumatic movement disorders: Review. *Movement Disorders*. 1989;4:20–36.

89. Katz DI. Movement disorders following traumatic head injury. *J Head Trauma Rehabil.* 1990;5(1):86–90.

90. Biary N, Cleeves L, Findley L, et al. Post-traumatic tremor. *Neurology*. 1989;1:103–106.

91. Ellison PH. Propranolol for severe post-head injury action tremor. *Neurology*. 1978;28:197–199.

92. Samie MR, Selhorst JB, Koller WC. Post-traumatic midbrain tremor. *Neurology*. 1990;40:62–66.

93. Zdonczk D, Royse V, Koller WC. Nicotine and tremor. *Clin Neuropharmacol*. 1988;11:282–286.

94. Eames P. The use of Sinemet and bromocriptine. *Brain Injury*. 1989;3:319–320.

95. Stewart JT. Akathisia following traumatic brain injury: Treatment with bromocriptine. *J Neurol Neurosurg Psychiatry.* 1989;52:1200–1203.

96. Adler LA, Reiter S, Corwin J, et al. Neuroleptic-induced akathisia: Propranolol versus benztropine. *Biol Psychiatry*. 1988;23:211–213.

97. Watanabe K, Kuroiwa Y, Toyokura Y. Epilepsia partialis continua: Epileptogenic focus in motor cortex and its participation in transcortical reflexes. *Arch Neurol.* 1984;41:1040–1044.

98. Obeso JA, Artieda J, Rothwell JC, et al. The treatment of severe action myoclonus. *Brain.* 1989;112:765–777.

99. Jankovic J, Pardo R. Segmental myoclonus: Clinical and pharmacologic study. *Arch Neurol.* 1986;43:1025–1031.

100. Robin JJ. Paroxysmal choreoathetosis following head injury. *Ann Neurol.* 1977;2:447–448.

101. Richardson JC, Howes JL, Celniski MJ, et al. Kinesigenic choreoathetosis due to brain injury. *Can J Neurol Sci.* 1987;14:626–628.

102. Bontke CF. Medical complications related to traumatic brain injury. In: Horn LJ, Cope DN, eds. *State of the Art Reviews: Physical Medicine and Rehabilitation*. Philadelphia, Pa: Hanley & Belfus; 1989: 43–59.

103. Temkin NR, Dikmen SS, Wilensky AJ, et al. A randomized, double blind study of phenytoin for the prevention of post-traumatic seizures. *N Engl J Med*. 1990;323:497–502.

104. Kuhl DA, Boucher BA, Muhlbauer MS. Prophylaxis of post-traumatic seizures. *DICP Ann Pharmacotherapy.* 1990;24:277–285.

105. Dalmady-Israel C, Zasler ND. Post-traumatic seizures: A critical review. *Brain Injury*. 1993;7(3):263–273.

106. Glenn MB, Wroblewski B. Anticonvulsants for prophylaxis of post-traumatic seizures. *J Head Trauma Rehabil.* 1986;1:73–74.

107. Jones GL, Matsuo F, Baringer JR, et al. Valproic acid-associated encephalopathy. *West J Med.* 1990;153:199–202.

108. Collaborative Group for Epidemiology of Epilepsy. Adverse reactions to anti-epileptic drugs: A multicenter survey of clinical practice. *Epilepsia*. 1986;27:323–330.

109. Thompson PJ, Trimble MR. Comparative effects of anticonvulsant drugs on cognitive functioning. *Br J Clin Pract*. 1982;18:154–156.

110. Trimble MR. Anticonvulsant drugs and cognitive function: A review of the literature. *Epilepsia*. 1987;28:Suppl 37–45.

111. Meador KJ, Loring DW, Huh K, et al. Comparative cognitive effects of the anticonvulsants. *Neurology*. 1990;40:391–394.

112. Levy RH, ed. *Antiepileptic Drugs*. New York: Raven Press; 1989.

113. Kreutzer J, Zasler ND. Psychosexual consequences of traumatic brain injury: Methodology and preliminary findings. *Brain Injury*. 1989;3:177–186.

114. Zasler ND, Horn LJ. Rehabilitative management of sexual dysfunction. *J Head Trauma Rehabil.* 1990;5:14–24.

115. Zasler ND. Sexual dysfunction and traumatic brain injury. In: Silver JM, Yudofsky SC, Hales RE, eds. *Psychiatric Aspects of Traumatic Brain Injury*. Washington, DC: American Psychiatric Press; 1994: 443-470.

116. Stewart JT. Carbamazepine treatment of a patient with Kluver-Bucy Syndrome. *J Clin Psychiatry.* 1985;46:496–497.

117. Crismon ML, Childs A, Wilcox RE, et al. The effect of bromocriptine on speech dysfunction in patients with diffuse brain injury (akinetic mutism). *Clin Neuropharmacol*. 1988;11:462–466.

118. Bachman DL, Morgan A. The role of pharmacotherapy in the treatment of aphasia: Preliminary result. *Aphasiology*. 1988;2:225–228.

119. Albert ML, Bachman DL, Morgan A, et al. Pharmacotherapy for aphasia. *Neurology*. 1988;38:877–879.

120. Bieger D, Giles SA, Hockman CH. Dopaminergic influences on swallowing. *Neuropharmacology*. 1977;16:245–252.

121. Bieger D, Weerasuriya A, Hockman CH. The emetic action of L-dopa and its effect on the swallowing reflex in the cat. *J Neural Transm*. 1978;42:82–87.

122. Rucker KS, Zasler ND, Narasimhachari N, et al. Resolution of swallowing disorder with L-dopa and evaluation of CSF neurotransmitters in brain injury. *Arch Phys Med Rehabil.* 1988;69:734.

123. Biary N, Pimental PA, Langenberg PW. A double-blind trial of clonazepam in the treatment of parkinsonian dysarthria. *Neurology*. 1988;38:255–258.

124. Baratz R, Mesulam MM. Adult-onset stuttering treated with anticonvulsants. *Arch Neurol.* 1981;38:132.

125. Berrol S. Cranial nerve dysfunction. In: Horn LJ, Cope DN, eds. *State of the Art Reviews: Physical Medicine and Rehabilitation*. Philadelphia: Hanley & Belfus; 1989: 85–94.

126. Hilten Van JJ, Roos RAC. Posttraumatic hyperthermia: A possible result of frontodiencephalic dysfunction. *Clin Neurol Neurosurg*. 1991;93(3):223–225.

127. Paul SM, Barlow JR. Neurogenic heterotopic ossification. *NeuroRehabilitation*. 1993;3(3):66–78.

128. Katz RT. Mechanisms, measurement, and management of spastic hypertonia after head injury. In: Berrol S, ed. *Phys Med Rehabil Clin North Am.* 1992;3(2):319–336.

129. Guanche C, Keenan MAE. Principles of orthopedic rehabilitation. In: Berrol S, ed. *Phys Med Rehabil Clin North Am.* 1992;3(2):417–426.

130. Young B, Ott L, Phillips R, McClain C. Metabolic management of the patient with head injury. In: Eisenberg HM, Aldrich EF, eds. *Neurosurg Clin N Am.* 1991;2(2):301–320.

131. Pacult A, Gudeman SK. Medical management of head injuries. In: Becker DP, Gudeman SK, eds. *Textbook of Head Injury*. Philadelphia, Pa: WB Saunders; 1989: 192–220.

132. Bachman DL. The diagnosis and management of common neurologic sequelae of closed head injury. *J Head Trauma Rehabil.* 1992;7(2):50–59.

133. Narayan RK. Head injury. In: Grossman RG, ed. *Principles of Neurosurgery*. New York: Raven Press; 1991: 235–291.

134. Nelson VS. Pediatric head injury. In: Berrol S, ed. *Phys Med Rehabil Clin North Am.* 1992;3(2):461–474.

135. Jaffe KM, Brink JD, Hays RM, Chorazy AJL. Specific problems associated with pediatric head injury. In: Rosenthal M, Griffith ER, Bond MR, Miller JD, eds. *Rehabilitation of the Adult and Child with Traumatic Brain Injury*. Philadelphia, Pa: FA Davis; 1990: 539–557.

136. Goldman H, Englander J. Traumatic brain injury in elderly individuals. In: Berrol S, ed. *Phys Med Rehabil Clin North Am.* 1992;3(2):441–460.

137. Doherty D. Post-traumatic hydrocephalus. In: Berrol S, ed. *Phys Med Rehabil Clin North Am.* 1992;3(2):389–405.

138. Cope DN, Date ES, Mar EY. Serial computerized tomographic evaluations in traumatic head injury. *Arch Phys Med Rehab*. 1988;69:483–486.

139. Temkin NR, Dikmen SS, Winn HR. Post-traumatic seizures. In: Eisenberg HM, Aldrich EF, eds. *Neurosurg Clin North Am.* 1991;2(2):425–436.

140. Kneipp S. Cognitive remediation within the context of a community reentry program. In: Kreutzer JS, Wehman PH, eds. *Cognitive Rehabilitation for Persons with Traumatic Brain Injury: A Functional Approach*. Baltimore, Md: Paul H Brooks; 1991: 239–250.

141. McLean A, Cardenas DD, Hasekorn JK, Peters M. Cognitive psychopharmacology. *NeuroRehabilitation*. 1993;3(2):1–14.

142. Spitzer R, Williams JB, Gibbon M. *Structured Clinical Interview for DSM-III-R*. New York: Biometrics Research Department at New York State Psychiatric Institute; 1986.

143. Levin HS, High WM, Goethe KE, et al. The Neurobehavioral Rating Scale: Assessment of the behavioral sequelae of head injury by the clinician. *J Neurol Neurosurg Psychiatry*. 1987;50:183.

144. Hamilton M. A rating scale for depression. *J Neurol Neurosurg Psychiatry*. 1960;23:56–62.

145. Yudofsky SC, Silver JM, Jackson W, et al. The Overt Aggression Scale for the objective rating of verbal and physical aggression. *Am J Psychiatry*. 1986;143:35–39.

146. Silver JM, Hales RE, Yudofsky SC. Neuropsychiatric aspects of traumatic brain injury. In: Yudofsky SC, Hales RE, eds. *The American Psychiatric Press Textbook of Neuropsychiatry*. Washington, DC: American Psychiatric Press; 1992: 363–395.

147. Wood RLI. *Neurobehavioral Sequelae of Traumatic Brain Injury*. New York: Taylor & Francis; 1990.

148. Hall KM, Hamilton BB, Gordon WA, Zasler ND. Characteristics and comparisons of functional assessment indices: Disability Rating Scale, Functional Independence Measure, and Functional Assessment Measure. *J Head Trauma Rehabil.* 1993;8(2):60–74.

149. Hall KM. Overview of functional assessment scales in brain injury rehabilitation. *NeuroRehabilitation*. 1992;2(4):98–113.

150. Uomoto J, McLean A. Care continuum in traumatic brain injury. *Rehabil Psych.* 1989;34:71–80.

151. Mackay LE, Bernstein BA, Chapman PE, et al. Early intervention in severe head injury: Long-term benefits of a formalized program. *Arch Phys Med Rehabil*. 1992;73:635–641.

152. Ragnaarson KT, Thomas JP, Zasler ND. Model systems of care for individuals with traumatic brain injury. *J Head Trauma Rehabil.* 1993;8(2):1–11.

153. Zasler ND. A medical perspective on physician training and brain injury rehabilitation. In: Durgin CJ, Schmidt ND, Fryer LJ, eds. *Staff Development and Clinical Intervention in Brain Injury Rehabilitation*. Baltimore, Md: Aspen Publishers; 1993:257–269.

154. Bontke CF, Baize CM, Boake C. Coma Management and Sensory Stimulation. In: Berrol S, ed. *Phys Med Rehabil Clin North Am.* 1992;3(2):259–272.

155. Sandel ME. Rehabilitation management in the acute care setting. In: Horn LJ, Cope DN, eds. *State of the Art Reviews: Physical Medicine and Rehabilitation*. Philadelphia, Pa: Hanley & Belfus; 1989: 27–42.

156. Deaton AV, Holland A, Stonnington H, Veach S, Wilkins SL. Postacute rehabilitation: The day rehabilitation model. *NeuroRehabilitation*. 1991;1(3):62–69.

157. Blackerby WF, Gualtieri T. Recent advances in neurobehavioral rehabilitation. *NeuroRehabilitation*. 1991;1(3):53–61.

158. Cope DN, Cole JR, Hall KM, Barkan H. Brain injury: Analysis of outcomes in a post-acute rehabilitation system: General analysis. *Brain Injury*. 1991;5(2 part 1):111–125.

159. Fraser RT, Clemmons DC, McMahon BT. Vocational rehabilitation counseling. In: Kreutzer JS, Wehman P, eds. *Community Integration Following Traumatic Brain Injury*. Baltimore, Md: Paul H Brooks; 1990: 169–184.

160. McMahon BT, Shaw LR, eds. *Work Worth Doing: Advances in Brain Injury Rehabilitation*. Orlando, Fla: Paul M Deutsch; 1991.

161. Cervelli L. Re-entry into the community and systems of post-hospital care. In: Rosenthal M, Griffith ER, Bond MR, Miller JD, eds. *Rehabilitation of the Adult and Child with Traumatic Brain Injury*. Philadelphia, Pa; FA Davis; 1990: 463–475.

162. Cervelli L, Berrol S. Description of a model care system. In: *Head Injury Rehabilitation Project Final Report*. Grant #13-P-59156/9. San Jose, Calif: Santa Clara Valley Medical Center; 1982:4.

163. Van Der Schaaf S. *Personal communication*. 1992.

164. Dixon TP. Systems of care for the head-injured. In: Horn LJ, Cope DN, eds. *State of the Art Reviews: Physical Medicine and Rehabilitation*. Philadelphia, PA: Hanley & Belfus; 1989: 169–181.

165. Horn LJ. Systems of care for the person with traumatic brain injury. In: Berrol S, ed. *Phys Med Rehabil Clin North Am.* 1992;3(2):475–492.

166. Anderson DW, McLaurin RL. The national head and spinal cord injury survey. *J Neurosurgery.* 1980;53:Suppl 1.

167. Miller JD, Jones PA. The work of a regional head injury service. *Lancet*. 1985;1:1141.

168. Rimel RW, Giordani B, Barth JT, et al: Clinical and scientific communications: Disability caused by minor head injury. *Neurosurgery*. 1981;9:221–228.

169. Rimel RW, Jane JA. Minor head injury: Management and outcome. In: Williams RH, Rengachery SS, eds. *Neurosurgery*. New York: McGraw-Hill; 1985.

170. Horn LJ, Zasler ND. Rehabilitation of post-concussive Disorders. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus; 1992;6(1).

171. Lehmkuhl LD. *Brain injury glossary*. Houston, Tex: Traumatic Brain Injury Model System Research Program: The Institute for Rehabilitation and Research; 1990.

172. Leininger BE, Gramling SE, Farrell AD, et al. Neuropsychological deficits in symptomatic minor head injury patients after concussion and mildconcussion. *J Neurol Neurosurg Psychiatry*. 1990;53:293–296.

173. Wilkinson HA, Binder LM. Commentary. *J Neurol Neurosurg Psychiatry.* 1991;54:846–847.

174. Gennarelli TA. Mechansims and pathophysiology of cerebral consussion. *J Head Trauma Rehabil.* 1986;1:23–29.

175. Zasler ND. Mild traumatic brain injury: Medical assessment and intervention. *J Head Trauma Rehabil.* 1993.

176. Travell JG, Simons OG. *Myofascial Pain and Dysfunction: The Trigger Point Manual*. Baltimore, Md: Williams & Wilkins; 1983.

177. Vernon H, ed. *Upper Cervical Syndrome: Chiropractic Diagnosis and Treatment*. Baltimore, Md: Williams & Wilkins; 1988.

178. Gronwall D. Rehabilitation programs for patients with mild head injury: Components, problems and evaluation. *J Head Trauma Rehabil.* 1986;1:53–62.

179. Kay T. Neuropsychological diagnosis: Disentangling the multiple determinants of functional disability after mild traumatic brain injury. In: Horn LJ, Zasler ND, eds. Rehabilitation of Post-Concussive Disorders. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus: 1992;6(1):109–127.

180. Binder LM. Malingering following minor head trauma. *Clin Neuropsychologist.* 1990;4(1):25–36.

181. Plumb F, Posner JB. *The Diagnosis of Stupor and Coma*. Philadelphia, Pa: FA Davis; 1980.

182. Giacino JT, Zasler ND, Katz D, Kelly JP, Rosenberg J, Filley C. Practice guidelines for assessment and management of the vegetative and minimally conscious states: Proceedings of the Aspen Neurobehavioral Consensus Conference. *J Head Trauma Rehabil.* In press.

183. American Academy of Neurology. Multisociety task force on PVS. Medical aspects of the persistent vegetative state: Statement of a multi-society task force. *N Engl J Med.* 1994;330(21):1499–1508.

184. Weiner JD. Legal issues regarding patients in coma or in persistent vegetative state. In: Sandel ME, Ellis DW, eds. The Coma Emerging Patient. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus: 1990;4(3):569–578.

185. Banja JD. Ethical aspects of treatment for coma and the persistent vegetative state. In: Sandel ME, Ellis DW, eds. The Coma Emerging Patient. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus: 1990;4(3):579–592.

186. Council on Scientific Affairs and Council on Ethical and Judicial Affairs. Persistent vegetative state and the decision to withdraw or withhold life support. *JAMA*. 1990;263:426–430.

187. Ethics and Humanities Sub-committe of the American Academy of Neurology. Position Statement: Certain aspects of the care and management of profoundly and irreversibly paralyzed patients with retained consciousness and cognition. *Neurology*. 1993;43:222–223.

188. McLellan DR. The structural basis of coma and recovery: Insights from brain injury in humans and experimental animals. In: Sandel ME, Ellis DW, eds. The Coma Emerging Patient. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus; 1990;4(3):389–408.

189. DeVolder AG, Goffinet AM, Bol A, et al. Brain glucose metabolism in postanoxic syndrome: positron emission tomographic study. *Arch Neurol.* 1990;47:197–204.

190. Levy DE, Sidtis JJ, Rottenberg DA, et al. Differences in cerebral blood flow and glucose utilization in vegetative versus locked-in patients. *Ann Neurol.* 1987;22:673–682.

191. Ellis DW, Rader MA. Structured sensory stimulation. In: Sandel ME, Ellis DW, eds. The Coma Emerging Patient. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus; 1990;4(3):465–478.

192. Pierce JP, Lyle DM, Quine S, et al. The effectiveness of coma arousal intervention. *Brain Injury*. 1990;4:191–197.

193. Rader MA, Alston JB, Ellis DW. Sensory stimulation of severely brain injured patients. *Brain Injury*. 1989;3:141–147.

194. Giacino JT, Zasler ND. Outcome following severe brain injury: The comatose, vegetative and minimally responsive patient. *J Head Trauma Rehabil.* 1995;10(1);40–56.

195. Whyte J, Glenn MB. The care and rehabilitation of the patient in a persistent vegetative state. *J Head Trauma Rehabil.* 1986;1:39–53.

196. Sandel ME, Ellis DW, eds. The coma emerging patient. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus; 1990.

197. Brooks N, McKinlay W, Symington C, Beattie A, Campsie L. Return to work within the first seven years of severe head injury. *Brain Injury*. 1987;1:5–19.

198. Heaton RK, Pendleton MG. Use of neuropsychological tests to predict adult patients' everyday functioning. *J Consulting Clin Psychology.* 1981;49:807–821.

199. Dikmen S, Reitan RM, Temkin NR. Neuropsychological recovery in head injury. *Arch Neurol.* 1983;40:333–338.

200. Jennett B, Snoek J, Bond MR, Brooks N. Disability after severe head injury: Observations on the use of the Glasgow Outcome Scale. *J Neurol Neurosurg Psychiatry*. 1981;50:1613–1618.

201. Jennett B, Teasdale G. *Management of Head Injuries*. Philadelphia, Pa: FA Davis; 1981.

202. Jennett B, Bond M. Assessment of outcome after severe brain damage: A practical scale. *Lancet*. 1975;1:480–487.

203. Dikmen S, Machamer J, Temkin N, McLean A. Neuropsychological recovery in patients with moderate to severe head injury: 2 year follow-up. *J Clin Experimental Neuropsychology.* 1990;12:507–519.

204. Oddy M, Coughlan T, Tyerman A, Jenkins D. Social adjustment after closed head injury: A further follow-up 7 years after injury. *J Neurol Neurosurg Psychiatry*. 1985;48:564–568.

205. Brooks N, Campsie L, Symington C, Beattie A, McKinlay W. The effects of severe head injury on patient and relative within seven years of injury. *J Head Trauma Rehabil.* 1987;2:1–13.

206. Leininger BL, Kreutzer JS, Hill MR. Comparison of minor and severe head injury emotional sequelae using the MMPI. *Brain Injury*. 1991;5:199–205.

207. Thomsen IV. Late outcome of severe blunt head trauma: A 10–15 year follow-up. *J Neurol Neurosurg Psychiatry.* 1984;47:260–268.

208. Lezak MD. Relationships between personality disorders, social disturbances, and physical disability following traumatic brain injury. *J Head Trauma Rehabil.* 1987;2:57–69.

209. Ben-Yishay Y, Silver S, Piasetsky E, Rattok J. Relationship between employability and vocational outcome after intensive holistic cognitive rehabilitation. *J Head Trauma Rehabil.* 1987;2:35–48.

210. Rappaport M, Herrero-Backe C, Rappaport ML, Winterfield KM. Head injury outcome up to ten years later. *Arch Phys Med Rehabil.* 1989;70:885–892.

211. Thomsen IV. The patient with severe head injury and his family. *Scand J Rehabil Med.* 1974;6:180–183.

212. McKinlay WW, Brooks DN, Bond MR, Martinage DP, Marshall MM. The short-term outcome of severe blunt head injury as reported by relatives of injured persons. *J Neurol Neurosurg Psychiatry*. 1981;44:527–533.

213. Hathaway SR, McKinley JC. *Manual for the Minnesota Multiphasic Personality Inventory*. Minneapolis: University of Minnesota Press; 1982.

214. Clark W, Midanik L. *Alcohol use and alcohol problems among U.S. adults: Results of the 1979 national survey*. In: US Department of Health and Human Services, Alcohol and Health Monograph. No. 1: Alcohol consumption and related problems. Washington, DC: US Government Printing Office; 1982. DHHS Pub No ADM 82-1190.

215. Institute of Medicine, National Academy of Sciences. *Alcoholism, Alcohol Abuse, and Related Problems: Opportunities for Research.* Washington, DC: National Academy Press; 1980.

216. Greer BG, Roberts R, May G, Jenkins WM. Identification of substance abuse in a vocational evaluation setting. *J Rehabil.* 1988;54:42–45.

217. Johnson P, Armor D, Polich S, Stambol H. *U.S. adult drinking practices: Time, trends, social correlates, and sex roles*. Draft report prepared for the National Insitute on Alcohol Abuse and Alcoholism under Contract No ADM 281-76-0200. Santa Monica, Calif: Rand Corporation; 1977.

218. Rimel R, Giordani B, Barth J, Jane J. Moderate head injury: Completing the clinical spectrum of brain trauma. *Neurosurgery*. 1982;11(3):344–351.

219. Zasler ND. Neuromedical aspects of alcohol use following traumatic brain injury. *J Head Trauma Rehabil.* 1991;6(4):78–80.

220. Sparadeo FR, Gill D. Effects of prior alcohol use on head injury recovery. *J Head Trauma Rehabil.* 1989;4:75–82.

221. Galbraith S, Murray W, Patel A, Knill-Jones R. The relationship between blood alcohol and head injury and its effect on the consciousness level. *Br J Surg.* 1976;63:128–130.

222. Murray PK. Clinical pharmacology in rehabilitation. In: Caplan B, ed. *Rehabilitation Psychology Desk Reference*. Rockville, MD: Aspen Publishers; 1987.

223. Greer BG. Substance abuse among people with disabilites: A problem of too much accessibility. *J Disability.* 1986;14:34–38.

224. Hackler E, Tobis JS. Re-integration into the community. In: Rosenthal M, Griffith E, Bond M, Miller J, eds. *Rehabilitation of the Head Injured Adult*. Philadelphia, Pa: FA Davis; 1983.

225. Rohe D, DePompolo R. Substance abuse policies in rehabilitation medicine departments. *Arch Phys Med Rehabil.* 1985;66:619–624.

226. Brooks N, Campsie L, Symington C, Beattie A, McKinlay W. The five year outcome of severe blunt head injury: A relative's view. *J Neurol Neurosurg Psychiatry.* 1986;49:764–770.

227. Devany CW, Kreutzer JS, Halberstadt LJ, West DD. Referrals for supported employment after brain injury: Neuropsychological, behavioral, and emotional characteristics. *J Head Trauma Rehabil.* 1991;6(3):59–70.

228. Lezak MD. *Neuropsychological Assessment*. 2nd ed. New York: Oxford University Press; 1983.

229. Ruff RM, Baser CA, Johnston JW, et al. Neuropsychological rehabilitation: An experimental study with head-injured patients. *J Head Trauma Rehabil.* 1989;4(3):20–36.

230. Hart T, Hayden ME. The ecological validity of neuropsychological assessment and remediation. In: Uzzell BP, Gross Y, eds. *Clinical Neuropsychology of Intervention*. Boston: Martinus Nijhoff; 1986: 21–50.

231. Kreutzer JS, Leininger BE, Sherron PD, Groah CH. Managing psychosocial dysfunction. In: Wehman P, Kreutzer JS, eds. *Vocational Rehabilitation for Persons with Traumatic Brain Injury*. Rockville, MD: Aspen Publishers; 1990.

232. Barth JT, Macciocchi SN, Giordani B, Rimel R, Jane JA, Boll TJ. Neuropsychological sequelae of minor head injury. *Neurosurgery*. 1983;13:529–533.

233. Leininger BE, JS Kreutzer. Neuropsychological outcome of adults with mild traumatic brain injury: Implications for clinical practice and research. In: Horn LJ, Zasler ND, eds. Rehabilitation of Post-concussive Disorders. *Physical Medicine and Rehabilitation: State of the Art Reviews*. Philadelphia, Pa: Hanley & Belfus. 1992;6(1):169–182.

234. Anastasi A. *Psychological Testing*. 6th ed. New York: Macmillan; 1988.

235. Kreutzer JS, Devany CW, Myers SL, Marwitz JH. Neurobehavioral outcome following traumatic brain injury: Review, methodology, and implications for cognitive rehabilitation. In: Kreutzer J, Wehman P, eds. *Cognitive Rehabilitation for Persons with Traumatic Brain Injury: A Functional Approach*. Baltimore, Md: Paul H Brooks; 1991: 55–71.

236. Beck AT. *Depression: Clinical, Experimental, and Theoretical Aspects*. New York: Harper & Row; 1967.

237. Beck AT, Ward C, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. *Arch Gen Psychiatry.* 1961;4:561–571.

238. Dahlstrom WG, Welsh GS, Dahlstrom LE. *An MMPI Handbook, Volume 1: Clinical Interpretation*. Minneapolis: University of Minnesota Press; 1972.

239. Graham JR. *The MMPI: A Practical Guide*. 2nd ed. New York: Oxford University Press; 1987.

240. Kreutzer JS, Leininger BE, Harris JA. The evolving role of neuropsychology in community integration. In: Kreutzer J, Wehman P, eds. *Community Integration Following Traumatic Brain Injury*. Baltimore, Md: Paul H Brooks; 1990.

241. Novack TA, Daniel MS, Long CJ. Factors related to emotional adjustment following head injury. *Int J Clin Neuropsychology*. 1984;6:139–142.

242. Kreutzer JS, Wehman P. *Community Integration Following Traumatic Brain Injury*. Baltimore, Md: Paul H Brooks; 1990.

243. Gordon WA, Hibbard MR. The theory and practice of cognitive remediation. In: Kreutzer J, Wehman P, eds. *Community Integration Following Traumatic Brain Injury*. Baltimore, Md: Paul H Brooks; 1990: 13–22.

244. McMordie WR, Barker SL, Paolo TM. Return to work after head injury. *Brain Injury*. 1990;4:57–69.

245. Wrightson P, Gronwall D. Time off work and symptoms after minor head injury. *Brain Injury*. 1981;12:445–454.

246. Humphrey M, Oddy M. Return to work after head injury: A review of post-war studies. *Brain Injury*. 1980;12: 107–114.

247. Rao N, Rosenthal M, Cronin-Stubbs D, Lambert R, Barnes P, Swanson B. Return to work after rehabilitation following traumatic brain injury. *Brain Injury*. 1990;4:49–56.

248. Dresser A, Meirowsky A, Weiss G, McNeel M, Simon G, Caveness W. Gainful employment following head injury: Prognostic factors. *Arch Neurol.* 1973;29:111–116.

249. Kreutzer JS, Wehman P, Morton MV, Stonnington HH. Supported employment and compensatory strategies for enhancing vocational outcome following traumatic brain injury. *Brain Injury*. 1988;2:205–223.

250. Kreutzer JS, Morton MV. Supported employment for persons with head injury. In: Wehman PS, Moon MS, eds. *Vocational Rehabilitation and Supported Employment*. Baltimore, Md: Paul H Brooks; 1988.

251. Wood W. Supported employment for persons with physical disabilities. In: Wehman PS, Moon MS, eds. *Vocational Rehabilitation and Supported Employment*. Baltimore, Md: Paul H Brooks; 1988.

252. Wehman P, Moon MS. *Vocational Rehabilitation and Supported Employment*. Baltimore, Md: Paul H Brooks; 1988.

253. Kozloff R. Networks of social support and the outcome from severe head injury. *J Head Trauma Rehabil.* 1987;2:14–23.

254. Jacobs HE. The Los Angeles Head Injury Survey: Procedures and initial findings. *Arch Phys Med Rehabil.* 1988;69:425–431.

255. Lezak MD. Living with the characterologically altered brain injured patient. *J Clin Psychiatry.* 1978;39:111–123.

256. Mauss-Clum N, Ryan M. Brain injury and the family. *J Neurosci Nurs.* 1981;13:165–169.

257. Oddy M, Humphrey M, Uttley D. Stresses upon the relatives of head-injured patients. *Br J Psychiatry.* 1978;133:507–513.

258. Florian V, Katz S, Lahav V. Impact of traumatic brain damage on family dynamics and functioning: A review. *Brain Injury*. 1989;3:219–233.

259. Kreutzer JS, Marwitz JH, Kepler K. Traumatic brain injury: Family response and outcome. *Arch Phys Med Rehabil.* 1992;73(8):771–778.

260. Lezak M. Brain damage is a family affair. *J Clin Exp Neuropsychol.* 1988;10:111–123.

261. Rosenbaum M, Najenson T. Changes in life patterns and symptoms of low mood reported by wives of severely brain-injured soldiers. *J Consulting Clin Psychology.* 1976;44:881–888.

262. Panting A, Merry PH. The long term rehabilitation of severe head injuries with particular reference to the need for social and medical support for the patient's family. *Rehabilitation*. 1972;38:33–37.

263. Livingston MG, Brooks DN, Bond MR. Three months after severe head injury: Psychiatric and social impact on relatives. *J Neurol Neurosurg Psychiatry.* 1985;48:870–875.

264. Snaith RP, Ahmed SN, Mehta S, Hamilton M. Assessment of the severity of primary depressive illness. *Psychol Med.* 1971;1:143–149.

265. Snaith RP, Bridge GW, Hamilton M. The Leeds Scales for the self-assessment of anxiety and depression. *Br J Psychiatry.* 1976;128:156–165.

266. Lezak MD. Psychological implications of traumatic brain damage for the patient's family. *Rehabil Psych.* 1986;31:241–250.

267. Bishop DS, Miller IW. Traumatic brain injury: Empirical family assessment techniques. *J Head Trauma Rehabil.* 1988;3:16–30.

268. Epstein NB, Baldwin LM, Bishop DA. The McMaster family assessment devise. *J Marital Fam Ther*. 1983;9:171–180.

269. Derogatis LR. The *SCL-90 Manual: Scoring, Administration, and Procedures for the SCL-90.* Baltimore, Md: Johns Hopkins University School of Medicine; 1977.

270. Kanner A, Coyne J, Schaefer D, Lazarus R. Comparison of two modes of stress measurement: Daily hassles and uplifts versus major life events. *J Behav Med.* 1981;4:1–39.

271. Gilson B, Bergner M, Bobbitt R, Carter W. *The Sickness Impact Profile: Final Development and Testing.* Seattle, Wash: Center for Health Services, University of Washington; 1979.

272. Tarter SB. Factors affecting adjustment of parents of head trauma victims. *Arch Clin Neuropsychology.* 1990;5:15–22.

273. Auerbach SM. Assumptions of crisis theory and a temporal model of crisis intervention. In: Auerbach SM, Stolberg A, eds. *Crisis Intervention with Children and Families*. Washington: Harper & Row; 1986.

274. Peters LC, Stambrook M, Moore AD, Esses L. Psychosocial sequelae of closed head injury: Effects on the marital relationship. *Brain Injury*. 1990;4:39–47.

275. Walker AE. Long term evaluation of the social and family adjustment to head injuries. *Scand J Rehabil Med*. 1972;4:5–8.

276. Livingston MG, Brooks DN, Bond MR. Patient outcome in the year following severe head injury and relatives' psychiatric and social functioning. *J Neurol Neurosurg Psychiatry.* 1985;48:876–881.

277. Brooks N, McKinlay W. Personality and behavioural change after severe blunt head injury a relative's view. *J Neurol Neurosurg Psychiatry.* 1983;46:336–344.

278. Olson DH, Portner J, Lavee Y. *FACES-III: Family Adaptability and Cohesion Scales*. St. Paul, Minn: Family Social Science, University of Minnesota; 1985.

279. Moos R, Moos B. *Family Environment Scale: Manual*. Palo Alto, Calif: Consulting Psychologists Press; 1974.

280. Kreutzer JS. *Family Needs Questionnaire*. Richmond, VA: Rehabilitation Research and Training Center on Severe Traumatic Brain Injury, Medical College of Virginia; 1988.

281. Rosenthal M, Young T. Effective family intervention after traumatic brain injury: Theory and practice. *J Head Trauma Rehabil.* 1988;3:42–51.

THE VIETNAM HEAD INJURY STUDY: OVERVIEW OF RESULTS TO DATE

Excerpted from Andres M. Salazar, M.D., Colonel, Medical Corps, U.S. Army (Ret); H. M. Brown; Karen Schwab, Ph.D.; Jordan Grafman, Ph.D. Unpublished report, 1997. VHIS Collaborative Group, Vietnam Head Injury Study, Building 1, Walter Reed Army Medical Center, Washington, DC 20307-5001.

INTRODUCTION

The Vietnam Head Injury Study (VHIS), a collaborative project of the Department of Defense (DOD), the Veterans Administrations (VA, now the Department of Veterans Affairs [DVA]), National Institutes of Health (NIH), and the American Red Cross (ARC), involves a detailed follow-up evaluation of 1,221 veterans who survived brain wounds during the Vietnam War. The VHIS was conceived and developed by William F. Caveness at NIH in Bethesda, Maryland, who had also been responsible for similar studies of Korean War veterans. At the beginning of the Vietnam conflict he developed a one-page head and spinal injury registry form and secured the support for disseminating it throughout the Vietnam theater. Registry forms were completed by neurosurgeons in field hospitals throughout Vietnam on their head-injured patients who had survived the first week postinjury. Completed forms were returned to Dr. Caveness at NIH on patients injured from 1967 to 1970, when the initial registry was closed at 2,000 cases.

The primary intent was to register a large number of wounded men for a prospective follow-up study, and not necessarily to obtain a random sample of all head wounds incurred in combat. Nevertheless, patients were entered from army, navy, marine, and air force units throughout the theater of operations and represented a wide spectrum of injury types and severity; the sample amounts to about 7% of all casualties with head injuries in the Vietnam War. The registry forms included demographic data, as well as information from the field on the circumstances of the casualty's injury, wound location, initial status, early treatment, and initial hospital course. Complete military medical and VA follow-up records were subsequently obtained on 1,221 registry cases; of these, 77% had suffered missile fragments wounds, 15% had gunshot wounds, and only 8% had a closed head injury.

The VHIS registry consists of a population with unique advantages for the study of head injury outcomes. Prior to their injuries, these men were healthy, young, and employed. In addition, preinjury intelligence test data were available for these casualties, having been collected as part of their evaluation on entering the military.

EVALUATION AND ANALYSIS OF DATA

The Vietnam Head Injury Study is a rich source of knowledge about the nature of militarily relevant head injuries. This brief review of the study to date cites some of the important findings of the VHIS group, with special emphasis placed on findings and conclusions that are relevant to the rehabilitation of military personnel with head injuries. Analysis of the VHIS data was carried out in two phases, the second of which is still ongoing:

- Phase I of the VHIS consisted of a detailed, standardized review and computerization of all medical records by a small group of experienced neurosurgeons and neurologists.
- In Phase II, an unbiased sample of 520 of these casualties, some 15 years postinjury, along with 86 uninjured Vietnam veterans as controls, underwent an extensive, standardized, 1-week-long, multidisciplinary, inpatient evaluation at Walter Reed Army Medical Center (WRAMC), Washington, DC. By the end of this evaluation, over 22,000 datapoints had been gathered and computerized on each Phase II participant.

Phase I: Records Review

Phase I of the VHIS began in 1975, some 5 years after the end of data collection. Medical records of all military and VA hospitalizations and all outpatient care visits were collected. After excluding patients with associated spinal cord injury and those for whom adequate medical records could not be located, the formal registry was reduced to 1,221 men (1,133 with penetrating head injury and 88 with closed head injury). A detailed review and codification of these records was undertaken by a small group of experienced neurologists and neurosurgeons under the direction of W. F. Caveness. About 2,000 datapoints were recorded on each patient and organized into various domains for analysis. Several reports have been published based on this data: on posttraumatic epilepsy,[1] retained bone fragments and abscess,[2,3] motor recovery,[4] and other neurosurgical issues.[5]

Phase II: Follow-up Evaluation

Phase II of the VHIS, organized in 1981 by Dr. Caveness, was made possible by a unique collaboration of the three military services, the DVA, and the ARC. The ARC contacted patients and conducted the initial in-home interviews;

the US Air Force provided transportation; WRAMC provided the hospital beds and some personnel; the National Naval Medical Center, Bethesda, Maryland, provided the computed tomography (CT) scans; and the DVA provided operational funds for the project.

Registry patients were located by the ARC and were invited to participate in the follow-up study. Trained ARC workers conducted a social work and family history interview, and volunteers who were able then came to WRAMC for a week-long hospital evaluation. Of the 1,125 men still alive at the time, more than 750 initially agreed to participate and underwent the ARC interview, although only 520 were actually able to come to WRAMC over the next 3 years (1981–1984).

Control subjects were uninjured Vietnam veterans selected from DVA beneficiary files. Of those invited to participate, 86 came to WRAMC and received the full VHIS evaluation battery (except for the CT scan).

The extensive multidisciplinary evaluation included the following:

- neurology history and examination (2 h),
- neuropsychological evaluation (16 h),
- speech and language evaluation (6 h),
- rehabilitation/motor function evaluation (4 h),
- audiology examination (2 h),
- CT scan, and
- electroencephalography (EEG) and visual and auditory evoked potentials.

CT scans were performed on a General Electric 8800 Scanner in standard cuts. Involvement of specific brain areas was coded for computer entry by using templates prepared for each slice, assigning code numbers to specific brain structures or areas. Structures were coded as normal, partly involved, fully involved, or unreadable due to metal artifact.

RESULTS: PHASE II FOLLOW-UP STUDY

Perhaps the most encouraging finding to date has been the amazing ability of many of these young men to compensate for their injuries despite the large size of many of their brain wounds. CT scanning showed that 80% of the registry patients had injuries involving multiple lobes of the brain, and 33% had bilateral injury (the injuries were thus much larger than had previously been estimated from surgical reports and X-ray examinations of the skull alone, the only means of assessing brain wound size in the Vietnam era). To the casual observer, almost two thirds of these patients might appear to be functioning normally. Nevertheless, careful examination almost invariably revealed some neurological or neurobehavioral functional deficit. Unrecognized cognitive and especially memory deficits often resulted in a failure to seek medical help or veteran's benefits; many patients with large brain wounds had been returned to duty after cranioplasty and had eventually received nonmedical discharges from the military services. Thirty-eight percent of our brain-injured patients (vs 28% of controls) received a recommendation for psychological intervention or therapy, although many had previously undergone such therapy before participation in the VHIS. Overall, recommendations for neurological or psychological follow-up were made in 72% of the brain-injured patients and 52% of the controls; the brain-injured group received more recommendations per person than the controls. Much of the analysis made possible the formulation of guidelines, which may be useful in (1) predicting the eventual outcome in brain-injured patients, (2) providing such patients and their families some insight into difficulties that they may expect, and (3) targeting specific therapies for them early in their convalescence.[6]

Mortality

A 15-year mortality study on men in the registry showed 90 deaths (8%). Most of the deaths occurred early in the first year after trauma and were secondary to the direct effects of brain injury or the sequelae of coma. Complications, particularly infections, were significant mortality factors. Coma was the best prognostic predictor of early death. Posttraumatic epilepsy was not related to mortality except for the risks accompanying each seizure. The population began to approach the actuarial mortality norm of their peers within about 3 years of injury.[5]

Neurology and Neurosurgery

Thanks in large part to helicopter evacuation and the deployment of neurosurgeons close to the battlefront, a wounded soldier in Vietnam usually received prompter and better medical care for such wounds than was available anywhere else in the world at that time. Most men had definitive neurosurgery within 6 hours of injury, but a preliminary analysis of complication rates by delay in provision of initial surgery suggests that mortality and morbidity begin to rise significantly only with delays of longer than 24 to 48 hours. Combined with data on early hospital mortality, this type of information may be important for establishing military medical logistical and evacuation policy

in future conflicts (in which battlefield conditions and distance from hospitals, for example, might make such prompt care impossible to provide).

Retained Bone Fragments

One important and controversial neurosurgical issue addressed has been the significance of retained bone within the intracerebral wound tract. The experience of previous wars had suggested that retained bone fragments increased morbidity and mortality. It thus became standard operating procedure in Vietnam to remove such fragments surgically, even if this called for repeated brain operations in otherwise healthy, convalescing patients. Over 10% of our patients thus underwent repeat surgery for this purpose, some of them multiple times. Retrospective analysis of CT scans now shows that more than 20% of the VHIS population still have retained bone fragments, including almost 75% of those who had had secondary surgery for removal of such fragments. Moreover, a detailed review of the medical records of each of these men shows that in this population, retained bone, per se, has no significant effect on mortality, morbidity (including infection rate), or sequelae of brain injury. This strongly suggests that repeat operations for retained bone, in the absence of complications, are not warranted and may be detrimental.[7]

Other neurosurgical questions that are currently being addressed in the data include the relation of ventricular enlargement to intraventricular wounds, clinical and cognitive deficits, and eventual community adjustment; and the relation of surgical complications such as infection to wound type, fragment type and size, surgical procedure, spinal fluid leaks, and eventual outcome.

Posttraumatic Epilepsy

The incidence of posttraumatic epilepsy (PTE) some 15 years after injury in the VHIS was 51%. This overall incidence appears to be somewhat higher than figures reported for previous wars (World War I, 38%; World War II, 34%, 43%; Korea, 36%), the Iran–Iraq and Lebanese conflicts, and even for these same patients at an average 5-year follow-up (34%).[1] Explanations of this apparent discrepancy include the longer follow-up, the fact that detailed histories were available in person from the patient and family, and inclusion in the VHIS cohort of patients with injuries so severe that they would not have survived in previous wars. In 57% of the Vietnam group with seizures, attacks began within 1 year of injury; in 18%, 5 to 10 years after injury; and in 7%, 10 or more years after injury. When compared with a normal age-matched population, the relative risk of epilepsy in the Vietnam cohort was 520 in the first year after injury, 90 in years 2 to 5, and 36 in years 5 to 10. At years 10 to 15 postinjury, the relative risk of developing PTE was still 25 times higher than normal.

A number of clinical and injury factors were found to be associated with PTE. As expected from prior studies, total brain volume loss on CT was significantly associated with PTE ($P = .0001$), as was the presence of hematoma ($P = .01$) or retained metal fragments ($P = .02$). However, tangential high-velocity gunshot wounds, retained bone fragments, use of a dural graft for closure, cranioplasty, and brain abscess showed no relationship to PTE. Similarly, preexisting factors such as family history of epilepsy or preinjury intelligence as measured by the Armed Forces Qualification Test had no impact on incidence of PTE. Among neurological outcomes, hemiparesis ($P = .03$), aphasia ($P = .009$), organic mental disorder (DSM III) ($P = .01$), visual field loss ($P = .01$), or headache ($P = .001$) were all associated with seizures, but traumatic loss of consciousness, either immediate or at first neurological examination, was not. Neither subsequent head injury, other encephalopathy, nor alcohol abuse played important roles in occurrence, particularly in late-onset cases.[8]

Motor Function

Forty-seven percent of our patients were recorded as having a paralysis early after injury, and about half of those have now recovered. Analysis of the clinical and anatomical correlates of recovery from hemiparesis has resulted in a simple initial model that may allow us to predict which patients will recover. Clinical findings significantly ($P \leq .05$) associated with nonrecovery were sensory loss, organic mental disorder, abnormal EEG, partial simple seizures, and an initial extensor plantar response. Anatomical correlates included large, total brain volume loss and involvement of the following anatomical structures on CT: sensory-motor cortex, supplementary motor area, posterior temporal cortex, temporal white matter, and the posterior limb of the internal capsule. Clinical and anatomical factors were then allowed to interact in a stepwise logistical regression model comparing unrecovered patients to those with delayed recovery (> 1 mo postinjury). Items significantly ($P < .05$) predicting recovery in this model were involvement, seen on CT scan, of (1) vertex or medial sensory motor cortex, (2) central corona radiata and caudate body, (3) extensor plantar response, and (4) sensory loss, in that order. Probability of recovery was .05 for patients with all items present and .97 when all were absent. This model was 82% accurate.[9] Most patients who are going to recover

motor functions will do so within the first 6 months after injury (15% recover within 1 mo), but a small percentage may not do so for several years.

Considerable ipsilateral as well as contralateral deficits in complex hand motor functions can be found in patients with lesions in the frontal and parieto-occipital lobes, even in the absence of an overt hemiparesis. This is most pronounced in patients with right hemisphere brain injuries and in right-handed individuals.[10] Preliminary analysis of our data also shows that the relation of persistent hemiparesis to eventual successful community adjustment is not direct and that other factors, primarily cognitive status, may play a more important role than paralysis per se. Ongoing follow-up studies will clarify this relationship. Further analyses of the pattern of motor recovery, the relation of paresis to language function, and the relation of spasticity to lesion location are also planned.

Traumatic Unconsciousness and Amnesia

Analysis of traumatic unconsciousness and amnesia in the VHIS casualty database showed that only 15% of the patients had prolonged unconsciousness and 53% had no or only momentary unconsciousness after injury, emphasizing the focal nature of these wounds. There was a clear dominance of the left (or language-dominant) hemisphere for the "wakefulness" or vigilance component of consciousness. The areas of the posterior limb of the left internal capsule, the left basal forebrain, midbrain, and hypothalamus were most associated with unconsciousness. Left dominance is not seen for posttraumatic amnesia after elimination of the "wakefulness" variable, suggesting that the latter may be linked to the well-recognized role of the left hemisphere in certain verbal memory processes.[11] This particular analysis illustrates another example of the functional asymmetry of the two halves of the brain and has also helped to sharpen the distinction between the two major aspects of the arousal component of consciousness: "wakefulness" (left hemisphere) and attention (right hemisphere).

Basal Forebrain Lesions and Cognition

The neurological and cognitive performance of 15 young veterans who suffered unilateral penetrating missile wounds of the basal forebrain was compared with that of patients without basal forebrain lesions and uninjured controls.[12] They did somewhat more poorly on tests of episodic memory, reasoning, and arithmetic, and had more prolonged unconsciousness than patients with lesions elsewhere in the brain. However, their performance on tests of intelligence, attention, and language was not consistent with that of demented patients. These results suggest that the basal forebrain may be a component of limbic-hippocampal memory processing systems, but it is not responsible for the maintenance of cognitive processing in general.

Electrophysiological Studies

The relationship between EEG findings and clinical and radiological features was studied in the first 300 VHIS subjects. EEGs were performed on 16- and 18-channel Grass equipment using the international 10–20 system. Fifty age-matched Vietnam veterans were used as controls. The EEG was abnormal in 48% of the patients. Epileptiform findings (EF; spikes or spikes wave) were found in 15% of the records and focal slowing (FS) in 38%. Of the patients with EF, 80% had one or more seizures after head injury, compared with 64% of patients with FS and 41% of patients with normal EEGs. Epileptiform findings were seen in 16% of patients who had had their initial seizures during the first year following head injury but in only 7% of those with onset after 5 years. EEG was normal in 31% of the former group and 71% of the latter. No correlation was found between EF and family history of epilepsy, seizure frequency in first year after injury, or seizure persistence. Both EF and FS correlated significantly with hemiparesis ($P = .0001$), aphasia ($P = .00074$), and CT scan revealed evidence of deep cerebral injury ($P = .0004$).[13]

Another analysis studied the relationship between visual evoked potentials (VEPs), perimetry, clinical, and CT findings of the first 150 patients in our study. Full field (FF) and half field (HF) responses were obtained via a television screen that delivered checkerboard-pattern reversal stimuli at the rate of 2.1 per second. Responses were recorded on four medial and lateral occipital electrodes simultaneously, placed 5 cm and 10 cm from the midline. Visual fields were obtained by Goldmann perimetry and CT scans by a General Electric 8800 scanner. Fifty age-matched Vietnam veterans served as controls. Fourteen patients (9%) showed a mono-ocular delay of VEP on the side of head injury. Seven of these patients had no visual complaints, suggesting that VEP detected a subclinical traumatic macular or optic nerve dysfunction. HF stimulation and perimetry produced concordant data in 88% of the patients. When abnormal, both tests correlated highly with a parieto-occipital site of injury. In six patients, abnormality of HF-VEP pointed correctly to the side of head injury but perimetry was normal; while in a few patients, perimetry showed small hemianopic field defects and HF-VEP missed them. These data indicate that HF-VEP is a sensitive measure of optic radiation dysfunction in penetrating head injury. Information derived from HF-VEP and perimetry complement each other in retrochiasmatic brain lesions.[14]

Audiology

Analysis of central auditory testing on 250 VHIS subjects was done, including correlations with CT evidence of damage to eight different regions of the temporal lobe. The location and degree of temporal lobe injury was compared with dichotic speech test results in an effort to establish auditory correlates of physical damage. Results indicate that speech test scores are significantly affected by injury site. In addition, three dichotic speech tests were administered to 300 individuals with brain injury in various locations. The sensitivity of each test was studied relative to the percentage of normal/abnormal scores for specific injury groups. A high rate of false-negative and false-positive results was present for all measures. The three dichotic tests did not vary substantially in their ability to detect damage to the right or left temporal lobes.

Separate analyses have also studied the relationship of lesion site to loss of perception of time-compressed speech, which has been reported to be a useful test in the identification and differentiation of central auditory deficits.[15]

Speech and Language

Initial analyses of the speech and language data have included studies of recovery from Broca's aphasia,[16] speech discrimination deficits,[17] and acquired stuttering.[18] The first was designed to determine which language faculties are retained in the chronic form of expressive aphasia, and what characteristics of brain lesions differentiated between patients who recovered and those who did not recover from expressive aphasia within 15 years following penetrating head injury. Two groups of men who sustained penetrating head injuries and had an expressive aphasia during the first 6 months following injury were examined 15 years later. One group had a chronic expressive nonfluent aphasia; the other had recovered and was without symptoms of aphasia. On a comprehensive battery of speech and language tests, the patients with chronic expressive aphasia demonstrated specific deficits in syntactic processing in all language modalities, while they were within the normal range in other language faculties. The recovered group demonstrated syntactic deficits only in written expressive syntax. The CT lesions of the two groups differed in the extent of left hemisphere lesion volume and the degree of posterior and deep lesion extension. Broca's area was equally involved in both groups but was not involved in all patients in either group. All the nonrecovered group had posterior extension of the lesion to involve Wernicke's area, with some involvement of the underlying white matter and basal ganglia in the left hemisphere.[16]

Speech discrimination and identification tasks assessing voicing and place distinctions were given to 16 unilaterally brain-injured subjects free of aphasic or dysarthric symptoms 12 to 15 years postinjury. Seven subjects did not demonstrate any difficulty with these speech tasks, while five subjects who had been injured on the left side of the brain and four who had been injured on the right showed moderate difficulties. These difficulties were more pronounced on the discrimination than on the identification tasks. Analysis of CT scans demonstrated that the lesion locations most clearly associated with the speech discrimination deficits were upper levels of the white matter subjacent to cortical regions in either hemisphere.[17] Other analyses of the VHIS database now underway will study recovery from Wernicke's aphasia and patients with dysprosody.

Neuropsychology

The Neuropsychology Section of the VHIS was developed to broadly address certain critical issues regarding brain–behavior relationships, the conceptual validity of specific cognitive theories, and the persistence of cognitive deficits and their effect on the clinical course of a patient. One unique characteristic of the VHIS population is the availability of preinjury intelligence testing in the Armed Forces Qualification Test (AFQT). The same test was then administered at follow-up to all patients. We assessed the impact of education, preinjury intelligence, brain volume loss, and lesion location on postinjury intelligence level.[19] We found that, in general, the most important determinant of postinjury intelligence was preinjury performance on the AFQT. One exception was seen in patients with severe left hemisphere lesions, which was not surprising given the linguistic processing demands of the AFQT. In addition, we discovered that the more global a cognitive process, the greater the effect of brain loss volume; that is, specific cognitive processes were affected relatively more by lesion location.[20] This methodological approach will continue to guide our research effort: distinguishing between effects on global versus specific cognitive processes and mood presentations, and their interactions, by considering both anatomical and behavioral variables.

Exploitation of the VHIS data has been proceeding in several separate areas: cognition, mood, injury characteristics, and functional and clinical outcome utilizing lesion location, brain loss volume, and preinjury intelligence as covariates. We believe that the initial studies in each area not only will contribute to the scientific and clinical literature but will also provide the basis for continuing analysis in the future. This continuing analysis is necessary to refine the models of brain–behavior relationships we have only barely begun to construct.

Cognitive Process

One example of a specific cognitive process is the ability to discriminate and recognize faces. Our analysis indicates that *both* hemispheres of the brain contribute to this process, with the left hemisphere storing face knowledge information and the right hemisphere storing procedures that allow for rapid face discrimination and form memory. Face recognition that requires transformation of features (eg, the person has to rely on specific facial features for recognition) seems to require the integrity of the frontal lobe.[21]

A second example of a specific cognitive process involves the semantic encoding of recently presented verbal information. We have tested an individual who presented with a restricted deficit in this process in contrast to superior skills in all other cognitive areas. We argue that of his brain lesions, the critical one for this cognitive process is the one in the columns of the fornix.[22]

Mood Presentation

We have taken a parallel course in examining the mood presentation of our patients. A particularly interesting area of investigation is the effects of frontal lobe lesions on the maintenance of control of anxiety, fear, and hostility. We have demonstrated the rather profound and persistent effects of orbitofrontal lesions on the modulation of feelings of anxiety, dorsofrontal lesions on feelings of sluggishness, and the acute effects of frontal lobe lesions in general on control of anger and hostility.[23] Patients with left dorsofrontal and right orbitofrontal lesions were most disinhibited, edgy, angry, and depressed. Ongoing studies are investigating single cases with limited orbitofrontal lesions, Beck Depression Inventory group profiles, Minnesota Multiphasic Personality Inventory group profiles, and factor analysis of the Bear-Fedio Trait Scales. Our purpose is to develop a rudimentary model of mood state representation and to discover how mood state interacts with cognitive processes.

Although for many years anecdotal reports have linked violent and aggressive behavior to frontal lobe injury, the VHIS gives us an opportunity to examine this issue in a large cohort of well-characterized survivors with injuries to the frontal lobe and other parts of the brain. Using factor analysis and other statistical techniques, VHIS psychosocial and other data were used to generate two indices of violence or aggression in a cohort of 336 subjects and controls. These were then correlated with anatomical lesion location. Results indicate that it is ventromedial frontal lesions that correlate with violence and aggression, which, in this cohort, was usually manifested verbally in family situations.[24]

Psychosocial Outcome

A final analysis scheme addresses the impact of penetrating brain wounds on ultimate functional psychosocial outcome, the major attribute, for purposes of this study, being the subject's ability to return to work. Earlier analyses in Korean War veterans with head injuries indicated that a considerable proportion of such men were able to return to gainful employment. We have investigated the effects of residual impairments resulting from head injury on the work status of the VHIS population as well. Extensive standardized testing of neurological and neuropsychological and social functioning was done some 15 years postinjury on the VHIS subjects (N = 520), as well as on the uninjured controls (N = 85). Fifty-six percent of the head-injured subjects were working at follow-up, compared with 82% of the uninjured controls. Up to 80% of the subjects with head injuries reported having worked at some time postinjury. Furthermore, the occupational distribution of our working, head-injured veterans does not differ significantly from that of the uninjured controls or the age-matched population of the United States.[25] After excluding several patients with severe triplegia or global aphasia, none of whom worked, we investigated the relationship of selected neurological, neuropsychological, and social impairments to the ability to return to work, using factor analysis and multiple logistic regression.

In the final model, we identified seven systematically defined impairments that were significantly related to the ability to return to work. These were posttraumatic epilepsy, hemiparesis, visual field loss, verbal memory loss, visual memory loss, psychological problems (anxiety and depression), and violent behavior. These disabilities had a cumulative and nearly equipotent effect on the likelihood of returning to work. A simple summed score of the number of these seven disabilities can yield a residual "disability score," which may prove to be a practical tool for assessing the likelihood that patients in the VHIS population, and perhaps in other brain-injured populations, will return to work. Patients with up to any three of these impairments had a 65% or better likelihood of returning to work. However, the presence of five or more impairments was associated with only a 20% likelihood of returning to work. These findings may also help to focus rehabilitation efforts on those disabilities most likely to affect the ability to return to work.[26]

SUMMARY

The VHIS contains a rich database on a well-defined group of casualties with penetrating head injuries. This ongoing study will continue to provide material for analysis for years. While many of the questions posed in the

original planning for the study have already been answered, new questions have arisen and will continue to arise as investigators explore the data. The VHIS evaluation has also helped us identify subsets of patients with specific types of wounds or deficits who can be invited to return for more detailed experimental testing that concentrates on their specific disabilities, or on hypothesized functions of the brain areas involved in their injuries. Many of the questions have immediate practical implications for prediction of outcome, therapy, and determination of disability status. However, perhaps the most valuable aspect of the study will be the long-term benefits resulting from a better scientific understanding of brain function and its localization.

REFERENCES

1. Caveness WF, Meirowsky AM, Rish BL, et al. The nature of posttraumatic epilepsy. *J Neurosurg*. 1979;50:545–553.

2. Rish BL, Caveness WF, Dillon JD, Kistler JP, Mohr JP, Weiss GH. Analysis of brain abscess after penetrating craniocerebral injuries in Vietnam. *Neurosurgery*. 1981;9(5):535–541.

3. Rish BL, Dillon JD, Meirowsky AM, et al. Cranioplasty: A review of 1,030 cases of penetrating head injury. *Neurosurgery*. 1979;4(5):381–385.

4. Mohr JP, Weiss GH, Caveness WF, et al. Language and motor disorders after penetrating head injury in Vietnam. *Neurology*. 1980;30(12):1273–1279.

5. Rish BL, Dillon JD, Weiss GH. Mortality following penetrating craniocerebral injuries: An analysis of the deaths in the Vietnam Head Injury Registry population. *J Neurosurg*. 1983;59:775–780.

6. Sweeny JK, Smutok MA. Preliminary analysis of the functional and anatomical sequelae of penetrating head trauma. *Phys Ther*. 1983;63(12):2018–2025.

7. Myers PW, Brophy J, Salazar AM, Jonas B. Retained bone fragments after penetrating brain wounds: Long term follow-up in Vietnam veterans. *J Neurosurg.* 1989;70:319A. Abstract.

8. Salazar AM, Jabbari B, Vance SC, Grafman J, Amin D, Dillon JD. Epilepsy after penetrating head injury, I: Clinical correlates: A report of the Vietnam Head Injury Study. *Neurology*. 1985;35(10):1406–1414.

9. Smutok MA, Vance SC, Salazar AM, Foulkes M, Grafman J. Neurologic and anatomic correlates of recovery from hemiparesis following penetrating head injury. *Neurology.* 1985;35(1):83.

10. Smutok MA, Grafman J, Salazar AM, Sweeney JK, DiRocco PJ. The effects of unilateral brain damage on contralateral and ipsilateral upper extremity function in hemiplegia. *Phys Ther*. 1989;69:195–203.

11. Salazar AM, Grafman J, Vance SC, Weingartner H, Dillon JD, Ludlow CL. Consciousness and amnesia after penetrating head injury: Neurology and anatomy. *Neurology*. 1986;36:178–187.

12. Salazar AM, Grafman J, Schlesselman S, et al. Penetrating war injuries of the basal forebrain: Neurology and cognition. *Neurology*. 1986;36(4):459–465.

13. Jabbari B, Vengrow MI, Salazar AM, Harper MG, Smutok MA, Amin D. Clinical and radiological correlates of EEG in the late phase of head injury: A study of 515 Vietnam veterans. *Electroencephalogr Clin Neurophysiol.* 1986;64:285–293.

14. Jabbari B, Vance SC, Harper MG, Salazar AM, Smutok AM, Amin D. Clinical and radiological correlates of somatosensory evoked potentials in the late phase of head injury: A study of 500 Vietnam veterans. *The EEG Journal*. 1987;67:289–297.

15. Mueller HG, Sedge RK, Salazar AM. Central auditory nervous system dysfunction: Preliminary findings of the Vietnam Head Injury Study. In: Miner ME, Wagner KA, eds. *Neurotrauma: Treatment, Rehabilitation and Related Issues*. Boston, Mass: Butterworth; 1985: 155–188.

16. Ludlow C, Rosenberg J, Fair C, Buck D, Schlesselman S, Salazar AM. Brain lesions associated with nonfluent aphasia 15 years following penetrating head injury. *Brain*. 1986;109:55–80.

17. Yeni-Komshian GH, Ludlow CL, Rosenberg J, Fair C, Salazar AM. Lesion locations associated with speech perception deficits following penetrating head injury. *Neuropsychologia*. 1986;24:631–647.

18. Ludlow CL, Rosenberg J, Salazar A, Grafman J, Smutok M. Site of penetrating brain lesions causing chronic acquired stuttering. *Ann Neurol*. 1987;22:60–66.

19. Grafman J, Jonas BS, Martin A, et al. Intellectual function following penetrating head injuries in Vietnam veterans. *Brain*. 1988;111:169–184.

20. Grafman J, Salazar AM, Weingartner H, Vance SC, Amin D. The relationship of brain-tissue loss volume and lesion location to cognitive deficit. *J Neurosci*. 1986;6:301–307.

21. Grafman J, Salazar AM, Weingartner H, Amin D. Face memory and discrimination: An analysis of the persistent effects of penetrating brain wounds. *Int J Neurosci*. 1986;29:125–139.

22. Grafman J, Salazar AM, Weingartner H, Vance SC, Ludlow CL. Isolated impairment of memory following a penetrating lesion of the fornix cerebri. *Arch Neurol*. 1985;42:1162–1168.

23. Grafman J, Vance SC, Weingartner H, Salazar AM, Amin D. The effects of lateralized frontal lesions upon mood regulation. *Brain*. 1986;109:1127–1148.

24. Grafman J, Schwab K, Warden D, Pridgen A, Brown HR, Salazar AM. Frontal lobe injuries, violence, and aggression: A report of the Vietnam Head Injury Study. *Neurology*. 1996;46:1231–1238.

25. Kraft JF, Schwab KA, Salazar AM, Brown HR. Occupational and educational achievements of head injured Vietnam veterans at 15-year follow-up. *Arch Phys Med Rehabil*. 1993;74:596–601.

26. Schwab K, Grafman J, Salazar AM, Kraft J. Residual impairments and work status 15 years after penetrating head injury: Report from the Vietnam head injury study. *Neurology*. 1993;43:95–103.

Chapter 7

CRANIAL NEUROPATHIES: ELECTRODIAGNOSIS AND MANAGEMENT

MARGARETE DI BENEDETTO, M.D.*

Colonel Medical Corps, US Army (Ret); formerly, Assistant to the Deputy Commanding General, US Army Training Command, Fort Monroe, Virginia; currently, Professor, Physical Medicine and Rehabilitation, University of Virginia, Box 3522 UVA Health Science Center, Charlottesville, VA 22908

INTRODUCTION

Injuries of the cranial nerves (CN) are relatively common, especially secondary to head trauma. Injuries may be direct or indirect, through damage caused by shear, distraction, or compression. Upper cervical lesions can involve the lower CN IX–XII, because the 1st and 2nd cervical vertebrae are in anatomical proximity to the base of the skull and to CN VI, whose intracranial course is precariously long.[1,2] The exact incidence is not known, but numerous reports discuss such injuries in specific patient populations.[3–9]

A large retrospective study by Dobson and associates[10] reviewed head and neck injuries, especially maxillofacial injuries, during 50 military conflicts between 1914 and 1986, and showed an overall mean incidence of 16% of all reported injuries. This exceeds the reported incidence concerning whole body surface area. There was little variation during the period, except for an incidence of 21% in terrorist conflicts.

Blunt trauma to the head mostly affects CN I, VII, and VIII, followed in frequency by injuries to CN III and IV. Injury to CN III is reported especially in pediatric populations.[11,12] Skull crushing injuries have a stretching effect on cranial nerves, with possible avulsion of nuclei from the brainstem and potential disruption of CN II in the optic canal. However, they cause relatively limited brain damage. Penetrating injuries such as gunshot wounds have varying effects, frequently involving CN II or the lower CN X, XI, and XII. CN V is more vulnerable to facial trauma. Orbital or jaw fractures can cause "atypical" (minor or secondary) neuralgia. Atypical neuralgia must be differentiated from "typical" (major or primary) trigeminal neuralgia because of the differing therapeutic approach to each. (See section in this chapter on CN V.)

Surgical trauma of cranial nerves occurs most often during removal of space-occupying lesions at the base of the skull. CN VII and VIII are vulnerable during cerebellopontine angle surgery (acoustic neuroma or meningioma). CN III, IV, V (branches 1 and 2), and VI may suffer injury during exploration of the cavernous sinus. CN III, V, VI, VII, X, XI, and XII are at risk during tumor removal at the petrous ridge. Endartarectomies may be followed by tongue weakness (CN XII) or minor transient facial muscle weakness (CN VII), or both. Vocal cord weakness (CN X) is also possible. Neuromonitoring during surgery employing cranial nerve stimulation and display of evoked potential amplitude changes may lead to decreased morbidity. The most common procedures are facial nerve stimulation, electromyography (EMG) of mimetic muscles, and auditory (brainstem) evoked potentials.[13–16]

If cranial nerves are injured during surgery, reconstruction through end-to-end anastomoses or insertion of nerve grafts may be undertaken. Sekhar and colleagues[17] reported a high success rate for reconstruction of CN III–VI traumatized during surgery.

Nontraumatic lesions can be degenerative, infectious, or vascular (sudden onset), but most frequently they are secondary to tumors (primary or metastatic). Ransom and associates[18] reported a series of patients with prostate cancer and metastases to the base of the skull who presented with symptoms and signs of different cranial nerve lesions.

Prognosis for spontaneous recovery after cranial nerve injury is best for the facial and oculomotor nerves, and poorest for the olfactory, optic, and audiovestibular nerves. Steroid therapy sometimes promotes recovery. Decompression of CN II or VII may result in return of function.[11]

In the comatose patient, early recognition of cranial nerve involvement may be difficult except for CN III, VI, and VII. An incompetent afferent arc of the pupillary reflex indicates a CN II lesion. Due to the effects of cerebral edema or hemorrhagic meningitis, close monitoring for any deterioration in function of CN VI and VII must be maintained, even when initial findings are normal. Transtentorial herniation may cause a delayed CN III lesion, and if this is not recognized early the result can be increased morbidity and mortality. Cranial nerve functions must be reevaluated frequently to ensure an optimal rehabilitation outcome.

The following sections will be concerned with the anatomy and function of the 12 pairs of cranial nerves, and the incidence, diagnosis, prognosis, and therapeutic measures for injuries or diseases affecting them. CN I and II can be regarded as extensions of the brain, and the spinal root of CN XI originating from the cervical spinal cord can be considered a spinal nerve. However, for this discussion these three pairs of nerves will be considered as cranial nerves (Figures 7-1 and 7-2).

Cranial nerves have one or more types of functional components. Special sensory fibers are those

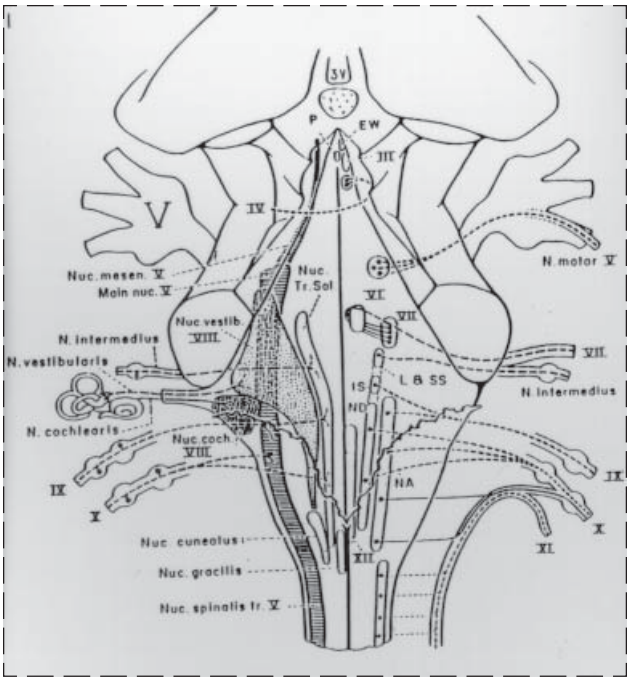


Fig. 7-1. Positions of cranial nerve nuclei III–XII and course of their respective fibers (dorsal view). 3V: Third ventricle; EW: Edinger-Westphal nucleus; P: Perlia's nucleus; Nuc. Tr. Sol.: nucleus tractus solitarius; L and SS: Lacrimal and superior salivatory nuclei; IS: Inferior salivatory nucleus; ND: Dorsal nucleus of vagus nerve; NA: Nucleus ambiguus. Reprinted with permission from Haymaker W, Kuhlenbeck H, Baker AB, Baker LH, eds. Disorders of the brainstem and its cranial nerves. *Clinical Neurology.* Vol 3. Hagerstown, Md: Harper and Row; 1976: 3.

from the special sense organs. Efferent fibers pass from the central nervous system, to skeletal muscle (striated muscle) derived from somites or branchial arches, to visceral (smooth) and cardiac muscle, and to the secretory cells of glands. Afferent fibers transmit sensation from mucous membranes, skin, blood vessels, and internal organs. These cranial nerve functional components are noted below.

1. Special sensory fibers
   - (I, II, VIII): from special sense organs.
2. Efferent fibers
   - Somatic (III, IV, VI, and XII): innervate striated muscle derived from somites;
   - Branchial (V, VII, IX, X, and XI): innervate striated muscle derived from branchial arches;
   - Visceral: parasympathetic preganglionic fibers travel through CN III to smooth muscle within the eye; through CN VII to salivary and lachrymal glands; through CN IX to the parotid gland; and through CN X to the heart, the smooth muscle and glands of the lung and bowel, and to the liver and pancreas.
3. Afferent fibers
   - Visceral: sensation from the heart, lungs, blood vessels, and alimentary tract through CN IX and X; and gustatory fibers through CN VII, IX, and X;
   - Somatic: sensation from skin and mucous membranes of the head through CN V. Some afferent fibers travel with CN VII, IX, and X, but then terminate centrally on the trigeminal nuclei in the brainstem.

Knowing the anatomy and function of each individual cranial nerve is essential for accurate diagnosis and effective treatment of injuries. A detailed discussion follows.

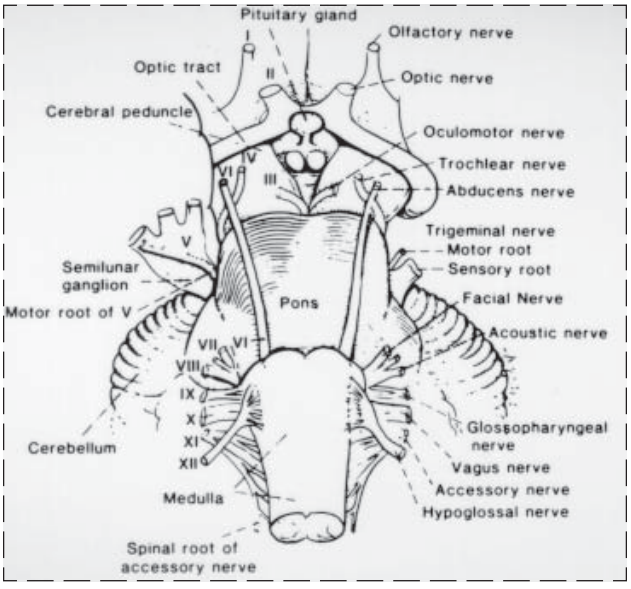


Fig. 7-2. Ventral view of the brain stem with cranial nerves. Reprinted with permission from Brazis P, Masdeu J, Biller JL. *Localization in Clinical Neurology*. 2nd ed. Boston, Mass: Little, Brown; 1990: 271.

CRANIAL NERVE I: OLFACTORY

The olfactory nerve is classified as special sensory and its anatomy is depicted in Figures 7-3 and 7-4.

Anatomy and Function

The primary neurons are bipolar sensory nerve cells located in the lateral wall of the nasal cavity and the posterior nasal septum. The dendrites extend to ciliated receptors in the upper part of the nasal mucosa. The unmyelinated axons (central processes) of these bipolar cells are gathered into bundles of approximately 20 filaments each, which then pass through the cribriform plate of the ethmoid bone to the olfactory bulb. There they synapse with secondary neurons and send myelinated processes to form the olfactory tract. At the anterior perforated substance, the tract divides into medial and lateral striae, forming the olfactory trigone. Some striae fibers decussate in the anterior commissure and terminate in the contralateral cerebral hemisphere, so as to provide bilateral cortical representation for smell. Most of the lateral striae fibers pass to the ipsilateral piriform lobe, the primary olfactory cortex (temporal cortex).

Here the secondary neurons synapse again with tertiary neurons that extend to the endorhinal cortex (area 28), the lateral preoptic area, the amygdaloid body, and the hypothalamus. The central connections of the olfactory nerve are complex. Association fibers to the tegmentum and pons pass directly from the anterior perforated substance and indirectly from the hippocampus via the fornix and olfactory projection tracts through the mamillary bodies and anterior nuclei of the thalamus. Close linkage of the prepiriform cortex and amygdala with the thalamus may enable integration of smell with affective behaviors. Certain reflex connections with the nuclei of other cranial and spinal nerves may be functionally significant for swallowing and digestion.

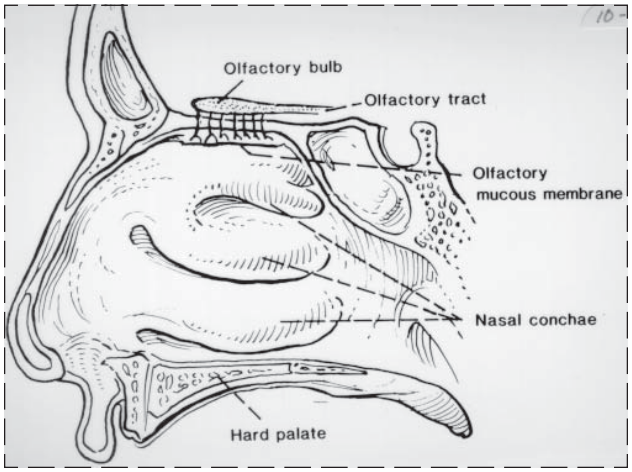


Fig. 7-3. The olfactory nerve (lateral view). Reprinted, with permission from Brazis P,Masdeu J, Biller JL. *Localization in Clinical Neurology.* 2nd ed. Boston, Mass: Little, Brown; 1990: 95.

Injuries and Lesions

The most common cause of injury or lesion is traumatic injury.[19,20] This includes fracture of the cribriform plate, closed head injury without fracture (shearing), or compression of vascular supply by increased intracranial pressure. In some cases a blow to the back of the head (contrecoup coup) may cause injury to CN I.

The incidence of olfactory nerve involvement for all head injuries is approximately 7%, but incidence increases to 20% or 25% following severe head trauma.[6,20] CN I involvement following head trauma is less common in children.[21] Another source of olfactory nerve injury is surgical trauma during procedures in the olfactory region. Nontraumatic lesions include tumors (olfactory groove meningioma may present with anosmia as the sole symptom in the beginning),[22] chronic basilar meningitis,[23] Korsakoff's syndrome,[24] Huntington's chorea,[25] Alzheimer's disease,[25,26] Parkinson's disease,[27] or Foster Kennedy syndrome[28] (ipsilateral anosmia, ipsilateral optic atrophy, and contralateral papilledema due to increasing intracranial pressure). With increasing age, smells are less intense and more difficult to identify and discriminate; the cause of this may be receptor site pathology, or neuronal, hormonal, or neurotransmitter abnormalities.[29]

The causes of loss of smell were studied by Deems and associates[30] in a comprehensive study of 750 consecutively evaluated patients at the University of Pennsylvania Smell and Taste Center. Inflammatory nasal disorders were reported to be responsible for 26%; 15% were due to nasal or paranasal sinusitis; 22% were idiopathic; 18% were due to head trauma; 4% were congenital; 2% were due to toxins; and several other causes each accounted for less than 2%. Intranasal cocaine free-

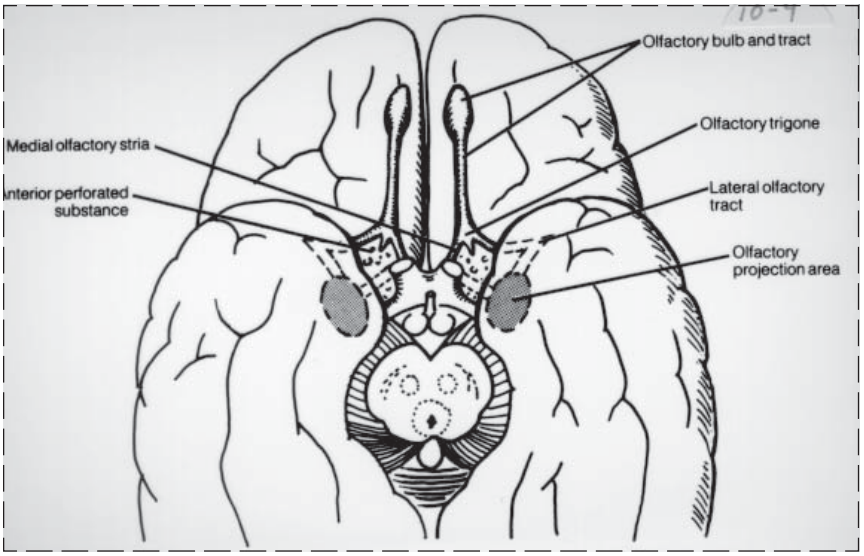


Fig. 7-4. The olfactory nerve (inferior view). Reprinted with permission from deGroot J, Chusid JG. *Correlative Neuroanatomy*. 20th ed. Norwalk, Conn: Appleton & Lange; 1988: 296.

basing (smoking the extracted and volatile form of the street drug) seems to have produced an increased frequency of bony septal and cartilaginous necrosis with osteolytic sinusitis as a cause for loss of smell in patients.[31]

Symptoms and Signs

Patients present with an altered sense of smell, which may be diminished detection and recognition, or heightened awareness.

1. Dysosmia
   - Anosmia: absence of smell. This is most significant when unilateral.
   - Hyposmia: decreased sense of smell.
   - Parosmia: perversion of smell. In addition to head injury, this is found with schizophrenia, uncinate gyrus lesions, hysteria, and other temporal lobe lesions. During recovery from injury, parosmia is often transitory, although it may become permanent.
   - Cacosmia: unpleasant odors, usually due to decomposition of tissues and noticed by the patient while breathing out. Cacosmia can also occur secondary to head injury.
   - Hyperosmia: increased sensitivity to odorants. This may be present in hysteria, cocaine addiction, or pregnancy.
2. Diminished olfactory recognition
   - This may be present despite a relatively preserved olfactory detection. It may occur with blunt trauma affecting the orbitofrontal and temporal lobes.[32]
3. Olfactory hallucination
   - This may be present in psychoses but also may result from structural nerve damage.

Evaluation

Every patient with mild head injury due to an occipital blow or frontal vault fracture should be evaluated carefully for olfactory dysfunction.[33] Peripheral causes of dysosmia such as fractures, nasal mucosal swelling, and other soft tissue nasal obstruction must be ruled out.

The psychophysical examination is a qualitative and quantitative testing of the ability to perceive different fragrances. There are several approaches of increasing sophistication. Tests 1 and 2 below are mainly qualitative in nature.

1. Presentation of nonirritating odorants (chocolate, vanilla, coffee, and the like) to one nostril at a time, while occluding the other nostril.
2. Odor identification test with forced choice format. The patient is offered a choice of four possible responses for each of 40 odorants located on microencapsulated strips.
3. Threshold tests add a quantitative element to the evaluation by offering known concentrations of odorant.
   The Connecticut Chemosensory Clinical Research Center[34] has developed a thresh-

old test that uses eight plastic squeeze bottles containing graded concentrations of butanol and distilled water.

Threshold tests are further refined in the Medical College of Virginia Olfactory Screening Test (MCVOST).[34] In this test, a stimulus such as chocolate, vanilla, or coffee is presented to each nasal cavity using a 20-mL plastic squeeze bottle. Scoring includes stimulus detection (1 point) and identification (1 point) for each stimulus. The maximum score is 6 points for each side or 12 points in all. Lateralization of olfactory function is considered when there is a difference in left and right scores. In one study[35] of 51 consecutive rehabilitation admissions, 60% of mild head injury patients were found to have normal olfactory function; 20% had impaired function; and 13% were anosmic. Patients with severe head injury showed only 8% with normal olfactory function; 67% were impaired; and 25% were anosmic. Care must be taken not to present strong aromatic odorants like ammonia because they will stimulate trigeminal nerve terminals.

Electrodiagnosis

Olfactory evoked potentials are a study of the late near-field event-related potentials during multichannel electroencephalogram (EEG) recordings.[36,37] The test is performed using an olfactometer, which delivers stimuli having the shape of a square wave (steep onset and decline). The stimulants are presented in a constantly flowing air stream of controlled temperature and humidity. Rise time of the stimulus must not be more than 20 millisecond. Cues from tactile, thermal, or acoustic sensation must be prevented. Total flow rate can be varied within a wide range but is generally about 140 mL/s. Delivery of the stimulus should be independent of breathing. This is done by velopharyngeal closure and avoiding the flow of respiratory air in the nose during stimulation. The stimulus interval should be approximately 6 to 8 millisecond and care should be taken to avoid habituation. The odorant presented is usually vanillin. Evoked responses are recorded from scalp electrodes placed on C_z referenced to A_1, according to *The International Ten-Twenty System of Electrode Placement* (International 10-20), as shown in Figures 7-5 through 7-7.)Normal values are a response latency of 300 to 400 millisecond and a response amplitude of 10 to 20 μV.

With stimulation of the left nostril, the more pleasant an odor is perceived to be, the longer the latencies and the larger the amplitudes observed.

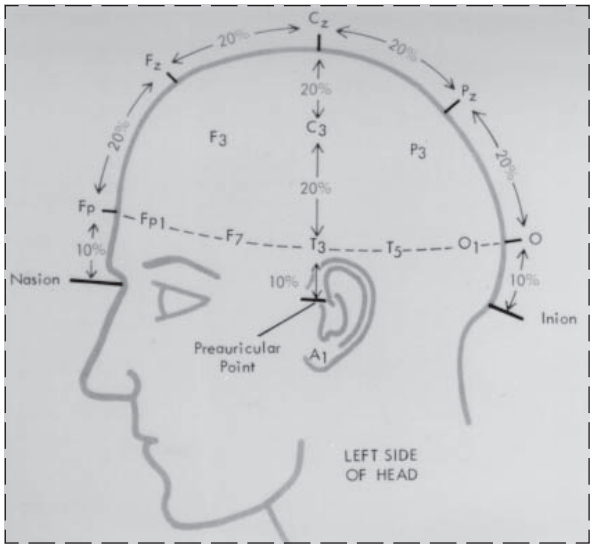


Fig. 7-5. Standard international 10-20 EEG electrode placement system (electrodes are placed either 10% or 20% of the total distance between skull landmarks). Lateral view. Adapted with permission from Grass Medical Instruments. *A Review of The International Ten–Twenty System of Eelectrode Placement*. Quincy, Mass: Grass Instrument Co; 1974: 1.

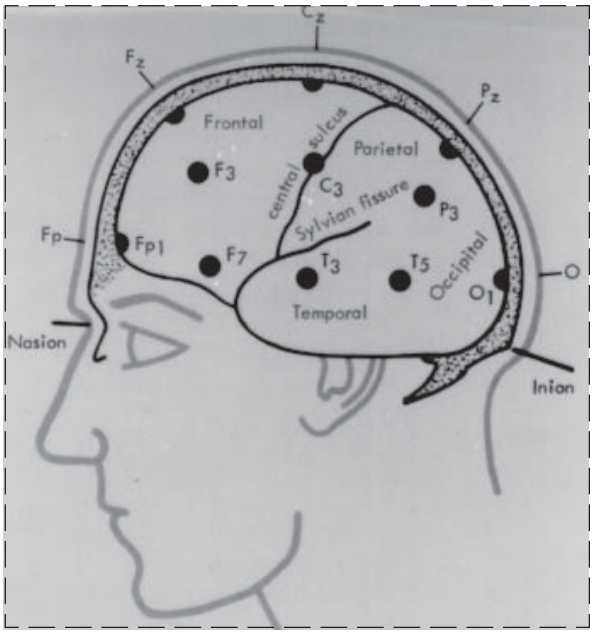


Fig. 7-6. Lateral skull showing location of electrode placement points in relationship to brain. Adapted with permission from Grass Medical Instruments. *A Review of The International Ten–Twenty System of Electrode Placement.* Quincy, Mass: Grass Instrument Co; 1974: 2.

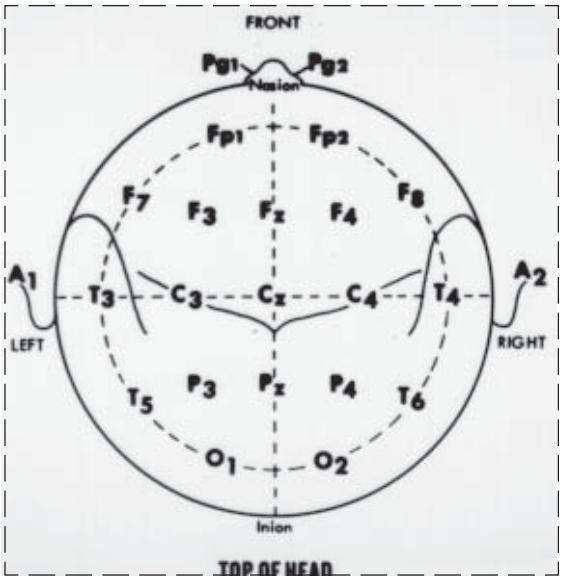


Fig. 7-7. Standard International 10-20 EEG electrode placement system, view from above. Adapted with permission from Grass Medical Instruments. *A Review of The International Ten–Twenty System of Electrode Placement*. Quincy, Mass: Grass Instrument Co; 1974: 19.

This variation is believed to be due to emotional responses to the odorant. Responses to carbon dioxide, menthol, and hydrogen sulfide (trigeminal stimulation) show significantly shorter latencies and smaller amplitudes after stimulation of the left side. Vanillin shows shorter latencies and smaller amplitudes after stimulation of the right side.

Imaging

Lesions identified by computed tomography (CT) and magnetic resonance imaging (MRI) correlate highly with olfactory impairments, especially when primary and secondary olfactory cortical centers are involved.[38]

Prognosis

In patients with CN I involvement secondary to head injury, Costanzo[6] reported a 33% recovery rate, 27% further deterioration, and 40% no change. Improvement may occur as late as 5 years after injury but is unlikely after 1 year.[4] Recent animal studies[39] have shown regeneration and functional reconnection of olfactory nerves, suggesting a similar possibility in humans.

Management

Contrary to common clinical opinion, dysosmias can have a significant impact on function.[6] Inability to smell the "warning signals" of fire, gas, or other dangerous substances may interfere with safety and present problems in some vocations. The American Medical Association[40] impairment rating scale allows a 3% disability for bilateral anosmia.

Rehabilitation strategies to normalize function must take account of the impact of dysosmia on emotions. The emotional component of smell appears to be confirmed by the variability of evoked olfactory responses, and the bilateral differences of latencies and amplitudes. This evidence also tends to support the concept of Dimond and colleagues [41] that pleasant emotions are predominantly processed by the left hemisphere and unpleasant feelings by the right. Many association fibers from the olfactory nucleus course through the hypothalamus to the limbic forebrain, influencing both sexual and nonsexual behaviors.

Therapeutic interventions consist mainly of counseling. They include the following:

- Olfactory evaluation: in the United States this is available in national centers providing chemosensory testing (chemosensory clinics).
- Teaching of hygiene routines.
- Nutritional counseling to avoid medically contraindicated excessive use of spices. Instead, increased attention to the texture, temperature, and visual appeal of foods is emphasized.
- Attention to appropriate food storage when there is a danger that spoilage will not be detected by odor.
- Concentration on visual and auditory cues while cooking.
- Observing fire safety measures, including extra smoke detectors.

CRANIAL NERVE II: OPTIC

Anatomy and Function

The optic nerve is classified as special sensory and its anatomy is shown in Figures 7-8 and 7-9.

The rods and cones are photoreceptors in the retina. They form the deepest layer of the retina and are oriented toward the pupillary opening. The pigment of the rods is rhodopsin, a glycoprotein that

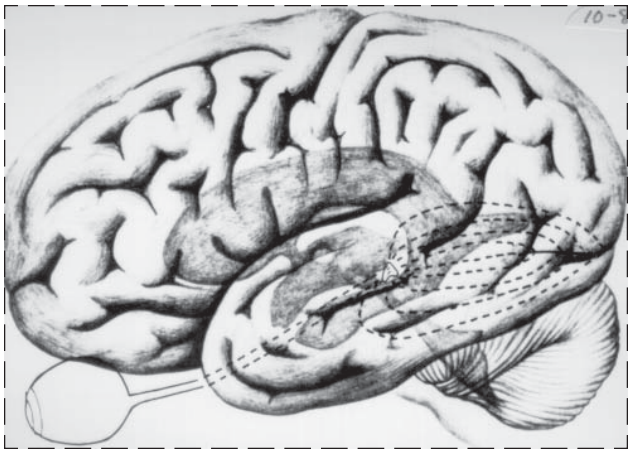


Fig. 7-8. Lateral view of the brain showing optic radiation in the parietal and temporal lobes, lateral to the ventricular system. Reprinted with permission from Brazis P, Masdeu J, Biller J. *Localization in Clinical Neurology*. Boston, Mass: Little, Brown; 1990: 109.

reacts to visible light. The receptors convert light energy to electrical signals. The retina contains approximately 100 million rods and 7 million cones. The cones are of three types, reacting maximally to red, green, or blue light. They are concentrated in the macula region and are especially dense in the fovea centralis. The fovea is the center of the macula and is tightly packed with cones to provide the most acute visual discrimination. The rods and cones connect with bipolar cells, the primary neurons. The primary neurons synapse with the secondary neurons, which are ganglion cells near the surface of the retina. The myelinated axons of ganglion cells form the optic nerve. Postchiasmatic fibers form the optic tract, consisting of ipsilateral temporal fibers of the retina and contralateral nasal fibers. Optic tract fibers pass to the tertiary neurons located in the lateral geniculate bodies of the thalamus. A small number of fibers ascend to terminate in the pretectal region as part of the pupillary light reflex pathway. Axons of the remaining neurons are contained in the geniculocalcarine tract (optic radiations), as seen in Figure 7-8. This tract goes from the lateral geniculate body through the internal capsule to the occipital (calcarine) primary visual cortex. Images from the upper visual field project to the lower area of the calcarine fissure and images from the lower visual field project to the upper area of the calcarine fissure. This compensates for the inverted and reversed image projected onto the retina by light rays converging and passing through the pupil. The central connections are from the pretectal region to the Edinger-Westphal nucleus via the posterior commissure, and from the superior colliculi via tectobulbar and tectospinal tracts to other cranial and spinal nuclei (for visual and body reflexes, such as turning the head in response to light).

Injuries and Lesions

The most common cause of injury or lesion is traumatic injury to the head. These injuries frequently cause basilar skull fractures, frontal lobe lesions, or increased intracranial pressure. Also, temporal bone fractures and blunt trauma to the outer orbital ridge may cause blindness in one eye. Most lesions are in the anterior visual pathways. Injuries can be intraocular, intraorbital, intracanicular, or intracranial. The incidence of CN II injuries in head trauma is reported to be 0.5% to 5.2%, and in children up to 6.0%.[42]

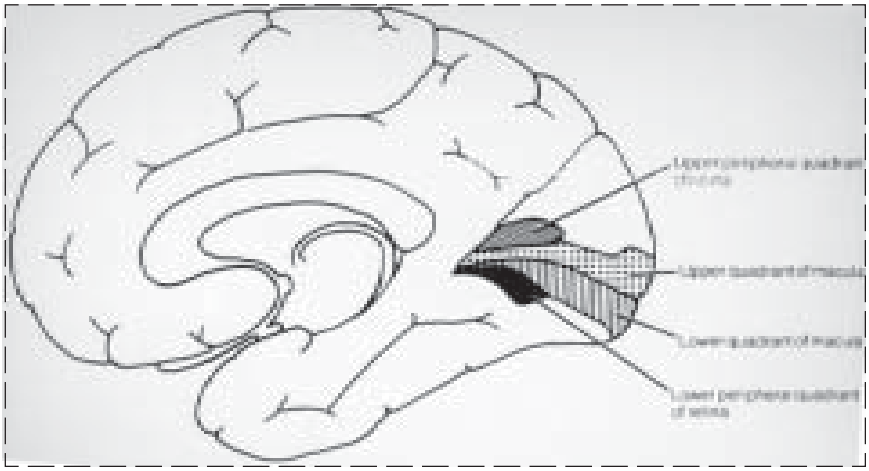


Fig. 7-9. Medial view of the right cerebral hemisphere, showing projection of the retina on the calcarine cortex. Reprinted with permission from deGroot J, Chusid JG. *Correlative Neuroanatomy.* 20th ed. Norwalk, Conn: Appleton & Lange; 1988: 286.

Penetrating gunshot wounds with entry in front of the ear frequently cause blindness despite often minimal brain damage. LaGrange[43] reported on war injuries with specific attention to the projectile entry site and the resulting orbital trauma. The report noted that entry above the orbit typically caused fractures of the orbital vault, affecting sensory, motor, and optic nerves near the optic foramen. Entry below the eyeball may fail to fracture the orbit at any point, but may still produce concussion of the eye in the macula region. Reduction of central acuity will then follow. If there is fracture of the orbit, findings are significantly more severe. Another observation was that a projectile may traverse the orbit without touching the eyeball, but still divide the optic nerve and avulse the papilla (optic disk).

Lesions of the optic nerve need not necessarily be severe. In a study by Wilbrand and Saenger (cited by LaGrange[44]), 50 out of 100 cases of unilateral optic nerve lesions following cranial nerve trauma had complete and permanent blindness, 4 had total blindness at onset but recovered fully, 17 recovered partially, and 34 had only partial involvement from the start. The incidence of optic nerve trauma through gunshot wounds is 25%. Keane and Baloh[11] report 231 cases in 21 years.

Nontraumatic lesions include tumors of the orbit, disease of the cavernous sinus, and cocaine abuse.[45] Consequences vary according to the location of the lesions, and several syndromes characterized by specific deficits must be recognized. Syndromes involving the optic nerve are as follows:

- Foster Kennedy syndrome: characterized by ipsilateral blindness and anosmia due to atrophy of optic and olfactory nerves and contralateral papilledema, caused variously by tumors at the base of the frontal lobe, arachnoiditis, syphilis, and occult trauma.
- Amaurotic familial idiocy (Tay-Sachs disease): caused by cerebromacular degeneration.
- Holmes-Adie syndrome: presents with tonic pupillary reaction and absence of one or more tendon reflexes. There is abnormal sensitivity to weak solutions of mecholyl (2.5%) instilled into the conjunctival sac. Normal eyes are not affected, but the response of tonic pupils is constriction (Adler-Scheie test).[46]

Symptoms and Signs

Optic nerve lesions are manifested by defects in visual field or acuity, or by objective alterations observed in an ophthalmoscopic examination. Relevant symptoms and signs are as follows:

1. Alteration of visual acuity
   - Amblyopia: markedly reduced vision
   - Amaurosis: complete blindness
   - Hemeralopia: day blindness
   - Fatigue syndrome: vision that is best in dim light
   - Nyctalopia: night blindness, sometimes associated with vitamin A deficiency
2. Visual field defects
3. Papilledema
4. Optic atrophy
5. Cortical blindness due to bilateral occipital infarction, caused by bilateral posterior cerebral artery involvement. The pupillary light reflex remains intact and total blindness is unusual.[47]

Visual field defects are illustrated in Figure 7-10. Signs and symptoms relative to the specific anatomic location of injury or lesion are presented in Table 7-1.

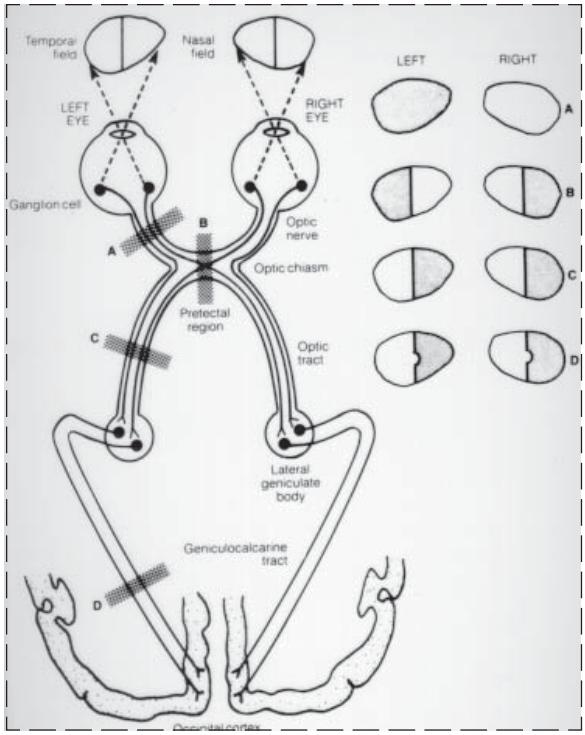


Fig. 7-10. Visual pathways and locations (indicated by letters) causing field defects shown in the diagrams on the right. Reprinted with permission from Ganong WF. *Review of Medical Physiology*. 16th ed. Norwalk, Conn: Appleton & Lange; 1993: 287.

TABLE 7-1

SIGNS AND SYMPTOMS: VISUAL PATHWAYS

	Retina	Optic Nerve	Optic Chiasm
Visual Acuity	Normal, if macula spared	Decreased	Medial chiasm: decreased vision bilateral Lateral chiasm: decreased vision ipsilateral
Visual Field Deficit	Corectopia central, arcuate or sectorial ring	Monocular in unilateral lesion shape as retinal lesions	Anterior angle ipsilateral (temporal or paracentral; contralateral) upper temporal
Pupillary Light Reflex	Unimpaired unless large lesion	Marcus-Gunn-pupil-lesion asymmetric afferent arc deficit	Afferent arc deficit. Ipsilateral impairment in lateral chiasmatic lesions

Adapted with permission from Masdeu GE. The localization of lesions affecting the visual pathways. In: Brazis PW, Masdeu JC, Biller J, eds. *Localization in Clinical Neurology*. 2nd ed. Boston, Mass: Little, Brown; 1990: 120–122.

Evaluation

Clinical evaluation consists mainly of visual acuity testing, which can be performed by presenting Snellen charts for distant vision and Jaeger cards for near vision. The patient is tested for ability to recognize the printed material at a specific distance, light, and contrast. If the patient is functioning at an extremely low level, saccadic responses (short, rapid movements of the eye made in order to scan the environment) may be obtained with an optokinetic stimulus. If a large stimulus is successfully followed, the size of the stimulus can be serially decreased, eventually reaching small letters. Convergence and accommodation can also be tested in this manner.

Visual field testing by confrontation can be used as a screening tool. If a defect is apparent, perimetry should be performed, preferably computer automated perimetry. When a cortical lesion is suspected, tests for visual inattention, neglect, and extinction are necessary. A lesion within a visual pathway can be localized by observing the different types of field defects. Monocular lesions are usually evidence of injury to the retina or the optic nerve. Binocular lesions can be localized at or beyond the optic chiasm. The shapes of different visual field defects and localization of the specific lesion are shown in Figure 7-10.

Trauma or infarction in bilateral occipital lesions is commonly responsible for bilateral altitudinal defects, in which the lower half of the visual field is affected and the macula is mostly spared. Such defects may also be caused by bilateral ischemic disease of the retinae or the optic nerves.[48,49] Color vision, often affected in patients with retrobulbar neuritis, is evaluated by testing ability to recognize figures hidden in a pattern of specially colored dots (Ishihara's chart).

Objective signs are observed during fundoscopy. Assessment for papilledema or optic atrophy is most important. Disk pallor will not be present before at least one month following injury. Vessels should be observed for size, regularity, and tortuosity. The examiner should look for hemorrhages and exudates and study the maculae carefully (patients should look at the examiner's light).

Another objective test for evaluating the visual pathways is the pupillary reflex (Figure 7-11; and see the discussion of CN III). The fibers that make up the afferent arc of the pupillary reflex exit the visual pathway before the lateral geniculate body and travel to the dorsal midbrain. Only large retinal lesions impair the light reflex. Asymmetric optic

Optic Tract	Lateral Geniculate	Optic Radiations	Calcarine Cortex
Normal in unilateral lesion	Normal in unilateral lesion	Normal in unilateral lesion	Normal in unilateral lesion
Contralateral homonymous hemianopia; incongruous	Contralateral homonymous may be incongruous; quadruple sectoranopia	Contralateral homonymous hemianopia (total lesion) or quadrantanopia (inferior with parietal lesion; superior with temporal lesion). Macular sparing with purely quadrantic defects	Contralateral homonymous hemianopia, congruous. Macular sparing. Involvement or sparing of contralateral unpaired temporal crescent. Ring shape or altitudinal with "vertical step" in bilateral lesions
Afferent deficit in contralateral eye	Normal	Normal	Normal

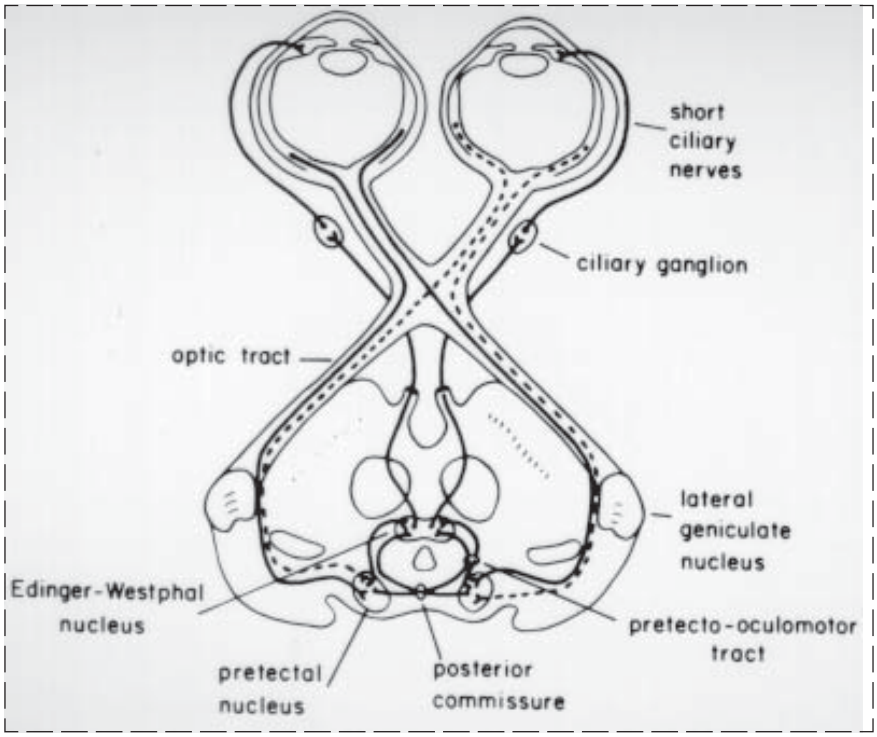


Fig. 7-11. Diagram of the path of the pupillary light reflex. Reprinted with permission from Brazis P, Masdeu J,Biller JL, eds. *Localization in Clinical Neurology.* Boston, Mass: Little, Brown; 1990: 145.

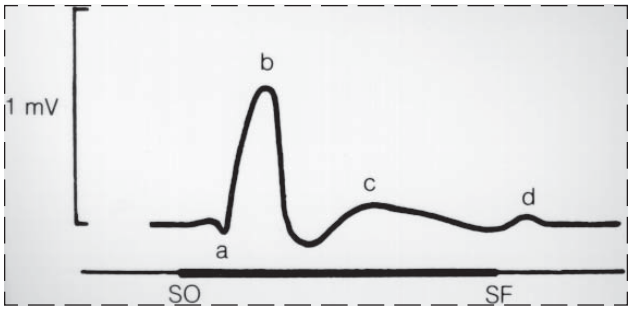


Fig. 7-12. Human electroretinogram (ERG). SO: Light stimulus on; SF: Light stimulus off. Adapted with permission from Ziv B. Electroretinography. *N Engl J Med*. 1961;264(11):546.

nerve or chiasmatic lesions can be detected by the Marcus-Gunn pupil. This is a relative afferent pupillary defect in which the abnormal pupil first dilates paradoxically as light is quickly moved from the normal to the abnormal eye. If one optic nerve is damaged, for example, by a temporal bone fracture, early diagnosis of blindness in that eye can be assessed by the pupillary light response. The pupil will be large and nonreactive to direct light but will respond to a light in the other eye (consensual reflex).

Electrodiagnosis

Two electrodiagnostic tests are available for objective assessment of the visual pathways. Electroretinography tests the function of the retina.[50] In this test, a light stimulus (stroboscope) or pattern reversal (reversing checkerboard) is presented to the eye. For recording, an electrode is placed in a contact lens over the cornea and an indifferent electrode is placed over the scalp. A 6 mV potential difference is observed between the two electrodes at rest. After onset of a light stimulus, a wave form (a, b, c) can be demonstrated, and another wave (d) follows when the light stimulus is turned off (Figure 7-12). Visually evoked potentials evaluate the complete visual pathway, including the retina. The parameters of the test are presented in the following list. Electrode placements are shown in Figures 7-5, 7-7, and 7-13.[51]

Visually Evoked Potentials

Stimulation
- Pattern reversal (reversing checkerboard)
- Size of squares, 30 to 50 minutes of arc
- Rate of change, 1 to 2 reversals per second
- Contrast and brightness held constant

Recording
- Active electrode placed on O_Z, reference on F_Z, ground electrode on F_{PZ} (International 10–20)
- Sensitivity: 10 μV per division
- Filters: low frequency 1 to 2 Hz; high frequency 100 to 200 Hz
- Averaging: 64 to 256 responses
- Analysis time: 300 to 500 millisecond

Measurements
- Absolute peak latencies
- Peak to peak amplitudes
- Duration of P100 (positive) (ie, N75 [nega-

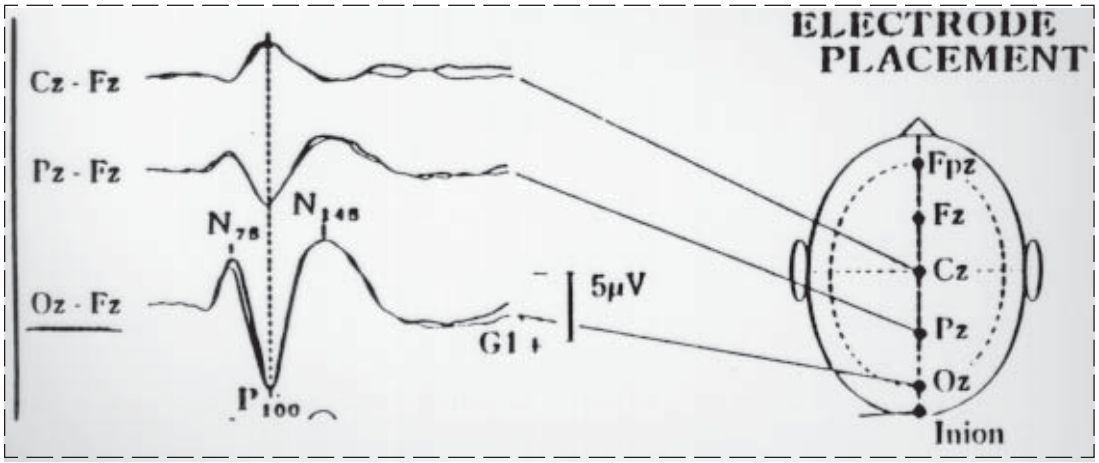


Fig. 7-13. Visual evoked potential electrode placement and evoked response. Reprinted with permission from TECA Corp. *Pattern reversal visual evoked potential VEPs*. In: TECA Applications Bulletin No. 1002. Pleasantville, NY: TECA Corp; 1981.

tive] to N145)
- Interwave latencies
- Interocular variation

Normal values
- Latency of P100: 102.3 ± 5.1 millisecond (range 89–114 ms)
- Mean ± 3 SD upper border of normal 117.6
- Latency difference between two sides: 2.3 ± 2.0 millisecond (range 0–6 ms)
- Amplitude of P100: 10.1 ± 4.2 μV (range 3.21 μV amplitude difference between two sides: 1.6 ± 1.6 μV (range 0–5.5 μV)
- Duration of P100: 63 ± 8.7 millisecond (range 47–86 ms)
- Duration difference between two sides: 2.8 ± 2.9 millisecond

Prognosis and Management

The prognosis for recovery after optic nerve injury is poor. Only one third of patients show significant improvement.[52]

Only in delayed visual loss are possible decompressive procedures indicated. There is hope that high dose steroid therapy may help counteract swelling and compression of the optic nerve in the optic canal. New drugs such as the GM (Monosialotetrahexosyl Ganglioside) gangliosides offer some hope for the future.[53] In case of cortical blindness with some sparing, training (utilizing low vision and other cues) seems to have promise.[47] Strong intensity stimulation of a defective visual field may help to improve function.

CRANIAL NERVES III, IV, AND VI: OCULOMOTOR, TROCHLEAR, AND ABDUCENS

Cranial Nerves III, IV, and VI are all motor cranial nerves. The anatomy is illustrated in Figure 7-14.

Anatomy and Function

Cranial Nerve III. The oculomotor nerve has its nuclei located in the central gray matter at the level of the superior colliculus (mesencephalon). These nuclei are arranged in one unpaired and four paired rostrocaudal columns. The unpaired rostrocaudal column shares bilateral nuclei, rostrally the Edinger-Westphal nuclei, and caudally the subnuclei for the levator palpebrae superioris. The most medially located nuclei of the paired columns are the subnuclei for the superior rectus muscles.

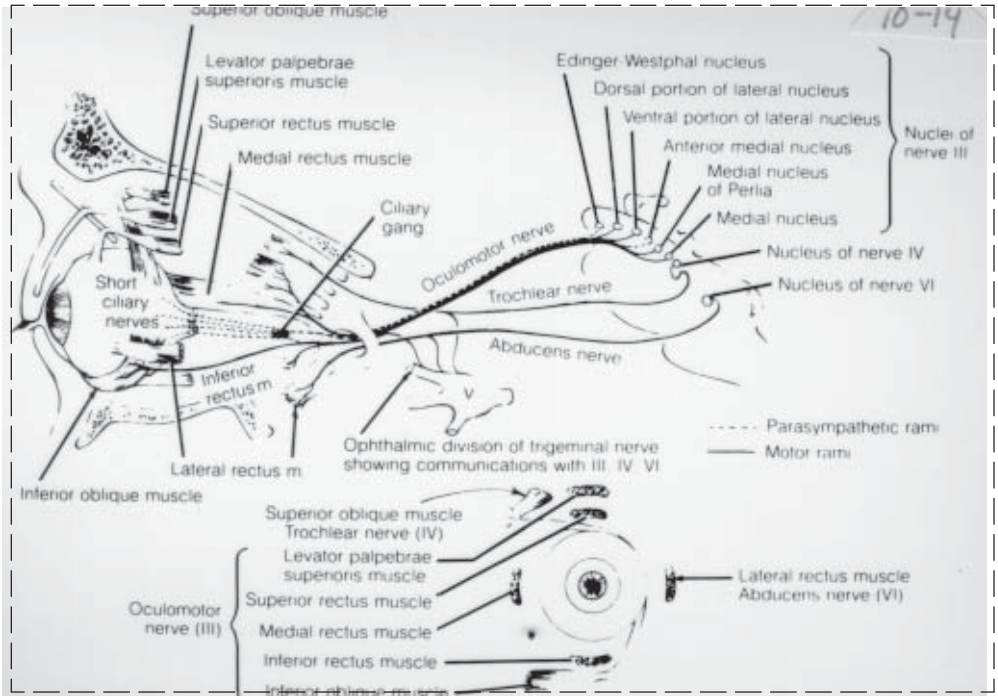


Fig. 7-14. The oculomotor, trochlear, and abducens nerves. Reprinted with permission from deGroot J, Chusid JG. *Correlative Neuroanatomy.* 20th ed. Norwalk, Conn: Appleton & Lange; 1988: 147.

The motor fibers (somatic efferent) from the superior rectus subnuclei immediately cross, actually through the opposite subnucleus. In the fascicular portion, oculomotor fibers diverge, and those to the levator palpebrae are located laterally. The oculomotor fibers travel through the red nucleus and the inner side of the substantia nigra and emerge on the sella turcica in the outer wall of the cavernous sinus. The fiber tracts then leave the cranium through the orbital fissure, where they separate into superior and inferior divisions. The superior division supplies the levator palpebrae and superior rectus muscles. The inferior division supplies the medial and inferior rectus and the inferior oblique muscles.

There is ipsilateral innervation of the medial rectus, inferior rectus, and inferior oblique muscles (CN III), and of the lateral rectus muscle (CN VI). Contralateral innervation supplies the superior rectus (CN III) and the superior oblique (CN IV) muscles.

The oculomotor nerve also carries parasympathetic fibers (visceral efferent), which arise from the Edinger-Westphal nucleus (preganglionic fibers) as part of the craniosacral division of the autonomic nervous system. These fibers end in the ciliary ganglion, from which postganglionic fibers emerge as short ciliary nerves to supply the ciliary muscle and the sphincter pupillae.

Cranial Nerve IV. The trochlear nerve nucleus is located just caudal to CN III at the level of the inferior colliculus (mesencephalon). The motor fibers (somatic efferent) in their entirety decussate in the anterior medullary velum and wind around the cerebral peduncles. The nerve then follows CN III and is situated in the lateral wall of the cavernous sinus. It enters the orbit through the superior orbital fissure and innervates the superior oblique muscle.

Cranial Nerve VI. The abducens nerve nucleus is located in the floor of the 4th ventricle in the lower portion of the pons near the internal genu of the facial nerve. Its special significance is its internuclear neurons, which send axons across the midline via the medial longitudinal fasciculus to the CN III medial rectus subnuclei. These fibers coordinate horizontal gaze. The abducens nucleus also relays impulses from the contralateral vestibular nucleus, which sends simultaneous impulses to the ipsilateral medial rectus subnucleus of the oculomotor nerve (vestibuloocular reflex).

The motor fibers (somatic efferent) to the lateral rectus muscle emerge anteriorly from the pontomedullary fissure and pass through the cavernous sinus in close proximity to the internal carotid artery. Abducens nerve fibers exit the cranium via the superior orbital fissure and supply the lateral rectus muscle. A few sensory (proprioceptive) fibers from the eye muscles are present in nerves III, IV, and VI. These sensory fibers terminate in the mesencephalic nucleus of CN V.

Central connections of the ocular motor nerve fibers are connected from the pretectal region near the posterior commissure to the Edinger-Westphal nucleus, in order to mediate the ipsilateral and consensual light reflexes. If this pathway is interrupted, an Argyll Robertson pupil occurs. From the supe-

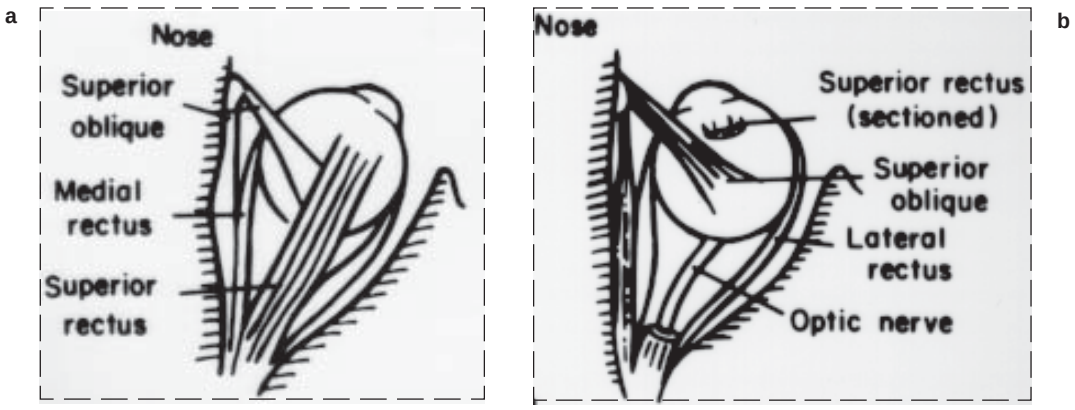


Fig. 7-15. Superior view of the right orbit. In the abduction (**a**), the superior rectus acts as an elevator and the superior oblique intorts the eye (brings the upper pole toward the nose). In adduction (**b**), the superior oblique acts as a depressor and the superior rectus intorts the eye. In (**b**), the superior rectus has been removed to show the position of the superior oblique. Reprinted with permission from Brazis, P, Masdeu, J. and Biller, JL. *Localization in Clinical Neurology.* Boston, Mass: Little, Brown; 1990: 129.

rior colliculi, fibers connect via the tectobulbar tract to nuclei III, IV, and VI, for mediation of accommodation and other reflexes. From the inferior colliculi, fibers connect via the tectobulbar tract to eye muscle nuclei, for reflexes correlated with hearing. From the vestibular nuclei, fibers connect via the medial longitudinal fasciculus, for reflex correlation with balance. From the cortex, fibers connect through the corticobulbar tract, for mediation of voluntary and conditioned movements of the eyes.

It is *imperative to understand fully* the anatomy and function of the ocular motor nerves and the innervated muscles associated with them (Figure 7-15). The recti muscles originate from the ligament of Zinn, which surrounds the circumference of the optic foramen except at the upper and outer part. All muscles attach with tendinous expansions to the sclera of the globe, at points above, below, medial, and lateral, as required.

The superior oblique arises above the inner margin of the optic foramen and in tendinous form passes through a pulley, a cartilaginous loop at the internal angular process of the frontal bone. It then reflects posterior, lateral, and inferior, passing beneath the superior rectus muscle to the lateral aspect of the globe. It inserts between the superior and lateral rectus muscles into the sclera, behind the equator.

The inferior oblique originates from the orbital plate of the maxilla. It passes lateral, posterior, and superior beneath the inferior rectus, and then inserts into the sclera behind the insertion of the superior oblique muscle.

The functions of the oculomotor nerve innervated muscles are illustrated in Figure 7-16 and listed here.

1. The medial and lateral rectus muscles move the globe horizontally.
2. The superior rectus and inferior oblique, as well as the inferior rectus and superior oblique, have complementary actions.
   With the globe in abduction:
   - the superior rectus elevates the globe,
   - the inferior rectus depresses the globe,
   - the inferior oblique extorts the eye (moves the left eye clockwise), and
   - the superior oblique intorts the eye (turns the left eye counterclockwise).

   If the globe is in adduction:
   - the inferior oblique elevates the globe,
   - the superior oblique depresses the globe,
   - the superior rectus intorts the eye (moves the left eye counterclockwise), and
   - the inferior rectus extorts the eye (moves the left eye clockwise).
3. The levator palpebrae elevates the lid. Fibers are intermingled with Müller's muscle (sympathetic innervation) and with the fibers of the orbicularis oculi (CN VII, which closes the lid).
4. The ciliary muscle decreases the tension of the lens capsule, increasing the convexity of the lens and adjusting the eye for near vision.

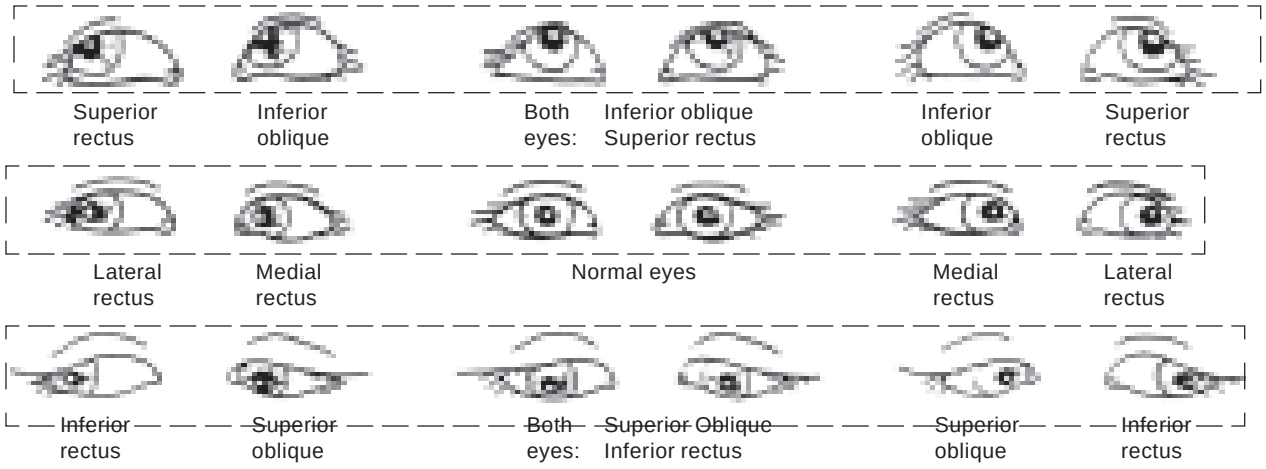


Fig. 7-16. Ocular muscles responsible for eye movement in different gaze positions. Drawing: Courtesy of artist: Dr. Anna Bettendorf, University of Virginia.

5. The sphincter pupillae constricts the pupil in response to a light stimulus and for accommodation.

The supranuclear control system of eye movement is as follows:

1. Vestibular system: The vestibuloocular reflex maintains focus on an object when the head moves; the eye moves within 10 millisecond in the opposite direction from head movement. Fibers from the vestibular nucleus travel to the contralateral abducens nucleus, and to the ipsilateral medial rectus subnucleus (oculomotor).
2. Optokinetic reflex: The optokinetic reflex maintains eyes on target during prolonged head movement in the same direction, after the vestibuloocular reflex has fatigued (ie, after approximately 30 s).
3. Smooth pursuit system: This system keeps a particular image in the fovea involuntarily. Images moving away from the fovea represent strong stimuli for smooth pursuit. The system can follow objects as rapidly as 30° to 40° per second. Ability decreases as a person grows older.[54] As the eyes and head move to follow an object, the vestibuloocular reflex is inhibited.
4. Saccadic system: Fast moving objects require rapid eye movements. These movements are called saccades and are under voluntary control. Objects of interest are registered by the peripheral vision and then focused in the fovea. Alertness is necessary to produce saccades and they are crucial for reading. Abnormal or inaccurate saccades, such as hypermetric (too fast), or hypometric (too slow), or purely initiated (unintentional) saccades, may occur with lesions in the structures that mediate the production of saccades. Elderly subjects and inattentive or medicated subjects have slower saccadic eye movements.[54]
5. Convergence system: Convergence and divergence work in tandem to permit binocular vision. Active eye muscle contraction is required to bring a single point within the visual field into homologous sites of both maculae. If there is a disparity of image registration between the retinas, diplopia will occur. During convergence the pupillary sphincter constricts. In a CN III lesion this accommodation reflex may be affected later than the light reflex because the pupillomotor fibers mediating convergence outnumber the fibers mediating the light reflex.[55]
6. Visual perception: Visual perception involves the interaction and integration of lower level abilities such as oculomotor control, visual field and acuity (the basis for visual attention), scanning, pattern recognition, and memory. Disruption of any of these skills, as is frequently observed in patients with head injury or stroke, causes significant deficits in daily activities. Severe visual scanning difficulties may contribute to language deficits in some aphasics because of the decreased information gathered.[56]

The anatomy and functions of the oculomotor nerves are as follows:

1. Innervation of muscles of the eyelid:
   - Levator palpebrae: CN III.
   - Orbicularis oculi: CN VII.
   - Müller's muscle: sympathetic innervation.
   - Sensation: CN V.
2. Innervation of ocular muscles:
   - Lateral rectus: CN VI.
   - Superior oblique: CN IV.
   - Inferior oblique: CN III.
   - Superior rectus: CN III.
   - Medial rectus: CN III.
   - Inferior rectus: CN III.
3. Innervation regulating size of pupil:
   - Pupillary constrictor: CN III (parasympathetic fibers).
   - Pupillary dilator: sympathetic nerve (from superior cervical ganglion).
   - Ciliary muscle: CN III (relaxes the lens for accommodation).
   - Corneal sensation: CN V (ophthalmic branch-upper cornea; maxillary branch-lower cornea).
4. Innervation of pupillary light reflex:
   - Afferent: CN II to pretectum to Edinger-Westphal nuclei.
   - Efferent: inferior division of CN III. Parasympathetic preganglionic fibers to bilateral ciliary ganglia, then through postganglionic ciliary nerves to constrictor pupillae muscles.
   - Reflex inhibition of dilator pupillae (sympathetic).

5. Innervation of accommodation reflex:
   - Afferent: CN II to visual cortex (occipital lobe), to pretectum.
   - Efferent: CN III, IV, VI (somatic efferents) for convergence by extraocular muscles and CN III (visceral efferents) for pupillary constriction.
6. Innervation of vestibuloocular reflex:
   - Afferent: CN VIII to contralateral nucleus of CN VI and ipsilateral CN III.
   - Efferent: CN VI to contralateral lateral rectus; and CN III to ipsilateral medial rectus.

Injuries and Lesions

The most common cause of injury or lesion is traumatic injury. In blunt head trauma, which causes closed head injuries, all three oculomotor nerves may be involved peripherally or centrally, primarily or secondarily through edema or herniation. Deviation of the eyes is commonly seen in early stages of brain injury, although it is often temporary. The incidence is reported to be 3% to 7% in all head injury populations.[57]Cerebral trauma most commonly affects CN III, especially in children.[42] The superior rectus appears to be most severely involved with blunt trauma. Blunt trauma may also damage the pupillary sphincter directly or through ischemia, causing mydriasis, poor response to light, and poor accommodation. CN IV is less frequently involved but may be affected in mild head injuries. CN VI has the longest intracranial course of all the cranial nerves; it is therefore vulnerable and is frequently involved. Bilateral lesions occur in many cases; often, injury is due to stretching of nerves after broad frontal impact.

Ophthalmoplegia is secondary to orbital fracture, which affects mostly CN II, III, IV, and VI; fracture may also cause a sensory defect by injuring the ophthalmic division of CN V. Ophthalmoplegia secondary to basal skull fracture (sphenoid, petrous ridge) involving the cavernous sinus may involve all oculomotor nerves. Here again, because of its long intracranial course, CN VI is most frequently impaired.

Oculomotor nerve lesions must be distinguished from orbital displacement observed during blowout fracture of the orbit. Entrapment of the inferior rectus muscle may cause restriction of upward gaze.[58] Old trauma or chronic progressive ophthalmoplegia also may limit the range of motion of the globe through shortening or fibrosis of the ocular muscles. These particular causes can be discovered or ruled out by the "forced duction" test, which moves the globe mechanically and, therefore, evaluates range of motion passively.

Penetrating gunshot wounds may involve oculomotor nerves as well as CN II. Injuries to the upper cervical spine may involve CN VI, in addition to CN IX, X, XI, and especially XII.[1]

Nontraumatic lesions include inflammatory cavernous sinus disease (Tolosa-Hunt syndrome), which may involve all oculomotor cranial nerves and branches 1 and 2 of CN V (Figure 7-17). An-

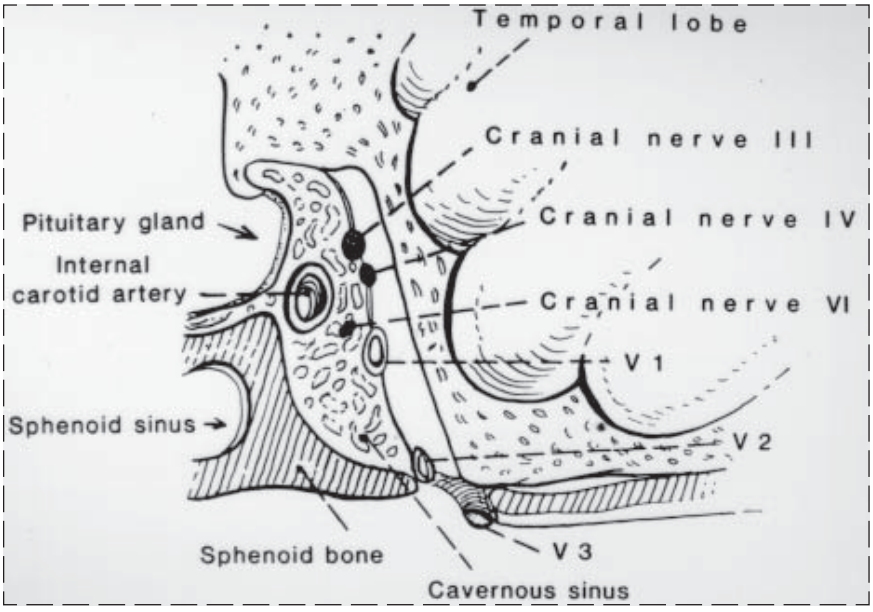


Fig. 7-17. Coronal diagram of the cavernous sinus. V_1: Ophthalmic division of cranial nerve V. V_2: Maxillary division of cranial nerve V. V_3: Mandibular division of cranial nerve V. Reprinted with permission from Brazis P, Masdeu J, Biller JL. *Localization in Clinical Neurology.* Boston, Mass: Little, Brown; 1990: 135.

other cause of lesion is septic thrombosis of the cavernous sinus.[59] In diabetic ischemic neuropathy, CN III and VI are most often affected. Frequently only one nerve is compromised. The pupillary light reflex is often reduced, although at times the pupil may be spared. Sparing can occur because ischemic lesions are frequently confined to the core of the nerve and spare the peripherally situated pupillary motor fibers.[55] In hypertension, CN VI fascicles can be infarcted and present as an isolated cranial nerve lesion.[60]

Inflammation and fibrosis are seen most frequently in thyroid ophthalmopathy, causing vertical diplopia because of asymmetric involvement of muscles with predilection of the inferior or superior rectus muscles. A myositis of the inferior oblique is common. Chronic progressive ophthalmoplegia is called Graefe disease. In myasthenia gravis, early involvement is seen in the medial rectus and levator palpebrae, monocular or binocular. The oculomotor nerve is also frequently involved in multiple sclerosis, more often causing internal rather than external ophthalmoplegia (60%); abducens nerve paresis has an incidence of 20%.[61] Involvement of oculomotor nerves by tumors is observed most commonly with pituitary adenomas that mainly involve CN III but is also common with meningiomas and nasal pharyngeal carcinomas. The most common lesions of the oculomotor nerves due to infections are seen in syphilis, scarlet fever, mumps, whooping cough, chicken pox, tuberculosis, and herpes zoster. Herpes zoster involves especially nerve III and the sphincter pupillae, levator palpebrae, and internal rectus muscles. Nutritional deficiencies, especially thiamin deficiency, may affect the function of the oculomotor nerves.

Other less common causes of ophthalmoplegia are Wernike's encephalopathy, internal carotid artery aneurysm or thrombosis, Paget's disease of the orbit, and Guillain-Barré syndrome. Occasionally there occurs a familial isolated oculomotor nerve lesion in association with Friedreich ataxia or Charcot-Marie Tooth disease.[62] It has been reported[63] that during dental anesthesia there may occasionally be paralysis of ocular muscles secondary to anesthetic injection into the inferior or superior dental artery. The anesthetic agent is carried through the maxillary artery, the middle meningeal artery, the lacrimal artery, and finally to the ophthalmic artery. As a postoperative complication following surgery or radiation therapy, ocular neuromyotonia may occur. The incidence was approximately 0.25% following spinal anesthesia.

Drugs or poisons such as phenytoin or phenobarbital may cause impairment of convergence and accommodation reflexes (watch for a diplopia occurring on near vision only). Lead may cause lateral rectus muscle paralysis, which develops rapidly; internal ophthalmoplegia may be observed. Methyl chloride and sodium fluoride poisoning may mimic botulism.

Syndromes Involving the Oculomotor Nerves

The following are syndromes involving the oculomotor nerves.

- Benedikt's syndrome: Ipsilateral ophthalmoplegia and contralateral hyperkinesia such as tremor, chorea, or athetosis resulting from a lesion of the tegmentum, which destroys the oculomotor nerve and the red nucleus on one side of the midbrain.
- Foville's syndrome: Pontine lesion causing contralateral hemiplegia with ipsilateral palsy of CN VII and ipsilateral paralysis of lateral gaze.
- Weber's syndrome: Ipsilateral ophthalmoplegia and contralateral hemiplegia. Ophthalmoplegia results from oculomotor nerve or nucleus interruption. Hemiparesis results from involvement of the cerebral peduncle with its corticospinal tract.
- Millard-Gubler syndrome: Ipsilateral facial weakness and contralateral hemiplegia, in many cases involving also CN VI, producing an internal strabismus. The lesion is in the pons.
- Duane's retraction syndrome: May follow paralysis of the lateral rectus muscle and is characterized by retraction of the eyeball on adduction of the eye with oblique upward movement of the eyeball and narrowing of the palpebral fissure.[64]
- Gradenigo's syndrome: Pain in the face caused by irritation of the semilumar ganglion; external rectus palsy. The syndrome is caused by meningitis at the tip of the petrous bone, usually secondary to purulent otitis media.[65]
- Wernicke's syndrome: Ocularmotor palsy due to involvement of the CN nuclei III or IV. Ptosis and pupillary changes are frequently observed, due to involvement of the red nucleus. Optic neuritis, retinal hemorrhages, ataxic gait, and muscular weakness may also occur.
- Möbius' syndrome: Ocular palsy in addition to facial palsy.

- Parinaud's syndrome: Conjugate ocular paralysis resulting in paralysis of upward gaze. This is mainly associated with lesions or disorders of the midbrain, especially the superior colliculi. It may be due to compression by pineal body tumor. Section of the posterior commissure can also produce this syndrome.

Symptoms and Signs

The symptoms and signs of oculomotor nerve lesions are described below. In addition, Table 7-2 reports them according to their location.

- Diplopia is the most common complaint associated with oculomotor nerve lesions. It is usually greatest in the direction of the weak muscle. In the position where the unfused images have their greatest separation, the more peripheral image will usually belong to the eye that has decreased mobility.
- Argyll Robertson pupil is miosis with loss of the light reflex and ciliospinal reflex, and with preservation of accommodation. Differential diagnosis includes neurosyphilis, multiple sclerosis, diabetes mellitus, pineal tumor, Wernicke-Korsakoff's syndrome, and midbrain encephalitis.
- Adie's pupil (myotonic pupil) is part of the Holmes-Adie syndrome. It is a benign condition in young women, often associated with absent deep tendon reflexes. It is of major importance to recognize the syndrome and so to prevent unnecessary investigation.[46]
- Pseudo-Graefe's syndrome is due to aberrant innervation (misdirection of regenerating nerve fibers). Most commonly it is observed after CN III and VI lesions, but it also occurs with CN III and X lesions. This is never caused by an ischemic neuropathy, but other lesions may cause nerve fibers to regenerate falsely.
- Exophthalmus may present secondary to extraocular muscle paralysis.
- Ocular neuromyotonia may be observed.

A complete isolated CN III lesion presents with the patient's eye being closed (ptosis). When opened manually the eye is found to be deviated outward and downward and the pupil is dilated. Unilateral, isolated oculomotor nerve palsy is most often related to ischemic diabetic neuropathy or to a lesion in the subarachnoid portion due to compression by an aneurysm. Isolated pupillary dilation is common in early uncal herniation. Migraine headaches may cause unilateral mydriasis lasting several hours. Seizures also can cause temporary unilateral mydriasis.

Other possible presentations of symptoms and signs secondary to CN III lesions are listed below.

- There may be paresis or paralysis of the superior rectus, the inferior rectus, the medial rectus, the inferior oblique muscles, and the levator palpebrae muscles (external ophthalmoplegia). One, a few, or all muscles may be affected.
- There may be dilation of pupils and reduction or absence of pupillary and accommodation reflexes (internal ophthalmoplegia).
- Bilateral large pupils may be seen normally in anxious young adults. Bilateral small pupils may be seen normally in the aged.
- There may be bilateral extraocular muscle weakness with sparing of the levator palpebrae muscle.[66]
- Bilateral ptosis is seen with nuclear involvement of CN III because of the midline position of the nucleus for the levator palpebrae muscle.
- Isolated bilateral ptosis with sparing of extraocular muscles and pupils may occur with encephalitis[67] or stroke.[68]
- Occasionally oculosympathetic spasms are associated with lesions of the cervical cord.
- Intermittent spasm of a portion of the pupillary sphincter may occur with recent onset CN III trauma or aberrant oculomotor reinnervation.

CN IV lesions in isolation are uncommon. The trochlear nerve may be involved in head injury, even in mild trauma. Other lesions occur mainly in conjunction with other oculomotor lesions. A complete CN IV lesion causes the eyeball to be turned upward and outward. Horner's syndrome may appear if the injury is near the sympathetic fibers. Symptoms and signs of CN IV lesions include

- weakness or paralysis of the superior oblique muscle, and
- vertical diplopia, which is greatest in down and inward gaze. The head tilts to the opposite side to compensate for diplopia. This is a characteristic sign.

TABLE 7-2

SYMPTOMS AND SIGNS ACCORDING TO LOCATION OF LESION–CRANIAL NERVES III, IV, VI

	Oculomotor	Trochlear	Abducens
Nuclear	Paresis/paralysis of oculomotor innervated muscles, contralateral superior rectus weakness, bilateral incomplete ptosis; complete one-sided CN III palsy rare; occasional isolated inferior rectus involvement[1]; possible bilateral paresis of CN III muscles. Occasional sparing of levator palpebrae[2]; pupillary constrictor weakness may be bilateral.	Contralateral superior oblique muscle weakness; lesion due to decussation.	Ipsilateral lateral rectus paresis; also ipsilateral gaze palsy (involvement of abducens interneurons), Möbius' syndrome, Duane's retraction syndrome.[3]
Fascicular	Complete CN III involvement unilateral; possible correctopia (irregularity of pupil); possible additional other neurological deficits (see midbrain syndrome).	Same as Nuclear. May in addition cause Horner's syndrome.[4,5]	Ipsilateral lateral rectus palsy and facial weakness and contralateral hemiparesis (Millard-Gubler syndrome); isolated lateral rectus palsy.[6]
Subarachnoid	Isolated unilateral CN III involvement; possible sparing of pupillary sphincter with incomplete lesions (pupillomotor fibers more dorsally located and smaller and more pressure resistant.[7]	None	Ipsilateral lateral rectus palsy; possible concomitant trigeminal nerve involvement (more so if lesion is in petrous bone.) Mostly secondary to chronic otitis media.
Cavernous Sinus	CN III innervated muscle weakness may involve also CN IV and VI and ophthalmic branch of CN V (combined ocular motor paresis, miosis, and poorly reactive pupil[8]); retroorbital pain.	Paresis superior oblique, may involve also CN III and VI and branch of CN V.	Paresis superior oblique, may involve also CN III and V (branch 1 and 2); retroorbital pain; occasional ipsilateral; Horner's syndrome.
Suborbital Fissure	Similar to cavernous sinus lesion; possible proptosis.	Similar to cavernous sinus lesion.	Similar to cavernous sinus lesion, except for *absence* of Horner's syndrome.
Orbit	Involvement of only superior or inferior branch of oculomotor nerve innervated muscles.	Weakness superior oblique muscle may be involved. CN III and VI also involved.	Paresis lateral rectus muscle; may also involve CN III and IV.

CN: cranial nerve

(1) Pusateri TJ, et al. Isolated inferior rectus muscle palsy from solitary metastasis to the oculomotor nucleus. *Arch Ophthalmol.* 1987;105:675. (2) Keane JR, Zaias B, Itabashi HH. Levator sparing oculomotor nerve palsy caused by a solitary midbrain metastasis. *Arch Neurol.* 1984;41:210–212. (3) Masdeu JC, Brazis PW. The localization of lesions in the oculomotor system. In: Brazis PW, Masdeu JC, Biller JL, eds. *Localization in Clinical Neurology*. 2nd ed. Boston, Mass: Little, Brown 1990;140–143. Chap 7. (4) Coppeto JR. Superior oblique paresis and contralateral Horner's syndrome. *Ann Ophthalmol.* 1983;15:681–683. (5) Guy J, Day AL, Mickle JP, Schatz NJ. Contralateral trochlear nerve paresis and ipsilateral Horner's syndrome. *Am J Ophthalmol.* 1989;107:73–76. (6) Donaldson D, Rosenberg NL. Infarction of abducens nerve fasicle as cause of isolated sixth nerve palsy related to hypertension. *Neurology.* 1988;38:1654. (7) Nadeau SE, Trobe JD. Pupil sparing in ocularmotor palsy: A brief review. *Ann Neurol.* 1983;13:143. (8) Spector RH, Smith JL, Chavis PS. Charcot-Marie-Tooth disease mimicking ocular myasthenia gravis. *Ann Ophthalmol.* 1978;10:1033–1038.

CN VI lesions are manifested by paresis or paralysis of the lateral rectus muscle. In a complete CN VI lesion the eye is turned inward (internal strabismus). There may be ipsilateral gaze palsy, which may present as conjugate gaze palsies, showing symmetrical restriction of gaze to one side, up, or down. Horizontal gaze palsies are manifested by unilateral restriction to one side, mostly due to contralateral frontal or ipsilateral pontine damage; vertical gaze palsies are due to bilateral involvement of structures in the commissure. Lesions in the upper pontine tegmentum can cause both horizontal and vertical gaze palsies.[69] Skew deviation results from supranuclear derangements.[70] Oculomotor nerve lesions may cause vertical misalignment and nystagmus. Because nystagmus has many different causes, often related to the vestibular system, this impairment will not be discussed here in detail.

Saccadic deficits commonly occur following brain injury.[71] There may be decreased saccadic accuracy, inability to fixate the gaze in the contralateral field, or decreased initiation of saccades toward the contralateral side of the lesion.[72]

Evaluation

Clinical examination observes the movements of the eye by having the patient follow a light stimulus. It is important to observe the reflection of light from the cornea to assess alignment (Hirschberg reflex).[73] Spontaneous voluntary and reflex gaze without a light stimulus must also be assessed. If diplopia is present, it must be determined whether it is a monocular or binocular diplopia. Physical evaluation should begin with examination of the eyelids; they should be checked for ptosis of the lid, upper, lower, or both. Pupils should then be examined for size (normally 2–6 mm in ambient light) and regularity (anisocoria up to 30% is normal).[74] Response to visual stimulus and accommodation must be observed. Further, clinical examination must rule out abnormal movement such as nystagmus.

It is important to look for a misdirection syndrome, which can be observed many months following CN III lesions, secondary to an aberrant regeneration. Fibers from the ocular muscles may regenerate aberrantly to the levator palpebrae, resulting in the pseudo-von Graefe's sign (lid elevation during an attempt to look down or lid winking while chewing). It is important to look for bilateral involvement because it is not uncommon in CN IV and VI lesions. The head tilt test demonstrates a CN IV lesion.[75] The observed degree of diplopia is greatest when looking down, and this causes the compensatory head tilt to the opposite side.

Miosis in darkness represents a paradoxical constriction (Flynn phenomenon) and may be congenital. It is seen in optic atrophy and bilateral optic neuritis.[76] In light, near dissociation, absent pupillary reflex to light and present convergence, is termed an Agyll Robertson pupil and may be seen with syphilis, sarcoidosis, diabetes, myotonic dystrophy, amyloidosis, or in aberrant regeneration.

Examination should include careful evaluation of gaze to rule out conjugate or dysconjugate gaze palsies. The lower motor neuron controls muscles; the upper motor neuron controls movement and gaze. The gaze must further be evaluated by observing automatic and planned eye movements. It is important to look for saccade deficits. These can be measured by having the patient look quickly from one object to another. The test objects should be held 6 in. apart and approximately 15 in. from the patient.[77] Mild traumatic brain injury can cause aberrant saccades and oscillations.[78]

Electrodiagnosis Tests

Electrodiagnostic tests for evaluating oculomotor nerve function utilize electronystagmography (ENG).[79] This is described in the discussion of CN VIII. Electrooculography may be of help in the diagnosis in eye movement disorders.[69] EMG of external ocular muscles is carried out according to standard electromyographic procedures.

Imaging and Management

CT scans and MRI scans are useful especially when delayed diplopia occurs. Spontaneous recovery within 9 to 12 months is not uncommon. In children, recovery up to 80% or 90% is reported.[7]

Diplopia is initially managed with eye patching. The sound eye is patched to encourage full excursion of the involved eye and to increase its function. Disuse amblyopia does not occur in adults and there is no need for alternate patching. However, during critical activities the involved eye should be patched to allow optimal performance. When the patient is able to suppress the second image, patching can be discontinued. Pleoptic exercises as well as stereoscopic training devices may be used to improve muscle excursion (Worth Four Dot flashlight).[80,81] Another recommended intervention is the use of Fresnel lenses[56] to preserve binocular vision. If after a prolonged time of observation (9 to 12 mo)

and appropriate exercises, no significant improvement is noted, surgical procedures may be considered for functional or cosmetic reasons. Surgical procedures frequently show excellent results, especially for persistent trochlear palsy, but less for abducens or oculomotor lesions. Another alternative is injection of botulin toxin into the antagonist of the paralyzed muscle.

Recent research[56] suggests that the best therapeutic approach to visual perceptual dysfunction is to aim at increasing the skills of visual attention, scanning, pattern recognition, visual memory, and ultimately cognition. To accomplish this goal the therapeutic approach employs strategies for remediating and compensating deficits in foundation skills, such as oculomotor control, visual field, and acuity. Visual field deficits are best evaluated with computerized automated perimetry.[82] Limited visual fields can be increased by training with repetitive intensive stimulation of the blind hemifield. Compensation for visual field deficits can also be increased with training. As already mentioned, training exercises can improve oculomotor control. Visual acuity has to be optimized with corrective lenses and improvement of lighting conditions. The patient is then taught an increased awareness of the deficit and how to "intellectually override" by repeated practice and meticulous planning of compensatory techniques for both self-care and academic activities.

CRANIAL NERVE V: TRIGEMINAL

The trigeminal nerve is a mixed nerve that is chiefly sensory. It has three major divisions, shown in Figures 7-18, 7-19, and 7-20.

Anatomy and Function

The three major divisions of the trigeminal nerve are the ophthalmic, the maxillary, and the mandibular (see Figures 7-18 and 7-19).

The *ophthalmic nerve* (CN V_1) supplies sensation to the upper part of the face, including the eyes (upper half of cornea, conjunctiva, and iris), paranasal sinuses, and part of the nasal mucosa and meninges. It is located in the lateral wall of the cavernous sinus near CN III, IV, and VI. The ophthalmic nerve and CN III, IV, and VI enter the orbit together through the superior orbital fissure. The ophthalmic nerve has three major branches: (1) the frontal nerve, which branches into supraorbital and supratrochlear nerves; (2) the lacrimal nerve; and (3) the nasociliary nerve.

The *maxillary nerve* (CN V_2) supplies sensation to the lower half of the cornea, the conjunctiva, and the iris; the upper jaw, teeth, lip, cheeks, and hard palate; and the maxillary sinuses and nasal mucosa. The maxillary nerve is located inferior to the ophthalmic division in the cavernous sinus. It leaves the cranium through the foramen rotundum and enters the orbit through the inferior orbital fissure. Its branches are the infraorbital, superior alveolar, zygomaticofacial and zygomaticotemporal, and the greater and lesser nasopalatine nerves.

The *mandibular nerve* (CN V_3) receives sensation from the lower jaw; teeth, lip, buccal mucosa, tongue; and part of the external ear, auditory meatus, and meninges. It supplies the skin of the chin, lower lip, and lower jaw, except for the area over the mandibular angle, which is supplied by the auricular nerve from the second and third cervical nerve roots. Sensory branches of CN V_3 are the buccal nerve, auriculotemporal nerve, lingual nerve, inferior alveolar nerve, and the meningeal branch of the mandibular nerve.

Trigeminal motor fibers supply the masseter muscle, the temporalis muscle, the medial and lat-

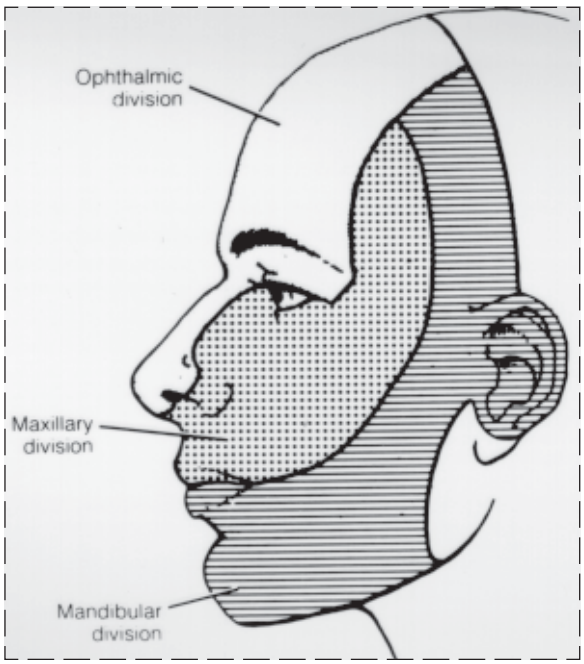


Fig. 7-18. Sensory distribution of cranial nerve V. Reprinted with permission from deGroot J, Chusid JG. *Correlative Neuroanatomy.* 21st ed. Norwalk, Conn: Appleton & Lange; 1991: 154.

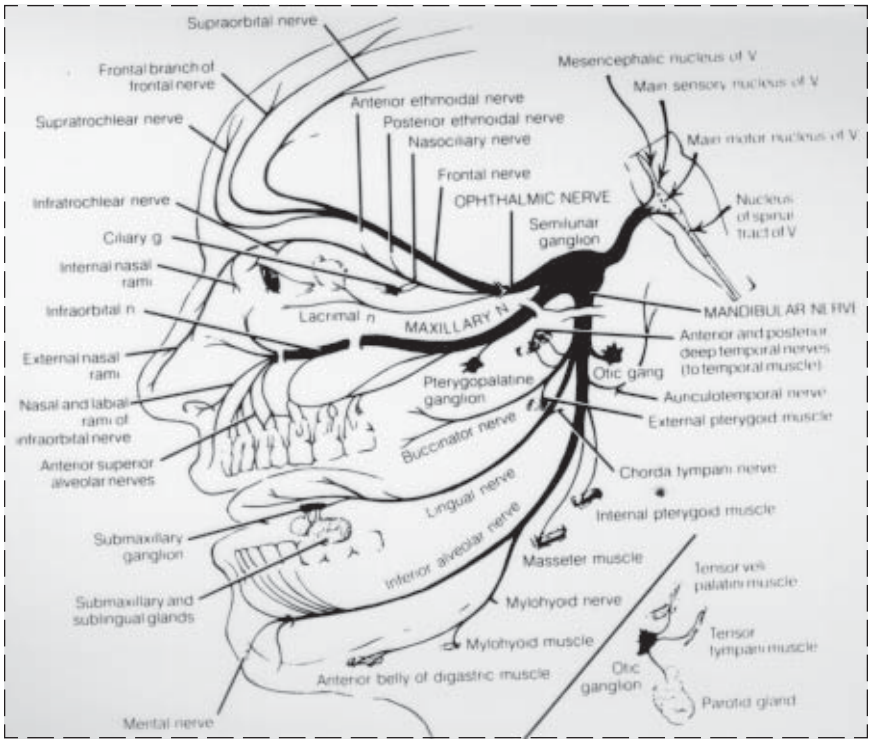


Fig. 7-19. The trigeminal nerve. Reprinted with permission from deGroot J, Chusid JG. *Correlative Neuroanatomy.* 21st ed. Norwalk, Conn: Appleton & Lange; 1991: 153.

eral pterygoid muscles, the mylohyoid muscle, and the anterior belly of the digastric muscle. Branches pass also to the tensor tympani and the tensor veli palatini (see Figure 7-19) The motor nucleus of CN V is located medial to the main sensory nucleus (see Figure 7-20) near the floor of the fourth ventricle. Motor fibers leave the midlevel pons on the ventricle surface, transverse the cranium through the foramen ovale, and join the mandibular nerve to reach and supply the muscles of mastication. Via the optic ganglion, fibers reach the tensor tympani and the tensor veli palatini. Via the mylohyoid nerve, fibers reach the mylohyoid muscle and the anterior belly of the digastric muscle.

Sensory receptors within the trigeminal nerve system include mechanoreceptors (rapidly and slowly adapting), thermoreceptors (warm and cold), nociceptors (responding to painful stimuli, possibly including chemoreceptors), and proprioceptors (responding to muscle or joint position).[83] The primary sensory neurons for tactile sensation (see Figure 7-20) have their cell bodies located in the gasserian (semilunar) ganglion, located in the middle cranial fossa near the cavernous sinus. Fibers terminate in the most lateral of the three sensory nuclei, the main sensory nucleus of the trigeminal nerve. Primary neurons for pain and temperature sensation have their cell bodies also located in the gasserian ganglion, but fibers terminate in the spinal nucleus, the most caudal of the trigeminal nerve. Primary neurons for proprioception (sensory

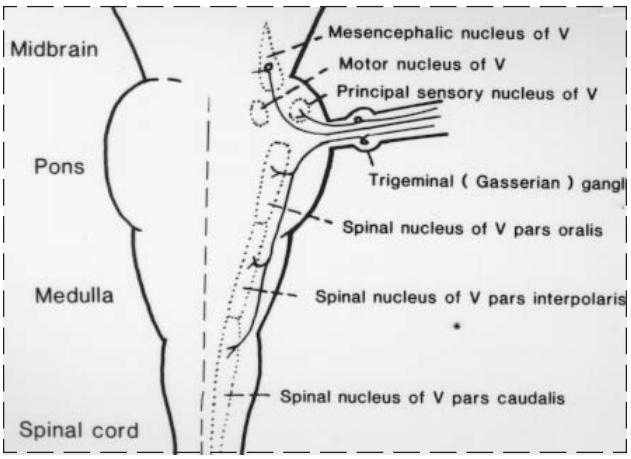


Fig. 7-20. Schematic diagram of the trigeminal nuclei. Reprinted with permission from Brazis P, Masdeu J, Biller JL. *Localization in Clinical Neurology.* Boston, Mass: Little, Brown; 1990: 191.

neurons for spindle stretch receptors in masseter and temporalis muscles) have their cell bodies *not* in the gasserian ganglion, but in the most superior trigeminal nucleus, that is, in the mesencephalic nucleus in the midbrain tegmentum (see Figure 7-20). Cell bodies of the golgi tendon organ for jaw closure muscles are located in the gasserian ganglion.

Central connections for CN V show sensory pathways from the spinal nucleus terminating in the thalamus. Reflex connections pass to the motor nuclei of CN V, VII, and IX. The motor nucleus receives bilateral, mainly crossed, cerebral connections, which originate as corticobulbar fibers in the lower frontal motor cortex and descend through the internal capsule; they decussate in the pons and supply the trigeminal motor nucleus. There also is input from extrapyramidal tracts.

The anatomic substrates for the reflexes involving CN V are as follows:

1. Masseter reflex:
   - Afferent: Muscle spindles to proprioceptive fibers from muscles of mastication through the mandibular nerve to the mesencephalic CN V motor nucleus, then monosynaptically to efferent limb.
   - Efferent: Trigeminal motor nucleus to mandibular nerve to extrafusal fibers in masseter and temporalis muscles.
2. Corneal reflex:
   - Afferent: A-delta fibers from the upper cornea (smaller than the fibers subserving the blink reflex) to the long ciliary nerve, to the ophthalmic nerve to the pons, to the spinal trigeminal nucleus; and A-delta fibers from the lower cornea to the maxillary nerve, to pons and also to spinal trigeminal nucleus; then multisynaptically to efferent limb.
   - Efferent: Direct response: ipsilateral facial nerve nucleus to nerve to orbicularis oculi. Consensual response: contralateral facial nerve nucleus to nerve to orbicularis oculi.
3. Blink reflex: (Figure 7-21)
   - The afferent limb consists of the supraorbital nerve to ophthalmic nerve to main sensory nucleus (CN V), then oligosynaptically to the efferent limbs, for which there are two possible efferent expressions. (1) Efferent: Facial nerve motor nucleus, ipsilateral facial nerve to orbicularis oculi—R1 response (early response) direct and polysynaptically from the spinal sensory nucleus of CN V. (2) Efferent: Bilateral facial nerve nuclei to nerve to orbicularis oculi muscle—R2 response direct and consensual. R2 coincides with eyelid closure.

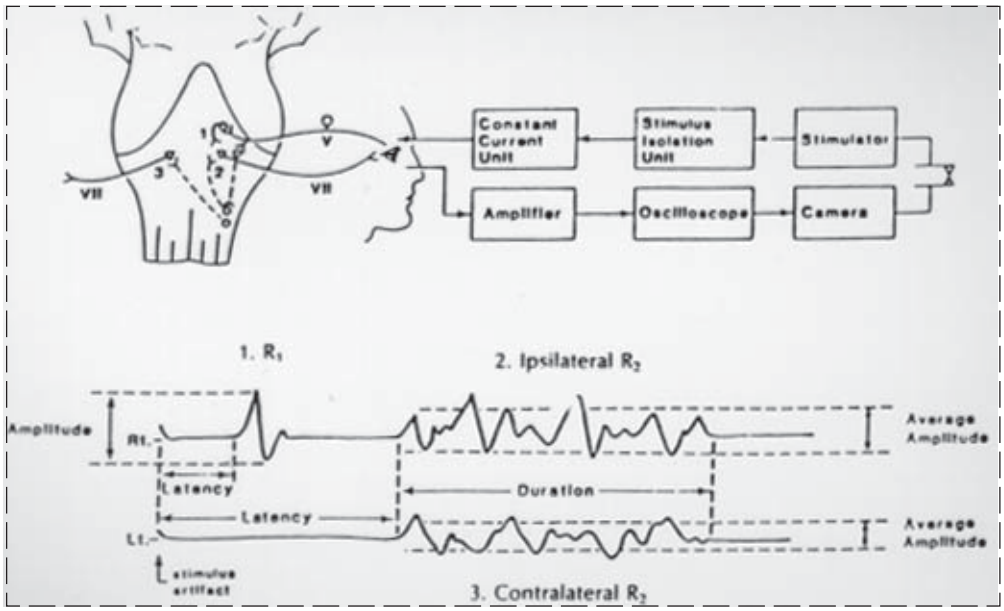


Fig. 7-21. Stimulation and recording arrangements for the blink reflex and its projected pathways. Reprinted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice.* 2nd ed.Philadelphia, Pa: FA Davis; 1989: 308.

The corneal reflex differs from the blink reflex. It uses different interneurons; there is no early response (R1) and there is a shorter latency to R2; there is less habituation than with the blink reflex.

Injuries and Lesions

The most common causes of injury or lesion are traumatic injuries, including skull fractures and orbital floor blow-out fracture, mainly injuring the maxillary nerve.[84,85] Blunt trauma often affects the supraorbital notch, injuring the supraorbital and supratrochlear nerves. Penetrating gunshot wounds may directly injure the semilunar ganglion. Nontraumatic lesions of CN V may be due to tumors such as meningioma, schwannoma, metastasis, nasopharyngeal carcinoma, or acoustic neuroma[86]; or to aneurysm, multiple sclerosis, syringobulbia, or infections.

In surgical trauma the most commonly affected nerve is the lingual nerve during lower third molar tooth extraction. Blackburn and Bramley[87] report an incidence of 11%; 0.5% of these patients experienced permanent sensory loss from the ipsilateral tongue.

A significant presentation of CN V involvement is trigeminal neuralgia (tic douloureux or Fothergill's disease), which may be idiopathic or due to multiple sclerosis, cerebellopontine angle tumor, or aberrant blood vessel.[88,89] A trigeminal sensory neuropathy may occur with Sjögren's syndrome. Other causes may be idiopathic, with possible full recovery within several months. Rheumatoid arthritis, systemic sclerosis, systemic lupus erythematosis, dermatomyositis,[90] and temporal arteritis[91] may present as sudden onset of unilateral or bilateral tongue numbness. Herpes zoster may affect the gasserian ganglion.[92]

Syndromes involving the trigeminal nerve are as follows:

- Tic douloureux (Fothergill's disease)[93] presents with severe pain in the trigeminal nerve distribution following irritation of a trigger zone. It is usually confined unilaterally to one division of CN V in adults over 40 years of age. Occasionally it is associated with dental or sinus disease.
- Paratrigeminal syndrome (Raeder's syndrome) is trigeminal neuralgia due to a semilunar (gasserian) ganglion tumor followed by facial anesthesia (rare). It presents also with paresis of muscles of mastication. CN III may be involved and there may be an ipsilateral Horner's syndrome.
- Auriculotemporal nerve syndrome (Frey's syndrome) presents with flushing and sweating during eating in the distribution of the ipsilateral auriculotemporal nerve. This may follow an injury or infection of the parotid gland area.
- Bonnier's syndrome involves CN III, V, VIII, IX, and X, with symptoms of Ménière's disease, contralateral hemiplegia, somnolence, apprehension, and weakness. The lesion is in the lateral vestibular nucleus and adjacent pathways.
- Gradenigo's syndrome presents with CN V_1 (ophthalmic branch) and CN VI deficits secondary to a lesion at the apex of the temporal lobe (osteitis or leptomeningitis secondary to otitis media); it also may be caused by trauma or tumor.
- Cavernous sinus syndrome may be secondary to trauma, carotid aneurysm, carotid-cavernous fistula, or infection. It may involve the ophthalmic as well as the maxillary branch of CN V, and in addition, CN III, IV, and VI.
- Superior orbital fissure syndrome may be caused by tumor, trauma, aneurysm, or infection. It may present as ophthalmoplegia associated with pain, paresthesias, and sensory loss (CN V, ophthalmic branch, and CN III, IV, and VI). There may be exophthalmos due to ophthalmic vein blockage and occasionally the maxillary branch is simultaneously involved. Except for exophthalmos it is difficult to differentiate a cavernous sinus syndrome from a superior orbital fissure syndrome.
- Horner's syndrome consists of miosis (paralysis of pupil dilator) and ptosis (Müller's muscle paralysis). There is often slight elevation of the lower lid because of paralysis of the lower tarsal muscle, which gives the appearance of an enophthalmus. Anhidrosis is possible, but is often not present if the lesion is beyond the carotid bifurcation.
- Neck and tongue syndrome presents with numbness of the tongue. This may be seen in temporal arteritis or it may be due to irritation of the cervical second dorsal nerve root, because proprioceptive fibers from the tongue via the hypoglossal nerve enter through the C2 nerve root.[94]
- Numb cheek syndrome may be caused by a maxillary fracture or lesion such as a recurrent squamous cell carcinoma of the

skin causing injury to the infraorbital nerve.[95] It may actually be the initial presentation of a nasopharyngeal tumor.
- Numb chin syndrome (Roger's sign) is caused by adhesions of the mandibular branch of CN V, which also may cause masticatory paralysis. Symptoms and signs are pain, swelling, and numbness of lower lip and chin (carcinoma of breast and lung, lymphoma).

Different symptoms correspond to different sites of lesions. Lesions located in the supranuclear area present with contralateral paresis of the muscles of mastication, causing deviation of the jaw away from the lesion. Even though innervation is predominantly from the contralateral hemisphere there is some bilateral control of CN V motor function. Usually this means that weakness is not too severe. Bilateral upper motor neuron lesions cause severe weakness of chewing muscles and an exaggerated jaw jerk. Lesions in the nuclear area are diagnosed by "the company they keep" (other cranial nerves or long tracts or both, as described with the anatomy of CN III, IV, VI, and the trigeminal). Cavernous sinus lesions may present with sensory deficits in the first (ophthalmic nerve) and second (maxillary nerve) divisions of CN V. In addition, there may be oculomotor nerve involvement (CN III, IV, and VI). Supraorbital fissure lesions show sensory loss in the ophthalmic nerve distribution (CN V), and oculomotor nerve lesions (specifically CN III, IV and VI).

The symptoms or signs of CN V lesions are pain, sensory loss, weakness of chewing muscles, and reflex changes, and more specifically as follows:

- Facial pain may be significant, especially with gasserian ganglion involvement.
- Loss of sensation may present with early corneal anesthesia. It may occur as isolated impairment of ipsilateral tongue sensation due to lingual nerve (mandibular nerve branch) involvement or lingual nerve injury. It may present with partial or complete loss of sensation of one, two, or all three branches of CN V.
- Dissociated anesthesia (trigeminal spinal tract involvement—syringobulbia) presents with loss of pain but not of touch; it may be present if the spinal tract of CN V is involved. Facial paresthesias, however, may also be seen in anemia or in nervous patients without cranial nerve lesion.
- Paralysis of muscles of mastication with deviation of jaw to the affected side.
- Loss of reflexes: Blink reflex, jaw jerk, sneeze, lid, conjunctiva, and corneal reflexes.
- Reduced hearing may be due to paralysis of the tensor tympani.
- Triasmus (lockjaw) is a tonic spasm of muscles of mastication (eg, in rabies, tetanus, epilepsy, and hysteria).
- Trophic and secondary disturbances such as dryness of nose, ulcerations of face, and loss of teeth may also occur (Herpes simplex, neurokeratitis).

Evaluation

The clinical examination consists of careful evaluation of sensory and motor functions and reflexes. The sensory exam must evaluate pinprick, light touch, and temperature over the three divisions of the trigeminal nerve on each side. The skin over the angle of the jaw is supplied by the auricular nerve.

Special attention must be paid to the sensation of the tongue, especially if the patient's history includes a recent lower third molar tooth extraction. Tongue numbness may cause difficulty in the oral phase of swallowing and in speech articulation, which is often noticed when the patient talks on the telephone. The patient may also accidentally bite the tongue.

To help identify lingual nerve compromise a moving two-point discrimination test was developed and recommended by Blackburn and Bramley.[87] The test is based on the observation of Mountcastle and colleagues[96] and Dellon[97] that touch sensation is mediated through quick and slow adapting fibers. Quick adapting fibers mediate moving touch, and they were found to greatly outnumber slow adapting fibers in the nerve to the tongue. The likelihood of identifying abnormalities is significantly increased by testing a larger number of fibers. Blackburn and Bramley[87] obtained two-point discrimination scores in the tongue of stimuli 1 mm and 3 mm apart. In normals there was a small difference between the two sides, with the threshold on the right being somewhat lower. Blackburn and Bramley also demonstrated that the test could predict recovery or nonrecovery correctly in approximately 90% of patients. Seven test stimuli were presented in a moving fashion. A score of one to three abnormal responses indicated a good prognosis for recovery, while four or more wrong responses indicated a poor prognosis. Test results may be helpful in considering possible surgical exploration.

Reflex testing should include the corneal test, in which a light touch of the cornea causes contraction of bilateral orbicularis oculi muscles. The blink reflex is tested by a tap applied to the glabella, which causes bilateral orbicularis oculi contractions.

Motor evaluation is carried out by testing the strength of jaw opening and lateral deviation. Weakness of one side will cause the open mouth to deviate toward that side. It is important to palpate for the contraction of the temporalis and masseter muscles while the patient bites on a tongue depressor. To test the jaw jerk, tap the mandible. If the jerk is visible or palpable, a bilateral upper motor neuron lesion above the level of CN V motor nucleus is suggested.

Electrodiagnosis

Electrodiagnostic testing can evaluate the masseter reflex,[98,99] the masseter inhibitory reflex,[100–102] the pterygoid reflex,[103] the blink reflex,[104–106] and the corneal reflex,[107] by means of reflex nerve conduction, trigeminal evoked responses, mandibular motor nerve conduction, and EMG of muscles of mastication. The masseter reflex is a myotatic reflex conducted through the midbrain. Afferent proprioceptor fibers innervate cell bodies in the mesencephalic nucleus and have monosynaptic connections with the motor nucleus of CN V for the efferent pathway to the masseter muscle. Unlike the spindle afferents in the limb musculature, it appears that the primary spindle afferents from the masseter muscle exert no direct inhibitory influence on the antagonistic muscles (those opening the jaw). Vibration of the jaw muscles facilitates the masseter reflex, while at the same time such vibration inhibits the "H" reflex.

The procedures for the electrically elicited masseter reflex and for the masseter inhibitory reflex follow.

Electrodiagnostic Evaluation of Masseter Reflex

Stimulation: A mechanical tap is applied with an electronic reflex hammer to the examiner's finger held to the subject's chin. A microswitch triggers the oscilloscope sweep.

Recording: This is accomplished by active surface or needle electrodes placed bilaterally on or in the lower third of the masseter muscles. Responses are recorded simultaneously from both sides. The reference electrodes are placed below the mandibular angle, and the ground is placed on the forehead.

Normal values: Because reflex latencies vary with successive trials, asymmetry of simultaneously recorded right and left values is more meaningful than absolute values.

- Amplitude: This is variable and depends on the weight supported by the mandible. The amplitude ratio between the two sides is clinically meaningful.
- Latency: Approximately 8.4 millisecond (range 6.4–9.2 ms). A difference of more than 0.5 millisecond between the two sides is abnormal, as is bilateral absent responses in persons below the age of 70 years.

The masseter inhibitory reflex is tested by stimulation of the infraorbital or mental nerve during maximal contraction of the masseter muscle (teeth clenching). Two silent periods ($SP_1$and SP_2) can be observed in the EMG interference pattern. This is believed to be a protective mechanism to prevent intraoral injury from excessive jaw movement during speech in patients with movement disorders.

Procedure to Evoke Masseter Inhibitory Reflex

Stimulation of the mental nerve is carried out in the mental foramen with a stimulus of 0.2 millisecond duration and approximately 30 to 40 mA.

Recording is bilateral on the masseter muscles.

Normal values:

- Latency: SP_1 10-15 millisecond, mean 11.4 $\pm$ 1.3 millisecond; SP_2 40-50 millisecond, mean 47 $\pm$ 6 millisecond
- Duration: SP_1 20 $\pm$ 4 millisecond; SP_2 40 $\pm$ 15 millisecond

Electrodiagnostic Evaluation of Blink Reflex

The blink reflex[104,105] (see Figure 7-21) can be elicited mechanically with an electronic hammer tapping the glabella (this is a cutaneous rather than a stretch reflex). A midline tap will cause an R1 component bilaterally. Latencies for R1 are a few milliseconds longer than those elicited with electrical stimulation. The electrically evoked blink reflex procedure is explained below.

Electrodiagnostic Evaluation of Pterygoid Reflex[103]

Stimulation: To elicit the masseter reflex, a mechanical cap is applied with an electronic reflex hammer to the examiner's finger held to the

subject's chin. A microswitch triggers the oscilloscope sweep.

Recording: To record, two monopolar needle electrodes are placed into the medial pterygoid muscle 6 to 10 mm apart, and 22 to 25 mm beneath the skin. Insertion is halfway between the angle of the mandible and the facial artery at its crossing of the mandible. Jaw closure can confirm adequate placement by observing the appropriate EMG signals. Since there is midline stimulation at the identical point for the masseter and pterygoid reflexes, all four can be elicited simultaneously if four channels are available.

Normal values: Values are slightly faster than that of the masseter reflex, which may have to do with the location of the subnuclei within the trigeminal mesencephalic nucleus; a cluster of neurons for the pterygoid muscles being located more caudally than those for the masseter.

- Amplitude: Amplitudes with needle recording are unreliable.
- Latency: 6.9 ± 0.43 millisecond, and side to side difference: 0.29 ± 0.21 millisecond. It is believed that testing both the masseter and the pterygoid reflexes provides a more precise localization of a small pontomesencephalic lesion.

Procedure to Evoke the Blink Reflex

Stimulation: The stimulation to evoke the blink reflex is carried out with cathode placement over the supraorbital fissure. The anode is placed laterally and above the cathode to avoid current spread to the other side. Optimal shock frequency is one shock every 7 seconds to avoid habituation. Stimulation is best applied between blinks.

Recording is best with superficial disks. The active electrode is placed 1 cm below lid margin, below the pupil, or slightly lateral over the orbicularis oculi. The reference is placed over the zygoma, or the nasalis muscle. The ground is placed on the chin.

Normal values:

Latencies in adults[104]:

- R1: 10.5 millisecond $\pm$ 0.8 millisecond (difference between two sides less than 1.2 ms)
- R2: 30.5 millisecond $\pm$ 3.4 ipsilateral (upper limit of normal 40 ms);
  30.5 millisecond $\pm$ 4.4 contralateral (upper limit of normal 41 ms)
- R1-D ratio: 3.6 $\pm$ 0.5 (D = direct facial nerve response)

The difference between R2 ipsilateral and R2 contralateral should not exceed 5 millisecond. The latency difference between R2 evoked by right side stimulation and corresponding R2 evoked by left side stimulation should be less than 7 millisecond.

Latencies in neonates[106]:

- R1: 12.1 millisecond $\pm$ 1.0 millisecond
- R2: 35.9 millisecond $\pm$ 2.5 millisecond ipsilateral;
  Contralateral often absent
- R1-D ratio: 3.7 $\pm$ 0.4

Stimulation: infraorbital fissure
Recording: same as above
Normal values:

Latency in adults:

- R1: none
- R2: 41 millisecond ipsilateral (upper limit of normal);
  42 millisecond contralateral (upper limit of normal)

Physiological variations observed in blink reflex recordings are variations during light sleep such that the R1 amplitude decreases and may disappear, and the R2-D ratio is of prolonged duration. During feelings of apprehension, R1 is decreased in amplitude and R2 is increased in amplitude. With repeated trials R1 is more stable than R2 and therefore better suited for assessing the trigeminal nerve. Analysis of R2 is essential to determine whether the afferent or efferent limb of the reflex is primarily involved. If R1 is unstable, paired shocks with 5-millisecond interstimulus intervals may be utilized to facilitate the response. The first shock should be simply a conditioning stimulus of lower amperage. Late responses are absent in coma.

The corneal reflex[107] and the blink reflex may help in evaluating brainstem interneuronal activity; disease or injury affecting the reflex arc; or lesions, such as compression or hemispheric, that indirectly affect the reflex arc. The procedure for eliciting the corneal reflex follows:

Electrodiagnostic Evaluation of Corneal Reflex

Stimulation is carried out through a thin, saline soaked cotton thread connected to a constant current stimulator. The anode is placed on the ipsilateral earlobe. A stimulus of 1 millisecond duration is employed, and responses are usually observed with a stimulus of 0.1 to 3 mA.

Recording is carried out from bilateral orbicularis oculi muscles.

Normal values are latencies of 35 to 50 millisecond.

The difference between the direct and consensual response is 5 millisecond or less. The difference between responses evoked from both corneas is 8 millisecond or less.

Sensory nerve conduction[108] can be determined by *stimulating* the supraorbital nerve at the supraorbital fissure and *recording* from a superficial electrode over the forehead 4 cm distal to the point of stimulation. Normal values are latencies from 1.3 to 1.5 millisecond and amplitudes of 5 to 8 μV.

Trigeminal evoked responses[109] (Figure 7-22) can be elicited over the scalp when superficially *stimulating* the lips (on the right and left sides independently), the gingiva, or the infraorbital nerve, with a current strength approximately 3-fold that of the sensory threshold. An averaging procedure is necessary as follows.

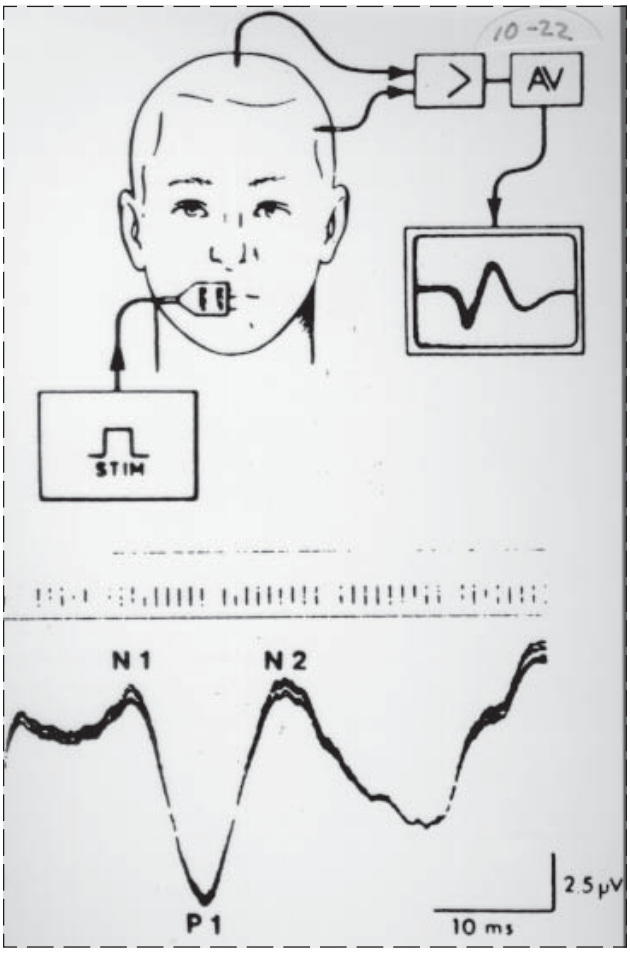


Fig. 7-22. A cortical sensory evoked potential of the trigeminal nerve elicited with stimulation of the lips. Reprinted with permission from Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice*. 2nd ed. Philadelphia, Pa: FA Davis; 1989: 396.

Procedure for Trigeminal Evoked Potential Studies[109]

Stimulation: This is supplied to lips, gingiva, or infraorbital nerve. Shocks between 1 and 2 Hz are averaged.

Recording: Electrodes are applied 1 or 2 cm behind C_5 or C_6, that is, between C_3 and T_3 or between C_4 and T_4 respectively (according to International 10-20; see Figure 7-5). The reference electrode is placed at F_Z.

Normal values: N1 is the first small negative response, often not easily distinguishable. P1 (second wave) can be used for measuring latency.

- Normal latency is 18.5 $\pm$ 1.5 millisecond. The mean difference between the two sides is 0.55 $\pm$ 0.55 millisecond.
- Amplitude N1 to P1 is 2.6 $\pm$ 1.05 μV. The difference between the two sides is 0.51 $\pm$ 0.54 μV.

Mandibular motor nerve conduction can be studied through magnetic stimulation to the central cortex, with recording electrodes placed over the muscles of mastication. This method can detect nerve conduction abnormality, but at present the appropriate equipment is not available for use in most laboratories.

Peripheral mandibular motor nerve conduction was reported by Dillingham and associates[110] Stimulation is carried out intraorally where the deep temporal nerve branches from the CN V_3. Recording is carried out from the temporalis muscle. The mylohyoid nerve (another branch of CN V_3) is also stimulated with recording from the mylohyoid muscle. Latencies for the deep temporal nerve are reported to be 2.1 $\pm$ 0.2 millisecond, and for the mylohyoid nerve they are 1.8 $\pm$ 0.2 millisecond.

EMG of the masseter muscles can easily be performed and evaluated in the usual manner. During voluntary jaw clenching, the elicited jaw reflex will cause a brief pause in the electromyographic activity of the masseter muscle. In normal subjects this pause lasts approximately 13 millisecond. It is prolonged in temporomandibular joint syndrome and decreased or absent in patients with tetanus.

The prognosis for tic douloureux is relatively good for pain relief with medications or surgery or

both. For secondary neuralgias the results are less satisfying.

Management

Facial pain or tic douloureux is successfully managed using the medications phenytoin, carbamazepine, and more recently also baclofen, chlorphenesin, and mephanesin. If no relief is obtained, surgery may have to be considered. Procedures most successful for relief of trigeminal pain are gangliolysis or microvascular decompression. Retrogasserian neurotomy or peripheral neurectomy may also be considered.[93]

Ablative trigeminal surgery is sometimes followed by anesthesia dolorosa, which is manifested by severe constant pain in the anesthetic area. Unfortunately, this condition is very resistant to treatment.[111,112]

CRANIAL NERVE VII: FACIAL

The facial nerve is mixed, but chiefly motor. It contains both somatic and visceral efferent components, and the afferents likewise are somatic and visceral. The anatomy is depicted in Figures 7-23 and 7-24.

Anatomy and Function

Motor branchial efferents are fibers from the motor nucleus of CN VII, located in the reticular formation of the caudal pons. The fibers loop around the nucleus of CN VI and then are joined by the visceral efferent fibers of the nervus intermedius of Wrisberg. The nervus intermedius carries sensory fibers to the nucleus tractus solitarius, and parasympathetic fibers from the superior salivatory nucleus. CN VII (motor) and nervus intermedius (the parasympathetic and sensory part of CN VII) emerge together from the brainstem in the region of the cerebellopontine angle just medial to the acoustic nerve (CN VIII). They cross the posterior cranial fossa and enter the internal auditory meatus of the temporal bone. Within the temporal bone the facial nerve can be divided into four parts: (1) the meatal segment, (2) the labyrinthine segment, (3) the horizontal segment, and (4) the mastoid segment.[113]

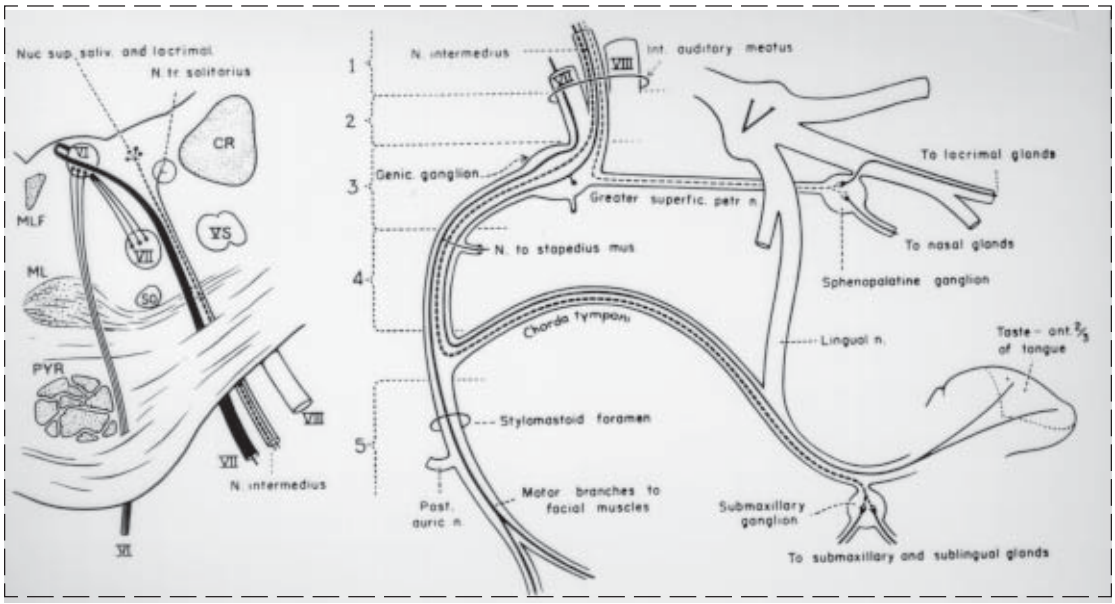


Fig. 7-23. Origin and distribution of various components of the facial nerve. The facial, intermedius, and acoustic nerves illustrated on the left continue in the drawing on the right. CR: Corpus restiforme; MLF: Medial longitudinal fasciculus; ML: Medial lemniscus coursing vertically through corpus trapezoideum; PYR: Pyramidal bundles in pars basilaris pontis; SO: Superior olivary nucleus; VS: Nucleus of spinal tract of the trigeminal nerve. Reprinted with permission from Haymaker W, Kuhlenbeck H. Disorders of the brainstem and its cranial nerves. In: Baker AB, Baker LH, eds. *Clinical Neurology.* Vol 3. Hagerstown, Md: Harper and Row; 1976: 28. (Used with permission from J.B. Lippincott Co.)

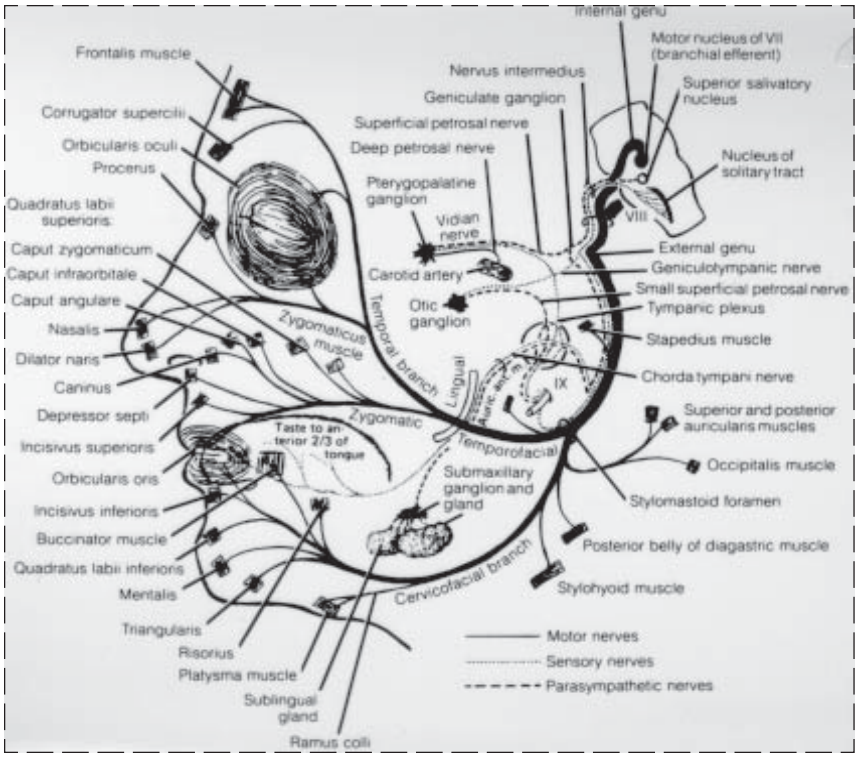


Fig. 7-24. The facial nerve. Reprinted with permission from deGroot J, Chusid JG. *Correlative Neuroanatomy***.** 21st ed. Norwalk, Conn: Appleton & Lange; 1991: 155.

The *meatal segment* (see Figure 7-23, area 1) gives off no major branches, but there is a close relationship with the position of CN VIII. The motor fibers of CN VII are superior and anterior; and the nervus intermedius, coming from the superior salivatory lacrimal nucleus and solitary tract nucleus, is between them.

In the *labyrinthine segment* (see Figure 7-23, areas 2 and 3), the motor division of CN VII and the nervus intermedius enter the fallopian (facial) canal in the petrous bone. They reach the geniculate ganglion from which the greater superficial petrosal nerve originates. This nerve contains preganglionic, parasympathetic efferents that innervate the lacrimal, palatal, and nasal glands via the sphenopalatine ganglion. Sensory cutaneous afferent fibers from the skin of the external auditory meatus, lateral pinna, and mastoid also travel through the greater superficial petrosal nerve (somatic afferents).

The *horizontal segment* (see Figure 7-23, area 3) distal to the geniculate ganglion) gives off no major branch. The *mastoid segment* (see Figure 7-23, areas 4 and 5) gives off the nerve to the stapedius muscle at the upper end of the segment. Further down, the chorda tympani branches off and joins the lingual nerve. The chorda tympani contains preganglionic parasympathetic fibers originating in the superior salivatory nucleus. The parasympathetic fibers innervate the submandibular and sublingual glands via the submaxillary ganglia. Afferent taste fibers from the anterior two thirds of the tongue also travel in the chorda tympani to reach the nucleus of the solitary tract.

The facial nerve fibers exit at the stylomastoid foramen (see Figure 7-24). The branches given off are the posterior auricular nerve, the digastric nerve (to posterior belly of digastric muscle), and the stylohyoid nerve. The facial nerve pierces the parotid gland and divides into temporofacial and cervicofacial branches. Its terminal branches are the temporal, zygomatic, and mandibular nerves supplying the facial mimetic muscles and the platysma.

The visceral efferent (parasympathetic) fibers of the nervus intermedius of Wrisberg supply all the major glands of the head except the parotid gland and glands of the skin. From the superior salivatory lacrimal nucleus, preganglionic fibers divide in the facial canal into the greater petrosal nerve and the chorda tympani. In the pterygoid canal, the greater petrosal nerve is joined by the deep petrosal nerve

(CN V_2). Together they reach the pterygopalatine (sphenopalatine) ganglion. Postganglionic fibers from the sphenopalatine ganglion supply the lacrimal, palatal, and nasal glands. The chorda tympani joins the lingual nerve (CN V_3) to reach the submaxillary ganglion. Postganglionic fibers subserve the submandibular and sublingual glands.

The visceral afferent primary neurons are located in the geniculate ganglion. The fibers of these neurons carry taste sensation (gustatory) from the anterior two thirds of the tongue. These gustatory afferents then travel in the nervus intermedius and end in the tractus solitarius and its nucleus in the medulla. Taste information is received from fungiform papillae on the anterior two thirds of the tongue and is transmitted via the chorda tympani (CN VII[114]; Figure 7-25). Taste information from the circumvallate papillae at the junction of the posterior third and anterior two thirds of the tongue, and from the foliate papillae at the rear edge of the tongue, is mediated by the lingual branches of CN IX. Fibers of both cranial nerves terminate centrally in the gustatory nucleus. Taste buds on the laryngeal surface of the epiglottis transmit impulses through the superior laryngeal nerve, a branch of CN X.

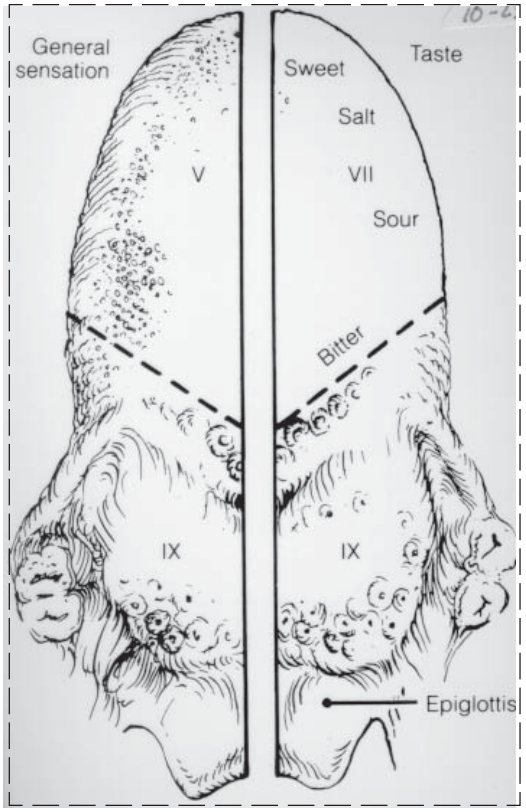


Fig. 7-25. Sensory innervation of the tongue. Roman numerals represent the cranial nerves innervating that area. Reprinted with permission from deGroot J, Chusid JG. *Correlative Neuroanatomy.* 20th ed. Norwalk, Conn: Appleton & Lange; 1988: 169.

The palatal taste buds located on the margin between the hard and soft palates transmit taste through the greater superficial prostrosal nerve, which lies deep to the semilunar ganglion and traverses the petrous portion of the temporal bone. The nerve exits the middle cranial fossa at the facial hiatus and joins the anterior margin of the geniculate ganglion. From that point fibers run in the nervus intermedius. Taste projections from the medulla terminate in the ventroposteromedial nucleus of the thalamus. Whether the projections are ipsilateral or contralateral is not known, for certain.

The somatic afferent primary neurons are located in the geniculate ganglion in the petrous temporal bone. They receive afferent fibers from the mucosa of the pharynx, nose, and palate, and from the skin of the external auditory meatus, lateral pinna, and mastoid. The sensory fibers enter the brainstem via the nervous intermedius, then descend in the spinal tract of the trigeminal nerve to synapse in the trigeminal spinal nucleus.

Proprioceptive fibers from the facial muscles travel in CN VII. They terminate in the mesencephalic nucleus of CN V.

Central connections for control of innervation of facial movements have fibers arising in the lower third of the precentral gyrus. The fibers course downward through the genu of the internal capsule (as the corticopontine tract) to the base of the peduncle, and terminate in the facial nucleus. The ventral part of the facial nucleus innervates the lower two thirds of the face. For the most part it has crossed supranuclear control. The dorsal portion supplies the upper third of the face and is controlled by bilateral supranuclear input. It is believed that the upper facial motor neurons controlling upper facial movements receive little direct cortical input, while the lower facial motor neurons have significant cortical innervation. Therefore, in supranuclear lesions there is sparing of the upper face.[115]

Ipsilateral innervation of the facial nucleus is also received from the extrapyramidal system originating cortically, that is, from areas anterior to the central sulcus and from areas behind the sulcus. From there, fibers proceed to the basal ganglia (mainly the putamen), then to the brainstem tegmental nuclei, and then to the facial nucleus. As the integrating center of the autonomic nervous system, the

hypothalamus has much influence on the parasympathetic superior salivatory nucleus. Impulses from the limbic system (emotional behavior) and the olfactory area reach the lacrimal nucleus via the dorsal longitudinal fasciculus. Visceral reflexes, such as salivation in response to smell, are mediated through these pathways.

With respect to motor innervation, the pathways for emotional movements may be different from those for voluntary movements. It appears that emotional pathways do not descend through the internal capsule. It was reported by Borod and colleagues[116] that the right hemisphere may be especially significant for emotional expressions of the face.

Sensory connections from the facial nucleus are to the thalamus and postcentral gyrus. The gustatory center is believed to be in the parietal sensory cortex. There is stimulation of the lacrimal nucleus by the gustatory nucleus to produce secretion from oral glands.

Taste information is received from fungiform papillae on the anterior two thirds of the tongue and is transmitted via the chorda tympany (CN VII). Taste information from the circumvallate papillae at the junction of the posterior third and anterior two thirds of the tongue and the foliate papillae at the rear edge of the tongue are mediated by the lingual branches of CN IX. Fibers also terminate in the gustatory nucleus. Taste buds on the laryngeal surface of the epiglottis transmit impulses through the superior laryngeal nerve, a branch of CN X. The palatal taste buds located on the margin between the hard and soft palate transmit taste throughout the greater superficial petrosal nerve, which lies deep to the semilunar ganglion and traverses the petrous portion of the temporal bone. It exits the middle cranial fossa at the facial hiatus and joins the anterior margin of the geniculater ganglion. From there on, fibers run in the nervus intermedius. Taste projections from the medulla terminate in the ventroposteromedial nucleus of the thalamus. Whether the projections are ipsi- or contralateral is not well known.

Injuries and Lesions

Taste awareness is present at birth and is not significantly reduced by aging. There are genetic differences in abilities to taste certain bitter or sweet substances. Taste may be lost partially (hypogeusia) or totally (ageusia), and there can occur taste phantoms (dysgeusia). The taste system functions by a balance of excitatory and inhibitory neuromessages. Loss of taste on one side of the tongue may cause hypersensitivity to taste on the other side. For this reason, total loss of taste due to unilateral nerve injury is rare.

The most common cause of injury or lesion of CN VII is trauma such as head injury, which very commonly affects CN VII.[114] Gustatory fibers may be involved. Central lesions may be bilateral,[117–119] but peripheral nerve damage is mostly unilateral. The incidence of involvement in head injuries is 0.5%, but 5% of patients with posttraumatic anosmia also have some deficit in taste sensation.[6]

An incidence of 10% to 15% facial paralysis was reported for patients with longitudinal fractures of the temporal bone, and 30% to 50% was reported for those with transverse fractures. The lesion is most often located at the geniculate ganglion. Fisch[120] reported that for 50% of the fracture patients facial palsy was due to an intraneural hematoma, 26% showed evidence of transection, and 17% bone impingement. Injuries to the ear or to the side of the face (mandible, parotid gland) also frequently result in peripheral facial nerve injury.

Nontraumatic lesions such as Bell's palsy are quite common. The incidence is clustered. Thirty percent of patients with Bell's palsy have a close family member with a history of Bell's palsy. Other nontraumatic lesions are neoplasms (especially cerebellopontine angle tumor), infections, cerebrovascular accident, and idiopathic. Bilateral Bell's palsy is suggestive of Lyme's disease.

Syndromes involving CN VII are mostly due to tumors, multiple sclerosis, infarction, poliomyelitis, or congenital abnormalities. Syndromes include the following:

- Millard-Gubler syndrome is caused by a lesion in the ventral pons. It presents with ipsilateral peripheral facial palsy, ipsilateral rectus palsy, and contralateral hemiplegia.
- Foville's syndrome is caused by a lesion in the pontine tegmentum and manifested by ipsilateral peripheral facial palsy, paralysis of conjugate gaze to the side of the lesion, and contralateral hemiplegia.
- Möbius' syndrome is congenital absence of facial muscles or nerves. It presents with facial diplegia, abducens palsy, and sometimes deafness.
- Ramsay-Hunt syndrome is caused by an injury to the geniculate ganglion or infection by herpes zoster. Symptoms and signs are ipsilateral facial paralysis, pain in the eardrum region, hyperacusis due to lack

of the stapedius muscle function that dampens sound, and loss of taste. In herpes there will be herpetic vesicles on the eardrum, external auditory meatus, and palate, in addition to the geniculate neuralgia.
- Bilateral facial palsies may be congenital, or due to infections such as *Mycoplasma pulmonalis*, infectious mononucleosis, or Lyme's disease; granuloma (sardoidosis); or Guillain-Barré syndrome. Other possible causes are allergic vascular disease, trauma, or birth injuries (forceps).
- Gustatory hyperhidrosis syndrome (auriculotemporal syndrome) is common after parotiditis, trauma, or surgery. It manifests as hemifacial redness and sweating during eating.
- Chorda tympani syndrome has the same expression as gustatory hyperhidrosis but spreads to involve the neck, back, and chest. Possible misdirected regeneration of nerve fibers is the cause.
- Crocodile tears are paroxysmal lacrimation during gustatory stimulation. Fibers that normally reach the submaxillary gland are traumatized and during regeneration are misdirected to the lacrimal gland.

The symptoms or signs of CN VII lesions are mainly mimetic facial muscle weakness or paralysis. Depending on the location, different afferent fibers may also be affected. Possible dysfunctions are

- Dysfunction of lacrimation.
- Hearing impairment (hyperacusis) due to loss of stapedius muscle function. Normally, strong acoustic stimuli cause stapedius muscle contraction that pulls the stapes out of the round window and so attenuates the loud sound. If this reflex is impaired, hyperacusis results.
- Loss of salivation.
- Loss of taste from the anterior two thirds of the tongue. From 10%[121] to 52%[122] of patients with Bell's palsy have gustatory problems.
- Minor sensory deficit at the external ear.
- Retroauricular pain (somatic pain fibers from the external ear canal and the skin between mastoid and pinna).
- Dysarthria, pooling of saliva, and excessive tearing. Food may collect between gum and cheek due to buccinator weakness.
- The jaw may deviate to the sound side when the mouth is opened wide because CN VII innervates the posterior belly of the digastric muscle.[123] In contrast, the jaw deviates to the involved side in CN V lesions, due to weakness of the pterygoid muscles.
- Mimetic muscle weakness.

In upper motor neuron lesions there may be weakness of lower facial muscles of expression with intact automatic or emotional movements. This may be possible through bilateral subcortical innervation from deep gray nuclei.[124] Abnormal facial movements may be classified as oral facial dyskinesis. Such movements include facial grimacing, twitching, puckering, lip smacking, protrusion of the tongue, or oromandibular dystonia. The cause may be extrapyramidal disease or it may be idiopathic. Other abnormal movements are hemifacial spasm or postparalytic spasm and synkinetic movements, mostly due to aberrant reinnervation or ephaptic transmission. Symptoms and signs according to the location of the lesion are reviewed in Table 7-3 and Figure 7-23.

Evaluation

There are several clinical tests available to localize a facial nerve lesion.[125] The *Schirmer lacrimation test* measures the production of tears and saliva. Filter papers 5.0 x 0.5 cm are bent on one end and inserted into each lower lid to collect tears. Normally the moisture migrates approximately 3.0 cm in 5 minutes.

The *stapedius muscle reflex* is examined when the patient complains of hyperacusis, especially for low tones (this suggests stapedius muscle weakness). A stethoscope is placed into the patient's ear with a gentle vibrating tuning fork on its bell. Normally the sound is heard equally in both ears. If stapedius reflex is absent the sound will be lateralized to the involved side.

The *salivary flow meter* measures production of saliva. *Gustometry* examines the sense of taste. A cotton swab is moistened with a dilute solution of sugar (4% glucose); salt (2.5% sodium chloride); vinegar; 1% citric acid solution; or 0.075% quinine hydrochloride. The solution is applied to the tongue outside, not inside, the mouth, separately to each side. Patients should drink water between stimuli to remove any traces of the previous application. The anterior two thirds of the tongue is tested.

Motor testing of the mimic facial musculature is done by clinical observation of possible asymme-

TABLE 7-3

SYMPTOMS, SIGNS, AND CAUSES ACCORDING TO LOCATION OF FACIAL NERVE LESION

	Symptoms and Signs	Possible Causes
Supranuclear	Paralysis of contralateral limbs and lower face No reaction of degeneration Taste and salivation not affected	CVA, head injury, trauma
Nuclear	May present with paralysis of conjugate gaze to ipsilateral side and contralateral hemiplegia Involvement of other cranial nerves (V, VI) Deafness (CN VIII) Impairment of lacrimations and salivation (nervus intermedius) Hyperacusis Mimic facial muscle paralysis with abnormal EMG	Pontine lesion, trauma, cerebello-pontine angle tumor
Meatal Segment	Impairment of lacrimation and salivation (nervus intermedius) Hyperacusis Loss of taste, anterior two thirds of tongue Mimic facial muscle paralysis with abnormal EMG	Fracture of petrous temporal bone; or pressure transition between meatal and labyrinthine segment may cause facial nerve compromise
Labyrinthine and Horizontal Segments	Often tympanic membrane pain Hyperacusis Loss of taste, anterior two thirds of tongue Mimic facial muscle paralysis with abnormal EMG	Trauma, mastoiditis, neoplasm (usually benign), herpes zoster
Suprachordal Mastoid Segment	Loss of taste, anterior two thirds of tongue Mimic facial muscle paralysis with abnormal EMG	Trauma, middle ear infections, measles, mumps, chicken pox, sarcoiditis
Infrachordal Mastoid Segment	Mimic facial muscle paralysis with abnormal EMG	Tissue swelling within canal of undetermined origin

CVA: cerebrovascular accident
CN: cranial nerve
EMG: electromyogram
Localization my not always be as precise as mentioned above. A general rule is that lesions proximal to or at the geniculate ganglion produce taste, lacrimation, and stapedial reflex abnormalities; lesions distal to the ganglion cause only muscle weakness.[1]
(1) Bartoshuk LM, Kveton JF, Karrer T. Taste. In: Bailey BJ, ed. *Head and neck surgery. Otolaryngology*. Philadelphia, Pa: JB Lippincott; 1993: 520–529.

try of the face during smiling, speaking, raising eyebrows, wrinkling forehead, and resistance given to grimacing. Upper motor neuron lesions spare the forehead and mostly the eyes. Lower motor neuron lesions may involve only the lower face if the lesion is restricted to the caudal portion of the nucleus, as may happen in polio.

Observation of any *reflex phenomenon* is important. Reflex eye closure is observed in response to threatening movements of a hand in front of the patient's face (menace reflex). A glabellar tap over the bridge of the nose elicits reflex blinking. Care is taken to hold the hand over the top of the head to avoid eliciting a visual blink reflex. After about three taps, blinking ceases. In patients with Parkinson's disease, blinking continues.

Testing of the *auditory palpebral reflex* is accomplished by observing the reflex bilateral eye closure secondary to presentation of a sudden loud noise. It is important to look for the possible Elve's phenomenon, which shows upward turning of the eyeball under the closed lid. In facial palsy the lid may not be fully closed and the turning of the eyeball will be visible. Facial muscles are observed also in

response to emotions. Occasionally in lower facial palsy there is a dissociation of volitional and emotional mimetic muscle function.[124]

Electrodiagnosis

Electrodiagnostic studies include electrogustometry, gustatory evoked potentials, nerve excitability studies, nerve conduction evaluation, reflex studies, and EMG.

Electrogustometry is a test of an anodal current threshold. The patient experiences a sour metallic taste. This test investigates loss or decrease of "sour" taste only (ageusia), not loss of other tastes, and not the distortion of taste (dysgeusia). These are better evaluated with clinical chemical taste testing.[126]

Gustatory evoked potentials[127] can be performed but, at the present time, these tests are mainly performed for purposes of research. The procedure is explained below.

Stimulation is achieved when an electrical stimulus or certain volatile tastants are presented to the taste buds. Because of habituation, presentation must be at random, with at least 5 seconds between each stimulus.

Recording electrodes are placed over C_7 and A_1 (International 10-20, as seen in Figures 7-5, 7-6, 7-7).

Normal values are a response in approximately 300–400 millisecond.

Nerve excitability studies have been performed often for early detection testing for threshold stimulation. Even after complete facial nerve section, the distal excitability remains normal for up to 4 days. There will no longer be any response at the end of the first week. If excitability remains, prognosis is good. The facial nerve is stimulated at the stylomastoid foramen with an electrical current of 1 millisecond duration. A minimal amount of current is applied to cause a minimal twitch in a facial muscle. The normal threshold is between 3 and 8 mA. There should be less than 2 mA difference between the two sides of the face.

Nerve conduction studies are the most commonly employed electrodiagnostic evaluations of the facial nerve. They are sensitive tests for evaluating axonal loss when the response amplitudes from side to side are compared. Some investigators recommend retesting every other day for the first 2 weeks for best prognostication.[128] If the evoked motor amplitude on the involved side is smaller than 10% of that on the good side, the prognosis for recovery is poor. The procedure for direct facial nerve conduction[129] is given below.

Procedure of Direct Facial Nerve Conduction Studies

Stimulation is carried out at the stylomastoid foramen.

Recording electrodes are placed in or over facial muscles. Preferred location is over the nasalis muscle. The lower orbicularis oculi muscle may be tested also when studying the blink reflex. The orbicularis oris, mentalis, or frontalis muscles may also be used for recording.

Normal values:

- Latency: 3.2 ± 0.34 millisecond (range, 2.2 to 4.0 ms)
- Amplitude: 2.0 ± 0.9 mV (range, 1 to 5 mV measured baseline to peak)
- Duration: 12.8 ± 2.9 millisecond (range, 6.2 to 22 ms)

Reflex studies can be utilized for indirect facial nerve conduction, especially to evaluate the proximal conduction velocity. This can be estimated by eliciting the blink reflex[106] (see trigeminal nerve). The clinical value of the blink reflex in Bell's palsy is used mainly for prognostication. If R1 has been absent, its return suggests a reasonable prognosis, even though the R1 may be delayed during the first 4 weeks. Delay indicates demyelination rather than physiological (functional) nerve block. The R1-D ratio (R1 vs direct facial response) is helpful to delineate whether a lesion is more proximal or distal. For example, the R-D ratio is slightly increased in Guillain-Barré Syndrome and multiple sclerosis, but decreased in Charcot-Marie-Tooth disease and diabetic polyneuropathy.

EMG is carried out in the usual fashion. A needle recording electrode is inserted into any one of the mimetic muscles. Evaluation is by observation of activity at rest, motor unit parameters during minor activation, and especially recruitment. The most commonly tested muscles are the orbicularis oris and oculi. The number of muscles having voluntary action potentials preserved for the first week provide an additional index for prognostication. Muscle over activity can present as:

- Facial myokymia, where motor units of normal shape and duration appear in a single, double, or group discharge about every 100 -to 200 millisecond, firing asynchronously. Continuous and discontinuous, but definitely rhythmic, patterns can be observed. Causes may be fatigue, mul-

tiple sclerosis, brainstem tumors, or injury. Myokymia is not affected by sleep or facial movements.
- Synkinesis, in which there is simultaneous motor unit activity in different facial muscles. Simultaneous activation of muscles innervated by different nerve trunks is also observed when a stimulus is given to one branch, or during a blink reflex study. This phenomenon is associated with regeneration or degeneration of the facial nerves (aberrant reinnervation or ephaptic transmission).
- Hemifacial spasm, as manifested by high frequency (150 to 400 Hz) rhythmic discharges with synchronization. The most common cause is compression of CN VII by an aberrant arterial loop. Therefore, for this lesion, decompression may be of help.[130] Hemifacial spasm is not of cortical origin and cannot be abolished by sleep or cerebral infarction. The most likely cause is ephaptic transmission.

The prognosis for recovery of facial nerve injuries as well as upper motor neuron facial palsy is in general good, without intervention.[131–135] Within 2 to 6 months, 60% to 70% of all Bell's palsy patients recover fully or have only minimal residual deficit. Thirty percent to 40% have slow or defective motor recovery after more than 6 months.

In a patient with incomplete facial palsy, if the evoked motor amplitude of an affected facial muscle is 10% or more of the amplitude of the unaffected side in the first 14 days after onset, the prognosis is good. Ninety percent of these patients will recover satisfactorily. By contrast, patients who show clinically complete palsy and an evoked response smaller than 10% within the first 14 days have an 80% chance of poor recovery.[128] Other criteria for possibly good prognosis reported in the literature are the presence of voluntary action potentials on EMG registered in two or more muscles on or after the third day, and return of the previously absent R_1 (or R_2) of the blink reflex.[131]

Management

Most clinicians promote early administration of prednisone, 1 mg/kg of body weight daily in two divided doses for 4 days, then a quick taper to a total of 5 mg/d within 10 days.[136] Dexamethasone, 10 mg intravenously every 12 hours, is also recommended, especially for facial palsy after head trauma.[137] This regimen may reduce edema and therefore compression of the facial nerve in the bony canal and may reduce synkinetic movements. If pain is present, steroids relieve symptoms promptly. If severe facial nerve paralysis persists, surgical intervention may be indicated. Hypoglossal facial nerve anastomosis may restore tone and function. A study[138] of 22 cases (out of a study sample of 245 surgical cases) of complete facial nerve palsy that occurred during cerebellopontine angle tumor removal reported good results in 63.6%, fair in 13.6%, and poor in 18.2%. One case (4.5%) was a failure, following hypoglossal-facial nerve anastomosis.

Electrical motor point or muscle stimulation to retard atrophy and promote early regain of function has been advocated and widely used. However, the effectiveness of this mode of treatment has not been proved scientifically and is not clinically convincing.

To prevent complication from possible corneal irritation secondary to incomplete eye closure, administration of artificial tears during the day and ophthalmic ointment at night, such as Lacrilube, are recommended. A protective plastic wrap taped around the eye may be helpful.

CRANIAL NERVE VIII: ACOUSTIC

The acoustic nerve is a composite sensory nerve with two separate parts, the cochlear (auditory) nerve and the vestibular nerve. The anatomy is shown in Figures 7-26 and 7-27.

Anatomy and Function

The cochlear nerve

The cochlear nerve is a special sensory nerve. The primary neurons are bipolar cells in the spiral ganglion of the cochlear nerve. The peripheral branches of the cells end in auditory receptors (hair cells) located in the cochlear duct. The entire sensory structure is called the organ of corti. Hair cells in the cochlear apex are stimulated by low frequencies, and basilar hair cells respond to high frequencies. The mechanical deformation of the hair cells is transduced into electrical signals. The central branches of the bipolar cells terminate in the ventral (low frequency) and dorsal (high frequency) cochlear nuclei (see Figure 7-27).

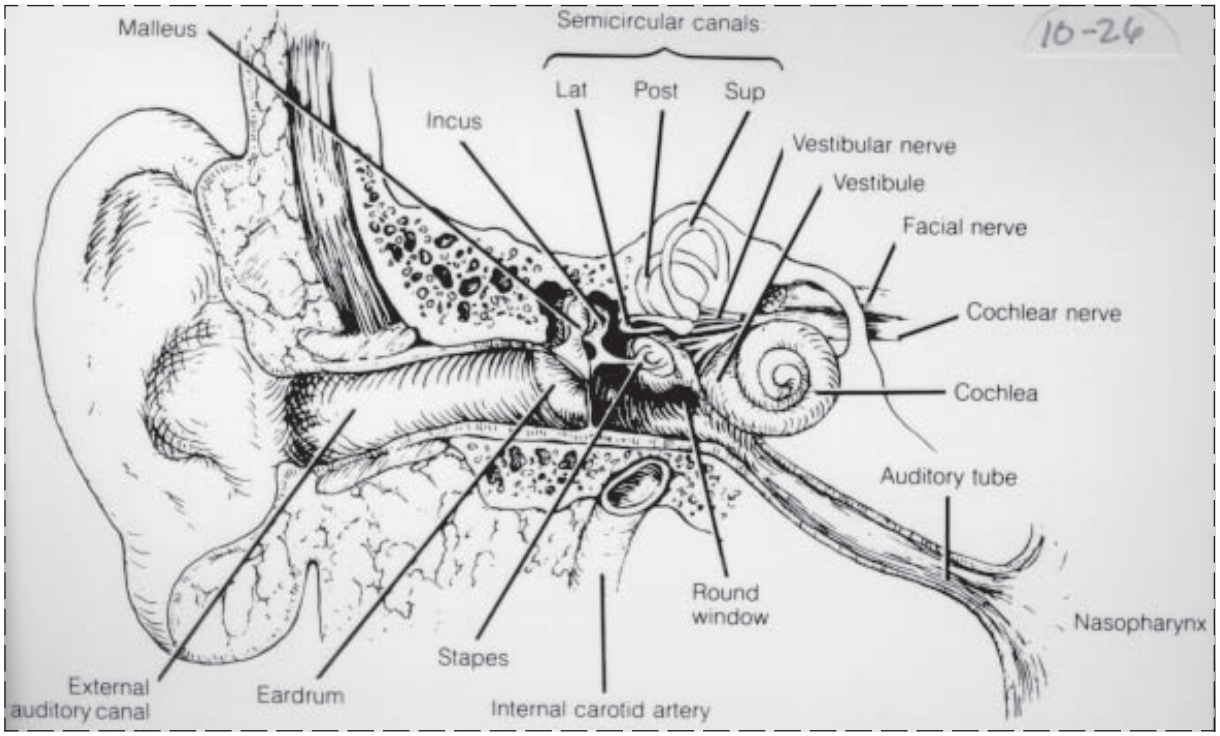


Fig. 7-26. The human ear. Middle ear muscles omitted. Reprinted with permission from Ganong WF. *Review of Medical Physiology.* 16th ed. Norwalk, Conn: Appleton & Lange; 1993; 157.

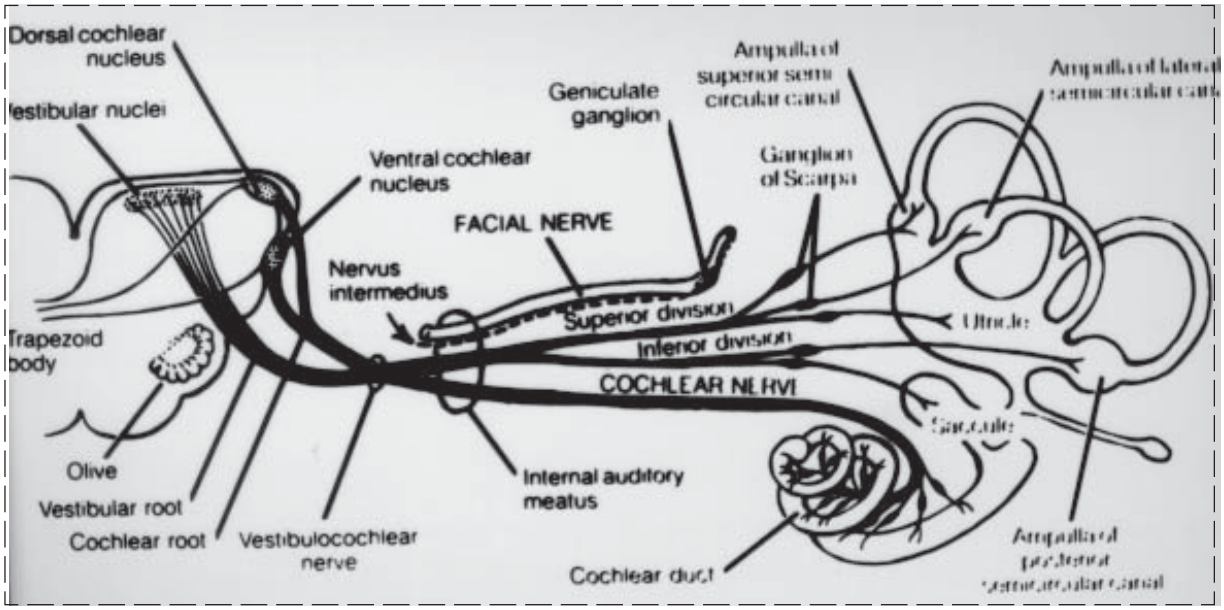


Fig. 7-27. Vestibulocochlear nerve. Reprinted with permission from deGroot J, Chusid JG. *Correlative Neuroanatomy.* 20th ed. Norwalk, Conn: Appleton & Lange; 1988: 157.

The secondary neurons are in the dorsal, anteroventral, and posteroventral cochlear nuclei. Innervation is tonotopically organized; the dorsal nuclei receive high frequency stimuli and the ventral aspects receive fibers conveying low frequency signals. From the dorsal and ventral cochlear nuclei nerve impulses are projected to the contralateral brainstem. Here they ascend in fibers of the lateral lemniscus to terminate in the inferior colliculus.

The tertiary neurons are in the inferior colliculus (midbrain tectum). Again, the fibers are located in tonotopic organization. From the inferior colliculus, fibers terminate in the medial geniculate body (thalamus). Fibers responding to low frequencies end in the apical lateral areas, and those responding to high frequencies end in the medial portions.

The central connections arise from the medial geniculate body. Auditory radiations are projected to the auditory cortex (area 41) located in the contralateral temporal gyri, and others terminate in the auditory association cortex (area 42). Reflex connections pass to the eye muscle nuclei and other motor nuclei of the cranial and spinal nerves via the tectobulbar and tectospinal tracts. Some fibers project bilaterally to the facial nucleus for reflex contractions of the stapedius muscle to dampen loud sounds.

There are also some inhibitory efferent axons with cell bodies in the superior olivary complex in the brainstem. A feedback loop function to suppress unwanted auditory signals is suggested.[139]

The vestibular nerve

The vestibular nerve also carries special sensory fibers. The primary neurons are in bipolar cells in the vestibular ganglion (ganglion of scarpa; see Figure 7-27). Peripheral fibers pass to the neuroepithelium in the ampullae of the semicircular canals and in the maculae of the utricle and saccule. The sensory receptors in the maculae are ciliated hair cells covered by gelatinous material. Otoliths (calcium carbonate crystals) are located in the gel and stimulate the hair cells in response to movement. Central branches enter the brainstem medial to the restiform body and end in the vestibular nuclei.

The vestibular system is concerned with maintaining equilibrium. The utricle, saccule, and semicircular canals make up the labyrinth. The main function of the utricle and the saccule is to detect the position of the head relative to gravity. Special receptors (hair cells) in the maculae of the utricle and saccule monitor linear acceleration, while angular acceleration is monitored by the cristae in the ampullae of the semicircular canals. Horizontal head movements stimulate the utricle, whereas tilting of the head activates the saccule. The superior portion of the vestibular nerve conducts impulses from the anterior and horizontal semicircular canals and from the utricle. The inferior portion of the nerve transmits information from the posterior semicircular canal and the saccule. The vestibular nuclei exert an influence on conjugate eye movements and on head and neck movements through the medial longitudinal fasciculus.

Pathways from the lateral vestibular nucleus project to the ipsilateral spinal cord. These fibers facilitate extensor trunk tone and the action of antigravity axial muscles.

Central connections of the vestibular system are mainly projections to the cerebellum. However, the medial longitudinal fasciculus projects ipsilaterally from the superior vestibular nucleus and contralaterally from the medial vestibular nucleus to the eye muscle nuclei. The cortical representation of the vestibular system is located in the postcentral gyrus (near areas 2 and 5).

Injuries and Lesions

The most common causes of injury and lesions are traumatic injuries, especially head trauma. It is reported[140] that the sensory organ most commonly injured is the ear. Blunt trauma causes inner ear concussion with symptoms of high frequency sensorineural hearing loss, transient positional nystagmus, and vertigo.[141] If the temporal bone is fractured, significant injury to the auditory and vestibular systems results. Eighty percent to 90% of patients with longitudinal fractures of the petrous portion of the temporal bone experience conductive hearing loss due to disruption of the ossicular chain in the middle ear, often due to dislocation of the incus. Ten percent to 20% of fractures of the temporal bone are transverse, causing sensorineural hearing loss and often involvement of the facial nerve. The vestibular system is frequently involved, and as many as 65% of traumatic brain injury patients are reported to demonstrate symptoms of vestibular dysfunction some time during their recovery.[142–145]

Nontraumatic lesions are mostly caused by tumors. Cerebellopontine angle tumors cause mostly unilateral sensorineural hearing loss with the main deficit in the high frequencies. The most important lesion to rule in or out with CN VIII testing is a cerebellopontine angle tumor, which is most often a vestibular schwannoma. The types of tumors ob-

served are mainly acoustic neuromas (mostly originating in the vestibular portion of CN VIII), schwannoma, meningioma, cholesteatoma, metastatic lesions, and posterior fossa lesions (possibly cerebellar junction hematoma). Other causes of CN VIII lesions are cranial neuropathy in association with systemic disorders; arteriovenous malformations; congenital and familial disorders; demyelinating diseases (multiple sclerosis); drug intoxications; excessive noise exposure; presbycusis (hearing loss secondary to aging); atherosclerosis; and viral diseases (mumps). Ménière's disease (fluctuating sensorimotor hearing loss, ear pressure, and tinnitus) may be a delayed consequence of conclusive injury.

Symptoms and Signs

The symptoms and signs observed with cochlear nerve lesions are mainly tinnitus, hearing loss, or both. Tinnitus is seen in up to 32% of adults visiting the audiologist or otologist.[146] Many patients experiencing tinnitus sustained a minor head injury or other injuries or diseases resulting in hearing loss. Tinnitus may precede, follow, or coincide with the onset of hearing loss. It may actually be an early symptom of a tumor in the internal auditory meatus or the cerebellopontine angle.[147] Tinnitus is characterized by humming, whistling, hissing, roaring, or throbbing auditory sensations.

Patients may hear these noises in one or both ears, or localized in the head. The most common cause is cochlear disease, but tinnitus can also result from a lesion in the external ear canal, tympanic membrane, ossicles, cochlea, auditory nerve, brainstem, or cortex. Six million persons in the United States reportedly experience tinnitus, which is often as disabling as hearing loss. Patients so afflicted are often willing to undergo multiple surgical procedures even if hearing in the involved ear may have to be sacrificed.[148] The examiner may be able to hear tinnitus caused by abnormal muscle contractions such as palatal myoclonus, tensor tympany, or stapedial myoclonus. Such mechanical tinnitus is an objective find.

Hearing impairment is the most prevalent chronic physical disability in the United States. Health statistics estimate that by the year 2050 more than 10% of the population will be hearing impaired.[148] The degree of hearing impairment as correlated with the inability to understand speech is shown in Table 7-4. Some of the causes of impairment and the corresponding effects are listed below.

- Conductive hearing loss due to lesions of the sound conducting apparatus of the middle ear affects mostly the lower frequencies.
- Sensorineural hearing loss is due to lesions central to the oval window, and affects mostly the higher frequencies.
- A lesion in the central auditory pathways beyond the cranial nerve causes central auditory dysfunction, but not complete deafness.

TABLE 7-4

AVERAGE HEARING THRESHOLD LEVEL FOR 500, 1,000, AND 2,000 Hz IN THE BETTER EAR

Class	Classification Category of Hearing Loss	More Than (dB)	Not More Than (dB)	Ability to Understand Speech
A	Within normal limits		25	No significant difficulty with speech
B	Slight or mild	26	40	Difficulty only with faint speech
C	Moderate	41	55	Frequent difficulty with normal speech
D	Moderately severe	56	70	Frequent difficulty with loud speech
E	Severe	71	90	Can understand only shouted or amplified speech
F	Profound	91		Usually cannot understand even amplified speech

Adapted with permission from Davis H. Guide for the classification and evaluation of hearing handicapped. *Trans Am Acad Ophthalmol Otolaryngol.* 1965;69:740–751.

- Hearing scotomas may be caused by hysteria, multiple sclerosis, schizophrenia, and at times trauma. This condition reveals deafness only to certain frequencies and noises.
- Auditory hallucinations may occur as an epileptic aura or during drug intoxications or psychoses.
- Hearing impairment may be secondary to lesion of CN V because of its innervation of the tensor tympany.
- Hearing impairment may be secondary to lesion of CN VII, because this nerve innervates the stapedius muscle. It supplies the efferent limb of the stapedial reflex.

Presbycusis may present with sensorineural hearing loss caused by loss of neurons in the central nervous system or by degeneration of the sensory and supportive cells in the cochlea. Presbycusis may also present with conductive hearing loss. This may be due to metabolic changes caused by atrophy of the stria vascularis (the site of endolymph production), or it may be mechanical, resulting from stiffness of the basilar membrane in the cochlea.

Vestibular nerve dysfunction symptoms and signs include dizziness, vertigo, nystagmus, ataxia, and dysequilibrium. The main symptoms and signs are dizziness, vertigo, and nystagmus, in any combination.

Dizziness is described as lightheadedness, faintness, dysequilibrium, or disturbance of consciousness. It is a disturbance in perception of bodily position in space. Dizziness may be confused with vertigo. Common systemic causes are cardiovascular disease, hematologic disorders, hypoglycemia, hypothyroidism, hyperventilation syndrome, ocular disorders, drugs, and psychiatric disorders.

Vertigo is the experience of movement of the self or the environment and is caused by lack of integration of information received by the visual, auditory, and vestibular systems. The basic functions of the vestibular system are spatial orientation at rest and during acceleration, visual fixation during head movement, body movement (vestibuloocular reflex), and feedback control of muscle tone to maintain posture. Vertigo reflects a disturbance in one or more of these systems.

Vertigo can have peripheral causes. Idiopathic (positional) vertigo is usually of short duration and not accompanied by cochlear or neurological symptoms, although nystagmus is frequently present. Matutinal vertigo (vertigo occurring on arising in the morning) may be peripheral or central. Other peripheral causes include peripheral vestibulopathy (extramedullary), vestibular neuronitis, acute labyrinthitis (associated with tinnitus and hearing loss), Ménière's disease (vestibular type), and vertigo secondary to middle ear disease or viral infections.

Posttraumatic vertigo and unsteadiness may be due to injury to the vestibular nuclei and cerebellum.[149] The incidence of vertigo after head trauma has been reported as 24% and 78%.[149–151] Centrally produced vertigo can also result from vascular causes (transient ischemic attacks mainly of the vertebrobasilar artery) and is mostly of prolonged duration. Other central causes are labyrinthine stroke, Wallenberg's syndrome, multiple sclerosis, cere-bellopontine angle tumor (causing hearing loss more frequently than vertigo), and vestibular epilepsy.

Nystagmus is an eye movement disorder consisting of a slow drift, which is quickly corrected by saccades to return to the desired focus. Nystagmus may be induced by central or peripheral vestibular lesions. The eyes drift parallel to the plane in which the diseased canal lies. Nystagmus may be horizontal or vertical.[152] A history of dysequilibrium (ataxia and unsteady gait) may be observed with vestibular nerve injury.

Evaluation

Clinical evaluation of the *cochlear nerve function* employs the following hearing tests[148]:

- The auditory screening test is performed by the examiner whispering into the ear or presenting a ticking watch at a distance of 60 cm, while the opposite ear is masked by gently occluding the external ear or pressing on the tragus. Sound should be perceived by a person of normal hearing.
- Pure tone audiometry (Figure 7-28) measures the hearing sensitivity to selected pure tone frequencies at calibrated sound pressure levels. The audiogram records the results graphically. It easily demonstrates low frequency or high frequency hearing loss and so may suggest the type of hearing deficit.
- The normal intensity of sound pressure reception is between 10 and 25 decibels (dB). The dB is a unit for expressing the difference between a given sound pressure level and a standard reference pressure of 0.0002 dyne/cm^2.

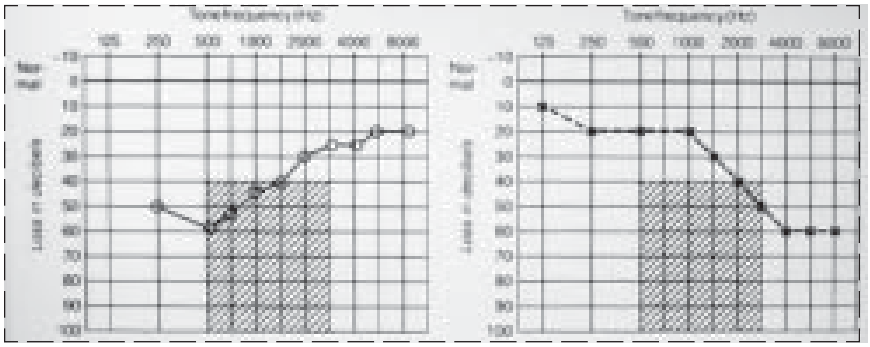


Fig. 7-28. Audiogram. Left: Middle ear (conduction) deafness. Right: perception (nerve) deafness. Reprinted with permission from deGroot J, Chusid JG. *Correlative Neuroanatomy.* 20th ed. Norwalk, Conn: Appleton & Lange; 1988: 161.

- The unit dB is used not only to describe sound-pressure level (SPL) but also to measure hearing sensitivity. The standard reference for hearing sensitivity is the amount of energy needed for a person of normal hearing to perceive that particular sound (the hearing threshold level—HTL). Threshold is defined as the level at which 50% correct responses are elicited. Although the frequencies perceived by the human ear can range from 20 to 20,000 Hz, the critical area is 500 to 3,000 Hz.
- Tympanometry (acoustic impedance—immittance evaluation) assesses the integrity and performance of the peripheral auditory system. The test uses a probe tip with three holes. One hole is for a probe tone at approximately 220 Hz; one is for cavity air pressure control; and one is for a microphone to record the sound pressure level in the ear canal. The probe tip is sealed into the external auditory meatus. A sound properly adjusted to the mass and stiffness of the system and the acoustical resistance of the ear is introduced, and the reflection from the tympanic membrane is measured.[147]

  Tympanometry provides an extremely sensitive measure of middle ear function. The normal middle ear absorbs and transmits energy easily, causing the tympanic membrane to have a low acoustic impedance. If the tympanic membrane is scarred or fluid is located in the middle ear, impedance is increased (static compliance is decreased).
- The stapedial reflex test may be performed if tympanometry is normal. Monaural stimulation causes bilateral contraction of stapedius muscles. Absent stapedial reflex strongly suggests a retrocochlear CN VIII lesion.
- A speech discrimination or word discrimination test evaluates reception of 50 phonetically balanced words at approximately 30 to 40 dB above the patient's speech reception threshold. Findings offer clues to the suspected severity of the handicap.
- The alternate binaural loudness balance test shows abnormal recruitment, an abnormal growth in the loudness of sound when intensity is increased above threshold levels. Sound is presented to both ears. The intensity to one side is held constant, while the other side is varied until the patient perceives the two as equal. Inequality of delivered sound intensities is identified. Abnormal recruitment suggests hearing loss due to cochlear disease.
- The test for hearing scotomas determines whether all frequencies can be heard. Testing is done with audiometry.
- An examination is made for sensory aphasia, or word deafness. This is an ability to hear but not comprehend words. Sensory aphasia is associated with lesions of the posterior portion of the superior temporal gyrus of the dominant cerebral hemisphere.
- Weber's test is performed by placing a tuning fork against the midline vertex of the scalp. Normally, no lateralization of sound to either ear is reported. If the sound is referred to the ear with hearing impairment, loss of hearing is due to impaired conduction in the external middle ear. If the sound is referred to the unimpaired hearing ear, loss of hearing is attributed to impaired function of the auditory nerve or cochlea.
- Rinne's test compares air and bone conduction. A vibrating tuning fork is placed first on the mastoid bone process, and then held with its tip beside the auricle. A patient with normal hearing or mild nerve deaf-

ness will report that it is louder when near the auricle. A patient with middle ear (conductive) deafness will report the tuning fork to be louder when placed on the mastoid bone. In severe nerve deafness no sound is heard in either position. Low frequency hearing impairment is mostly conductive hearing loss.

- Bine's test is conducted by placing a tuning fork (with pitch C, or 256 vibrations/s) on the vertex with one ear occluded. Normally the closed ear hears sound best by bone conduction. If no sound is heard in the closed ear, nerve deafness is suspected.
- An otoscopic examination examines the external canal to detect or rule out foreign bodies, congenital malformation, abnormal condition of the tympanic membrane, presence of wax, or evidence of infection. If a polyp is observed, care must be taken not to touch it because it may bleed disastrously.

Evaluation of vestibular nerve function includes inquiry about general symptoms such as history of diaphoresis, tachycardia, nausea, vomiting, and low blood pressure, such as may occur with labyrinthine disease. The following tests are performed:

- Nystagmus is observed and described as to type, direction of slow and fast components, and the head position in which it occurs. It may represent a disturbance in the reflex control of the ocular muscles, which is mainly a function of the semicircular canals. Other forms of nystagmus occur in central cerebellar and cerebral diseases.[152]
- In a positional test the patient is required to look to the left and then is assisted to recline quickly so that the head lies over the edge of the couch 45° below horizontal. The patient remains in this position with the eyes turned to the left for 30 seconds. Observation is made for possible nystagmus (peripheral type) and the patient is asked for complaints of vertigo. The patient then sits up with the head turned to the left for half a minute and is continuously observed. After any vertigo or nystagmus has ceased (in about a minute), the procedure is repeated with the head and eyes turned to the other side. Nystagmus and vertigo will normally occur after a latent period of 10 to 20 seconds. No latent period is observed with central lesion or habituation. With fatigue, signs and symptoms lessen after repetition of provocative maneuvers. With peripheral lesions, nystagmus and accompanying vertigo are significantly more severe, and often signs of brainstem involvement are seen.
- During caloric testing, cold water (to 10°C) is instilled into the right ear with the subject seated and head tilted backward 60°. Nausea responses normally occur, and there is horizontal nystagmus with a *slow* component to the right, pass-pointing to the left, and falling to the right; with warm water, the *quick* component is observed to the right. Complete interruption of vestibular nerve function is characterized by absence of the reaction to external irrigation; partial interruption of vestibular nerve function produces a diminished response. In the Hallpike caloric test, approximately 250 cm^3 of water at 30°C is applied to the external auditory canal for 40 seconds. The application is then repeated with water at 44°C. The normal response is nystagmus of 90 to 140 seconds duration.
- The Romberg test, which evaluates the ability to stand with feet together and eyes closed, is performed also with eyes open and the patient is observed for unsteadiness. Unilateral ataxia is almost always an indicator of focal posterior fossa abnormality (infarct, demyelination, abscess, or tumor).
- Posturography[153] is a quantification of the Romberg test. A recently developed dynamic posture platform is used to isolate the vestibular system for the purposes of the test. A movable visual field is provided with a special platform to remove visual and proprioceptive cues that otherwise would assist in maintaining posture. The patient's ability to maintain posture is evaluated. This procedure has not been properly validated, and further research is needed before scores can be adequately integrated.
- The examiner tests for presence of pass-pointing, which is the unidirectional drifting of outstretched fingers. Pass-pointing is a definite indication of tonic imbalance in the vestibular system.
- The examiner should test blood count, electrolytes, glucose, and thyroid function, and

should also check for hypercholesterolemia or increased triglycerides.

Electrodiagnosis

An electrical test can be performed to study the amount of galvanic current (mA) necessary to produce nystagmus, pass-pointing, and inclination of the head when current is passed between two saline pads placed one over each ear. The comparative effect of placing the cathode on the right and then on the left ear is determined.

In addition, electrodiagnostic evaluation includes ENG, electrocochleography, and brainstem auditory evoked potential (BAEP). BAEP is also called brainstem evoked response or auditory brainstem response (ABR).[154,155]

ENG offers an objective means of evaluating the optokinetic reflex, smooth pursuit, and saccades.[79] It can assist also in the diagnosis and interpretation of nystagmus by providing analysis of superficially recorded corneal-retinal potentials during gaze, positional changes, and simultaneous caloric testing. Bitemporal recording records only the sum of the movement of the two eyes. This simplifies the procedure but is unable to detect asymmetries between the two eyes, as may occur in internuclear ophthalmoplegia. A significant result is a difference of greater than 20% to 25% between the average slow component velocity (SCV) resulting from thermal stimulation of one ear compared to the SCV resulting from thermal stimulation of the other ear. This finding is an indication of hypofunction in one peripheral vestibular system (canal paresis).[79] Another benefit of ENG is that it can determine the presence of nystagmus through the eyelid in patients with ptosis or an inability to cooperate. The incidence of spontaneous nystagmus observed with ENG after head injury is reported as 23%.[154]

Electrocochleography measures electrophysiological activity that originates within the cochlea or the auditory nerve. This test studies the compound action potential of the auditory nerve. The recording electrodes must make contact with the promontory of the cochlea at the transtympanic membrane.

During BAEP studies, five main distinct wave forms representing different areas of the auditory pathways have been documented (Figure 7-29).[155] These important wave forms are believed to represent the following origins:

- wave I, the auditory nerve;
- wave II, the CN VIII or cochlear nuclei;
- wave III, the superior olive;
- wave IV, the lateral lemniscus; and
- wave V, the inferior colliculus.

It must be remembered that exact localization of the origin of these wave forms has not been determined at this time.

Cochlear nerve involvement in concussive states after closed head trauma may be objectified by BAEPs. Abnormal BAEPs were reported in 27.3%[156] of patients in postconcussive states, as manifested by unilateral or bilateral increase of interpeak latencies. These changes are often reversible.[156,157] Some conditions in which BAEPs are useful are listed below.

- In multiple sclerosis the most common abnormalities are an increase in waves I–V interpeak latencies and a reduction of amplitude of wave V.
- Acoustic neuroma may be associated with the absence of all waves, or the interpeak latencies of early waves I–III may be increased.

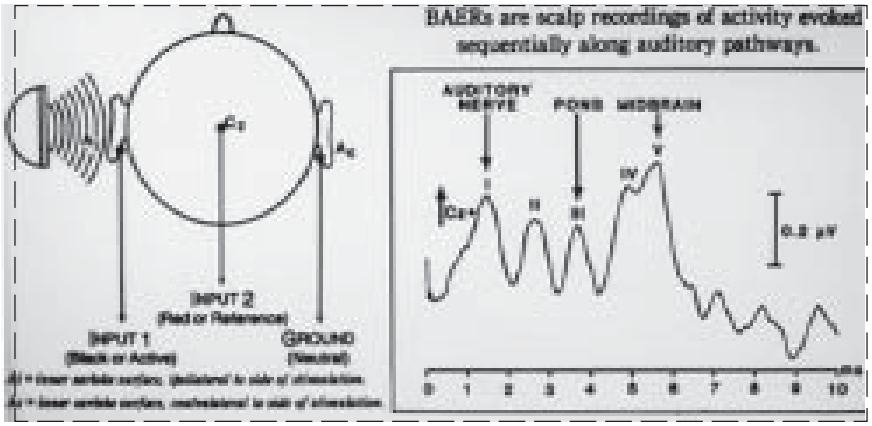


Fig. 7-29. Brain stem auditory evoked potential. Electrode placement and evoked response (schematic). Reprinted with permission from TECA Corp. *Brainstem Auditory Evoked Response (BAER).* In: TECA Applications Bulletin, No. 1001. Pleasantville, NY: TECA Corp: 1981.

- Brainstem tumors may show the absence of all waves after wave III. Multilevel lesions may abolish all waves except wave I.
- BAEPs may be used for intraoperative monitoring during brainstem surgical procedures.[14–16]
- In Sudden Infant Death Syndrome (SIDS), BAEPs may possibly be useful for detection of susceptibility.
- BAEPs can be used to assess brain death and coma, along with clinical and EEG assessment.

Procedure of BAEP Studies

Stimulations are monaural clicks presented to the ear under examination. Click polarity may be rarefaction or condensation. Duration of stimulus is 100 microsecond and the rate is 10/s. Intensity should be 60 to 80 dB above the patient's hearing threshold. The contralateral ear is masked by white noise of at least 40 dB above the patient's hearing threshold to prevent the stimulus from affecting the contralateral ear.

Recording electrodes are placed on the ear lobe and a reference electrode is placed on position C_Z (International 10–20, see Figure 7-7). Parameters and procedures are (*a*) sensitivity, 2.5 μV/division; (*b*) sweep speed, 10 ms/division; (*c*) filters: low frequency cut-off 100 to 200 Hz, high frequency cut-off 2 to 3 kHz; and (*d*) averaging number of responses is 1,000 to 4,000. Measurements taken are absolute peak latencies, interpeak latencies, peak-to-peak amplitudes (amplitudes are measured from peak to the following trough), relative peak-to-peak amplitudes, and inter-ear variations.

Normal values, with 10/s stimulation

- Latency to peak of wave, in milliseconds:
  wave I 1.7 ± 0.15
  wave II 2.8 ± 0.17
  wave III 3.9 ± 0.19
  wave IV 5.1 ± 0.24
  wave V 5.7 ± 0.25
  wave VI 7.3 ± 0.29
- Interwave latencies (interpeak), in milliseconds:
  I–III 2.1 ± 0.15
  I–V 4.0 ± 0.23
  III–V 1.9 ± 0.18
- Amplitudes peak to peak, in μV:
  wave I 0.28 ± 0.15
  wave III 0.23 ± 0.14
  wave IV 0.40 ± 0.13
  wave IV/V 0.47 ± 0.16 (highest peak)
  wave V 0.43 ± 0.16

The most significant abnormality is interwave separations (I–III or I–V), which are seen with cerebellopontine angle tumors.

The prognosis for improvement in auditory function is significantly greater for conductive than for sensorineural hearing loss. Patients with different types of presbycusis have varying degrees of success from the use of hearing aids. Patients with metabolic presbycusis have a significantly better prognosis than those with neural, sensory, or mechanical forms of presbycusis. The fact that hearing impairment is the most prevalent chronic physical disability certainly suggests limited possibilities of recovery.[148]

Recovery for patients with central vestibular damage is less complete than for patients with peripheral lesions. A study[143] of 321 patients with mild or moderate head injuries showed that 60% to 70% of patients with central vestibular dysfunction had persisting symptoms 5 years after the injury. Improvement in vestibular function is sometimes due to habituation of appropriate oculovestibular reflexes.

Prognosis for the recovery of CN VIII deficit secondary to ototoxic drugs is markedly improved if patients seek a physician's help at the earliest sign of tinnitus. Discontinuing or modifying the ototoxic medication may often preserve hearing.

Management

Cochlear Dysfunction

Tinnitus can be very disabling, as is often seen in patients who have had excessive noise exposure, head injury, or ototoxic medication. Medical management employs vasodilators, large doses of vitamin A, xylocaine, and carbamazepine. Biofeedback for tinnitus may be effective in some cases. Tinnitus maskers can be attached to the ear and a band of white noise introduced. The patient controls the intensity of the masking sound, which most likely occurs at the frequency of 2,000 Hz or higher. Another approach is external electrical stimulation. Electrodes are placed over the mastoid bone and different frequencies (which generate different size waves) are applied for progressively longer periods. Results have not been fully studied yet.[157,158]

The management of patients with hearing impairment varies with the etiology and severity of the impairment.[159]

Conductive hearing loss: Surgical or medical intervention or a combination of the two may be successful in treating conductive hearing loss. Pro-

cedures include repair and reconstruction of the tympanic membrane (myringoplasty), replacement of ossicles, plastic surgery on the outer ear, and early removal of CN VIII tumor.

Sensorineural hearing loss: Contrary to what is commonly believed, hearing aids can often improve sensorineural hearing loss, especially when caused by head injury. Audiological rehabilitation is necessary, in addition to using a hearing aid.

Cochlear implants: For selected patients with profound sensorineural hearing loss cochlear implants are a therapeutic option. Electrical stimulation of the auditory nerve affords some awareness of environmental noise signals, but not necessarily recognizable speech. Intensive audiological rehabilitation is necessary after the implants have been provided.

Hearing aids: Hearing aids are the major rehabilitation tool. They should be considered for all patients with irreversible forms of hearing impairment. Their function is to improve the delivery of sound to the ear. Modern electronic types allow speech to be presented at a comfortable level. The components of hearing aids are a microphone for converting sound energy to electrical energy, an amplifier, and a receiver (the earphone) that converts the amplified signal back to acoustical energy. Hearing aids are available in different types. Some are worn behind the ear or at other places on the body, and some are worn completely inserted in the ear. Digital hearing aids have been developed recently and represent a significant advance for persons with hearing deficits. These instruments amplify primarily the spoken word, not all signals indiscriminately.

Other assistive devices: Adaptive devices for the telephone may be extremely helpful in allowing the patient with a hearing impairment to maintain communication with friends and family. Also helpful are captioned television and home appliances that signal with light instead of sound.

Speech reading: This is the skill of observing the lips of a person speaking and using other clues as well to increase the speed and effectiveness of recognizing meaning, if not actually to "read" the words said. Some persons have significant difficulty learning this skill while others learn it quickly.[160] Speech readers require assistance to sharpen their visual perception and to become sensitive to nuances of language.

Vestibular Dysfunction

Vertigo. Positions that provoke dizziness or vertigo should be avoided. Medications used are mainly antihistamines, such as meclizine (Antivert 25–100 mg daily in divided doses), cyclizine (50 mg 1 to 2 times daily), or anticholinergics. Especially useful is the scopolomine transdermal patch, which is attached every 1 to 3 days. Other medications used are antiemetics, such as promethazine (25–50 mg/d); or tranquilizers, such as diazepam (5–10 mg one to three times a day), or oxazepam (10–60 mg/d), or halopredol (0.5–1 mg one to two times per day), or combined preparations.

Positional vertigo. Treatment for positional vertigo emphasizes the postural control underlying stability. Exercises include strengthening on isokinetic equipment, and strategies that address muscular dyscoordination. Such strategies employ EMG biofeedback, functional electrical stimulation, and facilitation techniques that use manual cues to improve motor performance. Exercises are mainly focused on improving balance by concentrating on visual and somatosensory surface inputs (eg, by walking on uneven surfaces). Patients with positional vertigo seem to have an increased sensitivity to visual motion cues, but these patients lose balance if the visual cues are not interpreted correctly. The patient is taught to maintain balance in progressively more difficult movement tasks.[161,162]

Gaze stabilization. Exercises to improve eye–head coordination for stabilizing gaze during head movements employ visual tracking tasks, first with the head stationary, and then with progressive head movement. The exercises are designed to improve visual modulation of the vestibuloocular reflex.[162]

Management

General rehabilitative measures for managing auditory dysfunction include the following:

Education and training: Teaching of sign language is especially appropriate for children and young adults. Teaching speech to children who were born deaf or became deaf at an early age is an awesome task. Proprioceptive, tactile, and visual cues must be used in the absence of auditory feedback. For vertigo, medications that suppress vestibular function can at times be detrimental because such medications also suppress central nervous system function. Patients are taught to avoid positions that provoke vertigo. This deliberate control of posture, however, must be considered carefully because it might delay recovery. The purposes of exercises are to habituate to dizziness, to improve eye–head coordination, and to retrain sensory and motor components of postural control. Treatment of diz-

ziness is based on repeated exposure of the patient to provocative positions or movements (5–10 times/d) in order to habituate to dizziness. Exercises are performed twice a day in the hospital and the patient continues these activities when discharged to home.

Emotional support: It is essential that the patient is assisted to achieve emotional acceptance of the new status imposed by the impairment.

Preventive measures: These extremely important measures include (*a*) protection from exposure to excessive noise by wearing earplugs in the firing range, in industrial environments, and in other places where noise exposure is a problem; (*b*) careful monitoring when ototoxic drugs have been administered; and (*c*) prevention of prenatal rubella.

Referrals to self-help groups: Persons with similar problems can often be of significant help to a patient who has difficulty adjusting to an impairment.

CRANIAL NERVE IX: GLOSSOPHARYNGEAL

The glossopharyngeal nerve is mixed. Branches contain various somatic and visceral afferents and efferents. The anatomy is shown in Figures 7-30 and 7-31.

Anatomy and Function

The motor branchial efferent fibers originate in the nucleus ambiguus, pass through the superior and petrous ganglia (within or distal to the jugular foramen), then leave the skull via the jugular foramen together with CN X and the bulbar fibers of CN XI. CN IX motor fibers are anterolateral to CN X, innervate the stylopharyngeus muscle (a pharyngeal elevator and the sole striated muscle supplied by CN IX), and then penetrate the pharyngeal constrictor muscles at the base of the tongue. The pharyngeal constrictors are supplied by CN IX and X.

The parasympathetic-visceral efferent fibers originate in the inferior salivatory nucleus. Preganglionic fibers reach the otic ganglion via the tympanic branch of CN IX and via the lesser superficial petrosal nerve. Postganglionic fibers supply the parotid gland via the auriculotemporal nerve (a branch of CN V) and stimulate secretion of saliva.

The sensory-somatic afferent fibers have their primary neurons in the petrous ganglion. The petrous ganglion receives sensory fibers (Jacobson's nerve)[141] from the area between the ear and mastoid cells, and from the lining of the tympanic membrane and the eustachian tube. The sensory fibers terminate eventually in the nucleus of the spinal tract of CN V.[163]

The sensory-visceral afferent fibers have their primary neurons in the superior or petrous ganglion, which receives fibers arising from unipolar cells. The sensory-visceral afferent fibers convey sensation from the posterior third of the tongue, epiglottis, posterior and lateral pharynx (including pain fibers), posterior part of the soft palate,[164] tonsillar region, uvula, and the eustachian tube. Centrally they terminate in the tractus solitarius.

The sensory-visceral-gustatory afferent fibers have their primary neurons in the petrous ganglion and terminate centrally in the nucleus solitarius.

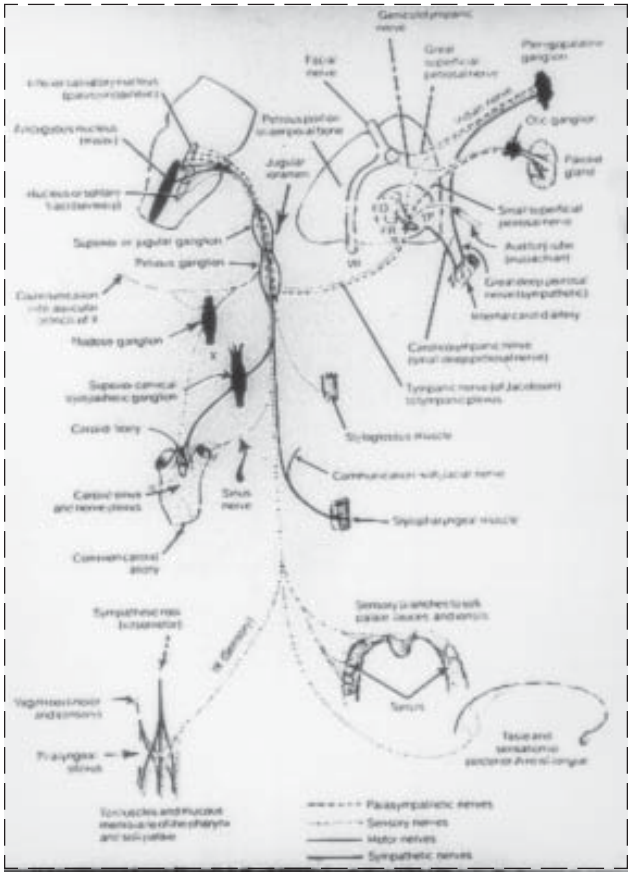


Fig. 7-30. The glossopharyngeal nerve. FO: Fenestra ovalis (oval window); FR: Fenestra rotunda (round window); TP: Tympanic plexus. Reprinted with permission from deGroot J, Chusid JG. *Correlative Neuroanatomy*. 20th ed. Norwalk, Conn: Appleton & Lange; 1988: 168.

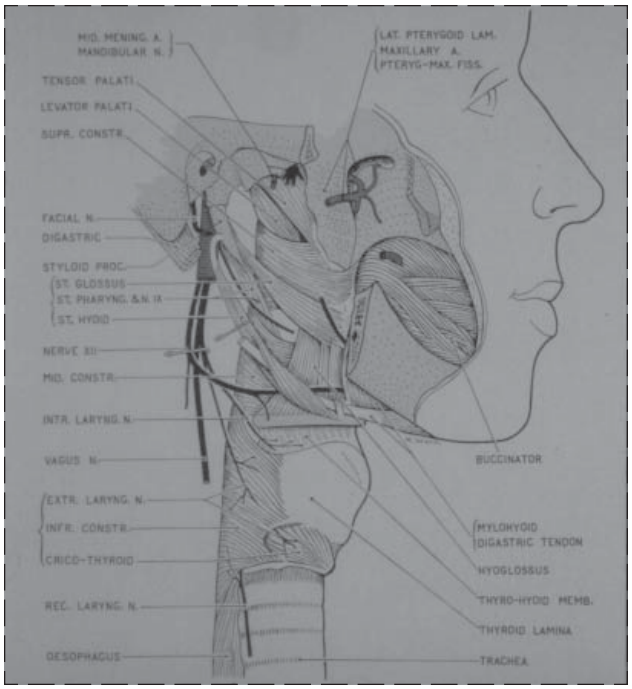


Fig. 7-31. Muscles and structures relevant to swallowing, lateral view. Reprinted with permission from Anderson J. *Grant's Atlas of Anatomy.* 7th ed. Baltimore, Md: Williams & Wilkins; 1978.

The petrous ganglion receives fibers conveying taste from the posterior third of the tongue.

The sensory-visceral, chemoreceptor and baroreceptor afferent fibers have their primary neurons in the petrous ganglion, which receives impulses conveyed through the sinus nerve fibers from the special chemoreceptors and baroreceptors in the carotid body and carotid sinus, respectively. The sensory-visceral chemoreceptor and baroreceptor afferent fibers are concerned with reflex control of respiration, blood pressure, and heart rate. Centrally, these fibers terminate in the nucleus solitarius.

The central connections from the nucleus ambiguus travel via the corticobulbar tract. They have reflex connections from the extrapyramidal and tectobulbar tracts as well as from the nucleus tractus solitarius. The inferior salivatory nucleus receives cortical impulses via the dorsal longitudinal tract and via reflex connections from the nucleus or the tractus solitarius. The sensory fibers are connected with the cortex via the medial lemnisci and thalamus, and reflexly with the salivatory nucleus, the nucleus ambiguus, and the motor nucleus of CN VII.

Injuries and Lesions

The most common cause of injury or lesion is trauma, especially head injury due to blunt trauma. Isolated injuries of CN IX are rare because CN IX, X, XI, and XII are in close anatomical relationship in the posterior cranial fossa, the nucleus ambiguus, and the jugular foramen. As noted previously, upper cervical lesions may also affect the last four cranial nerves and CN VI (the latter because of its long course through the base of the skull).

Penetrating gunshot wounds are the most common cause of traumatic injury to the last four cranial nerves.[165] Missile wounds or skull fractures at the jugular foramen are likely to damage CN IX and XI without involvement of CN X (Vernet's syndrome).

Nontraumatic lesions may be due to syringobulbia, tumors (meningioma,[166] chordoma,[167] glomus jugularis tumor[168]), multiple sclerosis, and Guillain-Barré syndrome (in almost all fatal and in many nonfatal cases, CN IX and X are affected). At times, even cervical lymphadenopathy, infections of the mastoid, and basal meningitis can cause CN IX lesions and glossopharyngeal neuralgia. The incidence of glossopharyngeal neuralgia as compared with trigeminal neuralgia is 1:100.[169,170]

Syndromes involving the glossopharyngeal nerve are:

- Bonnier's syndrome (CN VIII, IX, and X) is caused by a lesion at the lateral vestibular nucleus (Deiter's) and adjacent pathways. It is manifested by paroxysmal vertigo; symptoms and signs of CN IX, X, and occasionally, III and V lesions; contralateral hemiplegia; occasional somnolence; apprehension tachycardia; and weakness.
- Vernet's syndrome (jugular foramen syndrome, CN IX, X, XI) usually results from basilar skull fracture. It presents with paralysis of muscles innervated by ipsilateral glossopharyngeal, vagal, and accessory nerves. The *Vernet's Rideau* phenomenon, which normally has constriction of the posterior pharyngeal wall when saying "ah," is absent in CN IX lesions.
- Glossopharyngeal neuralgia (tic douloureux of the glossopharyngeal nerve) presents with painful attacks similar to trigeminal neuralgia, but in the glossopharyngeal nerve distribution.
- Reichert's syndrome is an incomplete "neuralgia" affecting the tympanic branch

(Jacobson's nerve) of the glossopharyngeal nerve. It may be relieved by intracranial section of the glossopharyngeal nerve. Pain is limited to the ear and eustachian tube.

The symptoms and signs of glossopharyngeal nerve lesions are:

- Loss of gag reflex.
- Slight dysphagia.
- Loss of taste in posterior third of tongue.
- Deviation of uvula to the uninvolved side.
- Loss of sensation in the pharynx, tonsils, fauces, back of tongue.
- Loss of constriction of the posterior pharyngeal wall when saying "ah."
- Increased salivation, involvement of the tympanic plexus in middle ear lesions.
- Possible nystagmus of the uvula in central inflammatory and respiratory lesions (rare).
- Tachycardia, probably from disturbance of the carotid sinus.
- Sharp, knife-like pains in the territory of the glossopharyngeal nerve (region of tonsillar fossa, pharynx, or base of tongue) in glossopharyngeal neuralgia; these are spontaneous or triggered by swallowing, yawning, clearing the throat, or talking.

Evaluation

Clinical evaluation begins with an observation of the palatal arch at rest and on vocalization. The affected site of the palatal arch may be somewhat lower at rest, causing a mild dysphagia. However, with vocalization the palate usually elevates symmetrically.[171]

Sensation of the posterior third of the tongue, the soft palate, tonsillary regions, and pharyngeal wall must be tested. Reflex function is tested by eliciting the pharyngeal (gag) reflex. The posterior pharyngeal wall or base of tongue is stimulated. The expected response is constriction and elevation of the pharyngeal musculature and retraction of the tongue. The afferent arc is carried by the glossopharyngeal nerve; the efferent arc by CN IX and X.

To test the palatal reflex, the soft palate is stimulated and elevation of the soft palate and ipsilateral deviation of the uvula is observed. Again, the afferent arc is carried by the glossopharyngeal nerve; the efferent arc by CN IX and X.

The sensory component of the carotid sinus reflex is tested by pressing over the carotid sinus. This normally produces slowing of heart rate and fall of blood pressure. The reflex may be deficient in lesions of CN IX or X.

The swallowing reflex is triggered by food or saliva at the base of the anterior faucial pillars. The afferent arc is carried by CN IX, X, and XI to the swallowing center in the medullary reticular formation located within the brainstem.[172] The efferent arc is carried by motor fibers of CN IX and X. CN VII, V, and XII may add components to this reflex. The reflex consists of three major phases: (1) oral, (2) pharyngeal, and (3) esophageal.

Swallowing during the three phases is preferably observed through a modified barium swallow (Figure 7-32). A barium swallow with cineesophagram (recorded on videotape) is modified by using three boluses of different consistencies (liquid, paste, and solid) to permit assessment of the oropharyngeal phases.

The *oral phase* is voluntary and accomplished by the CN VII innervated orbicularis oris and buccinator (lip seal); by CN V innervated muscles of mastication; and by CN XII innervated intrinsic and extrinsic muscles of the tongue.

Fig. 7-32. Schematic presentation of swallowing of a radio-opacified liquid bolus during fluoroscopy. First two pictures (A and B) represent the oral phase; the second two (C and D) represent the pharyngeal phase; these are followed by a picture of the esophageal phase (E). Reprinted with permission from Logemann T. *Evaluation and Treatment of Swallowing Disorders.* Austin, Tex: Pro-Ed; 1983.

The *pharyngeal phase* is short (0.5 second) and mainly involuntary. It is most critical for possibly life-threatening aspiration. In this phase, appropriate placement of the bolus is assured by preventing back flow (velar-nasopharyngeal seal, tongue-palate seal, lip closure seal) and inappropriate forward flow (tracheal seal). There is reflex inhibition of breathing. The CN V innervated tensor veli palatini assists the CN IX and X innervated levator veli palatini in creating the velar seal.

The CN V innervated anterior belly of the digastric, the CN VII innervated posterior belly, and the stylohyoid muscle (CN VII) assist the CN XII innervated hyoglossus and genioglossus muscles in laryngeal displacement. CN IX and X provide innervation to the aryepiglottis muscle (epiglottic tilt), to the lateral cricoarytenoid and thyroarytenoid (glottic seal), to the stylopharyngeus, and to the three pharyngeal constrictors to provide pharyngeal compression. The CN XII innervated styloglossus and hyoglossus assist in this function. Pharyngoesophageal relaxation is innervated by CN IX and X (cricopharyngeal inhibition). The *esophageal phase* is involuntary and is accomplished by CN X innervated muscles.[173]

The quantity of salivary secretion from the parotid gland can be measured. It may be absent, decreased, or occasionally increased, with glossopharyngeal lesions. Assessment for glossopharyngeal neuralgia requires checking the heart rate. Bradycardia or even asystole may cause syncopal episodes during neuralgic attacks, possibly due to stimulation of the nucleus solitarius and dorsal motor nucleus of the vagus by impulses originating in glossopharyngeal afferents.[171]

Electrodiagnosis and Imaging

Electrodiagnostic evaluation consists of standard EMG testing of the palate, evaluating pharyngeal constrictors supplied by CN IX and X. See the discussion of CN X below. Imaging by video fluoroscopy is the examination of choice for determining swallowing deficits.

Prognosis

The prognosis for recovery from swallowing problems secondary to stroke, closed head injury, neurosurgical procedures, or poliomyelitis is quite good. The prognosis is poor for swallowing problems secondary to degenerative disease such as Parkinson's, amyotrophic lateral sclerosis, multiple sclerosis, muscular dystrophy, and dermatomyositis. Prognosis for recovery from glossopharyngeal neuralgia is good if the patient responds to anticonvulsants, or if pathology can be identified during surgery and corrected. The stylopharyngeus is difficult to assess. Most other objective tests also examine CN X (see below).

Management

Any patient found to aspirate more than 10% of every bolus regardless of consistency of food should not receive oral feedings. Fluoroscopy examination (modified barium swallow) will assist the physician and speech therapist in deciding whether treatment should be directed toward appropriate adaptive behaviors, or whether exercises should be carried out to improve appropriate motor controls as prerequisites to normal swallowing. Therapeutic techniques may be applied during the fluoroscopic study to evaluate whether these would in fact reduce aspiration. Management also applies to CN X lesions.[173] Available techniques[174] of therapy are as follows:

- Exercises to improve motor control.
- Range of lip motion exercises, to improve labial closures.
- Tongue resistance exercises.
- Exercises to hold a cohesive bolus.
- Bolus propulsion exercises.
- Range of tongue motion exercises, to improve oral transit.
- Possible use of palatal reshaping prothesis to improve oral manipulation of food and speed of oral transit.
- Therapy to stimulate the swallowing reflex with a small, long handled, iced laryngeal mirror.
- Exercises to increase adduction of vocal folds to improve airway protection while swallowing.
- Special maneuvers to improve swallowing:
  - Close airway before and during the swallow (eg, supraglottic swallow); prolong laryngeal elevation and cricopharyngeal opening (Mendelsohn maneuver).
  - Postural changes to redirect bolus (eg, head rotated to damaged side for unilateral pharyngeal paresis); widen vallecula and place epiglottis in protection position (eg, head down "chin tuck" for delayed pharyngeal reflex and weak pharyngeal peristalsis); facilitate oral transit (eg, head back).[175]

  - Food and liquid consistency modifications are recommended after evaluation (clinical and/or with modified barium swallow fluoroscopy study) according to items patient can swallow without aspiration.
- Dilation of cricopharyngeus sphincter. Progressively larger bougies are gently passed through the sphincter gradually stretching the muscle. However, dilation is usually temporary, and eventually a myotomy will have to be performed.
- Teflon injection into damaged vocal folds, to improve closure and airway protection during swallowing .[176]
- Nasogastric feeding.

If no improvement is noted in 3 to 6 months, surgery, such as cricopharyngeal myotomy, may have to be considered.[177] If the impairment cannot be altered significantly, rehabilitation efforts should be aimed at reducing the effect of the impairment by providing alternate means of safe eating and drinking, possibly by modifying the diet[178,179] or swallowing patterns. Eventually a permanent gastrostomy may be necessary.

Medical management for glossopharyngeal neuralgia is the same as for tic douloureux (trigeminal nerve), using anticonvulsants (phenytoin, carbamazepine). Suboccipital craniectomy with exploration of the glossopharyngeal nerve is indicated in patients failing to respond to anticonvulsants. If a compressing blood vessel is found and mobilized, pain will stop. If no structural deficit can be identified, the nerve should be sectioned.[92]

CRANIAL NERVE X: VAGUS

The vagus nerve is mixed, containing somatic and visceral afferents and somatic and visceral efferents. See Figures 7-33 through 7-36.

Anatomy and Function

The branchial efferent vagal fibers derive from the nucleus ambiguus, which also contributes motor rootlets to the glossopharyngeal nerve and the internal ramus of the accessory nerve. These three cranial nerves leave the skull through the jugular foramen. The vagus nerve fibers supply all the striated muscles of the soft palate, pharynx, and larynx, except for the tensor veli palatini supplied by CN V and the stylopharyngeus supplied by CN IX. Other motor fibers of the vagus nerve, together with fibers of the accessory nerve, join outside the skull, and through the recurrent laryngeal nerve supply all of the intrinsic muscles of the larynx except for the cricothyroid. The left recurrent laryngeal nerve arises as a branch in the thorax and recurs by looping around the aortic arch; on the right side it arises in the root of the neck looping around the right subclavian artery to recur. Both recurrent nerves then ascend between the trachea and esophagus to reach the laryngeal muscles. The cricothyroid muscle as well as the inferior constrictor muscle of the pharynx are innervated by the external branch of the superior laryngeal nerve. The internal branch of this nerve gives sensory supply to the larynx.

The parasympathetic-visceral efferent preganglionic fibers arise in the dorsal motor nucleus of the vagus and terminate on ganglia close to the thoracic and abdominal viscera. The postganglionic fibers supply the smooth muscles and glandular structures of the pharynx, larynx, and viscera.

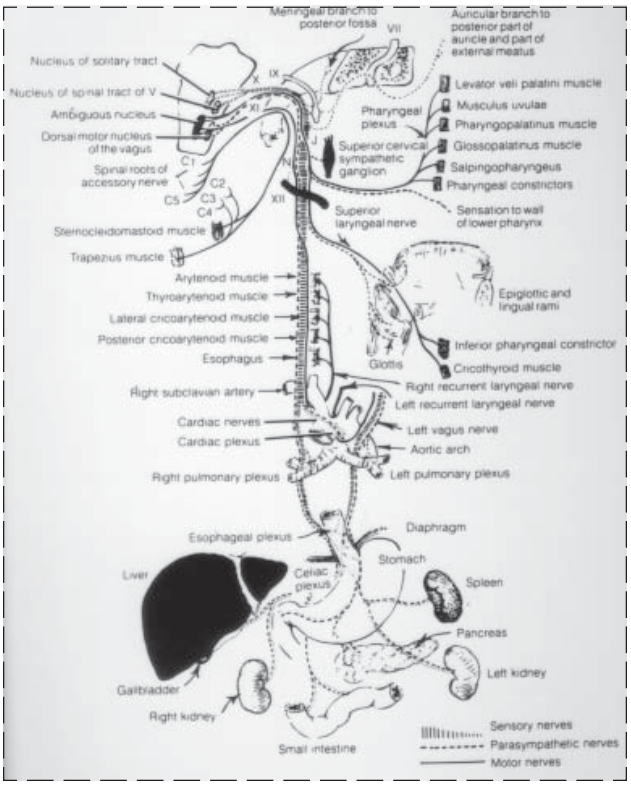


Fig. 7-33. The vagus nerve. N: Nodose (inferior) ganglion; J: Jugular (superior) ganglion. Reprinted with permission from deGroot J, Chusid JG. *Correlative Neuroanatomy*. 20th ed. Norwalk, Conn: Appleton & Lange; 1988: 171.

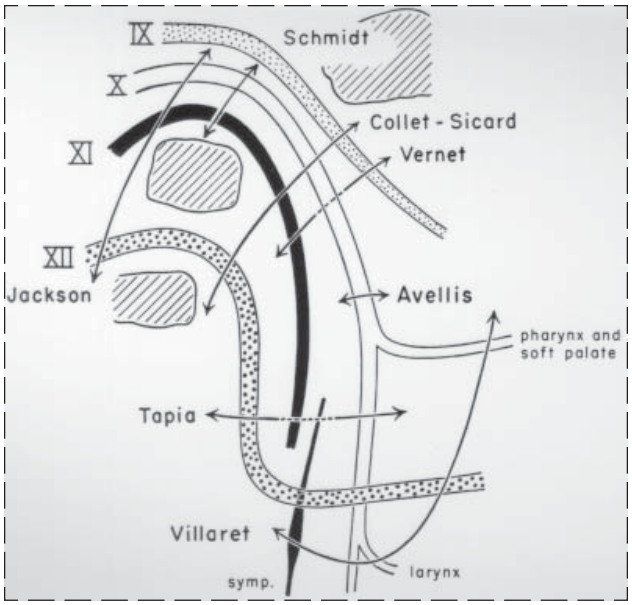


Fig. 7-34. Combinations of nerves affected in various syndromes. The broken lines over certain nerves indicate that they may or may not be affected. Reprinted with permission from Vernet M. *J Laryng Otologol*. 1918; 33:354.

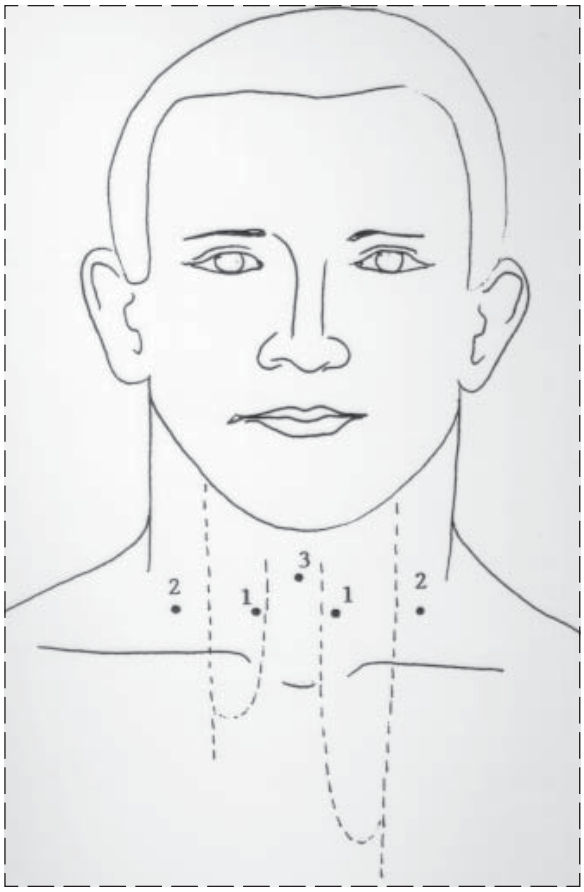


Fig. 7-35. Sites of electrode placement for paratracheal stimulation of the recurrent laryngeal nerve. 1) Recurrent laryngeal nerve; 2) Vagus nerve; 3) Thyroid cartilage. Drawing: Courtesy of artist: Dr. Anna Bettendorf, University of Virginia.

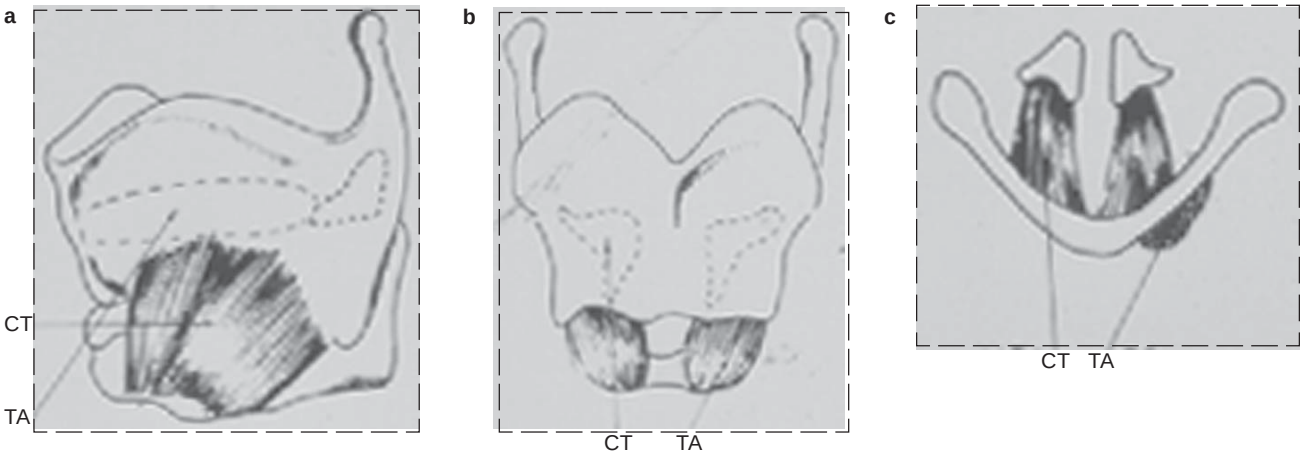


Fig. 7-36. (**a**) Lateral, (**b**) anterior, and (**c**) superior projection of the larynx and EMG needle insertion sites. CT: Cricothyroid muscle. TA: Thyroarytenoid muscle. Reprinted with permission from Simpson D, Sternman D. Vocal Cord Paralysis: Clinical and Electrophysiologic Features. *Muscle Nerve.* 1993;16:953**.**

Stimulation of the vagus nerve produces bradycardia, while vagus nerve paralysis results in tachycardia. CN X fibers also vasoconstrict the coronary arteries. There is inhibition of suprarenal secretions and stimulation of gastrointestinal peristalsis and gastric, hepatic, and pancreatic glandular activity. In general, the vagus nerve is the largest and most important parasympathetic nerve in the body, and also the longest and most widely distributed cranial nerve.

The somatic afferent fibers have their primary neurons in the jugular ganglion subserving fibers carrying sensation from the posterior portion of the external acoustic meatus, the adjacent part of the tympanic membrane, and a small area in the posterior aspect of the pinna. The fibers eventually terminate on the trigeminal nucleus. Afferent fibers carrying pain sensation from the dura mater of the posterior cranial fossa relay in the jugular ganglion and terminate in the spinal tract of the trigeminus and its nucleus. Fibers conveying sensory information from structures above the vocal cords travel in the internal laryngeal nerves; those below the vocal folds travel in the recurrent laryngeal nerves.

The visceral afferent fibers have their primary neurons in the ganglion nodosum carrying sensation from the lower pharynx, larynx, trachea, esophagus, and the thoracic and abdominal viscera. The fibers collect centrally as the fasciculus solitarius and terminate on its nucleus. Fibers that carry gustatory sensation from the epiglottis and the arytenoids also relay through the ganglion nodosum and terminate in the nucleus solitarius.[171] The central connections for the vagus nerve are as described under CN IX above.

The muscles supplied by the vagus nerve are

- Levator veli palatini: occludes the nasal passages in swallowing.
- Musculus uvulae: retracts uvula backward, helping to block off nasal passages.
- Palatoglossus: narrows the fauces in swallowing by elevating posterior tongue and depressing soft palate.
- Palatopharyngeus: approximates the pharyngopalatine arches and closes posterior nares and faucial orifice.
- Salpingopharyngeus: raises upper and lateral portions of pharynx.
- Superior, middle, and inferior constrictors of the pharynx: Together, these flatten and contract the pharynx in swallowing. The three constrictors help to force the food into the esophagus and initiate peristaltic waves. They also assist in production and modulation of speech.

The muscles supplied by the recurrent laryngeal nerve (branch of the vagus nerve) are

- Posterior cricoarytenoids: chief abductors that separate the vocal cords.
- Lateral cricoarytenoids: chief adductors.
- Thyroarytenoids: shorten and relax vocal cords (also adduct).
- Arytenoid (unpaired): closes the opening of the glottis, especially the posterior aspect. The arytenoid has two parts. The oblique arytenoid acts as a sphincter of the upper larynx, and the transverse arytenoid closes the posterior pad of the glottis.

The muscles supplied by the superior laryngeal nerve (external branch) are the cricothyroids, the chief tensors of the vocal chords. The extrinsic muscles of the larynx (functionally a part of the voice apparatus) include also those supplied by CN XII and by upper cervical nerves that insert on the hyoid bone.

Reflexes with vagus nerve participation are gag, swallowing, cough, sucking, hiccup, yawning, carotid sinus, and Hering-Brewer. The Hering-Brewer reflex is an overstretching of receptors in the visceral pleura and the tracheobronchial tree, causing inhibition of respiration to prevent overdistension.

Injuries and Lesions

The most common cause of injury is trauma, blunt and penetrating. Such traumas most frequently involve the posterior fossa. Injuries and other lesions also frequently involve CN IX, XI, and XII (Collet-Sicard syndrome). See Figure 7-34. The incidence of dysphagia following closed head trauma is reported as 26% to 27%.[178,179]

Injuries to CN VI, IX, X, XI, and XII may also be concomitants of high cervical lesions.[1,2] Neck and thorax injuries and lesions can affect the superior laryngeal and the recurrent laryngeal nerves. Vocal cord paralysis is a relatively common disorder. Adour[180] reported superior laryngeal nerve paralysis to have a higher incidence than facial palsy. Yamada et al[181] investigated 564 patients with recurrent laryngeal nerve paralysis in a 10-year period (1971–1980). The largest group of cases were

idiopathic (230 cases), 68 were caused by thyroid surgery, and 63 were due to endotracheal intubation. The last group had the best recovery rate.

There are reports from World War I of penetrating injury to the vagus nerve caused by shrapnel.[182] In 1909, an injury to the recurrent laryngeal nerve following a gunshot wound to the neck was successfully repaired.[183]

Surgical trauma is the most common cause of vocal cord paralysis.[184,185] The recurrent laryngeal nerve is especially vulnerable during thyroidectomies.[186] The incidence is reported as 0.3% to 13.2% depending on the experience of the surgeon.[187] Purposeful sacrifice of the recurrent laryngeal nerve during cancer surgery because of involvement in the tumor is no longer advocated by many surgeons if the vocal cord function is normal.[188]

Nontraumatic lesions are due to vascular insults (lateral medullary or Wallenberg's syndrome), motor neuron disease, tumors, syringobulbia, inflammatory disease, Guillain-Barré syndrome, carcinomatous meningitis, metastases, sarcoidosis, nasopharyngeal diphtheria, and chronic lead poisoning. Abductor paralysis in children is frequently associated with Arnold-Chiari malformation; other congenital malformations; birth trauma; or are idiopathic, possibly of viral origin. These nontraumatic lesions are mainly reported in the cranium. Lesions affecting the vagus nerve in the neck or thorax are mainly tumors, aneurysms of the internal carotid artery, enlarged lymph nodes, or the result of surgical trauma. The sites of lesions of CN IX, X, XI, and XII are shown in Figure 7-34. Syndromes involving the vagus nerve are

- Jugular foramen syndrome of Vernet, involving CN IX, X, XI;
- Schmidt's syndrome, involving CN X, XI;
- Hughlings-Jackson syndrome, involving CN X, XI, and XII;
- Collet-Sicard syndrome, involving CN IX, X, XI, and XII; and
- Avellis's syndrome, involving CN X and XI.

See also the discussion of syndromes for CN IX above.

Symptoms and Signs

The symptoms and signs of CN X malfunctions are motor disturbances within the pharynx and larynx; sensory disturbances within the pharynx and larynx; and autonomic disturbances involving heart rate, glandular dysfunction, and gastrointestinal functions. These symptoms and signs of CN X lesions are summarized below.

Motor Disturbances

- Aphonia (paralysis of vocal cords or hysteria).
- Dysphonia (impairment of voice, often with unilateral lesions producing nasal speech, hoarseness, weak voice).
- Position change of vocal cord, observed on examination (can be asymptomatic).
- Dysphagia (difficulty in swallowing) is caused by damage to the pharyngeal branches of the vagus resulting in paralysis of the palatini muscles and the pharyngeal constrictors. Dysphagia may be profound, especially for liquids, which tend to escape through the nasal cavity.
- Defective elevation of the palate on phonation and movement of the uvula toward the unaffected side (clinical symptoms may be absent).
- Unilateral vocal cord paresis; except for some snoring at night or minor nasal speech, the ipsilateral vocal cord will assume the cadaveric position (midway between adduction and abduction). There is little if any dyspnea.
- Bilateral vocal cord paresis; respiration may be severely affected.
- Stridor, especially in children.
- Vocal cord immobility (observed during laryngoscopy) can be caused by cricoarytenoid joint fixation, interarytenoid scarring, recurrent laryngeal nerve paralysis, or laryngeal synkinesis. To establish definitely the cause of immobility, laryngeal EMG must be performed.
- Esophageal, cardiac, or pyloric spasm not due to local causes or possibly of viral origin.
- Loss of gag reflex (paralysis of soft palate).
- Anesthesia of larynx, hoarseness, and voice fatigue (as secondary to unilateral superior laryngeal nerve paralysis, which is rare and usually traumatic).

Sensory Disturbances

- Pain or paresthesias of the pharynx, larynx, and external auditory meatus. In vagus or superior laryngeal neuralgia (tic douloureux), pain may be severe and knife-like.

- Anesthesia of the lower pharynx and larynx, as occurs in complete vagus nerve lesions.
- Cough, a constant symptom of vagus irritation.
- Dyspnea or pseudoasthma as may occur if the reflex vagal control of respiration is interrupted.
- Salivary hypersecretion as may occur with irritative lesions.
- Hypersecretion of acidic gastric fluids, which may cause gastric ulceration. Selective vagotomy may be necessary for recurring ulcers.

Parasympathetic (vegetative) disturbances

- Bradycardia occurs with irritative lesions.
- Tachycardia occurs with palsy of the vagus nerve.
- Gustatory sweating occurs after vagus nerve damage, possibly due to aberrant regeneration (see glossopharyngeal nerve).
- Intermittent diarrhea (lasting 1 to 2 days and recurring every 1 to 2 weeks) due to under activity of vagus (diabetes).
- Possible esophageal dysfunction, gastric dilation, and nocturnal diarrhea.

Evaluation

Clinical examinations should begin with observation of the soft palate and uvula at rest and in phonation. The palate should elevate symmetrically with no deviation of the uvula.[189] The character of the voice, breathing, and cough are evaluated. During laryngoscopy the laryngeal movements can be observed. Reflex testing should include the gag reflex (CN IX and X), the occulocardiac reflex (pressure on the orbit should cause cardiac slowing), the carotid sinus reflex (pressure on carotid sinus to produce cardiac slowing), sinus arrhythmia, and pulse retesting for variations during deep inhalations followed by slow deep exhalation.

Electrodiagnosis

One kind of electrodiagnostic testing consists of nerve conduction studies of the recurrent laryngeal nerve.[190] Because of the relative inaccessibility of the structures and the unreliability of distance measurements, the method has not found wide acceptance and is rarely performed. The procedure is described below. EMG[191–195] (see Figures 7-35 and 7-36) of the laryngeal muscles is carried out according to standard procedures.

Procedure for Recurrent Laryngeal Nerve Conduction Study

Stimulation: A needle stimulating electrode (5 cm) is inserted 2.5 cm below the most prominent point of the cricoid arch paratracheally. The anode is placed in the subcutaneous tissue of the jugular fossa. A second stimulus is applied at the same level as the first stimulus, but at the posterior edge of the sternocleidomastoid muscle. The depth of the cathode is adjusted to the lowest threshold that will produce an action potential. Again, the anode is placed in the subcutaneous tissue.

Recording: Electrodes inserted through a laryngoscope are placed in the thyroarytenoid muscle. Distance from point of stimulation averages 10 cm on the right and 21.5 cm on the left.

Normal values:

- Distal latency: 2.1 ± 0.05 millisecond (average distance 5.9 ± 0.05 cm)
- Conduction velocity: 60–70 m/s
- Amplitude: 6.8 ± 0.7 mV
- Duration: 6.6 ± 0.5 millisecond

The *cricothyroid muscle* is tested by inserting a small (approximately 2 cm) electrode approximately 1 cm from the midline over the lower aspect of the thyroid cartilage, to a depth of a few millimeters. The patient is instructed to phonate a low pitched vowel ("e" as in English "sea") and gradually raise the pitch. Increasingly more muscle activity is identified. To reduce discomfort, the skin may be infiltrated with 1% lidocaine (1 cm^3) with 1:100,000 epinephrine before insertion of needle.

The *thyroarytenoid muscle* can be reached by inserting a 7.5 cm needle through the cricothyroid membrane 0.5 cm laterally from midline. In males, the needle should then be angled 45° superiorly, while in females the angle should be approximately 30°. The needle is inserted to a depth of approximately 2.0 cm. The patient is required to say "ee" repeatedly; as the recording needle tip approaches the fibers of the thyroarytenoid or adjacent muscles (lateral cricoarytenoids), action potentials are detected. Observation is as with conventional EMG; at rest it should be silent even though in intrinsic laryngeal muscles there are often baseline normal action potentials observed. These potentials are diphasic or triphasic, of 3 to 6 millisecond duration and 100 to 300 μV in amplitude. Further activity summates into an interference pattern. Examination can be performed in most cases without difficulty, especially in the absence of laryngospasm, he-

matoma, or other complications. However, a laryngoscope, endotracheal tube, Ambu bag, and other resuscitative equipment should be available.

The most common conditions for which EMG is performed are unilateral recurrent laryngeal palsy, superior laryngeal palsy, recurrent laryngeal palsy, spastic dysphonia, chronic hoarseness, immobile vocal cords, incomplete recovery of cricoarytenoid joint fixation, or idiopathic vocal cord paralysis.

A recent report by Simpson and colleagues[192] discussed electromyographic findings in 52 vocal cords of 44 patients with idiopathic vocal cord paralysis. The most common complaints of these patients were hoarseness and shortness of breath (with bilateral involvement). Not as common were other symptoms suggesting laryngeal incompetence, such as loss of high pitch phonation. In 67.4% of vocal cords, evidence of acute or chronic denervation was shown (55% in the thyroarytenoids, 10% in the cricothyroids, and 2% involving both of these muscles); 33% were normal. Another study[194] by the same investigators reports on 48 patients (60 vocal cords) with laryngeal disorders due to different conditions. In 53% of vocal cords there was cranial nerve involvement (28% including the thyroarytenoid, 10% the cricothyroid, and 15% both). Patients with spastic dysphonia showed no spontaneous activity; action potential parameters were of normal values, but firing patterns were altered. There were some patients who showed normal position and activation of vocal cords during the laryngoscopic examination, but EMG detected some evidence of denervation. Conversely, there were patients whose laryngoscopic examinations were suggestive of cranial nerve lesions, but EMG changes were not observed. Involvement of the cricothyroid alone was mostly observed in professional singers.

Posterior criocarytenoid muscles can also be examined as described by Hiroto and colleagues.[193] These tests may be performed with a curved needle electrode or a hooked-wire electrode.

Since the criocarytenoid muscles are supplied by the recurrent laryngeal nerve, electromyography adds little to the diagnosis of a lower motor neuron process and is, therefore, only confirmatory. For kinesiological electrodiagnostic study, however, additional wire electrode placement may be of interest for observing the functional capacities and synergistic movements during swallowing and phonation. This may be especially applicable to the study of spastic dysphonias.

The superior constrictor muscles of the pharynx[196,197] and the cricopharyngeal muscle can be studied effectively with special bipolar suction electrodes.

Quantitative vagus parasympathetic fiber testing can be accomplished by assessment of sinus arrhythmia with an electrocardiograph, EMG, or other computer-assisted analog integrator. Normal vagus parasympathetic innervation of the heart mediates slowing of heart rate with exhalation, which follows an increasing heart rate during inhalation. Vagus denervation abolishes sinus arrhythmia, although exact reflex pathways are not known. During the test, 5 to 6 breaths per minute are taken and heart rate is recorded for 1 to 2 minutes. The maximal-to-minimal heart rate per respiratory cycle is determined, and the mean maximal-to-minimal rate variation is calculated. Test results are influenced by patient's body position, respiratory rate, age, and other factors such as changes in intrathoracic and intraabdominal pressures (Valsalva maneuver) or drugs (propanolol, atropine as autonomic blocking agents).[198]

Esophageal manometry can be used to identify disruptions in the peristaltic wave through the pharynx and esophagus. With the help of fluoroscopy, upper and lower esophageal sphincter disorders may be identified.

Prognosis

The prognosis of acute complete bilateral lesions of the vagus nerve is unfavorable without immediate emergency measures. Unilateral recurrent laryngeal nerve injury, which may simply cause hoarseness, is usually transient. Bilateral recurrent laryngeal palsies (postthyroidectomy, polyneuropathy, or carcinoma) may produce severe airway limitation, often necessitating a tracheostomy. Swallowing difficulties, especially after stroke and head injury, may be severe but often improve with time. Spastic dysphonia is quite resistant to treatment, and prognosis for recovery is poor. Unilateral recurrent laryngeal nerve sections have been advocated by Dedo and Izbedski.[199] Idiopathic paralysis (viral infection) usually recovers spontaneously within 6 months. The majority of patients showed long term improvement with only minor hoarseness as a complication.

Management

Management of dysphagia is described in the discussion of CN IX. Unilateral paralysis of laryngeal muscles if bothersome to the patient can be treated with speech therapy for better voice production. Bilateral paralysis usually needs tracheostomy or intralaryngeal surgery, such as arytenoidectomy or lateral fixation of the arytenoids.

Recurrent laryngeal nerve anastomosis, as well as regeneration without surgery, carries a significant risk for aberrant regeneration (synkinesis), which may cause spasticity during phonation (dysphonia), airway difficulty, or aspiration. It is reported[200] as a frequent problem following immediate repair. The reason for this aberrant regeneration is believed to be the composition of the recurrent laryngeal nerve. It consists of nerve fibers to the abductors, which are inspiratory muscles, and fibers that supply the adductors, which produce phonation. Therefore all options must be carefully considered. Crumley et al[201] describes the following choices: in unilateral vocal cord paralysis, an attempt is made to medialize the involved cord; while in bilateral paralysis one of the better vocal cords must be lateralized to open the airway.

Other treatment options include (1) recurrent laryngeal nerve neurorrhaphy; (2) ansa hypoglossi recurrent laryngeal nerve anastomosis; (3) neuromuscular pedicle transfer (a small piece of hypoglossal innervated omohyoid muscle); and (4) Isshiki thyroplasty (laryngoplasty).

Placement of a silastic block in a subperichondrial plane medializes the vocal cords.[202] Nonsurgical invasive procedures are gelfoam paste, phonagel, or Teflon vocal cord injections. Gelfoam is best used as a temporary measure when recovery is expected. Teflon paste is permanent, for use when recovery is not expected. Another option is to observe and wait.

Spastic dysphonia may be treated with section of the unilateral recurrent laryngeal nerve block or botulin toxin injections. Another treatment option is the electrolarynx, an artificial larynx that may assist in communication if no voice can be produced. The device introduces sound through the soft tissue of the neck, and the tongue and lips articulate this sound into intelligible speech. Instruction by a speech pathologist is mandatory in order to accomplish appropriate sound placement, timing, and articulation. If the vocal cords and the larynx are partially destroyed by a malignancy, laryngectomy followed by insertion of a voice prosthesis may be necessary. The most popular technique is the Singer and Blom technique.[202–204] Tracheoesophageal speech with a voice prosthesis offers the most acceptable communication, and importantly, avoidance of aspiration.[205]

Vagal or superior laryngeal neuralgia is treated with medications such as carbamazepine or other anticonvulsants. If there is no relief, suboccipital craniectomy with decompression should be considered. If no lesion can be identified, sectioning of vagal and glossopharyngeal nerves is recommended.[206] Deficits caused by involvement of parasympathetic fibers need medical management.

CRANIAL NERVE XI: ACCESSORY

The accessory nerve is all motor. The anatomy is given in Figure 7-37.

Anatomy and Function

The branchial efferent fibers have two nuclear origins. The cranial component of the accessory nerve arises from the caudal part of the nucleus ambiguus and is closely related to the radicles of the vagus nerve. The accessory and vagus nerves together supply the musculature of the pharynx and larynx. The spinal component arises in the anterior horn of the upper cervical spinal cord (C1–C5), enters the skull through the foramen magnum, and is somatotopically arranged. That is, C1–C2 predominantly innervate the sternocleidomastoid muscle and C3–C4 the trapezius. The cranial and spinal roots leave the skull through the jugular foramen together with CN IX and X, and then again separate. The external ramus penetrates and supplies the sternocleidomastoid muscle, then crosses the posterior cervical triangle and supplies the trapezius muscle. The internal ramus joins the vagus nerve to supply the pharynx and larynx. The lower part of the trapezius is said to have some innervation by the third and fourth cervical roots through the cervical plexus. However, these are mainly proprioceptive fibers. Muscles innervated by the accessory nerve are represented cortically between the elbow and the trunk in the homunculus of the precentral gyrus.

Central connections are mediated through fibers of the corticobulbar tract, which travel from the cortex, accompany the corticospinal tract to the midbrain, and eventually terminate in the brainstem as well as in the voluntary motor nucleus ambiguus. There are reflex connections with the tectospinal and vestibulbospinal tracts for postural reflexes. Corticobulbar fibers to the trapezius are crossed, and therefore lesions cause contralateral deficit. Corticobulbar fibers to the sternocleidomastoid are uncrossed, or more likely, are doubly decussated. Therefore, lesions cause ipsilateral deficit.[207,208] In the brainstem the fibers to the trapezius are located

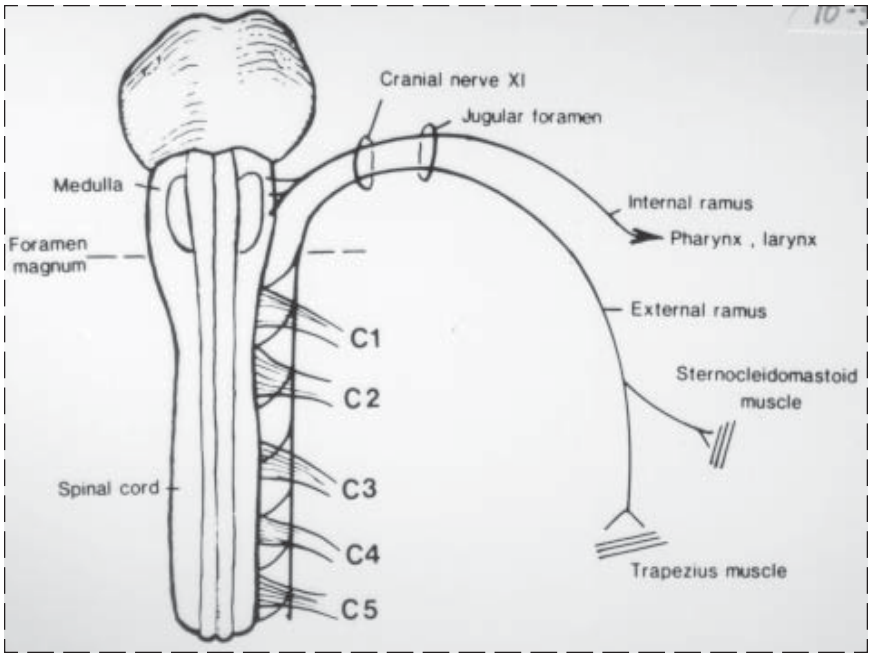


Fig. 7-37. The accessory nerve. Reprinted, with permission from Brazis P, Masdeu J, Biller JL. *Localization in Clinical Neurology*. Boston, Mass: Little, Brown; 1990: 251.

ventrally, and the fibers to the sternocleidomastoid are located in the brainstem tegmentum.[110] A ventral pontine lesion can therefore present with supranuclear paresis of the trapezius but not of the sternocleidomastoid muscle.

Injuries and Lesions

The most common injuries and lesions are the result of trauma such as head injury, especially basal skull fracture and posttraumatic syrinx following spinal cord injury. Neck injuries[209,210] may cause compression or percussion of the accessory nerve in the posterior cervical triangle (football injury),[211] where the accessory nerve is located quite superficially.

Other causes may be surgical trauma such as removal of lymph nodes, biopsy, or more commonly, radical neck dissections. Cannulation of the internal jugular vein[212] may be followed by weakness or paralysis of the trapezius muscle, the sternocleidomastoid, or both.

Nontraumatic lesions are mainly tumor, motor neuron disease, meningitis or other infections, spontaneous accessory nerve lesion[213] or other central lesions.[214,215] The causes and expressions of lesions according to location are given in Tables 7-5 and 7-6. Syndromes involving CN XI are as follows:

Vernet's syndrome is manifested by ipsilateral dysphagia (CN IX, X); diminished gag reflex (CN IX, X); palatal droop (CN IX); ipsilateral vocal cord paralysis (CN X); and weakness and atrophy of the sternocleidomastoid and trapezius muscles (CN XI).

Collet-Sicard syndrome[216] presents with ipsilateral paralysis of the trapezius and sternocleidomastoid (CN XI); paralysis of vocal cord (CN X); paralysis of the pharynx (CN IX); hemiparalysis of the tongue (CN XII); loss of taste from the posterior third of the tongue (CN IX); hemianesthesia of the palate, pharynx, and larynx (CN IX, X).

Symptoms and Signs

The symptoms or signs of supranuclear lesions of CN XI are ipsilateral sternocleidomastoid weakness, contralateral trapezius weakness, and hemiplegia. The symptoms or signs of lesions in the nuclear area are ipsilateral weakness of the sternocleidomastoid and trapezius muscles. Infranuclear lesions present with nuclear and CN IX, X, and XII deficits. Peripheral accessory nerve lesions show normal sternocleidomastoid muscle function, but weakness of the trapezius muscle, pain, atrophy, and drooping of shoulder. Other peripheral lesions may cause torticollis.

Evaluation

The clinical examination consists of strength evaluation of the sternocleidomastoid by applying resistance to head turning, opposite to the side of the muscle tested. Resistance is given also to head tilting. Resistance applied to head flexion tests the

TABLE 7-5

CAUSES AND EXPRESSION OF LESIONS BY LOCATION (CRANIAL NERVE XI)

	Traumatic Lesions	Nontraumatic Lesions	Symptoms/Signs
Supranuclear	Gunshot wounds, other trauma	Tumor	Contralateral hemiplegia including contralateral trapezius, and possible ipsilateral sternocleidomastoid weakness
Nuclear	Posttraumatic syrinx	Tumor, motor neuron disease	May have associated medullary or upper cervical cord dysfunction
Foramen Magnum	Skull fracture, other trauma	Neoplasm, meningitis	May also involve CN IX, X, and XII. Ipsilateral findings, dysphonia, dysphagia, loss of taste posttongue, vocal cord paralysis, tongue paresis, and atrophy
Juglar Foramen	Basal skull fracture	Tumor, infections	May also involve CN IX and X (Vernet's syndrome). Findings are ipsilateral dysphonia and dysphagia (paralysis of ipsilateral vocal cord), and loss of taste and sensation from posterior tongue
Retropharyngeal Space	Trauma	Intramedullary lesions	May also involve CN IX, X and XII. Collet-Sicard syndrome findings are ipsilateral.
Neck	Compression or percussion injury (football)[1,2] Shoulder dislocation[3]	Internal jugular cannulation in posterior triangle, radiation[4] therapy, adenopathy, postsurgery	Ipsilateral findings trapezius and/or sternocleidomastoid muscle weakness.

CN: cranial nerve

(1)Markey K, Di Benedetto M, Curl WW. Upper trunk brachial plexopathy: The stinger syndrome. *Am J Sports Med.* 1993;21:650–656. (2) Di Benedetto M, Markey K. Electrodiagnostic localization of traumatic upper trunk brachial plexopathy. *Arch Phys Med Rehabil.* 1984;65:15–17. (3) Mitchell SW. *Injuries of the Nerves and Their Consequences.* Philadelphia, Pa: JB Lippincott; 1872; 335. (4) Hoffman JC. Permanent paralysis of the accessory nerve after cannulation of the internal jugular vein. *Anesthesiology*. 1983;58:583.

TABLE 7-6

TRAPEZIUS AND STERNOCLEIDOMASTOID MUSCLE INVOLVEMENT

Lesion	Trapezius	Sternocleidomastoid
Upper motor neuron lesion,ipsilateral to sternocleidomastoid above oculomotor complex	Contralateral	Ipsilateral
Ventral brain stem lesion (supranuclear fibers to trapezius) or a lower cervical cord lesion (somatotopic arrangement)	Ipsilateral	
Lower brain stem tegmentum lesion or upper cervical accessory roots		Ipsilateral
Contralateral brainstem lesion or ipsilateral high cervical cord lesion (Peripheral accessory nerve lesion before the nerve divides into muscular branches.)	Ipsilateral	Ipsilateral

two sternocleidomastoid muscles simultaneously. Reflex can be elicited by tapping the insertion of the sternocleidomastoid muscle.

The strength of the trapezius muscle is tested by observing the position of the shoulders in lateral abduction of the arm from approximately 100° to 180° with the arm internally rotated and hand pronated.[217] The levator scapula and the rhomboids also elevate the shoulder. Therefore, the observer must be certain that the trapezius is isolated before judging normal. The position of the scapula is observed; the trapezius elevates, rotates, and retracts the scapula and abducts the arm above the horizontal. Scapular winging indicates a lesion of CN XI, especially the fibers to the middle trapezius. The scapula is more prominent at rest, with the superior angle moving away from the midline.

Electrodiagnosis

Electrodiagnostic evaluation consists of nerve conduction studies[208] of the accessory nerve and EMG of the sternocleidomastoid and trapezius muscles. Conduction studies are summarized below. EMG evaluates insertional activity, activity at rest, minimal and maximal contraction, and recruitment.

Procedure of Nerve Conduction Studies of CN XI

1. *Stimulation* in the posterior cervical triangle.
2. Upper trapezius (scapular elevator). The active electrode is placed on the upper trapezius muscle 10 cm inferior and lateral to the point of stimulation. The reference electrode is placed over the acromion.[211,218]
   Normal values:
   - Latency: 2.3 ± 0.3 millisecond
   - Amplitude: 1.5 ± 0.5 mV
3. Middle trapezius (scapular adductor). The active electrode is placed halfway between the midpoint of the ipsilateral scapular spine and the thoracic spine. The reference electrode is placed on the nearest spinous process.[219]
   Normal values:
   - Latency: 3.0 ± 0.2 millisecond
   - Amplitude: 2.5 ± 1.0 mV
4. Lower trapezius (scapular depressor). The active electrode is placed 2 finger breadths (4–5 cm) from the spinal column on the level of the inferior angle of the scapula.[220] The reference electrode is placed on the nearest spinous process.
   Normal values:
   - Latency: 4.6 ± 0.3 millisecond
   - Amplitude: 1.3 ± 0.9 mV

Imaging and Prognosis

Imaging studies such as high resolution CT through the jugular foramen and MRI are helpful.

The prognosis of CN XI lesions is generally favorable, but considerable time may be required for recovery. Central lesions may recover spontaneously especially if the lesion is nonprogressive.

Management

Range-of-motion exercises should be performed to prevent contractures. Resistive exercises promote strengthening. Preventive measures should be applied when possible. For example, properly fitted shoulder pads and neck rolls for players of American football have been shown to reduce occurrence of stingers (burns).[211]

CRANIAL NERVE XII: HYPOGLOSSAL

The hypoglossal nerve is all motor and is depicted in Figures 7-38 and 7-39.

Anatomy and Function

The somatic efferent fibers arise from the hypoglossal nucleus, which consists of a longitudinal cell column in the medulla. Approximately 10 to 15 rootlets emerge from the ventrolateral sulcus of the medulla along the lateral border of the pyramid. These rootlets are gathered into two bundles and pass through the dura mater and the hypoglossal canal of the skull. The two bundles then combine and descend toward the angle of the mandible, passing near the internal carotid artery and the internal jugular vein. A descending ramus is given off and joins the ansa hypoglossi. The hypoglossal nerve then supplies the intrinsic muscles of the tongue and also the hypoglossus, styloglossus, genioglossus, and geniohyoid muscles. It supplies all extrinsic muscles of the tongue except the palatoglossus, which is supplied by CN X. The intrinsic

muscles of the tongue alter the shape of the tongue, and the extrinsic muscles alter its shape and position. The hypoglossal nucleus contains some spindle afferents.

The central connections are fibers controlling tongue movement that originates from the lower portion of the precentral gyrus near the sylvian fissure. Corticobulbar tract fibers accompany the corticospinal tract and terminate in the brainstem on the motor hypoglossal nucleus. Fibers may be crossed or uncrossed. There are reflex connections with the cortex, extrapyramidal, and tectospinal tracts, and with sensory nuclei of CN V, IX, and X. Corticobulbar control of the genioglossus muscles is contralateral. Most of the other tongue muscles appear to have bilateral supranuclear control.[221]

Injuries and Lesions

Blunt trauma in head injury causing basilar skull fractures may affect CN XII in isolation or in combination with other cranial nerves. However, blunt trauma is not a common cause for CN XII injury. More common are penetrating missile or gunshot wounds in the submandibular region, reported as common causes of hypoglossal nerve injury during the U.S. Civil War (1861–1865),[222] World War I,[223] and in a recent study from Los Angeles County (California) Hospital.[224] Injury of CN XII can occur in the neck or near the nerve entry into the tongue. This is a common site of injury during surgery, especially injury involving endarterectomies. Dislocation or fracture of the upper cervical vertebrae may involve CN XII.

Nontraumatic lesions include vascular lesions, especially in the internal capsule; lacunar strokes; or tumors compressing the corticobulbar fibers to

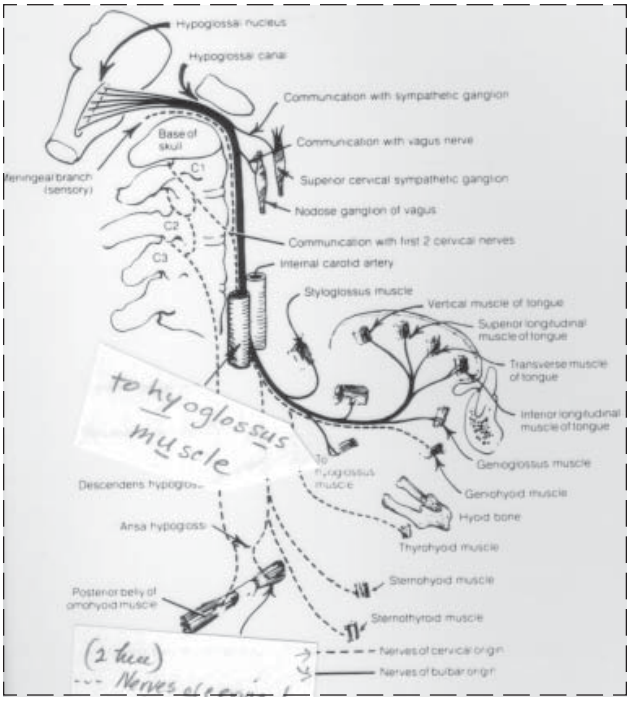


Fig. 7-38. The hypoglossal nerve. Reprinted with permission from deGroot J, Chusid JG. *Correlative Neuroanatomy.* 20th ed. Norwalk, Conn: Appleton & Lange; 1988: 175.

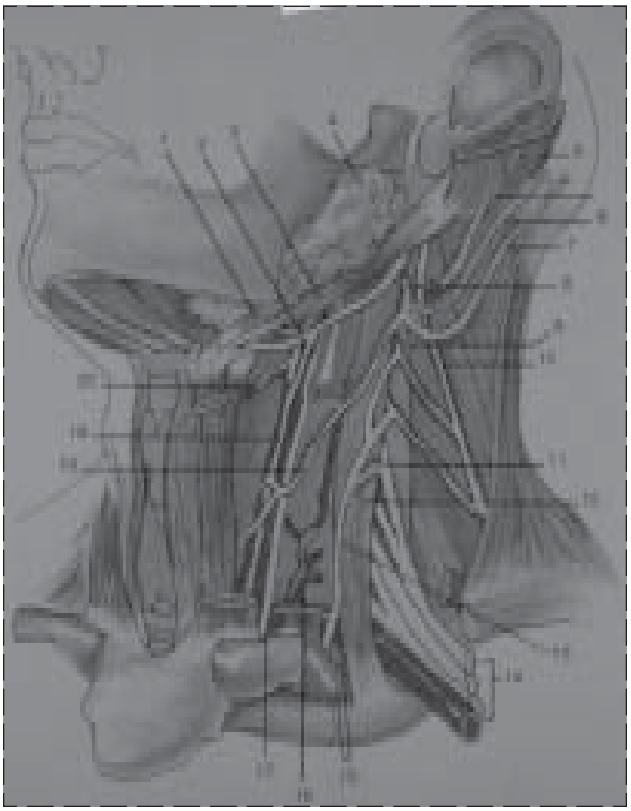


Fig. 7-39. Root of the neck showing location of the hypoglossal nerve (CN XII). 1: Stylohyoid muscle. 2: Cranial nerve XII. 3: Digastric muscle. 4: Parotid gland. 5: Sternocleidomastoid muscle. 6: Greater auricular nerve. 7: Lesser occipital nerve. 8: Ventral ramus C2. 9: Ventral ramus CC3. 10: Cranial nerve XI. 11: Ventral ramus C5. 12: Anterior scalene muscle. 13: Phrenic nerve. 14: Brachial plexus. 15: Subclavian artery and vein. 16: Thyrocervical trunk. 17: Cranial nerve X. 18: Inferior root ansa cervicalis. 19: Superior root ansa cervicalis. 20: Superior thyroid artery. Reprinted with permission from Maves MD. Surgical Anatomy of the Head and Neck. In: Byron E, Bailey J, eds. *Head & Neck Surgery: Otolaryngology*. Philadelphia, Pa: JB Lippincott; 1993: 20.

the hypoglossal nuclei in the nuclear and intramedullary area. Metastatic lesions invading the skull base may cause hypoglossal nerve lesions. Extracranial neurofibromas[225] or multiple sclerosis and syringobulbia may produce hypoglossal nerve lesions. Other nontraumatic causes are amyotrophic lateral sclerosis, poliomyelitis, and Dejerine's anterior bulbar syndrome. This is a rare syndrome that affects the hypoglossal nerve in its intramedullary course and is caused by occlusion of the anterior spinal artery. Still other nontraumatic lesions in the neck are carotid aneurysm and lesions of the parotid and other salivary glands, and the base of the tongue, with subsequent neck radiation.[226] Diseases such as myasthenia gravis, tuberculosis, and cerebral syphilis also must be considered. Common toxins that can interfere with hypoglossal nerve function are lead, alcohol, arsenic, and carbon monoxide. Certain tongue movement disorders may be induced by drugs or may be a form of seizure that injures the hypoglossal nerve.[227] Continuous lingual myoclonus may occur after head injury.[228]

Syndromes: Signs and Symptoms

Syndromes involving CN XII include Dejerine's anterior bulbar syndrome and Collet-Sicard syndrome.

Dejerine's anterior bulbar syndrome presents with ipsilateral paresis, atrophy, and fibrillations of the tongue. The protruded tongue deviates toward the side of the lesion. There also presents contralateral hemiplegia (pyramid involvement) with sparing of face, contralateral loss of position, and vibratory sense (medial lemniscus).

Collet-Sicard syndrome is manifested by paralysis of the trapezius and sternocleidomastoid muscles (CN XI); unilateral paralysis of vocal cord (CN X) and pharynx (CN IX); hemiparalysis of the tongue (CN XII); loss of taste on the posterior third of tongue (CN IX); and hemianesthesia of palate, pharynx and larynx (CN IX and X).[229]

Other syndromes involving lower cranial nerves are Jackson's syndrome (CN X, XI, and XII); Tapia's syndrome (CN X and XII); and Villaret's syndrome (CN IX, X, XI, and XII).

Signs and symptoms of CN XII lesions are secondary to weakness of the tongue. Specific observable signs and symptoms of CN XII lesions are listed below.

- Weakness and atrophy of the tongue (unilateral or bilateral depending on the lesion).
- Fibrillation of tongue (only in lower motor neuron lesions).
- Protruded tongue deviating to side of lesion (action of unopposed contralateral genioglossus muscle) in a lower motor neuron lesion.
- Nonprotruded tongue pointing to normal side (voluntary contraction, unopposed action of styloglossus).[230]
- Protruded tongue deviating to opposite side of lesion in an upper motor neuron lesion.
- Possible respiratory difficulties (in bilateral lesions, atrophic tongue slips back to throat).
- Dysarthria (difficulty with speech articulation, especially difficulty in pronouncing dental consonants). See Table 7-7.
- Possible Horner's syndrome (if cervical sympathetic trunk is interrupted in the region where the hypoglossal nerve emerges from the skull).
- Possible oral-buccal-lingual dyskinesia, athetosis, palatal myoclonus, tremor.

Evaluation

Clinical examination consists of observation of the tongue at rest, looking for fasciculations (motor neuron disease), atrophy, or furrowing. The tongue is then observed during movement within the mouth for intrinsic tongue muscle activity, and protruding from the mouth for extrinsic tongue muscle activity.

The strength of tongue muscles is assessed by providing resistance to the protruded tongue. Quickness of tongue movement is evaluated as well as the articulation of speech and tongue movements during eating. The paralyzed tongue has difficulty moving bolus toward the pharynx and from side-to-side in the preparatory phase of swallowing. Breathing must also be observed, to rule out obstruction of the pharynx with a paralyzed tongue.

Electrodiagnosis

Electrodiagnostic evaluation can be performed by nerve conduction studies[231] and EMG. The procedure for nerve conduction is as follows.

Stimulation of the hypoglossal nerve is conducted anterior and inferior to the corner of the mandible (one third the distance from the angle of the jaw to

TABLE 7-7

CRANIAL NERVE LESION AS CAUSE OF DYSARTHRIA

Involved CN	Laryngeal (Phonatory)	Velopharyngeal	Oral
XII			
Flaccid	Normal	Normal	Unilateral or bilateral atrophic weak tongue, fasciculations, decreased range-of-motion of tongue, drooling, imprecision of vowels and lingual consonants
X			
Flaccid	Hoarseness, breathiness, low volume, diplophoniae	Hypernasally nasal mission, if lesion is above pharyngeal branch	Normal
VII			
Flaccid	Normal	Normal	Unilateral or bilateral weak orbicularis oris, reduced range-of-motion in lips and cheek, imprecision of vowels and labial consonants
V			
Flaccid	Normal	Normal	Mandibular muscles weakness, loss of tongue sensation with lingual nerve involvement, imprecision of vowels and consonants
Multiple CN			
Flaccid	Breathiness, reduced volume, inhalatory, stridor, monopitch	Hypernasally, nasal emission	Imprecision of vowel and consonants
Bilateral Upper Motor Neuron			
Spastic	Hyperadduction of vocal folds, strained, strangled voice, hoarseness, low pitch, monopitch	Incompleteness of palatopharyngeal closure, hypernasality	Slowness, weakness of tongue and mimetic muscles, imprecision of consonants

CN: cranial nerve
Reprinted with permission from Aronson AE. Motor speech signs of neurologic disease. In: Darby JK Jr. *Speech Evaluation in Medicine.* New York: Grune & Stratton; 1981: 159–180.

the mental protuberance along the base of the mandible and 1 cm medial).

Recording is carried out with superficial electrodes (a bar embedded and placed on top of the tongue).

Normal values:

- Latency: 2.2 ± 0.4 millisecond
- Amplitude: 3.8 ± 1.6 mV.

EMG of tongue is carried out through direct insertion of electrodes into the tongue or into the mylohyoid muscle, through the skin beneath the chin. The examiner looks for possible evidence of denervation, action potential parameter abnormalities, reduced recruitment, or myoclonus.

Imaging and Prognosis

Imaging the skull base with CT, MRI, or both, is helpful. The prognosis for recovery is better for upper motor neuron lesions than for lower motor neuron lesions.

Management

Management of hypoglossal nerve lesions is assisted by speech therapy, using strengthening ex-

ercises for the tongue to facilitate speech and improve the oral phase of swallowing. See the discussion of CN IX for therapeutic interventions to improve the oral phase of swallowing.

Severe involvement makes tube feeding or gastrostomy necessary. Tracheostomy or a surgical procedure may be required to promote efficient breathing.

CONCLUSION

In this chapter, each of the 12 cranial nerves has been considered in detailed account as a single anatomical and functional entity. The signs and symptoms of deficits following peripheral and central injuries and lesions have been described and evaluated. Syndromes were tabulated and briefly discussed. Injuries and lesions of the head and neck frequently display conjoint nerve involvement due to the close proximity or origin of central tracts and nuclei of termination and the close anatomical intracranial and peripheral course of one nerve path with others. Such injuries and lesions then present a complexity of deficits, each attributable to the specific nerve (or nucleus or tract) involved. Keeping in mind that branches of nerves attach onto branches of other nerves and may course over several nerves from sensory origin to motor destination, such vulnerability and subsequent inclusion is far more frequent in injuries than single nerve involvement. Therefore, within the discussion of each cranial nerve as an entity, pertinent multiple involvement has been discussed.

In addition to more routine methods of examination and diagnosis, the use of the most current tools of electrodiagnosis and imaging have been emphasized. Techniques and procedures have been tabulated concisely. These sophisticated techniques offer advantages for use in surgical procedures in neuromonitoring as precautionary measures.

Lastly, prognosis for the various conditions and syndromes is integral to and supports the discussion of management and rehabilitative measures.

REFERENCES

1. Maclean JG, Taylor A. Combined lower cranial nerve injury: Complication of upper cervical or basal skull fracture. *J R Coll Surg Edinb.* 1991;36:188–189.

2. Hammer AJ. Lower cranial nerve palsies: Potentially lethal in association with upper cervical fracture dislocation. *Clin Orthop.* 1991;266:64–69.

3. Edmund J, Godtfredsen E. Unilateral optic atrophy following head injury. *Acta Ophthalmol.* 1963;41:693.

4. Roberts AH. *Severe Accidental Head Injury.* New York: Macmillan; 1979.

5. Russell WR. Injury to cranial nerves including the optic nerves and chiasma. In: Brock S, ed. *Injuries of the Skull, Brain, and Spinal Cord.* 2nd ed. Baltimore, Md: Williams & Wilkins; 1943:104.

6. Costanzo RN, Becker DFP. Sense of smell and taste disorders in head injury and neurosurgery patients. In: Meiselman HL, Rivlin RS, eds. *Clinical Measurement of Taste and Smell.* New York: Macmillan; 1986: 565–578.

7. Kitchins JL, Groff DB, Nagaraj HS, Fallat ME: Basilar skull fractures in childhood with cranial nerve involvement. *J Pediatr Surg.* 1991;26(8):992–994.

8. Friedman AP, Merritt HH. Damage to cranial nerves resulting from head injury. *Bull Los Angeles Neurol Soc.* 1944;9:135–139.

9. Hughes B. The results of injury to special parts of the brain and skull: The cranial nerves. In: Rowbotham GR, ed. *Acute Injuries of the Head.* Edinburgh, Scotland: Livingston; 1964: 410.

10. Dobson JE, Newell MJ, Shepherd JP: Trends in maxillofacial injuries in wartime (1914–1986). *Br J Oral Maxillofac Surg.* 1989;27:441–450.

11. Keane JR, Baloh RW. Post-traumatic cranial neuropathies. *Neurol Clin.* 1992;10(4):853.

12. Jacobi G, Ritz A, Emrich R. Cranial nerve damage after pediatric head trauma: A long term follow up study of 741 cases. *Acta Paediatr Hung.* 1986;27:173–187.

13. Harner SG, Danbe JR, Beatly CW, Ebersvold MJ. Intraoperative monitoring of the facial nerve. *Laryngoscope.* 1988;98:209–212.

14. Moller AR. Neuromonitoring in operations in the skull base. *KEIO J Med.* 1991;40(3):151–159.

15. Nelson KR, Phillips LH. Neurophysiologic monitoring during surgery of peripheral and cranial nerves and in selective dorsal rhizotomy. *Semin Neurol.* 1990;10(2):141–149.

16. Ruth RA. Neurophysiologic intraoperative monitoring. In: Bailey BJ, ed. *Head and Neck Surgery: Otolaryngology.* Philadelphia, Pa: JB Lippincott; 1993:1505–1516.

17. Sekhar LN, Lanzino G, Sen CN, Pomonis S. Reconstruction of the third through sixth cranial nerves during cavernous sinus surgery. *J Neurosurg.* 1992;76:935–943.

18. Ransom DT, Dinapoli RP, Richardson RL. Cranial nerve lesions due to base of the skull metastases in prostate carcinoma. *Cancer.* 1990;65(3):586–589.

19. Sumner D. Post traumatic anosmia. *Brain.* 1964;87:107–120.

20. Jennett B, Teasdale G, eds. *Management of Head Injuries.* Philadelphia, Pa: FA Davis; 1981: 272–278.

21. Meacy J. *Pediatric Head Injuries.* Springfield, Ill: Charles C Thomas; 1968: 88–136.

22. Bakay L. Olfactory meningioma, the missed diagnosis. *JAMA.* 1984;251:53–55.

23. Doty RL, Bartoshuk LM, Snow JB. Causes of olfactory and gustatory disorders. In: Getchell TV, Doty RL, Bartoshuk LM, Snow JB Jr, eds. *Smell and Taste in Health and Disease.* New York: Raven Press; 1991: 451.

24. Jones BP, Butter NM, Moskowitz HR, Montgomery K. Olfactory and gustatory capacities of alcoholic Korsakoff patients. *Neuropsychologia.* 1988;16:323.

25. Moberg PF, Pearlson GD, Speedie LJ, Lipsey JR, Strauss ME, Folstein SE. Olfactory recognition: Differential impairments in early and late Huntington's and Alzheimer's diseases. *J Clin Exp Neuro Psych.* 1987;9:650–664.

26. Warner MD, Peabody CA, Flattery JJ, et al. Olfactory deficits and Alzheimer's disease. *Biol Psychiatry.* 1986;21:116.

27. Schatz, NJ, Smith JL. Nontumor causes of the Foster Kennedy Syndrome. *J Neurosurg.* 1948;40:245.

28. Doty RL, Riklan M, Deems DA, Reynolds C, Stellar S. The olfactory and cognitive deficits of Parkinson's disease: Evidence for independence. *Ann Neurol.* 1989;25:166–171.

29. Schiffman SS. Age related damages in taste and smell and their possible causes. In: Meiselman HL, Rivlin RS, eds. *Clinical Measurement of Taste and Smell.* New York: Macmillan; 1986: 326–342.

30. Deems DA, Doty RL, Settle RG, et al. Smell and taste disorders: A study of 750 patients from the University of Pennsylvania smell and taste center. *Arch Otolaryngol Head Neck Surg.* 1991;117:519–528.

31. Schweitzer VG. Osteolytic sinusitis and pneumomediastinum: Deceptive otolaryngologic complications of cocaine abuse. *Laryngoscope.* 1986;96:206–210.

32. Levin HS, High WM, Elsenberg HM. Impairment of olfactory recognition after closed head injury. *Brain.* 1985;108:579–591.

33. Caruso V, Hagan J, Manning H. Quantitative olfactometry in measurement of post traumatic hyposmia. *Arch Otolaryngol.* 1968;90:500.

34. Cain WS, Gent JF, Goodspeed RB, Leonard G. Evaluation of olfactory dysfunction in the Connecticut chemosensory clinical research center. *Laryngoscope*. 1988;98:83–88.

35. Costanzo RM, Zasler ND. Head trauma. In: Getchell TV, Doty RL, Bartoshuk LM, Snow JB Jr, eds. *Smell and Taste in Health and Disease.* New York: Raven Press; 1991: 715.

36. Kobal G, Hummel T. Olfactory evoked potentials in humans. In: Getchell TV, Doty RL, Bartoshuk LM, Snow JB Jr, eds. *Smell and Taste in Health and Disease.* New York: Raven Press; 1991: 255–275.

37. Kobal G. *Elektrophysiologische Untersuchungen Des Menschlichen Geruchsinns.* Stuttgart: Thieme; 1981.

38. Zasler ND, Costanzo RM, Heywood PG. Neuroimaging correlates of olfactory dysfunction after traumatic brain injury. *Arch Phys Med Rehabil.* 1990;71:814.

39. Costanzo RM. Neural-regeneration and functional reconnection following olfactory nerve transsection in hamster. *Brain Res.* 1985;361:258–266.

40. American Medical Association. *Guides to the Evaluation of Permanent Impairment.* Chicago, Ill: AMA; 1988.

41. Dimond SJ, Farrington L, Johnson P. Differing emotional response from right and left hemisphere. *Nature.* 1976;261:690–692.

42. Shokunbi T, Agbeja A. Ocular complications of head injuries in children. *Childs Nerv Syst.* 1991;7:147–149.

43. LaGrange F. Fractures of the orbit and injuries to the eye in war. *Adv Ophthal Plast Reconstr Surg.* 1986;5:169–176.

44. LaGrange F. Fractures of the orbit with preservation of the eyeball. *Adv Ophthal Plast Reconstr Surg.* 1987;6:145–192.

45. Newman NM, Di Loreto DA, Ho JT, Klein JC, Birnbaum NS. Bilateral optic neuropathy and osteolytic sinusitis: Complications of cocaine abuse. *JAMA.* 1988;259:72–74.

46. Harriman DG, Garland H. The pathology of Adie's syndrome. *Brain.* 1968;91:401–418.

47. Zihl J, Von Cramon D. Restitution of visual function in patients with cerebral blindness. *J Neurol Neurosurg Psychiatry.* 1979;42:312–322.

48. Ross Russell RW, Bharucha N. Visual localization in patients with occipital infarction. *J Neurol Neurosurg Psychiatry.* 1984;47:153–158.

49. Spector RH, Glaser JS, David NJ, Vining DQ. Occipital lobe infarctions: Perimetry and computed tomography. *Neurology.* 1981;31:1098–1106.

50. Hennekes R. Clinical electroretinography. *Fortschr Ophthalmol*. 1989;86:146–150.

51. Chiappa KH. Pattern-shift visual evoked potentials. In: Chiappa KH, ed. *Evoked Potentials in Clinical Medicine.* 2nd ed. New York: Raven Press; 1989: 37–155.

52. Hughes B. Indirect injury of the optic nerves and chiasma. *Bull Johns Hopkins Hosp.* 1962;111:98–126.

53. Geisler FH, Dorsey FC, Coleman WP. Recovery of motor function after spinal cord injury: A randomized, placebo controlled trial with GM-1 ganglioside. *N Engl J Med.* 1991;324:1829–1838.

54. Spooner JW, Sakala SM, Baloh RW. Effect of aging on eye tracking. *Arch Neurol.* 1980;37:57–59.

55. Nadeau SE, Trobe JD. Pupil sparing in ocularmotor palsy: A brief review. *Ann Neurol.* 1983;13:143.

56. Warren M. A hierarchical model for evaluation and treatment of visual perceptual dysfunction in adult aquired brain injury. *Am J Occup Ther.* 1993;47:42–65.

57. Russell WR. Injury to cranial nerves and optic chiasm. In: Brock S, ed. *Injuries of the Brain and Spinal Cord and Their Coverings.* 4th ed. Baltimore, Md: Williams & Wilkins; 1960.

58. Kontroupas S, Meyerhoff NL. Surgical treatment of orbital floor fractures. *Arch Otolaryngol.* 1982;108:184–186.

59. DiNubile MJ. Septic thrombosis of the cavernous sinus. *Arch Neurol.* 1988;45:567.

60. Donaldson D, Rosenberg NL. Infarction of abducens nerve fasicle as cause of isolated sixth nerve palsy related to hypertension. *Neurology.* 1988;38:1654.

61. Carter S, Sciarra D, Merritt HH. The course of multiple sclerosis as determined by autopsy proven cases. *Res Publ Assoc Res Nerv Ment Dis*. 1950;28:471.

62. Spector RH, Smith JL, Chavis PS. Charcot-Marie-Tooth disease mimicking ocular myasthenia gravis. *Ann Ophthalmol.* 1978;10:1033–1038.

63. Hyams SW. Oculomotor palsy following dental anesthesia. *Arch Ophthalmol.* 1976;94:1281.

64. Masdeu J, Brazis PW. The localization of lesions in the ocularmotor system. In: Brazis PW, Masdeu JC, Biller J, eds. *Localization in Clinical Neurology*. 2nd ed. Boston, Mass: Little, Brown; 1990: 140–143.

65. Yaskin JC, Kornblum K. Neurologic aspects of petrositis. *Arch Neurol Psychiat.* 1937;37:307–333.

66. Keane JR, Zaias B, Itabashi HH. Levator sparing oculomotor nerve palsy caused by a solitary midbrain metastasis. *Arch Neurol.* 1984;41:210–212.

67. Conway VH, Rozdilsky B, Schneider RJ, Sundaram M. Isolated bilateral complete ptosis. *Can J Ophthalmol.* 1983;18:37–40.

68. Growden JH, Winkler GF, Wray SH. Midbrain ptosis. A case with clinico-pathologic correlation. *Arch Neurol.* 1974;30:179.

69. Leigh RJ, Zee DS. *The Neurology of Eye Movement.* 2nd ed. Philadelphia, Pa: FA Davis; 1991: 111.

70. Smith JL, David NJ, Klindworth G. Skew deviation. *Neurology.* 1964;14:96.

71. Neger RE. The evaluation of diplopia in head trauma. *J Head Trauma Rehabil.* 1989;4:27–34.

72. Warren M. Identification of visual scanning deficits in adults after cerebrovascular accident. *Am J Occup Ther.* 1990;44:391–399.

73. VonNoorden GK. *Burian-VonNoorden's Binocular Vision in Ocular Motility.* 2nd ed. St. Louis, Mo: Mosby; 1980.

74. Thompson BM, Corbett JJ, Kline LB, Thompson HS. Pseudo Horner's syndrome. *Arch Neurol.* 1982;39:108–111.

75. Sydnor CF, Seaber JH, Buckley EG. Traumatic superior oblique palsies. *Ophthalmology (Rochester).* 1982;89: 134–138.

76. Frank JW, Kushner BJ, France TD. Paradoxic pupillary phenomena: A review of patients with pupillary constriction to darkness. *Arch Ophthalmol.* 1988;106:1564–1566.

77. Bouska MJ, Kauffman MA, Marcus S. Disorders of the visual perceptual system. In: Umphred DA, ed. *Neurological Rehabilitation.* Vol 4. St. Louis, Mo: Mosby; 1990.

78. Cytowic R, Stump DA, Larned DC. Closed head trauma: Somatic, ophthalmic and cognitive impairment in nonhospitalized patients. In: Whitaker HA, ed. *Neuropsychological Studies of Nonfocal Brain Damage.* New York: Springer-Verlag; 1990.

79. Troost BT. Neuro-otology. In: Bradley WG, Daroff RB, Feinchel GM, Marsden CD, eds. *Neurology and Clin Practice.* Vol 1. Woburn, Mass: Butterworth-Heineman; 1991: 655.

80. Berrol S. Cranial nerve dysfunction. In: *Physical Medicine and Rehabilitation*: *State of the art reviews.* 1989;3:89.

81. Zasler ND, McClintic N. Functional orthoptic evaluation after traumatic brain injury. Presented at the Postgraduate Course on Rehabilitation of the Brain Injury in Adult and Child; June 1991; Williamsburg, Va.

82. Trobe JD, Acosta PC, Krischer JP, Trick GL. Confrontation visual field techniques in the detection of anterior visual pathway lesions. *Ann Neurol.* 1981;10:28–34.

83. Silver WL, Finger TE. The trigeminal system. In: Getchell TV, Doty RL, Bartoshuk LM, Snow JB Jr, eds. *Smell and Taste in Health and Disease.* New York: Raven Press; 1991: 97.

84. Greenward HS Jr, Keeney AH, Shannon GM. A review of 128 patients with orbital fracture. *Am J Ophthalmol* 1974;78:655.

85. Yadav YR, Khosla VK. Isolated 5th-10th cranial nerve palsy in closed head trauma. *Clin Neurol Neurosurg.* 1991;93:61–63.

86. Gonzalez Revilla A. Differential diagnosis of tumors at the cerebello-pontine recess. *Bull Johns Hopkins Hosp.* 1989,83:187–212.

87. Blackburn CW, Bramley PA. Lingual nerve damage associated with the removal of lower 3rd molars. *Br Dent J.* 1989;167:103–107.

88. Rushton JG, Olafson RA. Trigeminal neuralgia associated with multiple sclerosis. Report of 35 cases. *Arch Neurol.* 1965;13:383–386.

89. Jannetta PJ, Rand RW. Arterial compression of trigeminal nerve at the pons in patients with trigeminal neuralgias. *J Neurosurg.* 1967;26(Suppl):159.

90. Ashworth B, Tait GBW. Trigeminal neuropathy in connective tissue disease. *Neurology*. 1971;21:609–614.

91. Caselli RJ, Hunder GG, Whisnant JP. Neurologic disease in biopsy-proven giant cell (temporal) arteritis. *Neurology.* 1988;38:352–359.

92. Brazis PW. The localization of lesions effecting cranial nerve V. In: Brazis PW, Masdeu JC, Biller JL, eds. *Localization in Clinical Neurology*. 2nd ed. Boston, Mass: Little, Brown; 1990: 197.

93. Loeser JD. Cranial neuralgias. In: Bonica JJ, ed. *The Management of Pain*. 1990: 676–686.

94. Lance JW, Anthony M. Neck-tongue syndrome on sudden turning of the head. *J Neurol Neurosurg Psychiatry.* 1980;43:97–101.

95. Campbell WW. The numb cheek syndrome: A sign of infraorbital neuropathy. *Neurology.* 1986;36:421.

96. Mountcastle VB, Talbot WH, Darian-Smith I, Kornhuber HH. Neural basis the sense of flutter-vibration. *Science.* 1967;155:597–600.

97. Dellon AL. The moving two-point discrimination test. Clinical evaluation of the quickly adapting fibre/receptor system. *J Hand Surg.* 1978;3:474–481.

98. Ongerboer DE, Visser BW, Goor C. Jaw reflexes and masseter electro-myograms in mesencephalic and pontine lesions. An electrodiagnostic study. *J Neurol Neurosurg Psychiatry.* 1976;39:90–92.

99. Godoux E, Desmedt GE. Exteroceptive supression and motor control of the masseter and temporalis muscles in normal man. *Brain Res.* 1975;85:447–458.

100. Ongerboer DE, Visser BW, Goor C. Cutaneous silent period in masseter muscles: A clinical and electrodiagnostic evaluation. *J Neurol Neurosurg Psychiatry.* 1976;39:674–679.

101. Ongerboer DE, Visser BW, Cruccu G, Manfredi M, Koelman JH. Effects of brainstem lesions on the masseter inhibitory reflex. Functional mechanism of reflex pathways. *Brain.* 1990;113:781–792.

102. Cruccu G, Pauletti G, Agostino R, Berardelli A, Manfredi M. Masseter inhibitory reflex in movement disorders. Huntington's chorea, Parkinson's disease, dystonia, and unilateral masticatory spasm. *Electroencephalogr Clin Neurophysiol.* 1991;81:24–30.

103. Hopf HC, Ellrich J, Hundermer H. *The Pterygoid Reflex in Man and its Clinical Application Muscle and Nerve.* 1992;15:1278–1283.

104. Kimura J, Powers JM, Van Allen MW. Reflex response of orbicularis oculi muscle to supraorbital nerve stimulation: Study in normal subjects and in peripheral facial paresis. *Arch Neurol.* 1969;21:193–199.

105. Kimura J. Conduction abnormalities of the facial and trigeminal nerves in polyneuropathy. *Muscle Nerve.* 1982;5:139–144.

106. Kimura J, Bodensteiner J, Yamada T. Electrically elicited blink reflex in normal neonates. *Arch Neurol.* 1977;34:246–249.

107. Accornero N, Berardelli A, Cruccu G, Bini G, Manfredi M. Corneal reflex elicited by electrical stimulation of human cornea. *Neurology*. 1980;30:782–785.

108. Di Benedetto M. Trigeminal sensory nerve conduction. Unpublished data.

109. Stohr M, Petruch F. Somatosensory evoked potentials following stimulation of the trigeminal nerve in man. *J Neurol.* 1979;220:95–98.

110. Dillingham TR, Spellman NT, Belandres PV. Trigeminal motor nerve conduction: Deep temporal and mylohyoid nerves. *Muscle Nerve.* 1993;16:1115.

111. Jannetta PJ. Microsurgical approach to the trigeminal nerve for tic douloureux. *Prog Neurosurg*. 1976;7:180–200.

112. Roberts AM, Person P. Etiology and treatment of idiopathic trigeminal and atypical facial neuralgias. *Oral Surg.* 1979;48:298–308.

113. Brazis PW. The localization of lesions affecting cranial nerve VII (the facial nerve). In: Brazis PW, Masdeu JC, Biller JL, eds. *Localization in Clinical Neurology*. 2nd ed. Boston, Mass: Little, Brown; 1990: 206–207.

114. Bartoshuk LM, Kveton JF, Karrer T. Taste. In: Bailey BJ, ed. *Head and Neck Surgery: Otolaryngology.* Philadelphia, Pa: JB Lippincott; 1993: 520–529.

115. Jenny AB, Saper CB. Organization of the facial nucleus and cortico-facial projection in the monkey: A reconsideration of the upper motor neuron facial palsy. *Neurology.* 1987;37:930–939.

116. Borod JC, Koff E, Lorch MP, Nicholas M, Welkowitz J. Emotional and nonemotional facial behaviour in patients with unilateral brain damage. *J Neurol Neurosurg Psychiatry.* 1988;51:826–832.

117. Sumner D. Post traumatic ageusia. *Brain.* 1967;90:187–202.

118. Costanzo RM, Zasler ND. Epidemiology and pathophysiology of olfactory and gustatory dysfunction in head trauma. *J Head Trauma Rehabil.* 1992;7:15–24.

119. Schechter PJ, Henkin RI. Abnormalities of taste and smell after head trauma. *J Neurol Neurosurg Psychiatry*. 1974;37:802–810.

120. Fisch U. Facial paralysis in fractures of the petrous bone. *Laryngoscope.* 1974;84:2141–2154.

121. Miller H. Facial paralysis. *Br Med Bull.* 1967;3:815.

122. Park HW, Watkins AL. Facial paralysis: Analysis of 500 cases. *Arch Phys Med.* 1949;30:749–762.

123. Karnes WE. Diseases of the seventh cranial nerve. In: Dyck PJ, Thomas PK, Lambert EH, eds. *Peripheral Neuropathy.* Philadelphia, Pa: WB Saunders; 1975: 570–603.

124. Brown JW. Physiology and phylogenesis of emotional expression. *Brain Res.* 1967;5:1–14.

125. Hanson MR, Sweeney PJ. Lower cranial neuropathies. In: Bradley WG, Daroff RB, Feinchel GM, Marsden CD, eds. *Neurology and Clinical Practice.* Vol 1. Woburn, Mass: Butterworth-Heineman; 1991: 220.

126. Frank ME, Smith EV. Electrogustometry. In: Getchell TV, Doty RL, Bartoshuk LM, Snow JB Jr, eds. *Smell and Taste in Health and Disease.* New York: Raven Press; 1991: 503–514.

127. Plattig KH. Gustatory evoked brain potentials in humans. In: Getchell TV, Doty RL, Bartoshuk LM, Snow JB, eds. *Smell and Taste in Health and Disease*. New York: Raven Press; 1991;14:227–286.

128. Dumitru D, Walsh NE, Porter LD. Electrophysiologic evaluation of the facial nerve in Bell's palsy. A review. *Am J Phys Med Rehabil.* 1988;137–143.

129. Mitz M, Prakash AS, Melvin J, Piering W. Motor nerve conduction indicators in uremic neuropathy. *Arch Phys Med Rehabil.* 1980;61:45–48.

130. Jannetta PJ, Abbasy M, Maroon JC, Ramos FM, Albin MS. Etiology and definitive microsurgical treatment of hemifacial spasm. *J Neurosurg.* 1977;47:321–328.

131. Granger C. Prognosis in Bell's palsy. *Arch Phys Med Rehabil.* 1976;61:45–48.

132. Diamant H, Kirkstrand T, Wiberg A. Prognosis of idiopathic Bell's palsy. *Arch Otolaryngol.* 1972;95:431.

133. Drachman DA. Bell's palsy: A neurological point of view. *Arch Otolaryngol.* 1969;89:173.

134. El-Ebiary HM. Prognosis in Bell's palsy. *Rheumatol Phys Med.* 1971;11:111.

135. Langworth EP, Taverner D. The prognosis in facial palsy. *Brain*. 1963;86:465–480.

136. Schaumburg HH. Diseases of the peripheral nervous system. In: Wyngaarden JB, Smith LH, eds. *Cecil Text-book of Medicine.* Philadelphia, Pa: WB Saunders; 1988: 2267.

137. Crumley R, Robinson L. Traumatic facial nerve injury. In: Gates GG, ed. *Current Therapy. Head and Neck Surgery*. Philadelphia, Pa: BC Decker; 1990: 97–101.

138. Pitty LF, Tator CH. Hypoglossal-facial nerve anastomosis for facial nerve palsy following surgery for cerebellopontine angle tumors. *J Neurosurg.* 1992;77:724–731.

139. Wilson-Pawels L, Akesson EJ, Stewart PA. Cranial nerves. In: *Anatomy and Clinical Comments.* Hamilton, Ontario, Canada: BC Decker; 1988: 104.

140. Sakai CS, Mateer CA. Otological and audiological sequalae of closed head trauma. *Semin Hearing.* 1984;5:157–173.

141. Barber HO. Positional nystagmus especially after head injury. *Laryngoscope.* 1964;74:891–944.

142. Barber HO. Head injury: Audiological and vestibular findings. *Ann Otol Rhinol Laryngol.* 1969;78:239–252.

143. Berman J, Fredrickson J. Vertigo after head injury: A 5-year follow up. *J Otolaryngol.* 1978;7:237.

144. Healy GB. Hearing loss and vertigo secondary to head injury. *N Engl J Med.* 1982;306:1029.

145. Clark K, Rees TS. Post traumatic endolymphatic hydrops. *Arch Otolaryngol* 1983;103:1009–1012.

146. Vernon J, Northern JL, eds. *Tinnitus. Hearing Disorders.* Boston: Little, Brown; 1984.

147. Clark J, Yanick P. *Tinnitus and its Management.* Springfield, Ill: Charles C Thomas; 1984.

148. Schein JD, Miller MH. Diagnosis and rehabilitation of auditory disorders. In: Kottke FJ, Lehmann JF, eds. *Krusen's Handbook of Physical Medicine and Rehabilitation.* Philadelphia, Pa: WB Saunders; 1990: 953.

149. Tuohimaa P. Vestibular disturbances after acute mild head injury. *Acta Otolaryngol.* 1979;359(Suppl):1–67.

150. Windle WF, Groat RA, Fox CA. Experimental structural alterations in the brain during and after concussion. *Surg Gynecol Obstet.* 1944;79:561–572.

151. Griffiths MV. The incidence of auditory and vestibular concussion following minor head injury. *J Laryngol Otol.* 1979;9:253–265.

152. Masdeu JC, Brazis PW. The localization of lesions in the oculomotor system. In: Brazis PW, Masdeu JC, Biller J, eds. *Localization in Clinical Neurology*. 2nd ed. Boston, Mass: Little, Brown; 1990: 172.

153. Troost BT. Neuro-otology. In: Bradley WG, Daroff RB, Feinchel GM, Marsden CD, eds. *Neurology and Clinical Practice.* Vol 1. Woburn, Mass: Butterworth-Heineman; 1991: 657.

154. Chiappa KH. Brainstem auditory evoked potentials. Methodology. In: Chiappa KH, ed. *Evoked Potentials in Clinical Medicine,* 2nd ed. New York: Raven Press; 1989: 174–220.

155. Bergamasco BP, Gilli BC, Ferrero P, et al. Brainstem auditory evoked potentials in postconcussion syndromes. *Ital J Neurol Sci.* 1986;4:291–297.

156. Montgomery A, Penton GW, McClelland RJ. Delayed brainstem conduction time in post concussional syndrome. *Lancet.* 1984;1:1011.

157. Shulman A. External electrical stimulation in tinnitus control. *Am J Otolaryngol.* 1985;6:110–115.

158. Karnick PP, Kirtane MV, Wagh SP, et al. Otoneurologic problems in head injuries and their management. *Int Surg.* 1975;60:466.

159. Berger KW. *Speech Reading Principles and Methods.* Baltimore, Md: National Education Press; 1972.

160. Shumway-Cook A, Horak F. Rehabilitation strategies for patients with vestibular deficits. *Neurol Clin.* 1990;8:441.

161. Igarashi M, Ishixawa M, Yamane HI. Physical exercise and balance compensation after total ablation of vestibular organs. *Prog Brain Res.* 1988;76:395–401.

162. Herdman SJ. Treatment of vestibular disorders in traumatically brain injured patients. *J Head Trauma Rehabil.* 1990;5:63.

163. Hunt JR. Geniculate neuralgia: A further contribution to the sensory system of the facial nerve and its neuralgic conditions. *Arch Neurol Psychiatry.* 1937;37:253–285.

164. Kanagasuntheram R, Wong WC, Chan HL. Some observations on the innervation of the human nasopharynx. *J Anat.* 1969;104:361.

165. Mohanty SK, Barrios M, Fishbone H, Khatib R. Irreversible injury of cranial nerve IX–XII (Collet-Sicard Syndrome). *J Neurosurg.* 1973;30:86–88.

166. Russell JR, Buct PC. Meningiomas of the posterior fossa. *Surg Gynecol Obstet.* 1953;96:183.

167. Demir R, Steegmann AT. Intracranial chordoma. *Neurology*. 1959;9:514.

168. Henson RA, Crawford JV, Cavanagh JB. Tumours of the glomus jugulare. *J Neurol Neurosurg Psychiatry.* 1953;16:127–138.

169. Rushton JG, Stevens JC, Miller RH. Glossopharyngeal (vagoglossopharyngeal) neuralgia. *Arch Neurol.* 1981;38:201.

170. Laha RK, Jannetta PJ. Glossopharyngeal neuralgia. *J Neurosurg.* 1977;47:316.

171. Brazis PW. The localization of lesions affecting cranial nerves IX and X (the glossopharyngeal and vagus nerves). In: Brazis PW, Masdeu JC, Biller JL, eds. *Localization in Clinical Neurology*. 2nd ed. Boston, Mass: Little, Brown; 1990: 240–248.

172. Sumi T. Role of the pontine reticular formation in the neural organization of deglutition. *Japanese J Physiol.* 1972;22:295–314.

173. Siebens AA. Rehabilitation for swallowing impairment. In: Kottke FJ, Lehmann JE, eds. *Handbook of Physical Medicine and Rehabilitation*, 4th ed. Philadelphia, Pa: WB Saunders; 1990: 765–776.

174. Logemann J. *Evaluation and Treatment of Swallowing Disorders.* San Diego, Calif: Coll-Hill Press; 1983: 129–215.

175. Shanahan TK, Logemann JA, Rademaker AW, Palkoski BR, Kahrilas PJ. Chin-down posture effect on aspiration in dysphagic patients. *Arch Phys Med Rehabil.* 1993;74:736–739.

176. Sessions D, Zill R, Schwartz J. Deglutition after conservation surgery for cancer of the larynx and hyperpharynx. *Otolaryngol Head Neck Surg.* 1979;87:779–796.

177. Mitchell R, Armanini G. Cricopharyngeal myotomy: Treatment of dysphagia. *Ann Surg.* 1975;181:262–266.

178. Cherney LR, Halper AS. Recovery of oral nutrition after head injury in adults. *J Head Trauma Rehabil.* 1989;4:42.

179. Winstein CJ. Neurogenic dysphagia: Frequency, progression and outcome in adults following head injury. *Phys Ther.* 1983;12:1992.

180. Adour KK. Diagnosis and management of facial paralysis. *N Engl J Med.* 1982;307:348–351.

181. Yamada M, Hirano M, Ohkubo H. Recurrent laryngeal nerve paralysis: A 10-year review of 564 patients. *Auris Nasus Larynx*. 1983;10(Suppl):1–15.

182. Collet FJ. Surg un nouveau syndrome paralytique pharyngolarynge par blessure de guerre. *Lyon Med.* 1916;124:121–129.

183. Horsely J. Suture of the recurrent laryngeal nerve with reports of a case. *Trans South Surg Gynecol Assoc.* 1909;22:161.

184. Woodson GE, Miller RH. The timing of surgical intervention in vocal cord paralysis. *Otolaryngol Head Neck Surg.* 1981;89:264.

185. Holinger LD, Holinger PC, Holinger PH. Etiology of bilateral abductor vocal cord paralysis. *Ann Otol.* 1976;85:428.

186. Blackburn G, Salmon LF. Cord movements after thyroidectomy. *Br J Surg.* 1960;48:371.

187. Clark OH. Total thyroidectomy: The treatment of choice for patients with differentiated thyroid cancer. *Ann Surg.* 1982;196:361–370.

188. Clark OH. *Endocrine Surgery of the Thyroid and Parathyroid Glands.* St. Louis, Mo: Mosby; 1985.

189. Palmar ED. Disorders of the cricopharyngeus muscle: A review. *Gastroenterology*. 1976;71:510.

190. Peytz F, Rasmussen H, Buchtal F. Conduction time and velocity in human recurrent laryngeal nerve. *Dan Med Bull*. 1965;12:125–127.

191. Blair RL. Laryngeal electromyography: Techniques and application. *Otolaryngol Clin North Am*. 1978;11:2.

192. Simpson DM, Sternman D, Graves-Wright J, Sanders I. Vocal cord paralysis. Clinical and electrophysiologic features. *Muscle Nerve.* 1993;16:952–957.

193. Hiroto I, Hirano M, Tomita H. Electromyographic investigation of human vocal cord paralysis. *Am Otol Rhinol Laryngol.* 1968;77:246–304.

194. Simpson DM, Kaufmann H, Sanders I, Wolfe DE. Laryngeal dystonia in multiple system atrophy. *Muscle Nerve.* 1992;15:1213–1215.

195. Rodriguez A, Myers B, Ford C. Laryngeal electromyography in the diagnosis of laryngeal nerve injury. *Arch Phys Med Rehabil.* 1990;71:587–590.

196. Tanaka E, Palmer J, Siebens A. Bipolar suction electrodes for pharyngeal electromyography. *Dysphagia.* 1986;1:39–40.

197. Palmer JP, Holloway AM, Tanaka E. Detecting lower motor neuron dysfunction of the pharynx and larynx with electromyography. *Arch Phys Med Rehabil.* 1991;72:214–219.

198. Eckberg DL. Parasympathetic cardiovascular control in human disease. A critical review of methods and results. *Am J Physiol.* 1980;234:581–593.

199. Dedo HH, Izdebski K. Intermediate results of 306 recurrent laryngeal nerve sections for spastic dysphonia. *Laryngoscope.* 1983;93:9–15.

200. Crumley L. Repair of the recurrent laryngeal nerve. *Otolaryngol Clin North Am.* 1990;23:553–563.

201. Crumley L, Izdebski K, McMicken B. Nerve transfer versus teflon injection for vocal cord paralysis: A comparison. *Laryngoscope.* 1988;98:1200–1204.

202. Isshiki N, Morita H, Okamura H, et al. Thyroplasty, a new phono-surgical technique. *Acta Otolaryngol (Stockh).* 1974;78:451.

203. Johns ME, Cantrell RW. Voice restoration of the total laryngectomy patient: The Singer-Blom technique. *Otolaryngol Head Neck Surg.* 1981;89:82–86.

204. Johns ME. "How do I do it"—Head and neck. A targeted problem and its solution. The Panje button. *Laryngoscope.* 1982;92:204.

205. Singer MI. Voice rehabilitation after laryngectomy. In: Bailey BJ, ed. *Head and Neck Surgery: Otolaryngology.* Philadelphia, Pa: JB Lippincott; 1993: 1361–1372.

206. Kunc Z. Treatment of essential neuralgia of the IXth nerve by selective tractotomy. *J Neurosurg.* 1965;23:494.

207. Geschwind N. Nature of the decussated innervation of the sternocleidomastoid muscle. *Ann Neurol.* 1981;10:495.

208. DeToledo J, Smith DB, Kramer RE, Rosenbaum T, Stanulis R. Cortical innervation of the sternocleidomastoid in humans: Clinical and Neurophysiological Observations. *Ann Neurol.* 1989;26:171.

209. Patterson WR. Inferior dislocation of the distal end of the clavicle. *J Bone Joint Surg.* 1967;49A:1184.

210. Logigian EL. McInnes JM, Berger R, Busis NA, Lehrich JR: Stretch induced spinal accessory nerve palsy. *Muscle Nerve.* 1988;11:146–150.

211. Markey K, Di Benedetto M, Curl WW. Upper trunk brachial plexopathy: The stinger syndrome. *Am J Sports Med.* 1993;21:650–656.

212. Hoffman JC. Permanent paralysis of the accessory nerve after cannulation of the internal jugular vein. *Anesthesiology.* 1983;58:583.

213. Eisen A, Bertrand G. Isolated accessory nerve palsy of spontaneous origin. *Arch Neurol.* 1972;27:496–502.

214. Manon E, Espaillat R, Ruff RL. Dissociated weakness of sternocleidomastoid and trapezius muscles with lesions in the CNS. *Neurology.* 1989;38:796.

215. Mastaglia FL, Knezevic W, Thompson PD. Weakness of head turning in hemiplegia. *J Neurol Neurosurg Psychiatry.* 1986;49:195–197.

216. Mohanty SK, Barrios M, Fishbone H, Khatib R. Irreversible injury of cranial nerve IX–XII (Collet-Sicard Syndrome). *Neurosurgery.* 1973;38:86–88.

217. Anderson R, Flowers RS. Free grafts of the spinal accessory nerve during radical neck dissection. *Am J Surg.* 1969;118:796–799.

218. Di Benedetto M, Markey K. Electrodiagnostic localization of traumatic upper trunk brachial plexopathy. *Arch Phys Med Rehabil.* 1984;65:15–17.

219. Green RF, Brien M. Accessory nerve latency to the middla and lower trapezius. *Arch Phys Med Rehabil.* 1985;66:23–24.

220. Shankar K, Means KM. Accessory nerve conduction in neck dissection subjects. *Arch Phys Med Rehabil.* 1990;71:403–405.

221. Brodal A. *Neurological Anatomy in Relation to Clinical Medicine.* 3rd ed. New York: Oxford University Press; 1981: 453–459.

222. Mitchell SW. *Injuries of the Nerves and Their Consequences.* Philadelphia, Pa: JB Lippincott. 1872: 335.

223. Alrich EM, Baker GS. Injuries of the hypoglossal nerve. Report of ten cases. *Mil Surg.* 1948;103:20–25.

224. Keane JR, Baloh RW. Post-traumatic cranial neuropathies. *Neurologic Clin.* 1992;10:864.

225. Jgnelzi RJ, Bucy PC. Intracranial hypoglossal neurofibroma. *J Neurosurg.* 1967;26:352–356.

226. Berger PS, Bataini JP. Radiation induced cranial nerve palsy. *Cancer.* 1977;40:152–155.

227. Jabbari B, Coker SB. Paroxysmal rhythmic lingual movements and chronic epilepsy. *Neurology.* 1981;31: 1364–1367.

228. Troupin AS, Kamm RF. Lingual myoclonus: Case report and review. *Dis Nerve Syst.* 1974;35:378.

229. Brazis PW. The localization of lesions affecting cranial nerve XII (the hypoglossal nerve). In: Brazis PW, Masdeu JC, Biller J, eds. *Localization in Clinical Neurology*. 2nd ed. Boston, Mass: Little, Brown; 1990: 263.

230. Riggs JE. Distinguishing between extrinsic and intrinsic tongue muscle weakness in unilateral hypoglossal palsy. *Neurology.* 1984;34:1367.

231. Redmond MD, Di Benedetto M. Hypoglossal nerve conduction. *Muscle Nerve.* 1988;11:447–452.

Chapter 8

REHABILITATION OF THE COMBATANT WITH MUSCULOSKELETAL DISORDERS

JOEL M. PRESS, M.D.[*]; STANLEY A. HERRING, M.D.[†]; AND W. BEN KIBLER, M.D.[‡]

[*]*Assistant Clinical Professor of Physical Medicine and Rehabilitation, Northwestern University Medical School, Chicago, Illinios; Director, Sports rehabilitation Program, Rehabilitation Institute of Chicago, Chicago, Illinios.*

[†]*Clinical Associate Professor, Departmant of Rehabilitation Medicine, Clinical Associate Professor, Department of Orthopedics, University of Washington, Seattle, Washington; Puget Sound Sport and Spine Physicians, Seattle, Washington.*

[‡]*Clinical Associate Professor, Department of Physical Medicine and Rehabilitation, Clinical Associate Professor, Department of Family Practice, University of Kentucky, Lexington, Kentucky; Medical Director, Lexington Clinic Sports Medicine Ceenter, Lexington, Kentucky.*

INTRODUCTION

Musculoskeletal injuries are commonly encountered in the physiatrist's office. Most of these injuries occur as a result of occupational or sports related endeavors. Many of the rehabilitation principles and techniques for these disorders, as well as the overall approach, are also applicable to the injured soldier. In times of war, musculoskeletal injuries similar to those seen in civilian populations can and do occur, often at extremely high rates. General Pershing stated in 1918 during World War I that he wanted no more men with "flat feet, weak backs, and lack of muscular development" sent overseas until they had received special training to restore them to normal.[1] An aggressive, nonsurgical rehabilitative approach to these musculoskeletal ailments is essential in order to decrease injury time, to prevent long term disability, and to reconstitute our forces. The focus of this chapter will be to outline and discuss the pathophysiology of musculoskeletal injuries; the acute, subacute, and chronic management of musculoskeletal disorders; and the specifics of rehabilitation of these disorders as they particularly relate to wartime management.

PATHOPHYSIOLOGY OF MUSCULOSKELETAL INJURIES

In order to guide specific rehabilitation of a given disorder, some basic knowledge about injury and repair of damaged tissue is essential. In the military setting, musculoskeletal injuries may occur due to one of three mechanisms: (1) soft tissue failure, (2) overload, and (3) direct trauma. Musculoskeletal soft tissues include tendon, ligament, muscle, bursae, bone, and nerve. Each of these tissues has a specific function, mechanism of injury, and healing response to a given injury. We will first describe the response of specific tissues to injury and then detail in depth overload injuries. Direct trauma will be discussed as indicated under specific injuries and their rehabilitation. Bone and nerve injuries will be discussed in other chapters and, therefore, not described in this chapter.

Tendon Injuries

The primary function of tendons is to transmit muscle force to the skeletal system with limited elongation. Tendon tissue is composed of dense fibers of connective tissue with very high tensile strengths in which the fibers are arranged parallel to each other in the direction of the tensile force of the muscle.[2] A tendon is most likely to be injured when (*a*) tension is applied quickly, (*b*) tension is applied obliquely, (*c*) the tendon is under tension before loading, (*d*) the attached muscle is maximally innervated, (*e*) the muscle group is stretched by exterior stimuli, or (*f*) the tendon is weak in comparison with the muscle.[3,4] These are conditions that surface often during wartime, and in particular, during vigorous training or combat situations. Clinically, tendon injuries, especially tendinitis, are quite common, particularly those of the supraspinatus and bicep tendons used in overhead activities, patellar, iliotibial band (ITB), and Achilles tendinitises used in lower extremity activities.

Tendon healing after injury occurs in three phases (Table 8-1). The first phase is inflammatory, which occurs in the first 48 to 72 hours and is highlighted by influx of vasoactive substances, chemotactic factors, and degradative enzymes.[5] Details of the inflammatory reaction can be found elsewhere.[5–9] This phase is important in the healing process of injured tissue. Nonsteroidal antiinflammatory drugs (NSAIDs) are often used acutely in the treatment of soft tissue injuries. Carlstedt and associates[10,11] have found that in animal models, treatment with indomethacin increased the tensile strength in healing tendons possibly by increasing the cross linkage of collagen molecules.

The second phase is a reparative, or collagen production, stage. This phase starts within the first week and is characterized by collagen proliferation produced by newly infiltrated fibroblasts and cel-

TABLE 8-1

PHASES OF TENDON HEALING

Phase	Time	Predominant feature
Inflammatory	First 48–72 h	Acute inflammatory reaction
Reparative	72 h–3 wk	Collagen proliferation phase
Maturation	3+ wk	Maturation and remodeling

lular fibrin matrix. During this phase, collagen fibrils are laid down in a random pattern, and thus possess little strength.[2]

During the final phase of healing—maturation or remodeling—the mechanical strength of the healing tendon continues to increase because of remodeling and organization of the fiber architecture along the direction of muscle force. Unless specific stresses are placed upon the healing tissue, newly produced collagen will become useless scar tissue.[12–14] Such stresses can be accomplished even in the acute setting with continuous passive motion machines, or bracing, which allows some motion in one plane, that is, knee orthosis with free, or even limited, range of flexion and extension available.

Chronic repetitive microtrauma can advance beyond the state of inflammation and tendinitis to a condition of degenerative change and cell damage; this is the major component of the pathologic picture termed *tendinosis*.[15] Some common examples of clinical musculoskeletal injuries, where cell degeneration is more prevalent than inflammation, include elbow *epicondylitis* [16] plantar fasciitis,[17] patellar *tendinitis,* [18] and Achilles *tendinitis*.[16] The clinical manifestations of tendinosis are the result of a degenerative process, rather than an acute event.[19] The adaptive changes of the musculoskeletal system that occur can be clinically detected both locally and at the site of symptoms or injury, and distally in other links in the kinetic chain.[19]

The distinction between tendinitis and tendinosis has ramifications in the rehabilitation process. Antiinflammatory medications (to be discussed later) will have a greater role in tendinitis than they will in tendinosis. Local changes due to chronic tendon injury, that is, tendinosis, are usually clinically manifested as any or all of the following: (*a*) inflexibility in the involved muscle-tendon group, (*b*) weakness in the involved muscle or surrounding muscle, or (*c*) muscle strength imbalance between force generator and force regulator in the force couple.[19] All of these implications focus the goal of rehabilitation away from relief of the symptoms of the "itis," and toward restoration of function that is lost with the "osis."[1]

Ligament Injuries

Skeletal ligaments are highly specialized, dynamic, dense connective tissues that connect bones.[2] Ligaments function both as passive mechanical structures in stabilizing joints, and as neurosensory structures for providing proprioception to muscles and joints. Ligament injuries can occur by contact or noncontact mechanisms.[20] In either case the injury is often the result of a large force, often suddenly and rapidly applied, placed on a given ligament. These injuries result from acute overload at the insertion interface. Details on healing injured ligaments is reviewed elsewhere.[21] In general, however, the same phases of healing as described for tendon repair occur. Elastin fibrils, which are the most prominent component of ligaments and give ligaments their tensile strength, are stimulated to proliferate with stretching. Ligamentous tissue that is immobilized has poor tensile strength.[22,23] Emphasis on early motion and prevention of long term immobilization in the rehabilitation process will allow for greater ultimate strength of the healed ligament. Nevertheless, although the quantity of ligament may remain quite good after injury and healing, the quality of that tissue is never as good as it was preinjury.

Ligament injuries are the most common injuries to joints, most particularly to the knee; in most studies, they account for 25% to 40% of all knee injuries.[24,25] These injuries will be discussed in more detail in the section describing specific knee injuries. With the rehabilitation of ligament injuries, a point to keep in mind is that some studies have shown that increased activity level has a beneficial effect on the strength properties of bone–ligament complexes.[26,27] It stands to reason that the same effect of exercise may also result with bone-tendon complexes.[2]

Muscle Injuries

Muscle injuries can occur from a variety of mechanisms. Frequently muscles are injured during eccentric contraction, or activation of the muscle while it is being lengthened by an opposing force greater than the force in the muscle.[2] Therefore, strengthening muscles in both a concentric and eccentric mode is initially essential both in the prevention of muscle injuries, and postinjury during rehabilitation to prevent recurrent injury. Failure often occurs at the myotendinous junction.[28] Muscle tissue damage triggers an initial inflammatory phase followed by subsequent phases of tissue healing, repair, and remodeling, similar to that stated above for tendon injuries. If enough tissue damage occurs, clinical symptoms and signs (pain, swelling, and discoloration) develop.[29] In the overt or subclinical type of muscle injury, the tissue may repair and remodel, but concomitant changes in muscle function, that is, strength, strength balance (agonist vs antagonist), flexibility, and proprioception occur.[30] The signs and symptoms of injury

abate, but these functional deficits persist. The same functional changes may occur if there is a subclinical muscle injury. Other injuries to muscle can occur from lacerations and direct trauma or contusion. Such injuries can diminish muscle strength, limit joint motion, and lead to myositis ossificans. These types of injuries are quite common in the athletic and working populations and are often a cause of lost time from activity. The mechanism, pathophysiology, and location of muscle injury are well-described in other sources.[28,31–33]

Two important areas to consider in the rehabilitation of muscle injuries are (1) the effects of immobilization and (2) the effect of stretching and warm up. Muscle strength and loss of strength secondary to immobility is well recognized and described.[34,35] Loss of strength can delay the rehabilitation process and the injured soldier's return to active duty. In a study[36] using rabbits, it has been shown that muscle immobilized in a shortened position developed less force and stretched to a shorter length before tearing than did the nonimmobilized contralateral control muscle. Muscle immobilized in a lengthened position exhibited more force and needed more change in length to tear than in nonimmobilized controls. Therefore, when immobilization is necessary for any period of time, it should be done with the muscle in a lengthened, or at least a neutral, position. Secondly, a warm up or conditioning period has been shown to be effective in altering the biomechanical properties of muscle in a way that may be effective in avoiding injury.[37] A flexibility training program may have a beneficial effect on reducing the severity and cost of treating joint injuries.[38]

Bursae Injuries

Bursae are sacs formed by two layers of synovial tissue that are located at sites of friction between tendon and bone (pes anserinus bursa) or skin and bone (prepatellar, olecranon bursae). In their normal state, they contain a thin layer of synovial-like fluid and may actually communicate with an adjacent synovial sac (suprapatellar bursa). Bursae are typically injured with overuse, and repetitive trauma types of activities that cause either friction of the overlying tendon or external pressure. They may also become inflamed from degeneration and calcification of an overlying tendon, which leads to a chemical bursitis, as in subacromial bursitis secondary to calcific supraspinatus tendinitis.[3] When injured, the bursa will become inflamed, with resultant effusion and thickening of the bursal wall.

OVERVIEW OF REHABILITATION PRINCIPLES

The background for the rehabilitation of musculoskeletal disorders has been described in terms of the pathophysiology of musculoskeletal injuries. There are certain general principles that apply to the rehabilitation of musculoskeletal disorders. The rehabilitation plans for the injured soldier with a musculoskeletal disorder must be oriented toward restoration to function, not just relief of symptoms. The five goals of such a rehabilitation plan can be stated as (1) establishment of an accurate diagnosis, (2) minimization of deleterious local effects of the acute injury, (3) allowance for proper healing, (4) maintenance of other components of general fitness, and (5) return to normal combat function.

Establishment of Accurate Diagnosis

Without a precise diagnosis, thorough rehabilitation is not possible. Because of the varied presentation of symptoms, recognition is sometimes delayed. Even if recognition of the injury is made, the need for a complete and accurate diagnosis is still present. Symptoms of musculoskeletal injuries are usually pain, swelling, anatomical deformity, or decreased military performance. The symptoms or dysfunction that the soldier is experiencing may be directly related to the musculoskeletal injury, or may be a distant manifestation of a musculoskeletal injury in another part of the kinetic chain that accomplishes the activity.

Complete and accurate diagnosis of the injury can be established by identifying which of the five components of the musculoskeletal injury complex are present in each injury. In each injury, there are five separate areas that may be identified as contributing to the production or continuation of symptoms.[39] These components have an effect on either musculoskeletal anatomy or musculoskeletal functions; they are (1) *tissue complex injury* (the area of actual anatomical disruption); (2) *clinical symptom complex* (that group of symptoms that are causing acute pain, swelling, and dysfunction); (3) *functional biomechanical deficit* (the combination of muscle inflexibilities, weakness, and imbalance that causes inefficient mechanics); (4) *functional adaptation complex* (the functional substitutions that the soldier employs as a result of the injury in order to maintain performance); and (5) *tissue overload complex*

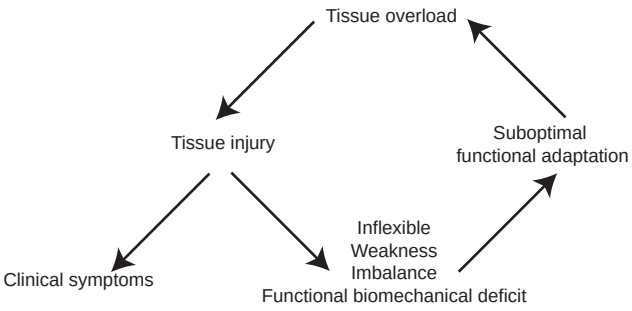


Fig. 8-1. Vicious overload cycle. Source: Kibler WB, Chandler TJ, Pace BK. Principles for rehabilitation after chronic tendon injuries. In: Renström PAFH, Leadbetter WB, eds. Tendinitis I: Basic concepts. *Clin Sports Med.* 1992; 11(3):663.

(that group of tissues that may be subject to tensile or eccentric overloads, which may cause or continue symptoms or disability) Figure 8-1.

These components are actually parts of a negative feedback loop, or vicious cycle, that is operative in muscle and musculoskeletal injuries (see Figure 8-1).[19] Depending on the soldier's intensity or duration of continued use, cycling within the loop may continue for varied periods of time before actual clinical symptoms are manifest. During this time, the soldier's function may be fairly normal, but his efficiency may not be optimal. A thorough evaluation of each soldier with respect to inflexibilities, weaknesses, or imbalances will demonstrate the deficits and allow the beginning of diagnostic and therapeutic processes. Specific diagnostic evaluation will guide specific rehabilitation, that is, anatomical diagnosis and diagnosis of functional deficits must be made. This will allow a holistic approach to the total effect an overload injury has on the entire kinetic chain and the total function of the soldier, and will guide rehabilitation back to normal function.

Minimization of Deleterious Local Effects of the Acute Injury

The initial steps in minimizing deleterious local effects of the acute injury are the control of inflammation and pain. Cryotherapy (the use of ice or other methods of cold application) decreases arteriolar and capillary blood flow and muscle spasm, and is applied to control edema and reduce pain.[40–49] Ice can be applied in the form of crushed ice in a plastic bag, an iced immersion tub, or an ice massage.[43,46,50–52] Limiting the initial development of joint effusion will speed the recovery process.[53] The length of time the cryotherapy must be administered to an injured muscle is directly dependent on the depth of overlying fat; it may vary from 10 to 30 minutes.[54,55] Ice and frozen gel may provide more consistent and longer duration cooling.[56] As with any modality, care must be employed to avoid complications such as burns to anesthetic areas or injury to superficial nerves.[57]

Compression must be concomitant with ice and elevation of the injured area. Ace bandages or other forms of local compression dressings will decrease the degree of the acute inflammatory response and the overdistension of soft tissues due to hemorrhage and exudate. Similarly, no weight bearing, or decreased weight bearing, may initially be important to decrease the degree of inflammatory response in lower extremity injuries. Crutches, canes, and walkers may be helpful in this manner. Likewise, the use of a sling or splint may be necessary for upper extremity injuries.

Early judicious use of antiinflammatory medications and pain medications may greatly speed recovery. The antiprostaglandin effect of NSAIDs has some potential benefit during the acute phase of musculoskeletal injuries. The drugs may minimize the local side effects of the injury by limiting the extent of the inflammatory response, as well as providing pain relief. The duration of the analgesic effect of the NSAID may be different from that of the antiinflammatory effect.[58] Acetylsalicylate may need to be avoided during the early phases of injury because its antiplatelet effects persist for the life of the platelets and may increase hemorrhage. NSAIDs also have antiplatelet effects; however, those are dose related.[14,59] Early use of NSAIDs in acute ankle inversion injuries has been shown to have no deleterious effect on the mechanical integrity of the healing tissue, and in fact, was shown to speed rehabilitation.[60] During the early inflammatory phase, potent glucocorticoids must be used cautiously, because their powerful antiinflammatory effect may inhibit the normal healing process and thereby prolong rehabilitation.[14,61–63] After 1 to 2 weeks, during the proliferative phases of healing, glucocorticoids may be helpful to reduce ongoing inflammation and edema. Glucocorticoids can be safely given orally in commercially available, tapering dose packets, or in a daily dose of prednisone, starting with 70 mg and decreasing by 10 mg per day for 7 days. When using prepackaged dose packets, the physician should evaluate for an adequate amount of corticosteroid in the preparations in order to get a good antiinflammatory response. Glucocorticoids should be used in situations where immediate an-

tiinflammatory response is critical, such as an acute combat situation or when standard antiinflammatory medications are not working. Ten to 20 mg of a corticosteroid (triamcinolone or equivalent) mixed with a short acting anesthetic can be injected into areas of inflammation, yielding a strong antiinflammatory response. Injection of corticosteroids into tendons should be avoided because of the risk of tendon rupture.[61,62] However, injection into tendon sheaths, bursae, or inflamed joints can rapidly decrease inflammation and give substantial pain relief. Some commonly injected areas are:

- Subacromial bursae.
- Lateral or medial epidondyle.
- Tendon sheath of abductor pollicis longus (APL), extensor pollicis brevis (EPB) (de Quervains tenosynovitis).
- Carpal tunnel.
- Greater trochanter.
- Knee joint (intrarticular).
- Plantar fascia.

Opiate and nonopiate analgesic medication can be very helpful in the acute phases of musculoskeletal injuries. To produce an analgesic response, it is important to properly administer the dose of medication, to give it at scheduled times, and to administer it for a predetermined length of time, such as 3 days or 1 week. Too often, inadequate doses of medication are given for fear of making the patient drug dependent.

The use of other therapeutic modalities besides ice can play a large role in the acute management of musculoskeletal disorders. Electrical galvanic stimulation can be very useful for reduction of edema.[57] Higher pulse rates of galvanic stimulation that will produce a tetanic muscle contraction can also be used for pain control. Transcutaneous electrical nerve stimulation (TENS) is helpful for acute pain problems.[57] In fact, TENS units have found the greatest utility in the treatment of acute, painful conditions. Therapeutic modalities, although of significant usefulness in the treatment of acute musculoskeletal disorders, probably have limited usefulness and availability in a combat situation. Occasionally, portable TENS units may be easily transported to the combat zone.

Allowance for Proper Healing

For proper healing to take place, immobilization for specific periods of time may occasionally be necessary. Immobilization may be accomplished by taping, bracing, padding, or casting. Prefabricated joint immobilizers and splints are quite useful (Figure 8-2). Slings, cervical collars, and back braces may also be helpful. The period of immobilization or protection of the injured structure will be individualized to fit the nature and extent of the injury. In general, however, gentle mobilization should be initiated even within the first 24 hours after an acute injury and increased as pain and swelling diminish.

Early motion and exercise are essential to proper rehabilitation of musculoskeletal disorders and to speed the healing process.[64–67] Collagen fiber growth and realignment can be stimulated by early tensile loading of muscle, tendon, and ligament.[68] The formation of adhesions between repairing tissue and adjacent structures can be limited by early motion.[69] Proprioception is better maintained and recovers faster with early motion.[68] Optimal conditions for healing depend on a very fine balance between protection from stress and return toward normal function at the earliest possible time.[64,70]

Regaining flexibility allows for proper healing. Decreased joint flexibility can result from muscle spasm, pain and resulting neural inhibition, connective tissue adhesions and contracture, or intraarticular blockade. Treatment of the decreased flexibility is predicated on the specific cause. Muscle spasm is often alleviated with cryotherapy and elec-

Fig. 8-2. Prefabricated joint immobilizers and splints for the knee and thumb or wrist.

trical stimulation. Pain and its consequent neural inhibition can be relieved with electrical stimulation modalities, soft tissue mobilization, and massage. Soft tissue and connective tissue changes can be alleviated with static stretch and proprioceptive neuromuscular facilitation (PNF) techniques.[71–73] Intraarticular blockade may be secondary to intraarticular fibrosis or mechanical blocking from such entities as a torn meniscus. Occasionally, increased flexibility can be obtained with intraarticular injection of a local anesthetic. Other times, surgery may be necessary.

Range-of-motion programs may be classified as passive, active, and active assistive. During the first 3 to 5 days after an injury, passive techniques are used while inflammation and edema are quite significant. The early controlled motion helps to decrease soft tissue edema and neural inhibition of tissues. Static stretch and PNF techniques (such as contract relax, the contraction of contralateral extremity, and contraction of antagonistic muscle) can all be effective in improving flexibility.[73–75]

Active assistive programs allow active motion to begin with the assistance of either another (ie, a therapist) or with devices (such as pulleys). The purpose of these activities is to enhance further mobilization of the injured tissues as active muscle firing starts to occur. Gradually these techniques are upgraded to include more active participation on the part of the injured soldier until he is completely active throughout the entire range-of-motion. Once full joint range-of-motion is attained, the injured soldier can proceed to a full flexibility program. Often the subjective complaints of the patient will determine the degree to which he progresses through the phases of mobilization.

The important points in restoring joint and soft tissue extensibility are to avoid overstressing healing tissue, and to recognize the effect of joint instability. It is essential to avoid excessive biomechanically induced stress on the healing structures while mobilizing and strengthening tissues appropriately. For example, an acutely injured and surgically repaired anterior cruciate ligament (ACL) should be mobilized as soon as possible to avoid articular degeneration. However, too rapid mobilization and early stressing of the extensor mechanism may hamper rehabilitation and proper tissue healing. Vigorous mobilization also needs to be avoided in situations where joint instability exists. For example, with acute anterior glenohumeral dislocations, it is often advantageous to avoid abduction and external rotation for the first 4 to 6 weeks after injury to allow some soft tissue healing to occur in the anterior glenohumeral joint. Later, flexibility techniques may be beneficial to avoid excess loss of external rotation of the shoulder.

The effects of immobilization on muscle is well documented.[76–78] There may be up to a 20% decrease of muscle strength after 1 week of muscle immobilization and another 20% decline in residual strength every subsequent week of immobilization.[79] Atrophy may occur even faster if the muscle is immobilized in a shortened position.[80] Type I fibers are particularly affected by immobilization, with up to a 47% decrease by the fifth week.[81–83]

The side effects from immobilization extend beyond the muscle. If an associated joint is excessively immobilized, significant joint capsule, cartilage, subchondral bone, and bone-ligament changes occur.[22] Protracted rehabilitation (greater than 1 year) may be necessary to regain function of the structures.[84] After 8 weeks of immobilization, some tissues, such as articular cartilage, may never completely return to normal.[80,85]

Once the joint and soft tissue extensibility has been attained, the injured soldier is ready to start strength training. Strength training can be started even when full range-of-motion has not been attained. However, any strength gains will be specific only for that range-of-motion. Strengthening programs are divided into isomet rics, manual resistance, isotonics, and isokinetics. These programs can be divided into concentric and eccentric contractions. Details of strength training can be found elsewhere.[86,87] The goal of all strengthening exercises is hypertrophy of muscle and the enhancement of recruitment, and firing of the motor units.[86]

The most important aspects of any strength program are specificity and overload. Muscles are activated for activities in a dynamic way with alteration of concentric and eccentric contractions. Training muscles with static contractions (isometrics) or against a set amount of resistance (isotonics) may improve the overall strength of the muscle group, but may not be transferable to specific activities in the combat field.[88–90] If hypertrophy is to occur, muscles must be subject to loads greater than the usual stresses of daily activity. The goal of a resistance program for increased strength is to overload, not overwhelm, the muscles.[87] Overloading the muscle too rapidly will result in reactive inflammation changes and associated synovitis. The four basic factors in overload are (1) intensity, (2) volume, (3) duration, and

(4) rest. Details of strength training are discussed elsewhere.[86,91–93]

Isometric exercises are used early in the acute injury phase. In isometric exercises, no joint motion occurs, therefore, strengthening can occur concomitantly with joint protection. This type of contraction helps maintain muscle tone and a pattern of contraction. Isometric contractions should be held for at least 6 seconds, with rest periods between 10 and 20 seconds to ensure proper muscle blood flow and to remove substrate of muscular contraction.[73] The isometric contractions should be carried out frequently during the day, utilizing sets of 10 to 20 repetitions.[88] Except in acute musculoskeletal injury rehabilitation, isometric exercises are not routinely used for strength training because of their ability to strengthen muscles only at one point of the range-of-motion.

Because of cross education of the neuromuscular system, exercise of the contralateral side is also important early after an injury. After exercising the contralateral side of the body, the immobilized (nonexercised) side has demonstrated strength increases of up to 30%.[94] Manual resistance exercises are begun as soon as the joint can be safely moved without threat of further injury. The therapist manually resists whatever effort the patient is able to exert. Contractions should be carried out in a pain-free range-of-motion.[95]

Since most strength training is done in a dynamic manner, isotonic and isokinetic exercises are integral in a proper rehabilitation program for musculoskeletal disorders. Sophisticated isokinetic equipment, which allows the control of speed while maintaining a constant force, will probably not be available in the combat zone.[96,97] However, isotonic exercises, where a constant resistance is applied, can be accomplished with free weights, sand bags, cans of food, water bottles, or whatever else is available in the field.

Maintenance of Other Components of General Fitness

Once an accurate diagnosis has been made, the deleterious local effects of tissue injury have been minimized, and allowances have been made for proper healing to occur, then the maintenance of other aspects of fitness need to be addressed. For complete rehabilitation to occur, changes in different parts of the kinetic chain after a musculoskeletal injury need to be dealt with. In the rehabilitation of wrist or elbow injuries, shoulders must be strengthened, because the shoulder is the primary stabilizer of the upper limb for distal joint functioning. Hip strength and flexibility may be altered because an ankle injury has caused modification in the gait cycle and resultant proximal limb substitution patterns. A prescription for substitute exercise to maintain general cardiovascular fitness, as well as general strength, will help decrease total rehabilitation time. Along with absolute strength gains, improvement of muscular endurance needs to be addressed. Gaining muscle endurance entails stressing the aerobic pathways to improve the oxidative enzyme capacity of slow-twitch muscle fibers.[98] High repetition, low-resistance exercises, which require greater degrees of muscle endurance, should be integrated into the rehabilitation program. The use of a stationary bicycle, cross-country ski machine, or rowing machine can all increase muscle endurance and are portable enough to be available in a combat hospital. In the field, aerobic conditioning may be accomplished by jumping rope, wind sprints, running hills, step climbing, and swimming, when water is available and safe.

Return to Normal Military Duty

After adequate flexibility, strength, endurance, and cardiovascular fitness are attained, the injured soldier is then ready to begin specific training or retraining in the development of biomechanical and neurophysiologic skill patterns for the specific activities he will need to perform. The neurophysiologic learning process for developing coordinated skill patterns is based on constant repetition, with focus on perfecting the movement.[73,99,100]

Criteria for return to active duty should include resolution of the tissue injury and clinical symptom complex, functional range-of-motion and adequate muscle strength, and ability to perform specific military duty activities. This usually occurs within 10 to 14 days for simple injuries, but can take up to 6 weeks for more severe injuries or for injuries that develop complications, such as myositis ossificans. Long-standing musculoskeletal problems may result in functional biomechanical deficits and concomitant substitution activity patterns. In these patients, the major focus of rehabilitation must extend well beyond symptom relief. If the functional biomechanical deficits and resultant activity patterns are not addressed, performance drop-off, recurrent injury, or both will occur.[29]

PRINCIPLES OF REHABILITATION FOR SPECIFIC DISORDERS

Cervicothoracic Disorders

Cervical Soft Tissue Injuries

Soft tissue injuries in the cervical region are usually classified with minimal precision. A sprain is an injury to a joint, with possible rupture of some of the ligaments or tendons, but without dislocation or fracture, and occurs from stretching of the supportive soft tissue. A cervical strain is an overload injury to the muscle-tendon unit in the cervical spine caused by excessive force, rotation, or eccentric loading. Most neck muscles do not terminate in tendons, but attach to bone by myofascial tissue that blends into the periosteum. Other cervical soft tissues include the sympathetic nervous system chain, the greater occipital nerve, the vertebral artery, and the interspinous and supraspinous ligaments. In the evaluation of cervical injuries, the most important conditions to rule out are bony lesions, that is, fractures or ligamentous instabilities. In acute severe trauma, plain radiographs of the cervical spine, including anteroposterior, lateral, obliques, and open mouth views, are essential when evaluating acute severe trauma cases for fractures or instability. Often flexion-extension views will also be necessary. On the battlefield, any soldier with severe neck pain or any neurological signs after an acute cervical injury must be assumed to have a fracture or instability until proven otherwise. This situation may require evacuating the soldier to a local hospital for radiographs under spinal precautions, that is, on a spine board with head and neck immobilized.

Method of presentation. A soft tissue injury usually presents as acute traumatic injury secondary to sudden jerking motion of the head and neck, or chronic overload from maintenance of one position of the cervical spine for prolonged periods of time, most often with poor posture.

Tissue injury complex. The tissue injury complex will include supraspinal and interspinal ligaments, cervical paraspinal or anterior cervical musculature (scalenes, sternocleidomastoid, trapezius, erector spinae, levator scapulae).

Clinical symptom complex. This complex presents as neck pain without radiation, exacerbated with movement and resistive motion testing, and relieved by rest and immobility.

Functional biomechanical deficit. With this, the manifestations are loss in the cervical spine of full flexion and rotation or lateral rotation, or both. There is loss of the normal coupled motions in the cervical spine with substitution patterns of movement.

Functional adaptation complex. Here, a clinician will observe marked restriction in cervical range-of-motion with most significant changes at specific segmental levels.

Tissue overload complex. This refers to the specific ligaments or muscles injured.

Rehabilitation (Table 8-2)**.** Early treatment involves control of the inflammatory process as discussed previously. Details of the specific parameters for the use of therapeutic modalities is beyond the scope of this chapter, but can be found in the referenced texts.[52,57] Judicious use of oral corticosteroids may be very helpful. Rarely, an injection of corticosteroid may be indicated in an involved and specifically identified cervical spine or ligamentous structure.[101] A soft cervical collar can improve comfort, assuming no cervical spine instability exists. The wearing of cervical collars should be sharply tapered to avoid dependence on them and prevent atrophy from prolonged use. Total wear of the collar probably should not exceed 10 to 14 days, and weaning should allow increasing daytime removal, with continued use at night to prevent injury during sleep.[102] Occasionally, when only one or two specific myofascial trigger points are present, injection with lidocaine or dry needling, or spray and stretch techniques, may be of benefit for reduction of local pain symptoms.[103,104] When multiple points are

TABLE 8-2

TREATMENT FOR CERVICAL SOFT TISSUE INJURIES

Time (d)	Treatment
0–3	NSAIDs, pain medication, oral corticosteroid
0–10	Soft cervical collar
3–7	ROM exercises initiated, isometric strengthening
10–21	Advanced strengthening and flexibility program

NSAID: nonsteroidal antiinflammatory drug
ROM: range-of-motion

Fig. 8-3. Passive neck stretches for cervical range of motion and flexibility.

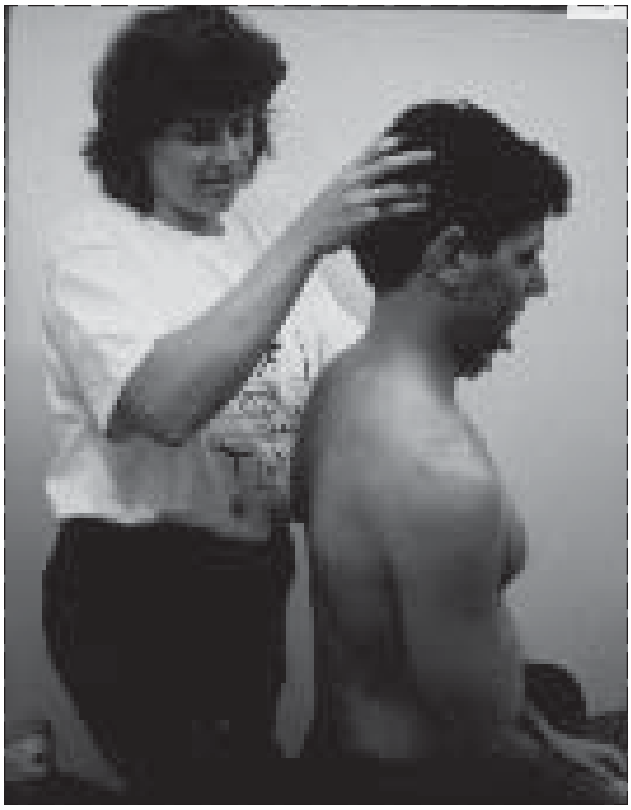


Fig. 8-4. Dorsal cervical glides to improve posture. The patient is instructed to tuck the chin to the back of the throat, not to the chest.

present, or symptoms are not well localized to specific, classical trigger points, success is quite variable with these techniques.

Once the acute phase passes (which should be within 3 to 7 days), gentle range-of-motion can begin. Dorsal glides and passive neck stretches are helpful (Figures 8-3, 8-4). Spinal manipulative therapy may be useful during the pain control phase and useful to improve segmental motion.[105,106] Manipulative therapy must be done with some caution, because of potential complications.[107] With sprain injuries, where there is a disruption or stretching of ligaments, traction should be avoided, because it can further stretch damaged ligaments and exacerbate symptoms.

Neck strengthening is an integral part of any cervical spine rehabilitation program. Initially, isometrics should be done with the head in midline only (the neutral position), and resisting forces should be applied perpendicularly to the head from every position. Very slowly, with strengthening in the midline, the head can be taken out of midline after there is no pain. Extremes of head flexion, either anteriorly, posteriorly, or laterally against resistance, are seldom indicated.[108] Of great importance in the overall cervical spine rehabilitation program is shoulder girdle stabilizer strength.[101] This includes the scapular protractors and retractors, as well as truncal/torso stabilizers. These are important to be able to maintain appropriate postural ergonomics by eliminating the head-forward posture associated with a stooped shoulder alignment (Figures 8-5, 8-6).[101]

Following initial cervical isometric exercises, progression to total upper-body isotonic exercises will improve strength and stability of the entire upper torso. Reinforcement of proper posture ergonomics, specifically including cervicothoracic and pectoral girdle posture mechanics, is imperative.[101]

Minor cervical soft tissue injuries should be resolved within 7 to 10 days. More severe injuries may take up to 3 to 4 weeks to resolve. When severe ligamentous injuries cause spinal instability, prolonged treatment with immobilization (up to 6 months), and possibly surgery, may be necessary.

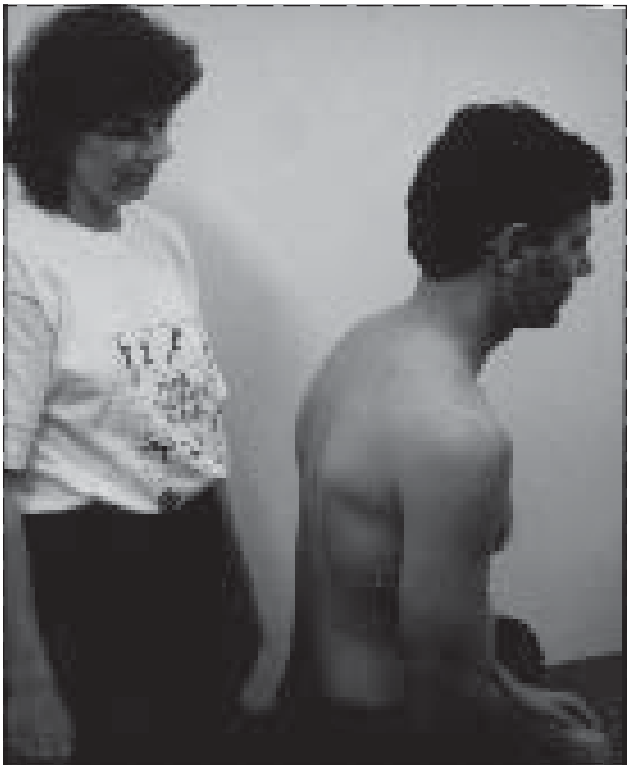


Fig. 8-5. Stooped posture with the head forward.

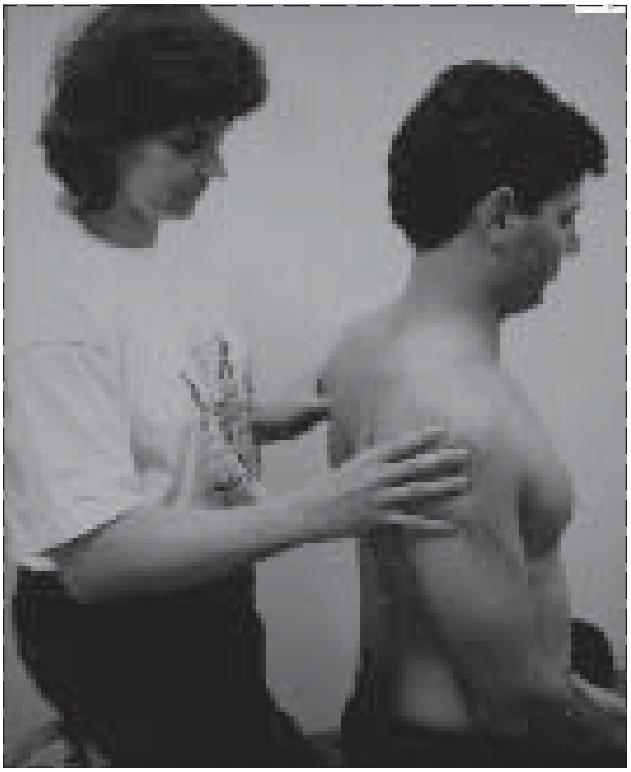


Fig. 8-6. Strengthening exercises for scapular retractors/stabilizers. The patient is instructed to squeeze the shoulder blades together with the chin tucked.

Cervical Radiculopathy

Radiculopathy in the cervical area is often the result of chronic stress to the bony, ligamentous, and muscular elements of the cervical spine. Symptoms may be quite subtle and are described as aching, dull, or diffuse in nature. More commonly, symptoms are sharp, piercing, and electric-shock–like with radiation into a specific dermatome of the upper extremity. The most important similar diagnoses to distinguish from cervical radiculopathy are (*a*) peripheral nerve entrapments, such as carpal tunnel syndrome (or high median neuropathy) vs C-6 radiculopathy; (*b*) ulnar neuropathy vs C-8 radiculopathy; (*c*) shoulder and hand tendinitises (ie, rotator cuff pathology) vs C-5 radiculopathy; or (*d*) de Quervain's and extensor tendinitis of the wrist vs C-6 or C-7 radiculopathy. Whenever symptoms of cervical radiculopathy (especially weakness) are progressive, or bowel or bladder dysfunction occur, urgent referral to either a neurosurgeon or orthopedic surgeon is mandatory.

Method of presentation. Radiculopathy may present as acute traumatic injury, or more commonly, chronic overload injury often related to repetitive activities stressing cervical spine musculature.

Tissue injury complex. Disruption of the annulus fibrosis with herniation of nucleus pulposus will cause a mechanical or chemical radiculitis, usually at the level of the foraminal canal or foraminal disease, or both caused by bony degenerative changes or other sources.

Clinical symptom complex. Arm pain or numbness and tingling will usually present in a dermatomal distribution, and be worse with lateral flexion and extension to the ipsilateral side (Spurling's maneuver, Figure 8-7). Also evident may be coughing and sneezing, focal weakness, sensory loss, and diminution of muscle stretch reflexes in a dermatomal distribution.

Functional biomechanical deficit. There will be altered weight distribution across the intervertebral disk. If this occurs gradually, loss of flexion, exten-

Fig. 8-7. Spurling's maneuver-axial compression applied to the cervical spine in a side bent and rotated position to close the neuroforamina and reproduce symptoms in a dermatomal distribution.

sion, and lateral rotation and bending motions are obvious, with segmental motion dysfunction.

Functional adaptation complex. Here will be observed the loss of normal coupled motion with lateral flexion and rotation of the cervical spine, abnormal segmental motion patterns (hyper- or hypomobility), and hunched forward posture.

Tissue overload complex. An indicator is fibrosis of annulus fibrosus.

Rehabilitation (Table 8-3). Initially, immobilization (and thus enforced relative rest) of the cervical spine structures should be initiated. If a cervical collar is used, the higher part of the collar should

TABLE 8-3

TREATMENT FOR CERVICAL RADICULOPATHY

Time (d)	Treatment
0-14	Relative rest with a cervical collar
0-7	Oral corticosteroids, NSAIDs, modalities
0-7+	Cervical traction
7-10	Cervical isometrics
10-14+	Advanced strengthening, cervicothoracic stabilization program

NSAID: nonsteroidal antiinflammatory drug

Fig. 8-8. Proper placement of cervical traction in 25°–30° of flexion.

be worn posteriorly, to maximally open the intervertebral foramen. As stated previously, early tapering should be initiated to prevent disuse atrophy. The collar should initially be worn at all times, except during baths and isometric strengthening exercises. Antiinflammatory medications, particularly oral glucocorticoids, may be used judiciously for an acute condition. Short courses of time-dependent doses of narcotic medications may be helpful, but prolonged use causes side effects. With cervical disk symptoms, a short course of cervical traction is also warranted. A clinical trial of manual cervical distraction is a useful diagnostic maneuver that can predict a successful response to mechanical cervical traction[109] and may be better tolerated than mechanical traction, making it therapeutically useful. Traction in the cervical spine may decrease lordosis, decrease muscle spasm, enlarge the foramina, and distract the vertebral bodies enough to give some pain relief. Proper placement of cervical traction is essential (Figure 8-8).[107] Traction should be initiated in 30° of flexion, starting with 10 to 15 lb, and then increased to 30 lb over time.[110] The reclining and sitting positions are equally therapeutic. However, continuous in-bed, low weight (5 lb) cervical traction, aside from the enforced bedrest, is relatively ineffective.[109]

A short course of passive modalities may help alleviate some pain and allow more aggressive active rehabilitation. These include electrical stimulation, heat packs, and massage. Active exercises are then begun, starting with cervical isometrics and continuing throughout pain free ranges-of-motions. Upgrading posture, that is, chest out-head back, is also an important consideration. Advancement to cervicothoracic stabilization exercises is then begun (Figure 8-9).[111]

Fig. 8-9. (**a**) Cervical stabilization exercises—poor positioning with the head and neck hunched forward. (**b**) Proper alignment for cervical posture. The chin is tucked in a gravity resisted position. This exercise is a progression from (**a**). The patient is to hold an isometric contraction in this position for 10 seconds. (**c**) Advancement to single extremity weight bearing while maintaining proper cervical and thoracic positioning. Hold for 10 seconds.

Cervical epidural corticosteriod injections may also be beneficial when radicular symptoms are prominent and oral antiinflammatory agents have not been successful.[112] Epidural corticosteroid injections can be given in the field, if necessary, under sterile conditions, by a physician trained in the appropriate techniques. Ideally, these injections should be done in a hospital situation with close monitoring.

Brachial Plexus Injuries

The major types of brachial plexus injuries that may be seen in the military population include penetrating trauma, traction injuries, and compression injuries.[101,113–115] "Stingers" or "burners" are probably not true anatomically defined plexus injuries, but rather root level injuries, from either traction or foraminal compression.[116] Details of these mechanisms are thoroughly reviewed elsewhere.[113,116]

Method of presentation. Acute traumatic injury will follow a forceful trauma to the head and neck region.

Tissue injury complex. Damage will occur at the cervical anterior and posterior horn nerve root fibers.

Clinical symptom complex. Severe neck and shoulder pain will be concomitant with radiation of burning and paresthesia into one of the upper extremities toward the hand. Weakness is occasionally associated, often in the shoulder musculature. The duration of symptoms is variable and frequently lasts less than 1 minute.[116]

Functional biomechanical deficit. There are none (these are acute injuries).

Functional adaptation complex. There are none (these are acute injuries).

Tissue overload complex. This will occur at the cervical nerve roots.

Rehabilitation (Table 8-4). Often symptoms of acute brachial plexus injuries (or stingers) resolve rapidly. When sensory symptoms persist, pain control with narcotic and nonnarcotic medications may be necessary to allow the soldier to function with the pain. However, motor findings tend to be the more persistent neurologic abnormalities following a stinger.[116] In any type of brachial plexus injury in a military situation, early rehabilitation focuses on prevention of secondary complications from prolonged immobilization of an injured and significantly weak upper limb. Some splinting or relative immobilization may be necessary when profound weakness is present. Range-of-motion exercises are necessary to prevent adhesive capsulitis. Distal strengthening, in the case of proximal injuries, will be helpful to prevent hand swelling and reflex sympathetic dystrophy. Specific strengthening of involved muscles, initially with isometric and then isotonic exercises, is important. This therapy may be necessary for many months as residual weakness can be prolonged.

TABLE 8-4

TREATMENT FOR BRACHIAL PLEXUS INJURIES

Time (d)	Treatment
0–1	Initiate splinting (if necessary)
0–3	Early ROM, Codman, and wand exercises
0–7	Pain medications
3–10+	Strengthening shoulder girdle, cervical and thoracic spine

ROM: range-of-motion

Thoracic Outlet Syndrome

Thoracic outlet syndrome (TOS) is a group of disorders attributed to the compression of the neurovascular bundle in the region of the cervical-thoracic dorsal outlet. The neurovascular bundle is a grouping of the brachial plexus nerve fibers and the subclavian vein and artery. Most symptoms of TOS affect the C-8 and T-1 nerve roots, as opposed to cervical nerve root syndromes, which most commonly involve the C-5, C-6, and C-7 levels. Details of the specific disorders that fall under the category of TOS are referenced.[117–121] Contributing factors to TOS include poor posture; muscle strength imbalances, such as weak scapular stabilizers and spinal extensors; tight pectoralis muscles; flexion extension injuries with associated muscle spasm (especially in anterior cervical and shoulder musculature); and emotional stress. The differential diagnosis of TOS includes cervical spondylosis and radiculopathy, shoulder disorders, entrapment mononeuropathies (carpal tunnel syndrome), ulnar neuropathy at the elbow, and myofascial syndromes.

Method of presentation. TOS is usually manifested after chronic overuse in soldiers who use their upper extremities most of the day for activities.

Tissue injury complex. Affected areas are the nerve root fibers of the brachial plexus, usually the lower trunk or medial cord, or less commonly, the subclavian artery and vein, or all three.

Clinical symptom complex. There will be numbness, tingling, and weakness in the affected upper

extremity. Symptoms can be very specific, from only the medial aspect of forearm to affecting the entire upper limb.

Functional biomechanical deficit. The patient will present with inflexibility of scalenes, pectoralis major and minor, and hypomobility of the first rib.

Functional adaptation complex. A hunched forward posture, increased thoracic kyphosis, and increased cervical lordosis may all be evident.

Tissue overload complex. Lumbar spine extensors, scapular stabilizers, and thoracic and cervical spinal extensor muscles are subject to increased stress to accommodate the functional adaptation complex.

Rehabilitation. Thoracic outlet problems are most often related to chronic overuse of the upper extremity, cervical and thoracic spine, and chest musculature with resultant soft tissue inflexibility, muscle imbalances, and altered postures. Treatment starts by correcting drooping shoulders, altering and improving sitting posture, and enhancing body mechanics to attain a "chest out, head back" position.[122,123] Stretching of the pectoralis and scalene muscles is essential.[124] Often soft tissue mobilization of these structures by a qualified therapist is necessary.[123] Increased joint and soft tissue mobilization of the scapula, and scapulothoracic motion is necessary. Joint mobilization of the first rib and clavicle to restore accessory motion of the sternoclavicular and acromioclavicular (AC) joints is necessary to obtain proper upper extremity motion.[125] Strengthening exercises focus on the scapular stabilizers, that is, the serratus anterior, mid trapezius fibers, rhomboids, and erector spinae muscles (Figure 8-10). Often weight reduction and stress reduction will also decrease symptoms. Surgical intervention of either first rib resection or scalenotomy, is necessary on rare occasions.[120] With proper treatment, most cases will start to show improvement within 3 to 6 weeks.

Shoulder Disorders

Rotator Cuff Injuries, Overload, and Tears

Rotator cuff pathology and associated lesions (such as labrum tears, bicipital tendinitis, and subacromial bursitis) are some of the most common upper extremity musculoskeletal problems seen in the military population. The pathomechanics of this syndrome implicate activities that repeatedly place the arm in overhead positions.[126] The diagnosis of rotator cuff pathology is often straightforward. However, other causes of shoulder pain that can be mistaken for rotator cuff disease include proximal limb nerve entrapments (ie, axillary nerve in the quadrilateral space, musculocutaneous nerve in the biceps muscle, and suprascapular nerve at the supraglenoid fossa), brachial neuritis, AC disease, referred pain from cardiac or gastrointestinal disorders, or cervical radiculopathy.

Method of presentation. Rotator cuff injuries will show subclinical functional alterations.

Tissue injury complex. The rotator cuff will exhibit tendinitis, a tear, or both.

Clinical symptom complex. There will be impingement with abduction and rotation, and pain with isolated resistance of the supraspinatus, both of which will cause a painful arc from 60° to 120° of abduction.

Functional biomechanical deficit. Deficits will present as internal rotation inflexibility, external rotator muscle weakness, and "lateral scapular slide."[39]

Functional adaptation complex. To compensate, there will be alteration of arm position for overhead activities, such as throwing and lifting; "short arming" of throw; and muscle recruitment from anterior shoulder, forearm, or trunk.

Tissue overload complex. Eccentric overload will occur in posterior shoulder capsule, posterior shoulder muscles, and scapular stabilizer muscles.

Rehabilitation (Table 8-5). The focus of a rotator cuff rehabilitation program is to decrease inflammation, to restore normal shoulder biomechanics, and to achieve adequate strength balance of shoulder girdle musculature. Reduction of inflammation through NSAIDs was discussed earlier in the section on tendon injuries. In a battlefield situation, subacromial bursa injection of corticosteroid can give excellent, rapid reduction of inflammation. However, corticosteroid injections can weaken musculotendinous structures and increase the risk of making a partial tear a complete one.[62] Although these risks are real, on the battlefield, the benefit of significant relief will often outweigh the risk of further damage to the tendon. Cryotherapy, TENS, and ultrasound have all been used successfully to expedite inflammation reduction of the bursa and rotator cuff tendons.[127] To discourage reinjury, the soldier can be advised to use the involved upper extremity only in positions under 90° of abduction and for light activities.

In subacute and chronic rotator cuff injuries, improvement of flexibility should be initiated as soon as any acute inflammation subsides. In particular, tightness of the external rotators of the shoulder, with resultant internal rotation deficits, needs to be

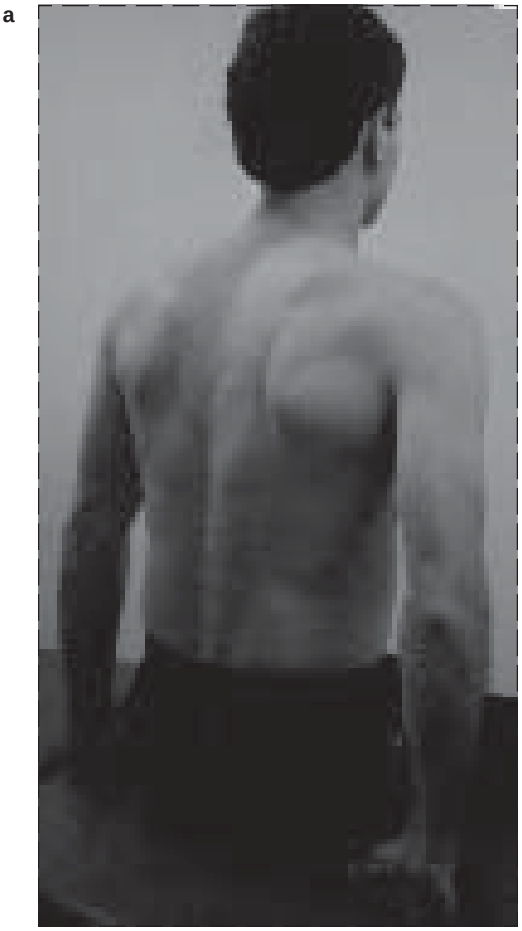
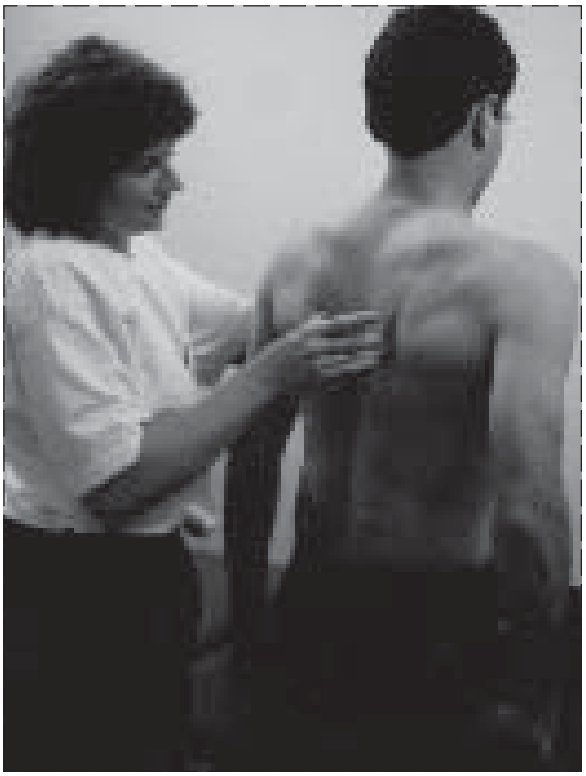


Fig. 8-10. (**a**) Poor scapular stabilizer strength with mild scapular winging/protraction. (**b**) Cueing the patient to activate scapular stabilizer muscles—midtrapezius and rhomboids. (**c**) Cueing the patient to activate scapular stabilizer muscles—serratus anterior.



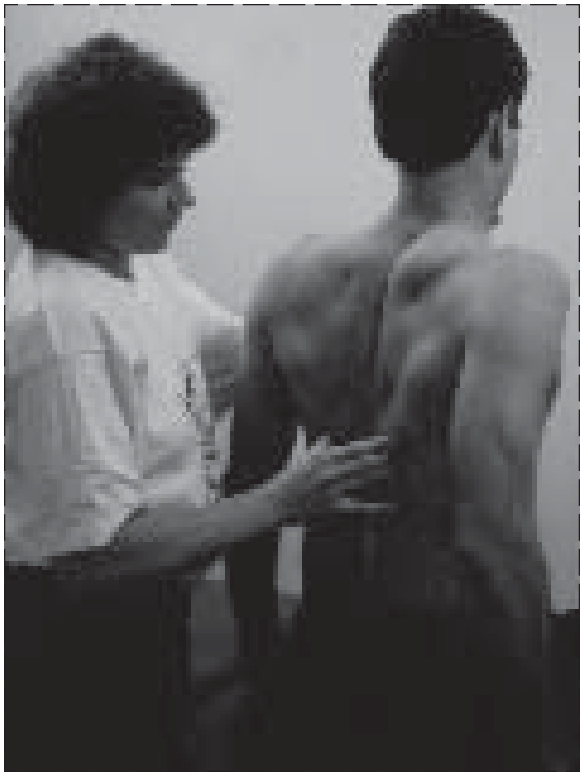


TABLE 8-5

TREATMENT FOR ROTATOR CUFF TENDINITIS

Time (d)	Treatment
0–7	NSAIDs, modalities, injection
3–7	Flexibility for internal rotation
3–7	Isometric strengthening of cuff and scapular stabilizers in positions under 90° abduction
7–14	Isotonic strengthening
21–28	Overhorizontal strengthening, PNF patterns

NSAID: nonsteroidal antiinflammatory drug
PNF: proprioceptive neuromuscular facilitation

addressed. Assessment should also be made of the motion of the sternoclavicular, AC, and scapulothoracic articulations, because movements at these joints greatly affect proper rotator cuff biomechanics and function. Deep friction massage may be of some benefit in improving range-of-motion when significant scarring has occurred and neither active nor passive stretching accomplishes the desired results.[128]

Strengthening exercises should start with shoulder isometrics in all planes. Progression is then to isotonic strengthening in the under-horizontal plane for internal and external rotation, scapular stabilizers (midtrapezius fibers, serratus anterior, rhomboids), and the biceps brachii (a humeral head depressor) (Figures 8-11 and 8-12). Emphasis on the scapular stabilizers must be appreciated because in rotator cuff injuries the clinical symptoms do not always correlate with function, and the point of clinical pathology is not always the site of muscle pathology. As described above, the tissue overload complex is the posterior shoulder muscles and the scapular stabilizer muscles. Over-horizontal exercises in nonpainful planes are begun when stretching exercises are pain free. Rehabilitation is then focused on activity specific training. Symptoms should show some improvement with acute measures in 5 to 7 days. Commonly, symptoms will take

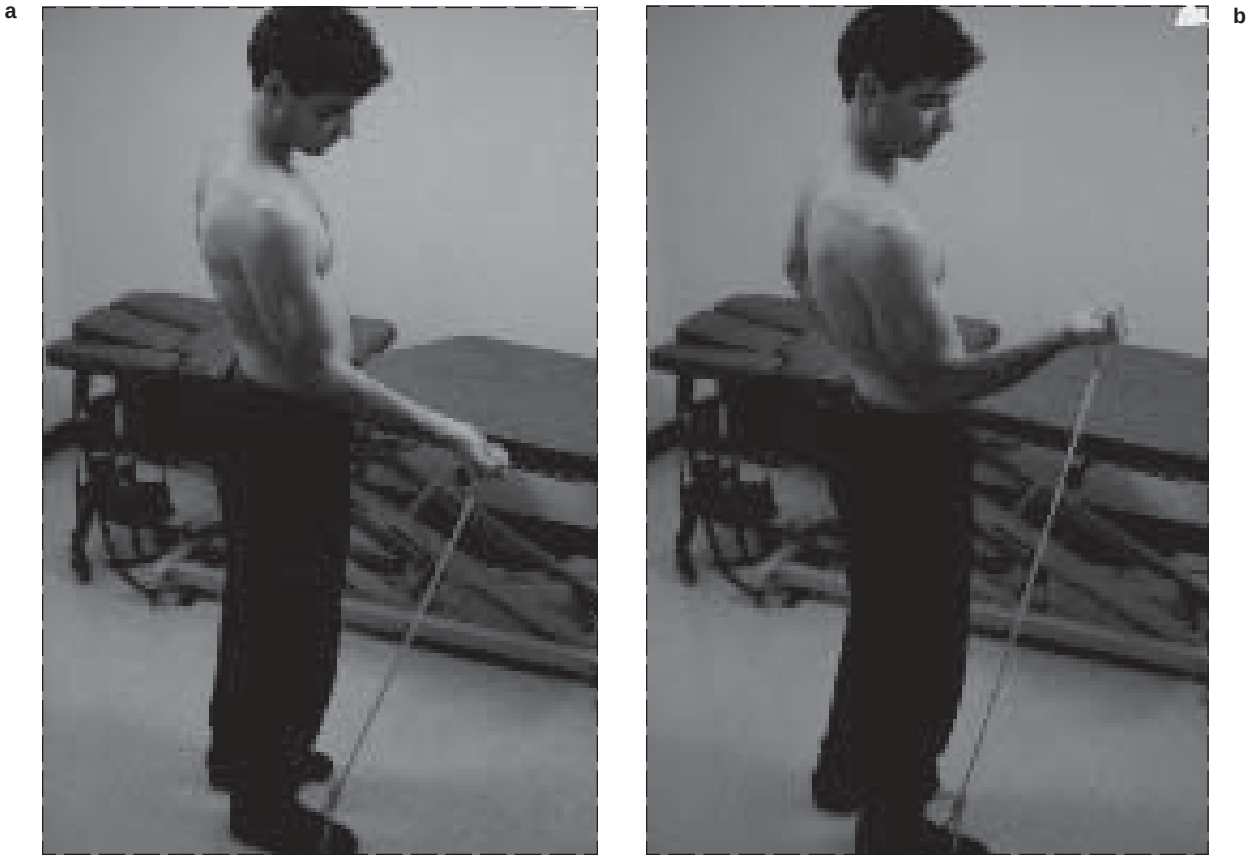


Fig. 8-11. Strengthening the biceps brachii muscle with a Theraband.

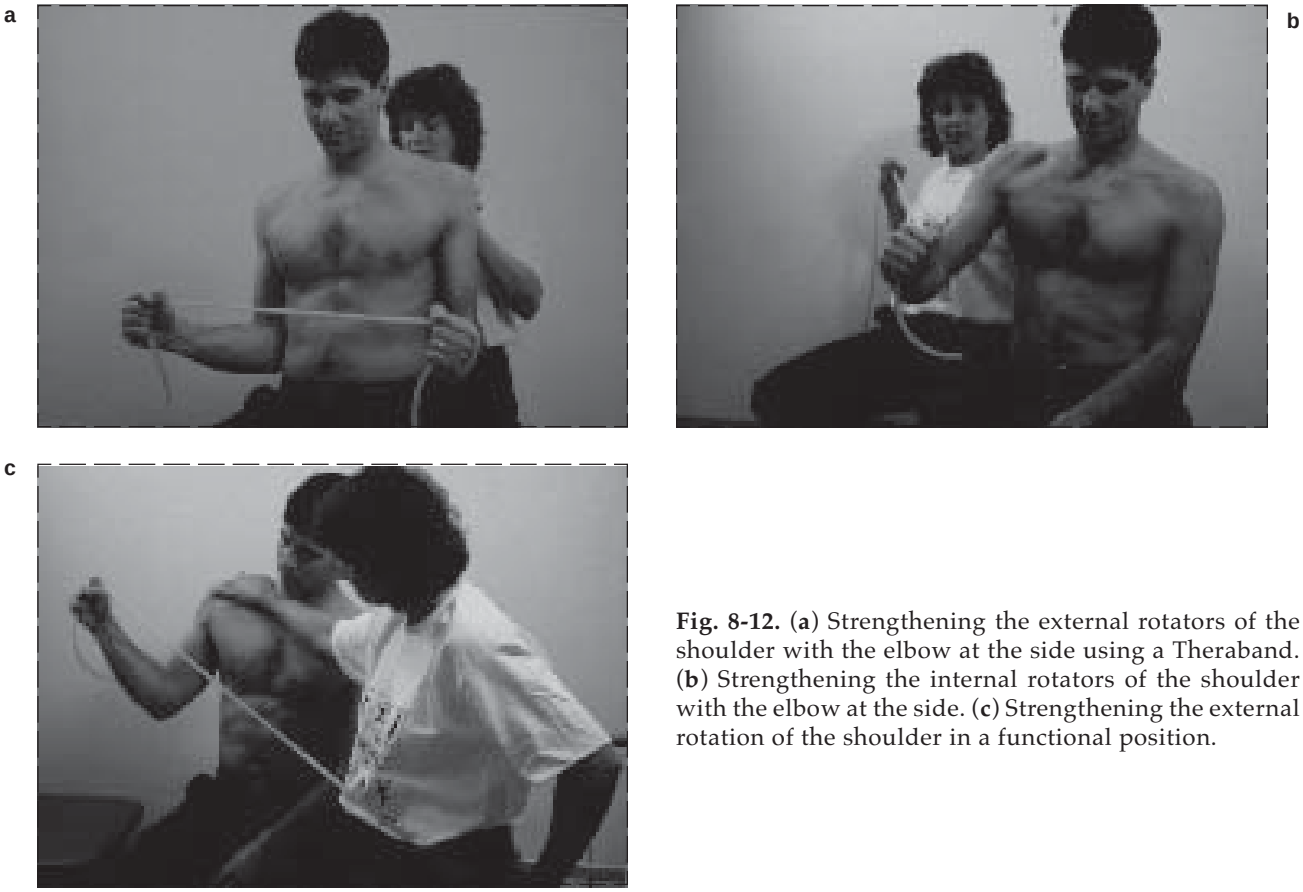


Fig. 8-12. (**a**) Strengthening the external rotators of the shoulder with the elbow at the side using a Theraband. (**b**) Strengthening the internal rotators of the shoulder with the elbow at the side. (**c**) Strengthening the external rotation of the shoulder in a functional position.

2 to 4 weeks of rehabilitation before significant changes in flexibility and strength are appreciated.

Shoulder Instability: Glenohumeral Joint

The three articulations of the shoulder girdle (the glenohumeral joint, the AC joint, and the sternoclavicular joint) are all relatively unstable because they allow a wide range of movement in the girdle as a whole. The supporting ligaments, therefore, are relatively lax and are easily stretched.[129]

Subluxation and dislocation of the glenohumeral joint can occur in a military setting when sufficient force is applied to overwhelm the muscular and capsuloligamentous apparatus. The inferior glenohumeral ligament complex is the prime static stabilizer for anterior, posterior, and inferior stability.[129] Often, with chronic subclinical instability, subtle changes occur in the subacromial bursa area and rotator cuff tendon, causing impingement symptoms and producing attritional tears of the glenoid labrum.

Method of presentation. Glenohumeral joint injuries present as an acute traumatic injury, or recurring injury to predisposed tissue, usually caused by a fall with the arm raised and outstretched.

Tissue injury complex. The anterior capsule will be affected, including the anterior glenoid labrum, and superior, middle, and inferior glenohumeral ligaments.

Clinical symptom complex. Pain and gross deformity of the shoulder profile with loss of normal fullness will be evident, along with the loss of mobility and the arm held away from the trunk.

Functional biomechanical deficit. Weakness of anterior shoulder stabilizers (subscapularis and pectoralis major muscles) will occur.

Functional adaptation complex. There is no functional adaptation complex.

Tissue overload complex. The anterior labrum and glenohumeral ligaments will be stressed, especially the inferior glenohumeral ligament.

Rehabilitation (Table 8-6). Restoration of normal structural integrity is the goal of rehabilitation. Descriptions for techniques of acute relocation of a dislocated shoulder are cited elsewhere.[130] Initial treatment for a dislocation will require a period of

TABLE 8-6

TREATMENT FOR SHOULDER DISLOCATION

Time	Treatment
0–6 wk	Immobilization in sling
0–3 d	Cryotherapy, modalities, pain medication
0–14 d	Isometrics for elbow, wrist, and hand
7–14 d	Isometrics for shoulder girdle muscles
4–6 wk	Wand, Codman exercises once splint removed
4–6 wk	Passive stretching
6+ wk	Strengthen isometrically and isotonically

immobilization to allow scarring to occur over the capsuloligamentous structures that were stretched. The optimal time for postreduction immobilization for the first dislocation is 3 to 6 weeks in a sling and swathe.[131,132] Application of ice decreases tissue edema and hemorrhage, and gentle isometrics for the elbow, wrist, and hand are initiated while the shoulder is immobilized. During the period of immobilization, isometric contractions of the shoulder musculature are initiated within the patient's tolerance. Throughout this period the soldier is instructed to maintain the axis of his arm anterior to the midcoronal plane of the body, so as not to encourage anterior instability.[133] Once the sling is removed, exercises to prevent extensive adhesive capsulitis should be started; they should concentrate on gentle, passive, assisted stretching exercises to regain range-of-motion. Strengthening exercises should begin from the under-horizontal (less than 90° abduction) position to encourage stability and diminish mechanical irritation from the injured capsule and ligaments.[134] In any strengthening program, emphasis is placed on the internal rotators of the shoulder because these are the most effective dynamic restraints against anterior instability in the middle to low ranges of abduction.[135,136] Later, strengthening of the external rotators and the remaining shoulder muscles is initiated.

The total period of rehabilitation may vary from 6 weeks to 4 to 5 months. Sufficient external rotation at 90° of abduction (the position at which the shoulder is generally most vulnerable to anterior instability) should be obtained without apprehension to allow comfortable participation in full military activities. Painless range-of-motion, strength, and endurance parity are also necessary for resumption of full military duty.

Shoulder Instability: Acromioclavicular Joint

The AC joint, because of its superficial position, is subject to frequent trauma. Dislocation of the AC joint follows damage to the AC and coracoclavicular ligaments. The cause is usually a fall onto the outstretched arm, elbow, or point of the shoulder so that the joint is forced inward and upward and the scapula is forced caudally.[137] Grade I injuries represent a mild sprain of the AC and coracoclavicular ligaments with no anatomic disruption of either ligament. Grade II injuries represent a partial displacement of the AC joint, less than the width of the clavicle. Grade III injuries represent complete loss of the integrity of the AC ligaments and coracoclavicular ligaments.[138]

Method of presentation. All grades present as acute traumatic injuries.

Tissue injury complex. The AC joint and supporting ligaments will be damaged.

Clinical symptom complex. Pain over the AC joint or proximal shoulder will occur with crossed adduction of the arm at 90° of abduction across the body (Figure 8-13).

Functional biomechanical deficit. There are none.

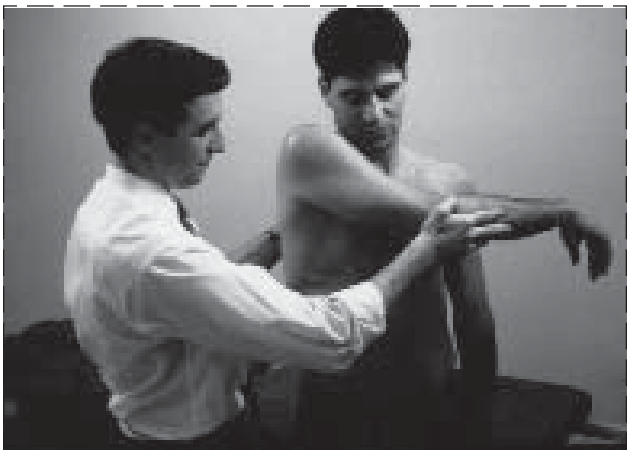


Fig. 8-13. Crossed adduction maneuver to check for acromioclavicular joint pathology. The arm is adducted horizontally and then some end range overpressure is exerted. Reproduction of pain over the acromioclavicular joint with this maneuver is a sign of acromioclavicular joint pathology.

Functional adaptation complex. Alteration in scapulothoracic and glenohumeral motion will be present.

Tissue overload complex. Stress will occur to the AC and coracoclavicular ligaments.

Rehabilitation (Table 8-7). To a large extent, rehabilitation will depend on the degree of injury to the AC joint. In any injury, the initial use of ice and antiinflammatory agents may be supplemented with a local injection of anesthetic into the injured joint. This injection may serve both a diagnostic and therapeutic effect. Usually 3 to 5 cm^3 of 1% lidocaine will suffice to give good relief of local pain from an AC joint injury. Local padding to prevent direct pressure over the joint can be helpful.

Grade I injuries usually respond to cryotherapy and antiinflammatory medications and padding. Return to full duty should be accomplished in 2 days to 2 weeks. Grade II injuries will require a sling to support the joint until it is asymptomatic, which may range from 1 to 4 weeks. The deformity that is initially present will remain a permanent deformity.[138] Once symptoms begin to abate, a muscle strengthening program, involving the trapezius and the remainder of the shoulder girdle muscles, is initiated; but repetitive activity with the arm above the shoulder should be kept to a minimum for the first 4 weeks of treatment.[139]

Grade III injuries have more limitations of motion and a higher potential for disability. In most cases, nonsurgical treatment is still the preferred course.[138,140] Overall, a 5% to 10% incidence of significant problems can be anticipated with a Grade III AC injury, whether it is treated by closed or open means.[138] Closed symptomatic treatment requires immobilization of the AC joint with a sling for comfort. Once the AC joint is reduced, the soldier is given pain medication and instructions to keep the shoulder quiet, not to remove the sling unless there is severe pain, and report periodically for adjustments of the sling. The sling is discontinued when the symptoms allow, usually in 7 to 10 days.[138] While the soldier is still in the sling, isometric exercises are begun; the sling is loosened for range-of-motion exercises, which are performed over the AC joint with support from the therapist. Progressive resistance exercises are done as tolerated. With closed aggressive treatment, an attempt is made to keep the joint reduced for 6 full weeks, during which time isometric exercises are instituted and periodic checks of sling position are made.[138] If closed techniques do not work, or the results are unacceptable to the patient, referral for surgery is indicated. If dislocation causes a major shift in the scapular position due to the loss of the support of the clavicle, more pain symptoms may be expected. In this case, early surgery may be indicated.

TABLE 8-7

TREATMENT FOR ACROMIOCLAVICULAR DISLOCATION

Time	Treatment
0–3 d	Cryotherapy, NSAIDs, pain medication
0–7 d–6 wk	Local padding, sling use
5–10 d	Strengthen trapezius, shoulder girdle

NSAID: nonsteroidal antiinflammatory drug

Elbow Disorders

Epicondylitis: Medial and Lateral

Medial and lateral epicondylitis are common chronic, repetitive overuse disorders seen in the military population. It is generally accepted that the primary pathology on the lateral aspect involves a microtear at the origin of the extensor carpi radialis brevis; and less commonly, the extensor carpi radialis longus and the anterior portion of the extensor communis tendon. With both, there is a formation of subsequent fibrosis and granulation tissue as a consequence of repeated trauma.[141–143] Repetitive concentric contractions of these muscles, shortening as they maintain tension to stabilize the wrist, produce chronic overload, which results in the symptoms of lateral epicondylitis.[141] Medial epicondylitis involves the pronator teres, flexor carpi radialis, and occasionally the flexor carpi ulnaris, all of which arise from the medial epicondyle of the humerus and from the fascia over it.[141] Differential diagnosis of medial and lateral epicondylitis includes cervical radiculopathy, nerve entrapment syndromes (particularly the radial nerve at the lateral elbow), or proximal radioulnar joint injuries.[141–143]

Method of presentation. The insidious onset will have gradually increasing symptoms, or acute exacerbation of chronic injury, or both.

Tissue injury complex. Microtears and tears will be evident in the extensor carpi radialis brevis and longus tendons, or in the flexor carpi radialis and pronator teres tendons, with angiofibromatous hyperplasia.

TABLE 8-8

TREATMENT FOR EPICONDYLITIS-MEDIAL AND LATERAL

Time (d)	Treatment
0–7	Cryotherapy, antiinflammatory modalities
3–5+	Counterforce bracing
3–14	Flexibility program for involved muscle
7–14	Eccentric and concentric strengthening
21–28	Corticosteroid injection (if necessary)

Clinical symptom complex. Pain at the epicondyle can radiate distally into the forearm. Occasionally there will be weakness of grip strength and tenderness to palpation over insertion points of involved muscles. Pain will occur with resisted motion of the involved muscle.

Functional biomechanical deficit. With lateral epicondylitis, there will be extensor muscle inflexibility, extensor weakness, pronation contracture, and decreased shoulder external rotation strength. The medial epicondylitis will show flexor-pronator inflexibility and weakness.

Functional adaptation complex. Alteration in grip positions on military equipment will be evident, as will more use of the shoulder in throwing motion.

Tissue overload complex. Lateral epicondylitis will have stress at wrist extensor (especially extensor carpi radialis brevis) and shoulder external rotators; with the medial epicondylitis, wrist flexor-pronator tendons will be affected from eccentric overload.

Rehabilitation (Table 8-8). Initially, copious use of cryotherapy and judicious use of antiinflammatory medication are indicated, usually for the first 2 to 3 weeks. With epicondylar pain, the flexibility of the involved tight muscles can be improved by fully extending the elbow and either palmar flexing or extending the wrist with increasing pressure against a table (Figure 8-14). Stretching should be done several times a day. Initial strengthening can be done isometrically, with resistance from the other hand at multiple angles of wrist flexion and exten-

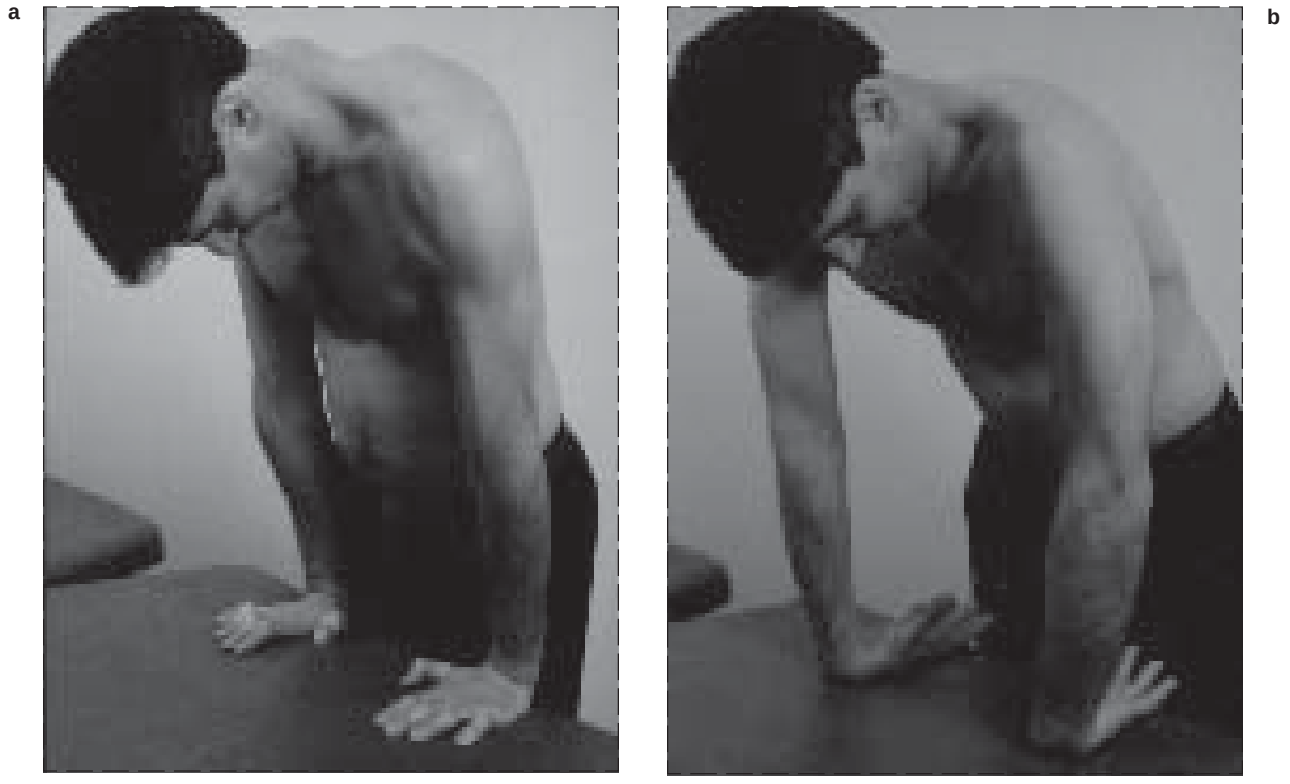


Fig. 8-14. (**a**) Stretching of the medial epicondylar muscles (wrist flexors and pronators). (**b**) Stretching of the lateral epicondylar muscles (wrist extensors and supinators).

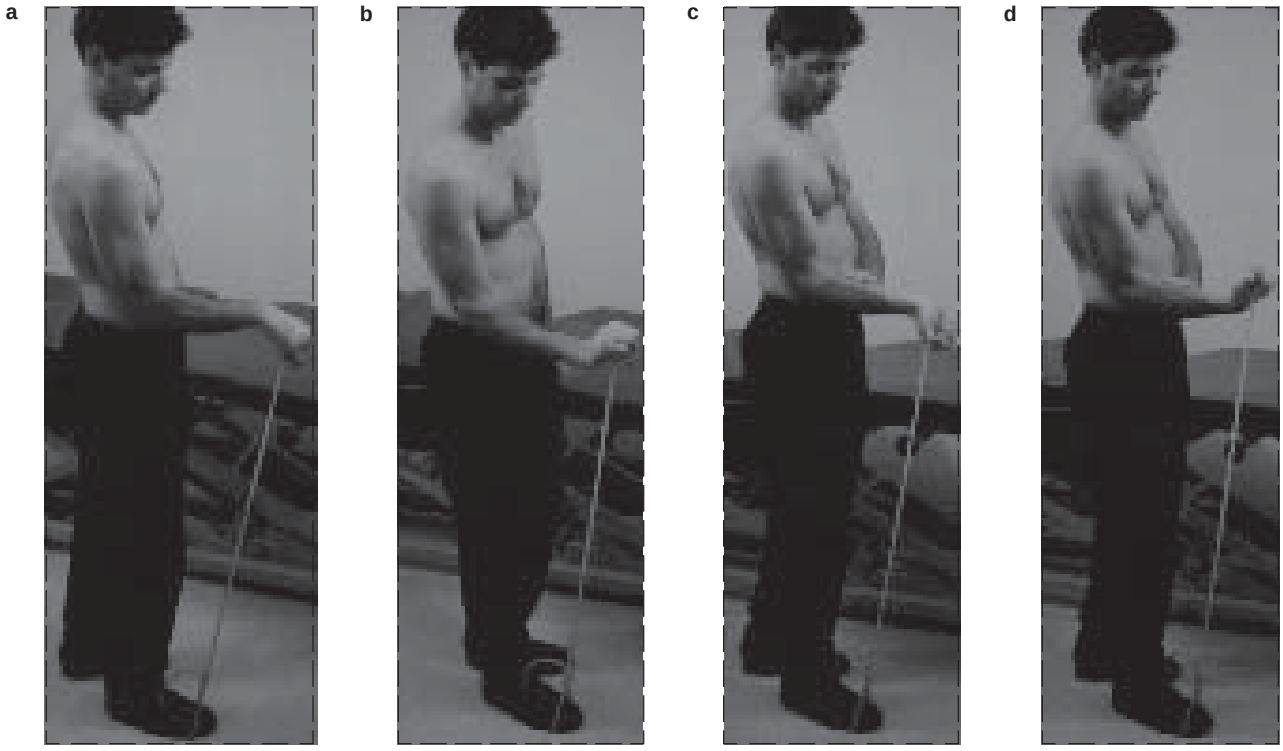


Fig. 8-15. (**a, b**) Strengthening wrist extensors using a Theraband. (**c**, **d**) Strengthening wrist flexors using a Theraband.

sion. Progression can then be made to wrist strengthening with the use of elastic bands or free weights (Figure 8-15). Ultimately, strengthening should be done both eccentrically and concentrically, such as with a weight tied to a piece of wood, which is slowly raised and lowered only with wrist motion (Figure 8-16).

Use of ultrasound or other heating modalities may give some pain relief, as well as loosen any scar tissue to allow better flexibility. Electric stimulation modalities may also help with pain reduction and prevention of muscle atrophy. Counterforce braces may also be helpful by distributing the forces around the elbow over a greater surface area.[144–146] For lateral epicondylitis, the brace is applied firmly around the forearm over the wrist extensor muscle mass at the elbow. It is tightened enough so that when the patient contracts the wrist extensors, they do not obtain a full contraction of the muscle; that may relieve tension on the attachment of the extensor tendon.[141]

If symptoms are not significantly reduced over a period of 3 to 4 weeks, a corticosteroid injection in the painful area may be helpful. The steroid is mixed with a local anesthetic and injected into the subaponeurotic space at the point of maximal tenderness. Vigorous activity of the involved forearm should be avoided for 2 weeks after an injection. If the patient receives some relief but still has pain that limits function, injections may be given once again. When conservative measures fail and the soldier is significantly disabled by epicondylitis, surgical release of the involved fibers should be considered.

Medial Capsuloligamentous Injuries

The medial aspect of the elbow is supported by the medial collateral ligament, the medial joint capsule, and the muscle mass. In overhead activities, such as throwing, the elbow may be subject to intense valgus-tension stress. Any of the structures on the medial aspect of the elbow may become injured. Tension on the medial aspect of the elbow is first resisted by the overlying flexor-pronator muscles. These muscles may tear, or a partial avulsion of one of the tendons or muscle insertions may occur, causing valgus overload injuries. Repetitive, violent stresses will involve the deeper capsule and ligament. Tension stress that the capsule and ligament put on the ulna and humerus can lead to spur formation and, ultimately, compression of the ulnar nerve. The differential diagnosis of medial capsuloligamentous injuries include ulnar neuropathies,

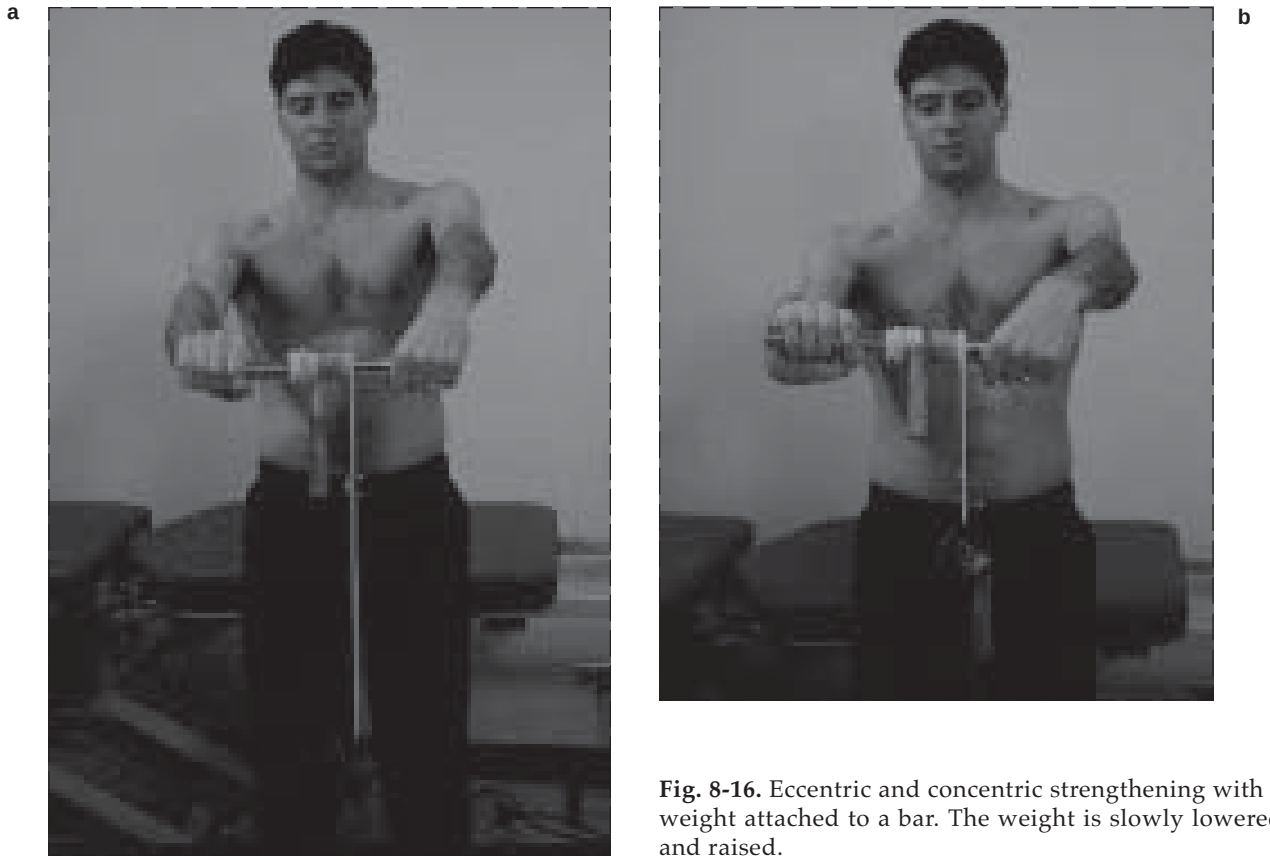


Fig. 8-16. Eccentric and concentric strengthening with a weight attached to a bar. The weight is slowly lowered and raised.

radioulnar joint lesions, degenerative joint disorders of the elbow, cervical radiculopathy (especially C-8 lesions), lower trunk brachial plexus lesions, and TOS.

Method of presentation. The method of presentation will be acute exacerbation of a chronic injury.

Tissue injury complex. Injury will occur in the medial collateral ligament, and the flexor-pronator muscle mass.

Clinical symptom complex. During overhead activities, such as throwing, pain will be present along the inner aspect of the elbow. Tenderness over the medial aspect of the elbow can be intensified by applying valgus stress to the elbow at 20° to 30° of flexion. Valgus laxity may exist.

Functional biomechanical deficit. The flexor-pronator will be inflexible and weak.

Functional adaptation complex. There will be a loss of terminal extension of elbow (inability to do a full push-up).

Tissue overload complex. Medial capsuloligamentous structures will be stressed, especially the anterior oblique band of the medial collateral ligament.

Rehabilitation (Table 8-9). Treatment will begin with relative rest and the judicious use of antiinflammatory medications. In most cases, symptoms should resolve in 7 to 14 days. Antiinflammatory modalities, such as ultrasound and electrical stimulation, are useful adjuncts to early treatment. Occasionally, a local injection of 3 to 5 cm^3 of anesthetic over the tender area may give temporary relief if immediate relief is necessary, such as in a battlefield situation. Therapy should be directed to stretching the flexor and pronator muscles of the forearm to improve range-of-motion. Regaining and maintaining a nor-

TABLE 8-9

TREATMENT FOR MEDIAL CAPSULOLIGAMENTOUS INJURIES

Time (d)	Treatment
0–3	Antiinflammatory medications and modalities, taping
3–7	Stretching flexor, pronator muscles
7–10	Strengthening eccentrically, concentrically elbow flexors

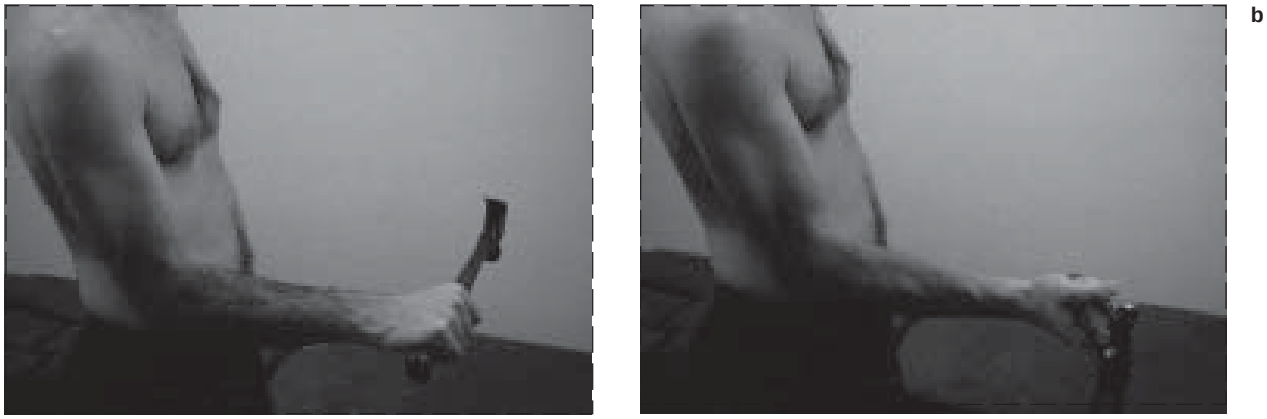


Fig. 8-17. Strengthening supination and pronation of the forearm by alternating forearm positions with a weighted object.

mal range-of-motion requires stretching in flexion, extension, pronation, and supination. Strengthening is also initiated, both concentrically and eccentrically. These type of strengthening exercises can be done as outlined before, with epicondylitis. Pronation and supination can be strengthened using a hammer or similar tool to produce torque throughout the full range-of-motion (Figure 8-17). Grip and shoulder exercises are also initiated.

Rehabilitation programs for elbow problems should always address proximal stability at the shoulder to allow the elbow to be properly placed in space to function appropriately. Ice may be applied immediately after exercises. If major soft tissue injuries have occurred, care must be taken to avoid early aggressive stretching because of the risk of traumatic myositis ossificans.[147] If a soldier's return to active duty is required before rehabilitation is completed, taping and bracing of the medial aspect of the elbow may be necessary for additional support. In the case of an acute rupture of the medial collateral ligament, with instability, surgical repair is indicated.

Hand and Wrist Disorders

De Quervain's Tenosynovitis

De Quervain's disease is a tenosynovitis of the first dorsal compartment. The first dorsal compartment contains the tendons of the abductor pollicus longus and the extensor pollicus brevis. These tendons are prone to inflammation from repetitive hand and wrist motions. Any of the tendons and muscles of the hand and wrist can become inflamed, especially with activities that require a forceful grasp coupled with ulnar deviation or repetitive use of the thumb.[148] Accurate and early diagnosis of tenosynovitis of the digital extensors is important, especially in the case of the extensor pollicus longus because this tendon tends to rupture.[149] The differential diagnosis of de Quervain's tenosynovitis includes distal radius stress fracture, radial neuropathy, cervical radiculopathy (especially C-6 lesions), and degenerative joint disorders of the wrist.

Method of presentation. This tenosynovitis shows the acute exacerbation of a chronic injury.

Tissue injury complex. Damage will occur to the synovial sheath of the abductor pollicus longus and to the extensor pollicus brevis.

Clinical symptom complex. Pain and swelling will be one-half inch proximal to the radial styloid, and over the radial aspect of the wrist with ulnar deviation. There will be a positive Finkelstein's test (Figure 8-18), and an occasional palpable nodule.

Fig. 8-18. Finkelstein's test. Ulnar deviation of the closed fist reproduces radial sided pain over the extensor pollicus brevis and the abductor pollicus longus.

TABLE 8-10

TREATMENT FOR DE QUERVAINS TENOSYNOVITIS

Time (d)	Treatment
0–3	Rest, splinting in slight extension, cryotherapy, NSAIDs
3–7	Corticosteroid injection
3+	ROM exercises within pain free range
4–7	Isometric strengthening around wrist
7–14	Isotonic strengthening

NSAID: nonsteroidal antiinflammatory drug
ROM: range-of-motion

Functional biomechanical deficit. Along with adduction, the thumb will be inflexible.

Functional adaptation complex. There are none.

Tissue overload complex. The abductor pollicus longus and extensor pollicus brevis tendons will have eccentric loading.

Rehabilitation (Table 8-10). Initial treatment for de Quervain's tenosynovitis (the same for any of the tendinitises of the wrist and hand) consists of rest, splinting (in mild extension), use of cryotherapy, and oral antiinflammatory agents. Corticosteroid injections into the tendon sheath, if coupled with rest, can also be effective. Soluble steroids such as dexamethasone are preferable to insoluble steroids, which tend to leave a deposit. Use of 0.5 mL of dexamethasone (40 or 80 mg/mL) and 0.5 mL of 2% lidocaine is suggested.[148] Repeated injections, that is, more than three, should be avoided.[150] Early, pain-free range-of-motion of the wrist is important to avoid soft tissue contracture and scarring. Strengthening should be started isometrically and progressed throughout the entire range-of-motion, concentrically and eccentrically, as tolerated. Ultrasound treatment, followed by stretching, may be particularly helpful in chronic cases where extensive soft tissue shortening has occurred. Most cases of de Quervain's tenosynovitis should respond to appropriate treatment within 14 to 21 days. If conservative treatment fails, or if the condition becomes chronic, surgical decompression may be necessary.[151]

Ulnar Collateral Ligament Injury

Injury to the ulnar collateral ligament of the thumb can be quite common. Abduction stress to the thumb while the metacarpophalangeal joint is near full extension can tear the ulnar collateral ligament. This type of injury is most often described in skiing accidents.[152] Early recognition and proper treatment is necessary to prevent instability and decreased functional use of the hand. Classifications of injury are as follows: grade I and grade II lesions are degrees of partial disruptions of the ligament; grade III lesions represent complete ligamentous disruptions. Joint stability is best evaluated by stress testing, but should always be preceded by conventional roentgenograms, when available, to determine if a large, undisplaced intraarticular fracture is present. Injuries are classified by stress testing the metacarpophalangeal joint in slight flexion and in full extension to see if any opening of the joint occurs. Comparison to the uninjured thumb is essential since there is a great variation in metacarpophalangeal range-of-motion from person to person. Differential diagnosis, in chronic cases where no history of acute injury is present, include carpometacarpal arthritis of the first digit, carpal tunnel syndrome, adductor pollicus brevis or flexor pollicus brevis strain, and C-6 radiculopathy.

Method of presentation. This is an acute injury.

Tissue injury complex. The tissue involved is the ulnar collateral ligament of the thumb, usually at the insertion point to the proximal interphalangeal joint.

Clinical symptom complex. Pain and swelling will occur in the medial aspect of the thumb.

Functional biomechanical deficit. Stability of the thumb will be lost.

Functional adaptation complex. There will be a decreased grip strength.

Tissue overload complex. The ulnar collateral ligament will be overloaded.

Rehabilitation (Table 8-11). For a partial grade I or grade II ligament injury in which there is no in-

TABLE 8-11

TREATMENT FOR HAND-ULNAR COLLATERAL LIGAMENT INJURIES

Time	Treatment
0–21 d	Immobilize in spica cast
21–28 d	Active ROM exercises outside of splint
6–12 wk	Protective taping or silicone cast for duty

ROM: range-of-motion

stability, treatment is immobilization in 20° of flexion in a spica cast for 3 to 4 weeks.[149,153] The interphalangeal joint is left free to allow for active motion to prevent scarring of the extensor mechanism. A removable splint is fabricated after 3 to 4 weeks, and active exercises are allowed several times a day.[153] The splint may be removed at 5 to 6 weeks for normal activity. If there is severe swelling after the initial injury, a molded volar gutter splint may be used for the first weeks until the swelling subsides (Figure 8-19). This may be followed by the application of a thumb spica cast. For participation in military duty, the thumb is protected for a total of 3 months by either taping it to the index finger in adduction or by fabricating a silicone cast.[153,154] Surgery can be reserved for cases in which there is later disability or in which the diagnosis of instability is delayed for weeks after injury[155]; or it can be performed on any unstable joint.[146,156] For many weeks after immobilization, range-of-motion exercises will be important because of soft tissue contracture.

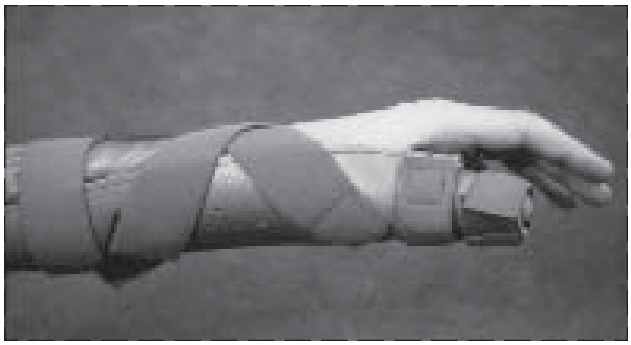


Fig. 8-19. Prefabricated volar gutter splint for ulnar collateral ligament injuries.

Lumbar Spine Disorders

Lumbar spine problems in the military population are quite common, and may parallel prevalence and incidence figures in the general population. Occupational and workplace factors clearly contribute to the development of low back pain, and in some cases, low back disability. Intrinsic to most occupations with a high risk of low back pain is a composite of repetition and force, and workers in occupations requiring high repetition/high force activities are more likely to sustain injuries. These types of activities are quite common in the theater of combat. The physical demands and potential injury mechanism of any particular job can be segregated into chronic repetitive overload such as bending, twisting, and vibration; or acute dynamic overload such as heavy lifting, slips, and falls. Details of these mechanisms of injury can be found in numerous publications, which are cited in the references section of this chapter.[157–166]

Diagnosis

Establishing a specific diagnosis in the acutely or subacutely injured soldier can lead to directed treatment that may allow quick, nonsurgical resolution of symptoms and early return to duty. In general, an adequate assessment of back pain requires (*a*) an intrinsic knowledge of spinal biomechanics and the degenerative spinal cascade, (*b*) integration and proper interpretation of imaging and electrophysiologic studies with clinical decision making, and (*c*) a healthy index of suspicion for both missed diagnoses, and signs and symptoms of disability and nonorganicity. An excellent review of these issues can be found in an article by Weinstein and Herring.[167] Identification of the tissue injury complex and the method of presentation of the injury are some initial steps. A complete history and physical examination should be performed for all soldiers with back pain to ensure that referred intraabdominal or intrathoracic pain is not the cause of the symptoms. Likewise, a history of fevers, chills, or constitutional symptoms should alert the clinician to the possibility of a tumor or infection of the spine. Iritis, uveitis, urethritis, a history of recent sexually transmitted disease (chlamydia) or a history of psoriasis raises the possibility of a spondyloarthropathy (Reiter's syndrome, ankylosing spondylitis). However, the vast majority of spine problems result from biomechanical and anatomic factors as well as repetitive overload, and are described below.

To categorize lumbar spine dysfunctions in a clinically useful way, the classification system of Kirkaldy-Willis and associates[168] may be helpful. Separating the three-joint complex into its component parts, namely discogenic and posterior element (joint), allows appreciation of different clinical presentations in the various phases of degeneration (dysfunction, instability, stability), and an understanding of the interaction of the individual components that lead to various types of spinal stenosis (Figure 8-20). Each phase will be discussed in regards to pathomechanics of the disk and joint, presenting symptoms and signs, and pertinent clinical correlates. It should be noted that the initial presentation of symptoms can occur anywhere along this continuum, implying that subpain threshold

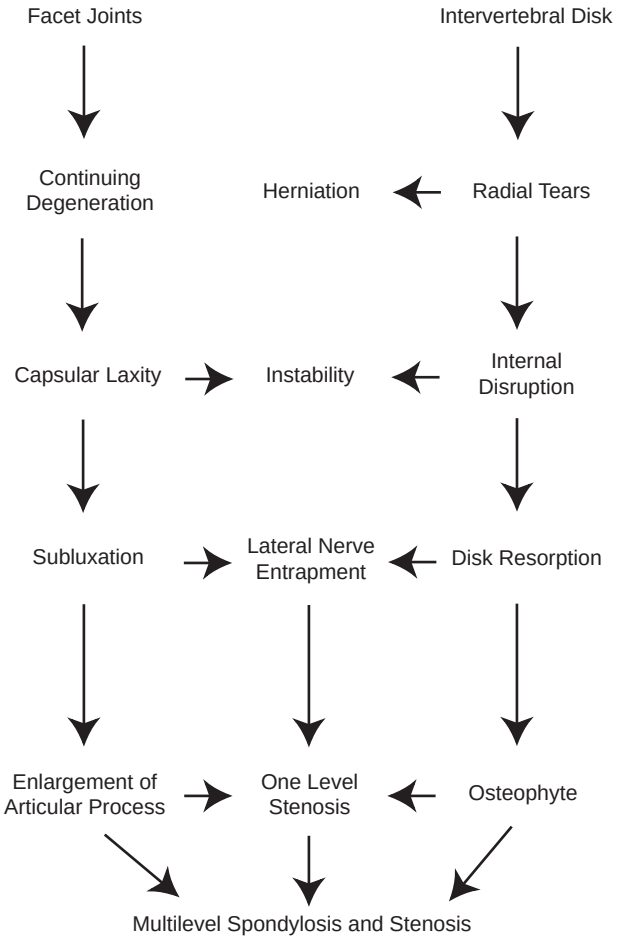


Fig. 8-20. Kirkaldy-Willis' degenerative cascade of the lumbar spine. Adapted with permission from Kirkaldy-Willis WH, Wedge JH, Yong-Hing K, Reily J. Pathology and pathogenesis of lumbar spondylosis and stenosis. *Spine*. 1978;3(4):320.

degenerative changes occur throughout life.[169–171] Also, degenerative changes in one motion segment may predispose to a similar process in adjacent segments. In one individual, different phases of the degenerative cascade can be found in different motion segments of the lumbar spine. Not uncommonly, dysfunctional (or hypomobile) segments might occur adjacent to a hypermobile segment. Lastly, the aging process does not always correlate with the clinical phase of degeneration.

Phase I. Phase I of the degenerative cascade is described as segmental dysfunction: a state of abnormally reduced, albeit subtle, movement of the motion segment. The initial clinical presentation in this phase usually reflects joint dysfunction as opposed to disk herniation. Facet joint dysfunction can include reactive synovitis and articular cartilage degeneration, resulting in joint pain, inflammation, and hypomobility. Abnormally sustained muscle contraction, especially of the short segmental extensors and rotators, can contribute to limited joint motion. The clinical presentation may be one of the classic sprain-strain syndrome. Low back pain may be worse with static standing, walking, extension, or extension combined with rotation; it may be somewhat relieved with flexion. However, even flexion may irritate a restricted joint capsule or inflexible musculotendinous unit in spasm. Local tenderness and muscle spasm, limited range-of-motion, and normal neurological examination are usual findings. Typically, posterior element dysfunction results in either nonradiating low back pain or referred pain to the buttock or proximal thigh.[172–174]

In this first phase, disk degeneration, including annular fiber tears, does occur. Joint dysfunction may not allow adequate load bearing and thus, transfers increased stress across the intervertebral disk. Radial tears are more likely to result in disk protrusion or herniation in the latter stages of dysfunction, but circumferential tears may also be painful because the outer annulus is supplied with nociceptive fibers. Classic discogenic radiculopathy can occur acutely, however, especially with a sudden, dynamic overload.

The typical presentation of discogenic low back pain is pain exacerbated by flexion oriented activities (sitting and bending,) activities that increase shear stress across the annulus (twisting), and activities that cause a valsalva-type maneuver (coughing or sneezing). The disk protrusion or herniation is usually in the posterolateral direction where the annular fibers are not well protected by the posterior longitudinal ligament, and where shear forces are greatest with forward and lateral bending. This clinical picture is not always present, however, and experience has shown that atypical discogenic pain can be seen, especially in young recruits and people with central disk protrusions or herniations. The usual clinical features of lumbar radiculopathy include leg pain greater than back pain, and paresthesias or weakness or both in a given dermatomal distribution; these are exacerbated by flexion activities, and relieved with extension activities. The degree of low back pain associated with disk protrusion or central herniations may be substantial, possibly due to the large number of free nerve endings in the posterior longitudinal ligament. The ratio of back pain to lower extremity pain varies, but with an extruded or free fragment resulting in radic-ulopathy, unless it is massive, low back pain may be minimal or absent. Lower extremity pain

may also diminish, and a neurologic deficit, that is, motor, reflex or sensory (or both) will become the predominant clinical feature.

Phase II. Phase II refers to a condition of excessive segmental motion, segmental hypermobility and, occasionally, frank segmental instability. This is a phase that is clinically more difficult to conceptualize. The objective findings of a "degenerative lumbar disk" by imaging studies presents a challenge to the practitioner in defining the source of pain, especially lumbar radicular pain or radiculopathy. As the border between these phases is somewhat arbitrary, a patient may who has never had discogenic low back pain (or for that matter, has never before been symptomatic) may enter Phase II.

Abnormalities of the facet joints include capsular laxity and joint subluxation as defined by Kirkaldy-Willis and associates.[168] Such movement may not be detectable by standard radiography, including lateral flexion and extension roentgenograms as no translation may occur, but the instantaneous center of rotation may move abnormally.[175] This also relates to our clinical impression that the quality of motion is more representative of pathology than is the quantity. Measuring gross range-of-motion allows evaluation of soft tissue extensibility and an ability to monitor the effects of treatment but does not provide information about segmental motion that could be obtained by a skilled manual examination. Observing dysrhythmia, a catching sensation or painful arc with recovery phase from forward bending, can be indicative of hypermobility. Another sign of a hypermobile segment is tenderness or spasm, or both, that is elicited by applying a torsional stress across a specific segment, such as, lateral springing on the spinous process (Figure 8-21).

Fig. 8-21. When lateral springing of a spinous process reproduces the patient's low back pain, it is suggestive of a hypermobile segment.

Disk related abnormalities in Phase II can include internal disk disruption and narrowing of the intervertebral disk space. An internally disrupted disk evolves as the process of degeneration progresses. Multiple annular tears develop that allow random distribution of nuclear material throughout the disk. A disk that is internally disrupted may be a pain generator. The source of the pain may result from hyperexcitability of the annular nociceptive fibers, or from neurobiomechanical factors. In this phase the disk is much less tolerant of torsional stress. Typically, torsional and compressive loads occur simultaneously and the compressive force assists with stabilizing the three-joint complex by loading the annular fibers.[176] As the annular fibers become less competent, torsional load results in a greater degree of "free play," and potentially a higher degree of symptoms. The reduction in disk space height promotes facet joint laxity, reduction in the size of the intervertebral foramen, and lateral recess narrowing. Radiculopathy can occur secondary to a variety of causes, including direct impingement on the lumbar or sacral nerve roots from a herniated disk, dynamic lateral entrapment due to narrowing of the lateral recess, and primary radiculitis due to neurobiochemical factors associated with an internally disrupted disk.

Radicular pain from any of these causes can occur in the absence of back pain. Hip girdle pain (buttock or trochanteric area, or both), thigh, or leg pain all may be precipitated through a number of dynamic clinical tests that cause forward displacement of the superior articular facet toward the vertebral body, thus narrowing the lateral recess medial to the intervertebral foramen.

Phase III. The process of segmental stabilization occurs over time. Pathomechanically, the facet joints become fibrosed, enlarged, and arthrosed. The intervertebral disk becomes increasingly degenerated and desiccated, allowing approximation of the ver-

tebral end plates and osteophyte formation. This combination of anterior and posterior changes can manifest in ankylosis of the motion segment, although lesser degrees of spondylosis are common. Limited range-of-motion and stiffness may become the predominant features, not unlike those observed in the dysfunctional stage. Low back pain may no longer be prominent, although severe degenerative changes may not permit this process of autofusion and, therefore, continuation of symptoms can occur. Symptomatic degenerative facet joints usually cause a low degree of aching low back pain, possibly bilaterally and sometimes at multiple levels.

In this phase, spinal nerve root entrapment is relatively common. Fixed lateral stenosis, central stenosis, and degenerative spondylolisthesis can all result in radicular signs and symptoms. Bilateral and multilevel radiculopathy may be seen. Central and lateral stenoses can coexist at the same or different levels. Neurogenic or pseudoclaudication is the typical presentation of lumbar radiculopathy in this phase.[177] The dermatomal symptoms of paresthesias, dysesthesias and myotomal distribution of muscle cramping and weakness, consistently occur with erect posture or exercise, including standing and walking. Typically these symptoms resolve with flexion maneuvers, such as forward bending or sitting. The mechanism of symptoms may represent venous engorgement of the cauda equina. Differentiation from true vascular claudication of the lower extremities is possible by the lack of (or the delayed onset of) symptoms when the lower extremities are exercised in a flexion posture, such as stationary bicycle riding. Unlike radiculopathy associated with disk herniation, straight leg raising is usually unremarkable and may be a clinical sign to differentiate spinal stenosis from the more unusual presentation of a disk herniation in this phase. The prevalence of disk herniations diminishes in people over the age 55[178] and these are typically far lateral herniations reflecting biochemical alterations of the disk, reduction in elasticity, and degenerative bony constraints. Far lateral herniations may have clinical features not unlike spinal stenosis.

On the battlefield, imaging techniques are not available for assisting in diagnosis of lumbar pain. However, in combat hospitals some imaging is available, and its greatest usefulness will be to rule out associated fractures as a cause of or contributing factor to low back pain. As with any diagnostic tool, imaging techniques of the lumbar spine must be utilized and interpreted in the context of clinical relevance.[179] More advanced imaging techniques may be available at corps level hospitals. In evaluating and treating low back disorders, it is very useful to have knowledge of the relative specificity and sensitivity of various techniques, including plain radiographs, computed tomography (CT) scans, multiplanar reformatted CT scan, myelograms, myelograms combined with CT scans, magnetic resonance imaging (MRI), discography with and without CT scans, and bone scans and single photon emission computed tomography scans (SPECT).[169,171,180–194] Although no radiographic equipment is available in the field of combat, knowledge of the radiographic tests that are useful for specific disorders is helpful in treating low back disorders, including military personnel, as well as injured soldiers in triage hospitals.

In general, plain radiographs of the lumbar spine have low specificity and sensitivity in regards to intrinsic lumbar spine pathology.[195] As previously reviewed, lateral flexion-extension roentgenograms may not provide additional information regarding lumbar hypermobility[183,187] or instability, except in cases of spondylolysis. Facet fracture or spondylolysis may not be appreciated without oblique radiographs, and although plain radiographs may not determine the age of such bony abnormalities, further specific imaging (ie, a SPECT bone scan)[182] can be directed.

The role of noncontrast enhanced CT scans is probably limited in the evaluation of clinically significant abnormalities, unless fracture or other primary bone pathology is being considered. CT scans have low specificity, as shown by the fact that 35% of asymptomatic individuals had radiologist-determined abnormalities on CT scans.[171] Although CT scans may reveal disk herniations, occasionally the distinction between disk material and thecal sac is difficult. Further, extruded disk fragments can be missed and the source of a migrated fragment may be difficult to determine. Typical lumbar CT scans are limited to axial images through the lowest three disk spaces, often skipping the lateral recesses and rarely evaluating the higher lumbar disks or conus medullaris. Myelography, combined with postmyelogram CT imaging, is an effective means of evaluating lateral stenosis, assuming the images are obtained in an overlapping manner, and can also evaluate for lateral disk herniations.[196] Myelography alone will typically miss any lesion that is lateral to the pedicle, such as a far lateral herniation or impinging lateral osteophyte.

With estimates of up to 99% sensitivity, high resolution MRI is extremely reliable in identifying lumbar disk herniations.[184,192] The sensitivity may diminish when evaluating for spinal stenosis, annular fi-

ber tears and even degenerative disks. MRI scans may have limited specificity, providing too many false positives, as demonstrated by Boden and colleagues[169] in a study of abnormal MRI findings in asymptomatic people. Nevertheless, MRI is an excellent tool for evaluating disk herniations at multiple levels, and it frequently supports the possibility of internal disk disruption as an etiology to chronic pain.

Selective injections, including epidural injection, facet joint and selective nerve blocks, are an extremely useful adjunct to other clinical diagnostic evaluation tools. These injections can also potentially provide therapeutic benefit if administered with a corticosteroid. From a clearly diagnostic viewpoint, selective injections can aid in identifying clinically significant lumbar spine pain generators. Differentiating the qualitative and quantitative contribution of the posterior element, discogenic, or radicular pain is the goal of diagnostic selective blocks. Determining a specific pain generator requires precise needle localization, by use of fluoroscopy and contrast dye.[197–202] However, in a combat area, using proper technique and a skilled clinician, these injections can be done with fairly accurate localization to allow some useful information as to the relative proportion of pain generated by a given structure. If necessary, a soldier who requires further evaluation can have these studies done with fluoroscopy and contrast agents in a major medical center.

Another type of injection that can be helpful, particularly in a combat situation, is a trigger point injection. When specific, reproducible myofascial tender (trigger) points are prominent and appear to be causing significant pain and disability, the pain-spasm cycle can be broken to provide temporary relief by either dry needling or a local injection of 1 to 3 cm^3 of lidocaine. These injections work best when the number of tender or trigger points are limited (fewer than three), are well localized, and easily reproducible. Trigger point injections are generally very safe and easy to perform and can be done in a combat field situation.

Rehabilitation

Rehabilitation principles can be applied to all spinal disorders whether these occur in acutely injured or chronic pain patients, injured soldiers in the field of combat, nonsurgical, or postsurgical patients. The benefits of appropriate rehabilitation techniques become more apparent with evidence that even large, extruded disk herniations have, in some circumstances, spontaneously resolved over 1 to 2 years without surgery.[203,204] In field hospitals in the communication zone, aggressive nonsurgical rehabilitation of spine disorders takes on even greater importance in reconstituting our forces in a timely manner.

Any successful spine rehabilitation program relies on adequate diagnoses, which address (*a*) pathoanatomy, (*b*) stage of healing, (*c*) functional phase of spinal degeneration, and (*d*) identification of secondary soft tissue changes. Selected spinal disorders will be discussed in terms of their presentation; tissue injury; and clinical symptom, functional biomechanical, adaptation, and tissue overload complexes. In this way appropriate specific rehabilitation issues will be discussed as predicated on specific anatomic, biomechanical, and functional diagnoses. The section on rehabilitation of acute muscle strain outlines the principles of spinal rehabilitation that are applicable to all disorders of the lumbar spine and should be included in the specific rehabilitation programs for the specific disorders listed below.

Acute discogenic pain. The method of presentation is as an acute injury, occurring with flexion and rotation of the lumbar spine with a patient history of previous episodes of low back pain that have usually resolved unremarkably within 3 to 5 days.

Tissue injury complex includes annular fibers of the disk and chemical or mechanical (or both) irritation of the anterior and dorsal spinal roots, or posterior longitudinal ligament and other pain-sensitive intercanal and foraminal structures, or all of these.

The ***clinical symptom complex*** will be low back and leg pain. Leg pain is usually much more pronounced than back pain when herniation of the disk occurs, whereas more back symptoms occur when there is an annular tear without protrusion of the disk. Pain can occur at any level of the lumbar spine, most commonly at L-4–L-5 and L-5–S-1 areas. Pain is usually worse with flexion and flexion and rotation, and is often relieved with extension.

Functional biomechanical deficit will show soft tissue inflexibilities (muscle, fascia, ligament) due to spasm or tightness, and segmental hypomobility.

Functional adaptation complex will include the loss of normal lumbopelvic rhythm, increased lumbar lordosis, increased loading of posterior elements, and lateral pelvic shift. This will result in ***tissue overload complex,*** including annulus fibrosis, nucleus pulposus, and to the supporting paraspinal ligaments and musculature.

Rehabilitation procedures are shown in Table 8-12, but education is probably the most important com-

TABLE 8-12

TREATMENT FOR LUMBAR SPINE-ACUTE DISCOGENIC PAIN

Time (d)	Treatment
0–1	Proper positioning, 90°/90° traction, education
0–3	Bedrest, antiinflammatory modalities
0–7	NSAIDs, pain medications
3–10	Begin extension exercises
3–10	Traction (often in extension)
10+	Flexibility and strength training progressed
7–14	Walking, cross-country ski machine

NSAID: nonsteroidal antiinflammatory drug

ponent in any back care program, including this acute injury.[205] Instruction in sitting and standing (maintenance of lordosis) and body mechanics is aimed at protecting injured structures and preventing further injury. Review of activities of daily living and nightly living allow practical application of these principles. A short course of bedrest may be appropriate. Prolonged absolute bedrest greater than 3 days has not been shown to reduce disability or dysfunction.[206] In the early stages, modalities may be helpful. Cryotherapy and electrical stimulation are effective for pain control and antiinflammatory effects. The use of NSAIDs and other analgesic medications is advocated early in the treatment of acute discogenic injuries, especially within the first 1 to 2 weeks, as has been previously discussed.

Initial exercises, movement into flexion or extension, depend upon which activity centralizes low back pain (ie, less radicular pain), or does not exacerbate low back pain. Usually, extension exercises are begun by lying prone, with support under the stomach to maintain a neutral position; and progress as tolerated to lying prone, unsupported, with support under the chest; to lying prone on elbows; and then to press-ups (Figure 8-22).[207–210] Lateral trunk shifts must be corrected before initiating extension exercises, or symptoms are likely to increase. Patients can be instructed in self-correction techniques. Theoretically, extension exercises may be effective in reducing pain by decreasing tension in the posterior annular fibers; increasing mechanoreceptor input, which activates the gate mechanism[211]; decreasing tension on the nerve root[212]; changing intradiscal pressure[213]; and allowing anterior migration of the nucleus pulposus.[208] Repeated extension posturing in standing, for use after sitting and forward bending activities should be taught. Contraindications to extension exercises include segmental hypermobility or instability; large or uncontained herniation; bilateral sensory or motor signs, or both; significant increase in low back pain unless associated with concomitant reduction in radicular pain; and increase in radicular sensory disturbance. If hypermobility exists at a segment adjacent to a disk herniation, manual blocking of extension at that level can be applied by the therapist, and patients can be taught to generally reduce motion at the lower lumbar segments. Care must be taken to prevent secondary hypermobility at the thoracolumbar segment, however, which can also occur if extension exercises are emphasized in a patient with lumbar segmental hypomobility. Hyper- and hypomobility are often clinically difficult to assess unless the examining physician has good manual and palpatory skills. Occasionally, hypermobility may be seen on flexion/extension radiographs of the spine where increased motion is occurring at specific segments with varying positions. However, in general, if the specific type of exercise program is increasing the severity and duration of symptoms, it should be avoided and other types of exercises should be contemplated.

The classic Williams flexion exercises[214] may decrease the compressive load to the posterior disk and open the intervertebral foramen, thus, its long history of use in acute disk presentations. Indeed, flexion exercises may be better tolerated in central

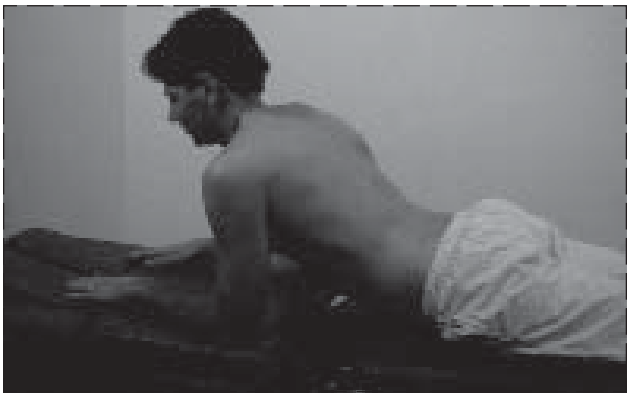


Fig. 8-22. Extension positioning of the lumbar spine with the patient prone and on the elbows.

disk herniations, but acute dural tension is most likely aggravated by flexion postures. Flexion does increase intradiscal pressure and may not be the best position for soldiers with acute disk symptoms. Therefore, in patients with acute discogenic pain, extension exercises, rather than Williams flexion exercises, would probably be the preferable regimen, unless a central disk protrusion is present.

Traction may be an effective treatment for acute discogenic low back pain.[215] Traction can be applied manually, mechanically (typically on an intermittent basis), through the use of gravity (either in an upright or inverted position), by means of autotraction (in which the patient controls the force), and in specific positions of side-lying, usually for far lateral disk herniations. Traction applied in a prone lying position, and neutral to extended lumbar postures can be maintained. Inversion traction has the added advantage of combining extension exercises with the benefits of traction.[215] Although originally felt to decrease intradiscal pressure, actual reduction in pressure is only to 20% to 30%.[213] Traction may be more effective in allowing vertebral body separation, decreasing compressive forces on the lumbar nerve roots by widening the foramen, improving blood flow to the nerve roots, and stretching spinal musculature. The applied force needed to adequately distract the vertebral bodies in a horizontal plane is 25% to 50% of body weight.[215] Less force does not overcome the friction component, and more force will pull the entire body. Inversion gravity traction can distract the lumbar vertebral bodies by 0.3 to 4.0 mm, which can reduce symptoms. Side effects of inversion therapy, however, are not benign and include hypertension, headaches, gastrointestinal reflux, and ruptured berry aneurysms.[216]

Early activity is encouraged. Walking, swimming, or using a cross-country ski machine helps to maintain muscle tone and cardiovascular endurance, provides extension for the lumbar spine, maintains joint mobility, and increases blood flow to the injured segment. Specifics of flexibility and strength training will be discussed later in this chapter.

Acute Posterior Element Pain. Acute posterior element pain is often an acute injury that occurs with the extension and rotation of the lumbar spine, and is usually related to a torsional load on the spine.

Tissue injury complex includes the zygapophyseal joints and surrounding synovium and joint capsule. Also, the posterior longitudinal ligament or intraspinous and supraspinous ligaments may be involved.

TABLE 8-13

TREATMENT FOR LUMBAR SPINE-POSTERIOR ELEMENT PAIN

Time (d)	Treatment
0–3	Cryotherapy, flexion postures, NSAIDs, pain medication
0–3	Relative rest
3–10	Progressive flexion exercises
3–10	Traction (in flexion postures)
7–14	Walking, exercise bike, treadmill

NSAID: nonsteroidal antiinflammatory drug

Clinical symptom complex includes nonradiating low back pain, or referred pain to the buttock or proximal thigh will be present, but rarely below the knee.

Functional biomechanical deficit will present as abnormal pelvic tilt and hip rotation secondary to tight hamstrings, hip rotators and quadratus, and weak erector spinae and hamstrings.

If the condition is chronic, the ***functional adaptation complex*** will appear as flattening of lumbar lordosis, rotation or side bending at the sacroiliac or thoracolumbar area.

Initial ***tissue overload complex*** will be at the synovium and capsule of zygapophyseal joints; when the injury is more chronic, the articular cartilage of zygapophyseal joints will be involved.

Rehabilitation for acute facet joint pain is outlined in Table 8-13, and the initial treatment stages are similar to acute disk pain, that is, education, ice, relative rest, and maintaining positions of comfort. These positions are typically accomplished through neutral to flexed postures. Greater relief may be obtained by hook lying, or 90°/90° positions, that is, hips and knees both flexed at 90° (Figure 8-23). Flexion exercises are theoretically effective by decreasing facet joint compressive forces, stretching hip flexors and lumbar extensors, and strengthening abdominal and gluteal muscles.

Pelvic tilts, or flattening the low back and decreasing the degree of lumbar lordosis, should be performed in multiple positions, including bent knees, straight legs, and standing. Pelvic tilts unload the facet joints and aid in pelvic awareness. Single knee-to-chest maneuvers help stretch the contralateral hip flexors and ipsilateral extensors,

Fig. 8-23. The 90°/90° position to decrease the load on the intervertebral disc and lumbar spine with the hips and knees flexed at 90°.

while double knee-to-chest positions promote stretching of the lumbar and hip extensors. Stretching the hamstring and internal and external rotator hip is also essential. Partial sit-ups strengthen the abdominal muscles. Contraindication to flexion exercises include segmental hypermobility or instability, increase in low back or peripherilization of pain into the lower extremity, or both.

Intermittent mechanical traction in a 90°/90° lying position or inversion gravity traction may be helpful in unloading the posterior elements, lubricating the joints, improving joint nutrition, and reducing pain through mechanoreceptor input. When traction equipment is not available, such as in a combat situation, simply positioning the patient in the 90°/90° position can be helpful. Sustained or static traction will often exacerbate symptoms due to stretch of the facet joint capsule. Prone lying positions should also be avoided.

Maintenance of aerobic activity is essential. It can be accomplished by a stair climbing machine, a treadmill at a slight incline, or a stationary bicycle, if available. These also help to maintain a neutral-to-flexed posture.

Pars Interarticularis Injury

Assessment of the soldier, particularly the young recruit, with localized lumbosacral discomfort should alert the physician to the possibility of spondylolysis or spondylolisthesis. Injury of the pars interarticularis is particularly common in active younger individuals who perform repetitive activities in lumbar extension, flexion, and rotation. These lesions include pars stress reaction, spondylolysis (isthmic) and spondylolisthesis. A pars stress reaction is a bony irritation to the pars interarticularis without the presence of a lytic lesion.[217] This prespondylolytic state may progress to a true spondylolysis. Spondylolisthesis occurs if this defect is bilateral, and forward slippage of the superior vertebral body on the one below occurs, particularly L-5 on S-1.

If a pars injury is clinically suspected (see *clinical symptom complex*, below), a radiographic assessment is indicated. Plain radiographs with an oblique view may show the spondylolytic defect with or without slippage. Not all pars interarticularis injuries will be apparent on radiograph.[218,219] A planar bone scan may demonstrate increased uptake in the pars region when plain radiography is normal. Even when the plain radiograph demonstrates a defect in the pars interarticularis, the planar bone scan is helpful to determine if the fracture is acute or chronic.

A pars interarticularis injury will present as a chronic overload injury, with the *tissue injury complex*, including pars interarticularis, either unilateral or bilateral, usually at L-4 and L-5, where maximum loading and shear occur.[220–222]

The ***clinical symptom complex*** will cause localized lumbosacral discomfort, which will be worse with extension and partially relieved with flexion. This is more common in soldiers whose activities require repetitive flexion, extension, and rotation. A normal neurologic examination is usually indicated.

Functional biomechanical deficit will be hamstring tightness, and the ***functional adaptation complex*** will be loss of lumbar lordosis. The ***tissue overload complex*** will be pars interarticularis.

Rehabilitation is outlined in Table 8-14. Management is often controversial and ranges from rest and restriction of activity to rigid immobilization. It is

TABLE 8-14

TREATMENT FOR LUMBAR SPINE-PARS INTERARTICULARIS INJURY

Time	Treatment
0–3 mo	Restrict repetitive extension exercises
0–10 d	Pain medication, NSAIDs
0–6 wk–6 mo	Antilordotic bracing
10+ d	Flexion exercises, stretching hamstrings

NSAID: nonsteroidal antiinflammatory drug

determined by the severity of symptoms. In cases of symptomatic spondylolysis, Micheli[223,224] favors a rigid polypropylene brace constructed with zero degrees of lumbar flexion (a modified Boston overlap brace) to be worn 23 hours a day for 6 months. Bracing from 6 weeks up to 3 months has also been suggested.[224] Unilateral defects may have a greater chance of healing than do bilateral defects. The presence of a bilateral pars defect on plain radiographs may indicate a significant decrease in the chance for bony healing, even with immobilization.[225] According to Wiltse (personal communication 1991), if the bone scan or SPECT scan demonstrates increased uptake in the *absence* of plain radiographic evidence of a pars defect, longer immobilization may be warranted due to a greater chance of bony healing. Whether relative rest alone or bracing, or both, are utilized, a thorough program of spinal rehabilitation is essential before the soldier's return to military activities.[226] Such programs emphasize hamstring stretching and antilordotic positioning and strengthening. If back pain persists despite bracing and rehabilitation, or if neurologic symptoms develop (or both), surgical intervention is most likely necessary.

TABLE 8-15

TREATMENT FOR LUMBAR SPINE-STRAIN/ CONTUSION

Time (d)	Treatment
0–2	Relative rest, back education
0–3	Antiinflammatory modalities, pain medications
3–10	Initiate activity, regain flexibility
10–21	Improve joint mobility
14–28	Therapeutic exercises
28+	Cardiovascular conditioning, maintenance program

Acute Muscles Strain and/or Contusion

The method of presentation will be acute traumatic injury, with the *tissue injury complex* including the muscle tissue or thoracolumbar fascia.

Clinical symptom complex will include localized lumbosacral discomfort, usually from a blow to the back; a tearing sensation while lifting or other traumatic event; or there will be a more subtle history of aggravation, such as a constant repetition of a new activity.

Functional biomechanical deficit will include segmental hypomobility of the three-joint complex secondary to muscle spasm and guarding.

Functional adaptation complex will show loss of active and passive segmental and combined motions; the ***tissue overload complex*** will be muscle myofilament.

Rehabilitation is outlined in Table 8-15. Initial treatment should focus on limited periods of rest (less than 3 days) with early gentle activity within the pain free range. Progression is then made to mild activities and mobilization in the subacute phase. Ice massage, cold packs, and electrical stimulation are often helpful. Restoration of full function (strength and mobility) and normal posture should be the most important aspects of treatment. Regaining soft tissue flexibility and segmental motion is the first goal in the subacute phase. This can be accomplished through a variety of manual therapy techniques, including myofascial release,[227] joint mobilization or manipulation,[228,229] muscle energy techniques,[227] and stretching.

In all lumbar problems, maximization of the lower extremity muscular flexibility is especially important to allow normal lumbar motion. Due to their attachments to the pelvis, the hip flexors, extensors, and rotators have a great influence on positioning the lumbar spine. Adequate hip muscle flexibility allows for hip joint motion independent of lumbar segmental motion and is essential for the use of proper body mechanics and posture. Poor flexibility will cause excessive stress to be transmitted to the lumbar motion segments and sacroiliac joints.[230] Typical patterns of lower extremity inflexibility include hamstring, gluteus maximus and gastrocnemius-soleus, hip flexor, tensor fascia, and quadriceps groups. Tight hip flexors (iliopsoas) and quadriceps (rectus femoris) often cause extension and rotation hypermobilities in the lumbar spine. If the iliopsoas is contracted in a shortened position, the pelvis is maintained in excessive anterior tilt, placing the hip extensors (gluteus maximus and hamstrings) at a mechanical disadvantage. Therefore, early recruitment of lumbar extensor muscles (erector spinae) occurs and will result in increased shear or torsion stress to the intervertebral disk. If hip rotators are tight, abnormal kinetic chain motions in rotation occur, causing more stress at the joints and sacroiliac area and over the extensor muscle attachments. Self-stretching techniques should be taught as early as possible to allow ac-

tive involvement in the rehabilitation program. It is essential to stretch with as neutral a pelvic position as possible, since excessive anterior and posterior pelvic tilt will diminish the benefits of these flexibility exercises.

After muscle flexibility is achieved, optimal joint mobility must be obtained. Assessing spinal segmental motion and restoring motion to restricted or hypomobile segments again demands precisely applied manual techniques.[228,229,231] Hypomobility can result from impairment of the soft tissue supporting structures (ie, muscle hypertonus or fascial restriction) or from the intrinsic components of the three-joint complex (ie, lumbar disk degeneration or facet joint dysfunction). Adjacent hypomobile segments can cause increased stress on injured segments, and injured segments may cause hypermobilities at adjacent noninjured joints. Mobilization techniques[105,227] do not provide long-term relief to painful spinal segments primarily caused by discogenic abnormalities, and do not reduce disk herniations. However, temporary relief may occur from mechanoreceptor stimulation, stretch of adhesions, or restoration of shortened muscle length. Muscle energy techniques can be utilized with mobilization to improve segmental mobility.[227] These techniques use the patient's own isometric muscle contraction to relax hypertonic muscles by resetting the gamma gain in the muscle spindles. A series of isometric muscle contractions is elicited at varying degrees of joint positioning. The series of isometric contractions can also be utilized to mobilize a joint and may be less painful than passive mobilizations.

Once appropriate muscle flexibility and joint mobilization is accomplished, appropriate exercise is prescribed. This can often be determined by comparing flexibility side to side with the asymptomatic extremity, or to what the initial degree of flexibility was in the spine when the soldier presented the complaints. Exercises can be used to control pain (possibly through endorphin release), optimize tissue repair and regeneration, and improve muscle performance. Exercise to improve the function of the spinal muscles is generally known as spinal stabilization.[232,233] Stability of the lumbar spine is provided by bony architecture, disk mechanics, ligamentous support, muscular strength, endurance, and coordination. Optimal muscle strength can protect the spinal motion segment from chronic repetitive shear stress or acute dynamic overload. The concept of spine stabilization implies a muscle fusion. Spine stabilization exercises use force couple muscles, acting in concert around a mobile area, to provide a stable base, smooth motion, and efficient force modulation and transfer in the lumbar spine. By flexing, extending, and rotating the spine, the various abdominal, pelvic, and trunk muscles that attach to the thoracolumbar fascia act as an abdominal corset. Increased intraabdominal pressure does not appear to be the mechanism of stabilizing the disk and joint, but rather the reduction of shear forces across the three-joint complex (the posterior elements and the intervertebral disk) through abdominal cocontraction and tension generation in the thoracolumbar fascia and midline ligaments.

The muscular stabilizers of the spine include the intersegmental muscles (ie, multifidi, rotatores, interspinalis) abdominal muscles (particularly the transversus abdominus and internal obliques), latissimus dorsi, erector spinae, iliopsoas, and quadratus lumborum. The role of the smaller intersegmental muscles in stabilizing the lumbar spine is controversial; however, it is felt that they are effective as stabilizers, can balance shear forces, and apparently produce rotation, although not as primary rotators.[116,234–236] The multifidi also secondarily maintain lumbar lordosis by the nature of the force vector posterior to the vertebral bodies. The multisegmental muscles of the spine have been shown to be more efficient prime movers.[235]

Any spinal exercise must address stability before movement, endurance, and strength. Following spinal injury, the intersegmental muscles act as postural stabilizers (or tonic) of the spine, administering to fatigue first and then atrophy. Therefore, the initial stabilization exercises are directed toward those muscles that can control individual segmental mobility. Typically, these are manually resisted exercises of the trunk and are limited to short arcs performed in rotation, flexion, extension, and sidebending. The next phase of stability training involves direct and indirect strengthening of muscle groups through a variety of exercises performed in a neutral spine posture. Neutral spine is defined as the midpoint of available range between anterior and posterior pelvic tilt, not the absence of lordosis. The advantages of neutral positioning are (*a*) its loose-packed position, which decreases tension on ligaments and joints; (*b*) its allowance of more balanced segmental force distribution between the disk and facet joints; (*c*) its closeness to the center of reaction, allowing movement into flexion or extension quickly; (*d*) it provides the greatest functional stability with axial loading; and (*e*) it is usually the position of greatest comfort.[116] Training begins with exercises designed to help locate neutral spine in a variety of body positions, which increases awareness of lumbar and pelvic motion. These are

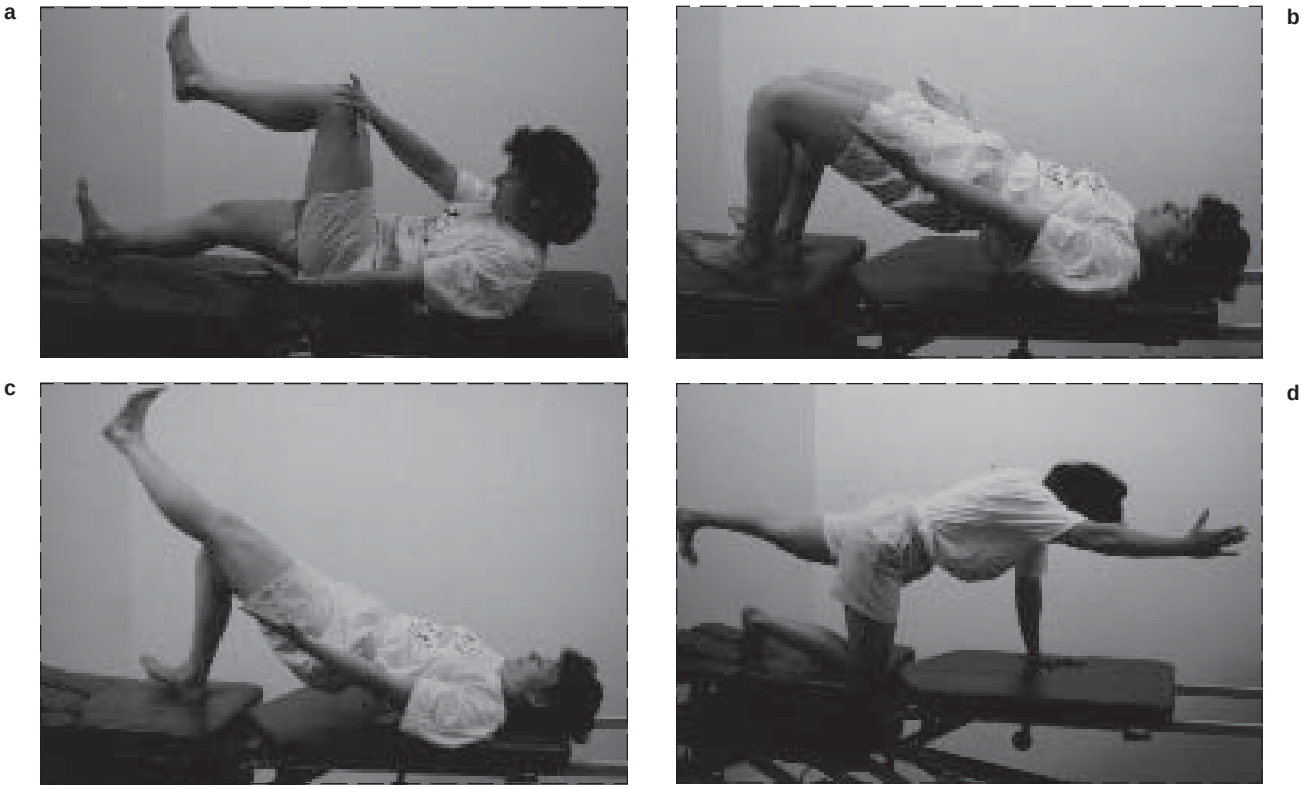


Fig. 8-24. Spine stabilization exercises for the low back. (**b**) **S**traight bridging of the back. The patient is instructed to keep the back straight to strengthen the gluteals and spine extensors, and using the abdominal muscles to maintain a neutral spine position. (**c**) Advanced bridging, progression to single leg stance for proximal stability. (**d**) Proximal stabilization of the "down" arm and leg with dynamic extensor strengthening of the "up" arm and leg.

followed by exercises of the extremities while maintaining a neutral spine, and later with the addition of resistance to the extremities, either manually or through use of weights. These exercises are performed slowly with the emphasis on precise pelvic control, which will facilitate neuromuscular coordination and enhance endurance and strength gains (Figure 8-24). These neutral spine stabilizing exercises will also emphasize the smaller postural stabilizers.

Strengthening prime movers, including abdominals and erector spinae, ultimately is required. Historically, abdominal exercises, especially sit-ups, have been emphasized as part of a low back exercise program. Sagittal plane sit-ups are utilized in the stabilization routine, but are limited to partial curl-ups lifting the head and upper body only. During this initial phase, the obliques and rectus abdominus are activated, whereas in the second half of a full sit-up, the iliacus and rectus femoris provide the main muscle force.[233] Lower extremity strength is necessary, as well as these muscles working in a coordinated manner with the trunk muscles for most functional activities. This is especially true during lifting, when the gluteal and hamstring muscles are the prime posterior rotators of the pelvis and trunk. Quadriceps strengthening is also important; it allows adequate support of body weight during use of proper body mechanics such as squatting. Torque to the lumbar spine is intrinsic to most activities, especially in work, athletic competition, and military duty.

After proper strength training and stabilization activities have been initiated, cardiovascular conditioning becomes essential. Improved cardiac fitness has been recognized to protect certain workers with high physical demand from back injury.[237] At hospitals in the Communication Zone (COMMZ), cardiovascular conditioning can be attained with aerobic exercises, such as running. Although not now available in the COMMZ, light portable cross-country ski machines could be used for this purpose.

The lumbar spine problems discussed in this section are in no way a comprehensive presentation of the myriad disorders seen in patients with lumbar spine complaints. Congenital disorders, metabolic

and neoplastic entities, as well as degenerative spinal conditions, such as spinal stenosis and spondylosis, have not been discussed. It is crucial to remember that if symptoms persist in a soldier who is believed to have a contusion or strain, the physician must reassess the diagnosis and consider other etiologies for the back symptoms (disk spondylolysis, posterior element dysfunction, and so forth). Most causes of low back pain resolve within 4 to 6 weeks. The aim of this section has been to focus on acute injuries that would be most commonly encountered in the field during war, and would need rapid evaluation and aggressive nonsurgical rehabilitation using limited resources. As stated previously, the discussion on specifics of strength, flexibility, and endurance pertains, in many respects, to all lumbar spine disorders and should be instituted in any spinal rehabilitation program.

TABLE 8-16

TREATMENT FOR HIP-TROCHANTERIC BURSITIS

Time (d)	Treatment
0–4	NSAIDs, modalities, ice, friction massage
2–14	Stretch ITB, hip external rotators, flexors, abductors, extensors
7–14	Strengthen hip adductors

ITB: iliotibial band
NSAID: nonsteroidal antiinflammatory drug

Hip Disorders

Trochanteric Bursitis

Bursitis over the greater trochanter is caused by friction where the ITB passes over the bony prominence of the greater trochanter. In response to friction, the potential space of the bursa becomes inflamed and fills with fluid. It is usually caused by excessive activity and insufficient stretching of the hip musculature. Femoral neck and ischial stress fractures, strain of the hip abductors, and referred back pain can mimic trochanteric bursitis.

Method of presentation. Bursitis is an acute injury or a chronic exacerbation of a preexisting condition.

Tissue injury complex. This will occur to the greater trochanteric bursa.

Clinical symptom complex. There will be tenderness just posterior to the greater trochanter, aggravated by contraction of the tensor fascia lata with the hip abducted against resistance.

Functional biomechanical deficit. There will be an inflexible ITB and hip external rotators, and weak adductors.

Functional adaptation complex. Increased external rotation at the hip will result in altered gait and running patterns.

Tissue overload complex. This will occur at the ITB proximally at the hip.

Rehabilitation (Table 8-16). Initial treatment consists of NSAID medication and modalities and stretching of the ITB, external rotators of the hip, quadriceps, and, often, the hip flexors. Stretching of the gluteus maximus, which inserts into the ITB, is also important. Strengthening the hip adductors and correcting an imbalance between abductors and adductors can decrease trochanteric bursitis symptoms. In severe cases, a few days of cane-assisted ambulation may be helpful. Ice and deep friction massage, as well as contrast baths, may be of some benefit.[238] Corticosteroid injections can be used in recalcitrant cases. Occasionally, a leg length discrepancy, causing abnormal pelvic tilt, can be an aggravating factor in trochanteric bursitis and may benefit from a shoe lift or orthotics. Also, correction of excessive posterolateral heel wear can decrease symptoms. In most cases, symptoms will usually respond to treatment in 7 to 10 days. Chronic cases may require extended treatment.

Iliotibial Band Syndrome

The iliotibial band or tract is a thickened portion of fascia lata that passes down the lateral aspect of the thigh and inserts into Gerdy's tubercle on the lateral tibial condyle.[23] Inflammation and symptoms can occur in any of three locations: (1) over the bony prominence of the greater trochanter, as discussed above; (2) most commonly, over the lateral femoral condyle; and (3) at the insertion at Gerdy's tubercle into the tibia. Factors associated with ITB syndrome are tibia vara, hyperpronation, cavus foot, worn outer soles of shoes, and ITB contracture.[239] The differential diagnosis includes hip pathology; meralgia paresthetica; upper lumbar radiculopthy; knee disorders, especially lateral collateral ligament injuries and lateral meniscal tears; and popliteus tenosynovitis.

ITB syndrome presents as an acute exacerbation of chronic overuse injury, and the ***tissue injury complex*** will include the proximal portion of the ITB

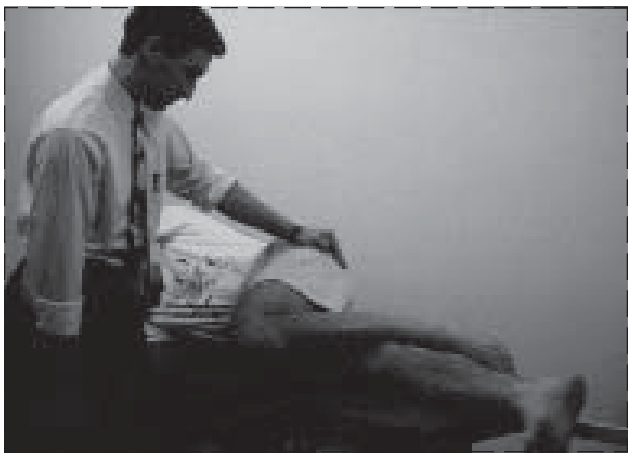


Fig. 8-25. Modified Ober test to evaluate for inflexibility of the iliotibial band (ITB). The bottom leg is extended. The top hip is flexed. The greater trochanter is perpendicular to the table surface. The degree of ITB tightness is measured as the number of fingerbreadths between the medial part of the knee and the table surface in this position.

over the greater tuberosity, the distal portion over the lateral femoral condyle, or insertion to the tibia.

Clinical symptom complex includes (*a*) localized pain, usually over the lateral femoral condyle, and worse with running activities, especially on banked surfaces or hills; (*b*) abduction against resistance; and (*c*) a positive Ober's test (Figure 8-25).

The *functional biomechanical deficit* will be at the inflexible ITB, with the ***functional adaptation complex*** presenting as increased lateral patellar tracking, external rotation at hip, internal rotation of the leg, and functional pronation of the foot.

Tissue overload complex will be varus loading on the lateral aspect of the knee.

Rehabilitation is outlined in Table 8-17. Therapy for ITB syndrome includes application of ice with ice massage for up to 20 minutes at a time, followed by active stretching of the ITB, the tensor fascia lata, the gluteus maximus, and often the hip flexor muscles (Figure 8-26). Antiinflammatory medications and modalities are used as available. Local injection of anesthetic and corticosteroid can be helpful, especially at the lateral femoral condyle, where the injection is made both anterior, posterior, and deep to the ITB.[239] Correction and support of the functionally pronated foot must be addressed. Strengthening exercises for the adductors of the hip are important to counteract the abduction moment caused by a tight ITB. Strengthening of the gluteus maximus and tensor fascia lata are also important to avoid overuse of these muscles that form the ITB.

TABLE 8-17

TREATMENT FOR ILIOTIBIAL BAND SYNDROME

Time (d)	Treatment
0–2	Ice massage, NSAIDs
2–14	Stretch ITB, TFL, gluteus maximus
7–10	Strengthen adductors, gluteus maximus, TFL
14–21	Local injection (if necessary)

ITB: iliotibial band
NSAID: nonsteroidal antiinflammatory drug
TFL: tensor fascia lata

Reduction in mileage running, along with a course of antiinflammatory medication, will frequently cause rapid reduction of pain within less than 1 week.[240] However, more difficult cases may take as long as 6 weeks to resolve.[241] Rarely, surgery may be necessary for recalcitrant cases.

Hamstring Strain

Hamstring strains are a frequent cause of disability in the military population. The injury may occur anywhere along the muscles, but is more common at the junction of the proximal muscles and tendons. The mechanism involves forced flexion of the hip with the knee extended.[242] Sudden, forced change in the musculotendinous length may result in strain or rupture at the junction of the muscle and tendon. Hamstring strains are associated with inadequate hamstring flexibility, inadequate warm-up, exercise fatigue, poor muscular coordination, abnormal or excessive pelvic tilt, previous injury, and imbalance between hamstring and quadriceps strength.[243–245]

Hamstring strain generally presents as an acute traumatic injury with a ***tissue injury complex*** involving the myotendinous junction disruption in the upper third of muscle secondary to eccentric overload.[28]

The *clinical symptom complex* includes pain in the upper third of thigh, swelling, bruising, mass in the thigh, muscle weakness, and tenderness on bending or prolonged sitting.

The *functional biomechanical deficit* will be decreased knee extension, increased hip flexion, and worsened hamstring, that is, quadriceps strength ratios.

Fig. 8-26. (**a**) Stretching exercise for the internal rotators of the hip and the hip extensors by placing the foot (ankle) on the knee and pulling it toward the chest. (**b**) Stretching exercises for the hip flexors. The patient is instructed to maintain a posterior pelvic tilt while actively extending the hip.

The *functional adaptation complex* will be decreased stride length with running, and the ***tissue overload complex*** will be at the myotendinous junction.

Rehabilitation is outlined in Table 8-18. Initial treatment for acute hamstring injury in the proximal midthigh or distal thigh should be rest and application of ice directly to the injured area, compression wraps with the knee in flexion to decrease edema and hemorrhage, medication, and non–weight bearing.[239] During the initial 24 to 48 hours, icing, with the knee in extension, should be done for 20 minutes at a time 3 to 4 times a day to put early, gentle stretch on the hamstrings. Isometric strengthening should also be instituted early, and advanced to progressive resistance exercises as tolerated. Within 3 to 5 days, electric stimulation and gentle passive massage and stretching should be initiated. Modified flexion exercises (Figure 8-27) are started as early as pain permits to progressively stretch the healing hamstring muscle. Activity can begin when the soldier can perform the modified Williams flexion exercise of the bent knee to straight leg raise and have the leg raised beyond a 90° angle from the perpendicular while keeping the knee perfectly extended and the toes dorsiflexed.[246] Generally, for first degree strains, where the injury is mild and there is only slight loss of extension, this point will be reached in 4 or 5 days. For a second degree strain, which has more significant swelling, pain, and loss of flexion, this point is usually reached in 7 to 10 days. Third degree injuries, where the loss of extension is greater than 45° and a more severe injury has occurred, may take 3 to 4 weeks to reach the point where activity can begin. To avoid recurrent injury, the musculotendi-

TABLE 8-18

TREATMENT FOR HAMSTRING STRAIN

Time (d)	Treatment
0–3	Ice, compression, decrease weight bearing
3–7	Electric stimulation, passive ROM exercises, modified Williams stretching
7–21	Progressive stretching of hamstrings
14–21	Begin isometrics of hamstrings, quadriceps
14–28	Stationary bicycling
21+	Eccentric and concentric strengthening of hamstrings

ROM: range-of-motion

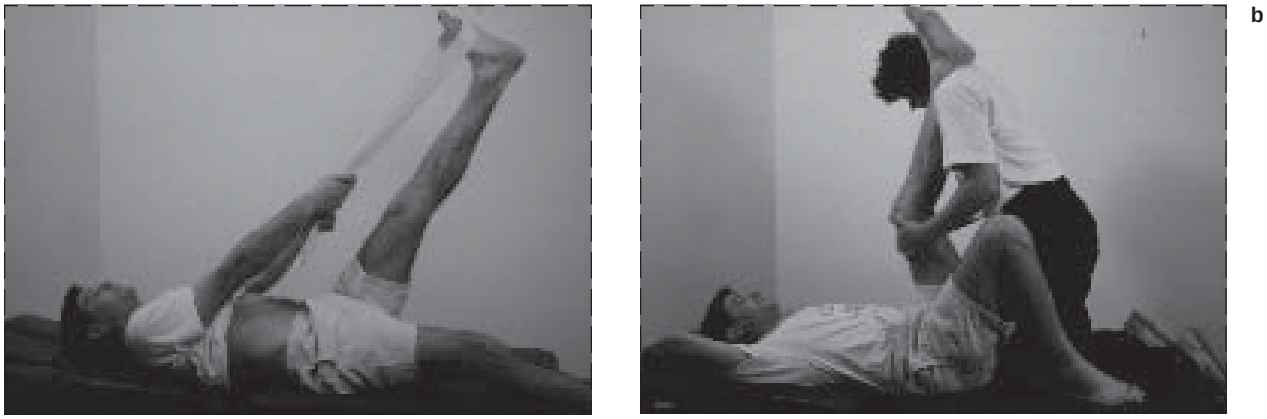


Fig. 8-27. Flexion exercises to stretch the hamstrings. (**a**) The straight leg raise as shown is difficult to hold for the 30 seconds required to have a beneficial effect. Alternatively, a useful exercise consists of lying in the doorway with the heel on the frame. (**b**) Therapist-assisted hamstring stretches. Ideally, the knee is not allowed to bend at all during this exercise.

nous unit must be slowly stretched to reestablish its full length before significant strengthening occurs. Stationary bicycle riding and more aggressive eccentric and concentric strengthening can proceed. Return to full activity requires restoration of muscle length and strength, with muscle strength to be at least 90% of the strength of the uninvolved side, and no pain experienced with isometric contraction.[247]

Knee Disorders

Ligament Injuries: Anterior Cruciate Ligament

The ACL functions to prevent both excessive anterior movement of the tibia on the femur, and abnormal tibial internal and external rotation. It is needed to maintain the normal biomechanical function of the knee; its loss not only produces abnormal kinematics, but frequently results in major degenerative changes.[248–250] The mechanism of injury can be by hyperextension of the knee, varus stress with internal rotation, and extremes of valgus and external rotation at the knee. Injuries are graded, from 1 to 4, by the degree of anterior displacement of the tibia with respect to the femur on the Lachman or Anterior Drawer tests. Grade 1 injuries have 0 to 5 mm of displacement; grade 2, 5 to 10 mm; grade 3, 10 to 15 mm; and grade 4, greater than 15 mm of displacement. Surgical vs nonsurgical treatment is controversial.[249–253] However, postoperative complications are more likely if surgery is done within the first 2 weeks after injury, when the knee is acutely inflamed and range-of-motion is restricted.[254] In a war situation, the initial important considerations are whether (*a*) the knee is stable enough to proceed with conservative, nonsurgical rehabilitation and (*b*) there are associated injuries, such as meniscus tear or tibial plateau fracture. The focus here will be on the nonsurgical and postsurgical issues that need to be addressed to adequately rehabilitate an anterior cruciate deficient (or reconstructed) knee.

The method of presentation is as an acute injury, with the ***tissue injury complex*** including the ACL part, or all, of the two major bundles (posterolateral, anteromedial).

The clinical symptom complex will be a sudden pop on the knee after rotational injury, occurring with either applied external force or simple axial loading. This will result in the acute onset of knee effusion, and a giving-way sensation.

The functional biomechanical deficit will be the loss of rotational stability of the knee, especially anterolaterally.

Functional adaptation complex will be the loss of ability to rotate on the foot in the midstance position; the ***tissue overload complex*** is the ACL.

Rehabilitation is outlined in Table 8-19. The anterior cruciate deficient knee has abnormal motion under stress or when involved in aggressive physical activities. This abnormal motion can be in a rotatory direction or in the anteroposterior direction, and in most cases, results in synovitis, which inhibits the rehabilitation program.[255] In any ACL rehabilitation program, the important factors to consider are the specificity of the treatment exercises, the positioning of the knee, the role of antagonist and agonist muscles, proprioceptive retraining, and the use of closed chain vs open chain exercises.[256,257]

TABLE 8-19

TREATMENT FOR ANTERIOR CRUCIATE LIGAMENT

Time	Treatment
0–7 d	Early ROM exercises, weight bearing as tolerated with immobilizer, isometric quadriceps and hamstring cocontraction
7–14 d	Full extension, hamstring curls, one-legged quarter squats, step-ups, calf raises
3–6 wk	Discontinue immobilizer, leg presses, stationary biking
6–8 wk	Jump rope, figure-8 drills, bracing for cutting or pivoting activity

ROM: range-of-motion

As soon as practical, acute injury of the ACL, without other major ligamentous or cartilage damage, can be treated with NSAIDs and physical therapy. This will reduce inflammation and restore full range-of-motion.[248] To control pain, crutches and immobilizing splints may be helpful, but are rarely necessary for more than a few days. When available, it may be helpful to use therapeutic modalities, such as interferential current or a TENS unit to decrease pain, cryotherapy to decrease edema and pain, and electric stimulation to retard atrophy and neuromuscular reeducation. Either postoperatively or acutely after injury, it is important to begin early passive full extension of the knee and weight bearing as tolerated.[254] Avoidance of hyperextension of the knee is sought to decrease the stress on the injured or repaired ligament. Postoperatively, or within the first week after injury, abduction and adduction straight leg raises, extension straight leg raises, and hamstring curls are initiated. Quadriceps exercises can be delayed for 2 to 3 weeks, and initial use should only be isometrically to avoid an increase in anterior translational forces on the torn ACL or the postoperative healing graft. Active extension exercises can be initiated at about 6 weeks after acute injury or postsurgery. Once swelling and pain have subsided, isometric hamstring and quadriceps strengthening are initiated with the knee resting on a rolled towel. Cocontraction of the quadriceps and hamstring muscles is needed to dampen the anterior shear force from the quadriceps contraction on the torn or healing ACL. Initially, straight leg raises are avoided because they may increase the anterior subluxation of the tibia on the femur.[258] Isometric internal and external tibial rotation exercises, which are added once the patient has 90° of knee flexion, have been shown to decrease abnormal tibial rotation.[259]

Isotonic exercises for the upper body, the contralateral leg, the hips, and the ankles, can be started early in the rehabilitation process. Also, cardiovascular endurance training with one-legged cycling and rowing, or upper extremity ergometry can be used. Neuromuscular and proprioceptive retraining become more important as good, closed chain kinetic strength is developed. Closed chain kinetic strengthening involves activities such as a partial squat, where the foot is fixed while leg muscles are being exercised. Active hamstring control, which reduces the pivot shift, was present in 95% of patients with ACL injuries who successfully avoided surgery.[251] Balance board activities are an excellent source of proprioceptive training. Later in the rehabilitation program, activity specific drills need to be incorporated to prepare the soldier for returning to full, unrestricted duty. Often a derotation type of brace may give the soldier more proprioceptive feedback when returning to active duty. Total rehabilitation time is variable, ranging from a few weeks for a mildly strained ACL to 6 to 8 months for a reconstructed ACL with concomitant injuries to the menisci, collateral ligaments, or other structures that complicate the rehabilitation process.

Ligament Injuries: Medial Collateral Ligament

The medial collateral (MCL) or tibial collateral ligament (TCL) is the primary restraint to medial joint opening.[260,261] The mid-third medial capsule is a secondary restraint to valgus stress and is usually torn with more force than necessary to tear the tibial collateral ligament alone.[262] The mechanism of injury to the MCL is most commonly a contact force to the lateral or posterolateral aspect of the knee. The collateral ligament complexes, due to their extracapsular environment with extensive blood supply, have a high potential for spontaneous healing. Lesions of the MCL are graded from 1 to 3, with grade 1 injuries being a stretch to the MCL fibers; grade 2, an incomplete tear of the MCL; and grade 3, a complete ligamentous disruption.

The method of presentation is as an acute, usually traumatic, injury, and the tissue injury (***tissue injury complex***) will be to the medial (tibial) collateral ligament.

The clinical symptom complex may include acute medial knee pain, swelling and tenderness over the site of injury, usually within 24 to 36 hours follow-

ing the injury. Occasionally, there will be complaints of the knee giving way into valgus, usually from a valgus force applied to the knee with external tibial rotation.

The functional biomechanical deficit will be a decrease or loss of medial stability of the knee when valgus force is applied.

Functional adaptation complex will present as ambulation with a flexed knee (avoidance of a full knee extension that stresses MCL fibers), and with the ***tissue overload complex;*** stress will occur on the MCL, and in the quadriceps and hamstring muscles.

Rehabilitation is outlined in Table 8-20. Following collateral ligament injury, there are time constraints for ligament healing. Appropriate restrained joint motion will allow physiologically tolerable stresses, improved cartilage nutrition, diminished muscular atrophy, and enhanced stimulation of collagen healing. All grades of medial collateral ligamentous injuries can be treated nonoperatively.[257, 263–270] Initially, ice is used for pain and edema reduction. For incomplete tears, early motion is advocated, usually in a hinged cast brace, which is initially set between 30˚ and 90˚ of flexion. Full extension is avoided during early treatment because this position puts increased tension on the healing ligament. Full weight bearing with the use of crutches can be employed for several days until the acute symptoms subside. Progression to full weight bearing and free range-of-motion is advocated as the swelling and pain decrease.[262] This may be within 3 to 5 days in a grade 1 injury, or 2 to 6 weeks with a grade 2 injury.[271,272] Grade 3 injuries can be immobilized in a cast for 2 weeks, followed by a cast brace treatment for 4 additional weeks.[271] Isometric quadriceps strengthening, straight leg raises and strengthening of the hip flexors, adductors, and abductors can be initiated early. Progressive resistive exercises are begun when full range-of-motion is obtained, and an isolated grade 3 lesion of the MCL will also respond well to nonoperative treatment and actually regain strength in a significantly shorter period of time than will patients who have had surgery.[272] Care must be taken during hip exercises to avoid valgus stress of the knee. During isotonic training of hip musculature, weights will need to be placed proximal to the knee to avoid such valgus stresses. Grade 3 MCL tears may require a cast brace from 30˚ to 90˚ of flexion for 2 to 6 weeks. Conditioning on a stationary bicycle, when available, is helpful in MCL rehabilitation, because of the varus moment at the knee with cycling and the subsequent reduced stress on the MCL. Active duty is resumed when the knee has full range-of-motion, good muscular control, and no tenderness. Total rehabilitation time can be as short as 2 weeks or as long as 8 to 10 weeks.[258]

TABLE 8-20

TREATMENT FOR KNEE-COLLATERAL LIGAMENT INJURY

Time (d)	Treatment
0–14	Cast brace 10º internal rotation of tibia, 30º–90º flexion (grades 2 and 3)
0–14	Multiple angle isometric strengthening of quadriceps, hamstrings, adductors, abductors, along with straight leg raises
14–21	Open hinge brace to 10º–90º (grades 2 and 3)
21–28	Remove brace, weight bearing, squats (partial), step-ups, toe raises, heel raises
28+	Light jogging

Patellofemoral Pain Syndrome

Patellofemoral pain syndrome is one of the most common musculoskeletal injuries seen in the athletic population. Similarly, these problems are seen in the military population. Repeated stresses applied to the patellofemoral joint in deceleration maneuvers are a common cause of injuries to this joint.[272] Many predisposing factors to patello-femoral pain exist, including vastus medialis obliquus dysplasia, vastus lateralis hypertrophy, extensor mechanism malalignment, high and lateral patellar posture, increased Q-angle, and bony deformity.[273–275] The differential diagnosis of patello-femoral pain includes referred pain from the hip and low back, osteochondritis dessicans of the femur or patella, bone tumors (especially in cases of unilateral symptoms), osteoarthritis, inflammatory joint disease, meniscal pathology, or a synovial plica.

The method of presentation is as a chronic overload injury, and ***the tissue injury complex*** will include the patellar cartilage and synovium, and tendon insertion into the patella.

The clinical symptom complex may include peripatellar pain (which will be worse with increased knee flexion), positive "theater" sign (pain upon arising after sitting for a prolonged period of time), crepitus, pain when descending stairs, and patellar compression.

The functional biomechanical deficit will include insufficiency of medial quadriceps musculature; inflexibility of the ITB, lateral retinaculum, hamstrings, and gastrocnemius muscles; alteration in patellofemoral tracking; and hamstring muscle weakness.

The functional adaptation complex may present as a knee flexion contracture, increased pain with running and axial loading of the knee, and jumping from the opposite leg, with the ***tissue overload complex*** being in the lateral retinaculum, and patellar tendon.

Rehabilitation is outlined in Table 8-21. Symptomatic treatment may start with antiinflammatory medications and modalities. Lower extremity malalignment problems, such as genu varum, tibia vara, hindfoot varus, and forefoot pronation, which if present, can cause a compensatory subtalar joint pronation and obligatory internal tibial rotation, must be corrected. Orthotics are often used for this. Flexibility exercises must focus on many lower extremity muscles, in particular the ITB, because of its insertion into the lateral aspect of the patella and laterally deviating forces on the patella. The hamstring and gastrocnemius muscle must also be exercised because of their propensity to shorten and increase the patellofemoral joint reaction forces with increasing flexion of the knee. This compression has been documented to rise sharply after 30° of knee flexion and can reach eight-fold the body weight with a full squat.[276] Manual medial glide and tilt of the patella may be employed specifically to stretch the tight lateral retinaculum.[273] Initial strengthening of the quadriceps and hip flexors is done isometrically and with straight leg raises.[277] Short arc (–30° extension to 0°) quadriceps exercises may be helpful, although the selectivity in strengthening only the vastus medialis obliquus is debatable. Often, multiple angle isometrics of the quadriceps can strengthen the quadriceps without the significant increase in patellofemoral joint reaction forces that occurs with isotonic exercises. Imbalances between the medial and lateral rotators, and adductors and abductors of the hip need to be addressed, because they may lead to excessive medial rotation and adduction of the hip during the stance phase of gait with an associated increased valgus vector at the patellofemoral joint. Closed chain kinetic exercises, with cocontraction of quadriceps, hamstring, and gastrocnemius-soleus muscles are important to reduce excessive forces across the patella. Furthermore, partial squats (one quarter of a full squat) will also eccentrically load the knee, which will be more physiologic in nature. MacConnell[278] has described an approach to neuromuscular reeducation of knee musculature combined with patellar taping that may be very effective for patellofemoral pain problems. The use of a knee sleeve with fenestration for the patella has also been occasionally helpful.[279]

TABLE 8-21

TREATMENT FOR KNEE-PATELLOFEMORAL PAIN

Time (d)	Treatment
0–3	NSAIDs, antiinflammatory modalities, correct alignment
3–14	Flexibility for ITB, hamstrings, gastrocnemius-soleus
7–14	Isometric strengthening, ie, SLR, multiple angle isometrics, adductors and abductors strengthening, taping
14–28	Closed chain kinetic exercises

ITB: iliotibial band
NSAID: nonsteroidal antiinflammatory drug
SLR: straight leg raise

Leg, Foot, and Ankle Disorders

Medial Tibial Stress Syndrome

Medial tibial stress syndrome, or shin splints, can result from a number of different factors that cause pain and discomfort in the anteromedial or posteromedial part of the lower leg.[241] This usually occurs after repetitive overuse in walking and running, and these conditions include tibial stress fractures (to be discussed subsequently), posterior tibial tendinitis, and periostitis, stress changes at the attachment of the soleus fascia, and compartment syndromes.[280–283] Factors contributing to shin splints include weak anterior leg muscles, improper footwear (hard heel and minimal cushion), varus foot, forefoot imbalance, weak posterior leg muscles, tight heel cords with equinovarus, hypermobile, pronated feet, increased heel eversion, tibia vara, subtalar varus, and forefoot supination.[284]

The method of presentation will be as a chronic overuse injury, and the ***tissue injury complex*** will include the posterior tibial tendon; attachment of soleus fascia, anterior, anterolateral, or deep posterior compartments of the leg; and the middle third of the tibia.

TABLE 8-22

TREATMENT FOR MEDIAL TIBIAL STRESS SYNDROME

Time (d)	Treatment
0–7	Cryotherapy, NSAIDs, evaluate biomechanics
3–10	Stretching gastrocnemius-soleus, posterior tibialis
7–14	Strengthen posterior tibialis, FHL, FDL, gastrocnemius-soleus; proximal limb strengthening

FDL: flexor digitorum longus
FHL: flexor hallucis longus
NSAID: nonsteroidal antiinflammatory drug

The *clinical symptom complex* will be anterior leg pain with activity, often localized over a 3 to 6 cm area of tenderness over the posteromedial edge of the distal third of the tibia, and is generally relieved with rest.

The functional biomechanical deficit may be inflexibility and weakness of ankle plantar flexion, and weakness of ankle dorsiflexors or invertors, or both.

The functional adaptation complex will present as a functional pronation of the foot, and the tissue overload complex will be at the plantar flexors and invertors of the feet, the attachment of soleus muscle to medial tibia, and the bony cortex of the tibia.

Rehabilitation is outlined in Table 8-22.The common denominator in all the potential causes of medial tibial stress syndrome is repetitive overload to leg structures; therefore, the cornerstone of rehabilitation is to modify activity and provide relative rest of the injured area. Relative rest may vary from a few days, in a very mild case, to 4 to 8 weeks in severe cases.[285] Modification of active duties is indicated; however, aerobic capacity and general fitness need to be maintained with other forms of activity, particularly nonweight bearing ones, such as the use of an exercise bicycle or cross-country ski machine. During the acute period, the area of pain and tenderness should receive ice treatments for 10 to 15 minutes, 2 to 3 times a day.[284] Antiinflammatory medications are also useful. Stretching of the gastrocnemius-soleus and tibialis posterior muscles is stressed. When pain and tenderness are diminished, strength needs to be developed, concentrically and eccentrically, in the posterior tibialis, anterior tibialis, flexor hallucis longus, and flexor digitorum longus.[284] Proximal limb strength at the hip and knee also needs to be maintained. When predisposing factors exist, evaluation of foot anatomy and mechanics is essential to prevent recurrence of problems.[286] Orthotics may be beneficial in such cases. An orthosis prevents or reduces compensatory pronation by use of a medial heel and forefoot wedge. With proper treatment, most symptoms will subside in 2 to 4 weeks. If symptoms persist, a further diagnostic work-up at a military hospital may be warranted.

Stress Fractures

Stress fractures are defined as a partial or complete fracture of bone caused by an inability to withstand nonviolent stress that is applied in a rhythmic, repeated, subthreshold manner.[287,288] Stress fractures can occur in any area where bony architecture is overloaded, causing an inability of the bone to remodel in response to the mechanical stress placed on it. The clinical manifestation is often pain. Running activities and sports cause stress fractures in the fibula and tibia; jumping activities affect the pelvis, femur, calcaneus, and patella.[284] In military recruits, the incidence of metatarsal stress fractures during basic training is 40%.[287,289] The fibula is the site of approximately 25% of the stress fractures in civilian athletes, as opposed to only 2% in military recruits. In the military population during basic training, tibial stress fractures are found in 20% of personnel, calcaneus fractures in 30%, femur fractures in 3%, and spine stress fractures in 5%.[287] A 4-year study on stress fractures of the lower extremity in basic training soldiers showed common male stress-fracture sites were the metatarsals (66%), calcaneus (20%), and lower leg (13%). In females, the common sites were the calcaneus (39%), metatarsals (31%), and lower leg (27%).[290] After the onset of stress fractures, roentgenograms taken before 3 to 4 weeks are almost always negative. During the early period of symptoms, a bone scan can be diagnostic. A positive result scan can be seen as early as 2 to 8 days after the onset of symptoms.

The method of presentation is as a chronic overuse injury, and the ***tissue injury complex*** will be the local bone.

The clinical symptom complex will include the gradual onset of local pain in the area of the fracture at the conclusion of activity. This is relieved with rest, but pain with percussion of the affected bone away from the fracture site generally produces pain at the fracture site.

The functional biomechanical deficit and the ***functional adaptation complex*** will vary, depending on the site of fracture.

The tissue overload complex will occur at the bone.

Rehabilitation of stress fractures will also vary, depending on the location of the fracture, but it generally involves decreasing the stress applied to the bone.[284,291] Pester and Smith[290] found that modification of the physical training program to eliminate continuous, high impact activities during high-risk weeks (ie, weeks 2, 4, and 6) resulted in a 12.9% drop in stress-fracture incidence. Other limited weight bearing activities can be substituted to maintain cardiovascular fitness, such as the use of a cross-country ski machine or bicycling, walking, and swimming.

Rehabilitation of fibular stress fractures should include no running for a minimum of 3 weeks, appropriate stretching of the heel cord and ankle, and lower extremity strengthening exercises to avoid ankle stiffness or weakness after the fracture has healed. In rare cases, cast immobilization or partial weight bearing may also be considered for 2 to 3 weeks until inflammation resolves.[284]

Tibial stress fractures usually involve the proximal or medial third of the tibia.[292] Since this bone bears five sixths of the body's weight, these fractures require longer rehabilitation time. Generally, it is necessary to stop running activities for at least 4 to 8 weeks. Flexibility and strength issues are similar to those for fibular stress fractures. Caution should be observed with anterior tibial cortical stress fractures because these frequently have delayed union, nonunion, and fracture completion.

Stress fractures are also common in the foot and ankle regions.[287,293–295] Treatment focuses on relative rest of the affected limb. However, movement should be maintained in surrounding joints, as well as the affected joints, to avoid effects of immobilization. An effective determinant for healing in foot and ankle stress fractures, is when a patient can hop on the affected limb without pain.[296]

Femoral neck and pubis stress fractures are common in females and people doing significant distance running. Femoral neck fractures will require many months of touch-down weight bearing with crutches. Occasionally this type of fracture will require prophylactic pin fixation. Due to persistent physical activity or inadequate treatment, completion of this fracture may lead to avascular necrosis of the femoral head.[238] Pubic rami stress fractures heal within 2 to 5 months.[288]

For most fractures, treatment should be from 6 to 8 weeks. Medication other than analgesics is rarely necessary. Ultrasound treatment is contraindicated because it aggravates the pain in the area of the fracture.[288] Ice and massage may be appropriate for pain reduction, and bracing may occasionally be necessary to limit any motion that would aggravate the injury. Surgery is considered in those bones in which a complete fracture would have serious consequences.[288] These include a displaced fracture at the tarsal navicular, a femoral neck fracture (as discussed above), or possibly a stress fracture of the fifth metatarsal shaft because of its frequent progression to a nonunion in an active soldier.

Inversion Sprains

Injuries to the ankle, particularly inversion sprains, are among the most common musculoskeletal problems seen in the military population. The ankle is much less stable in plantar flexion, owing to the narrowing, wedge-shaped posterosuperior surface of the talus and the distal extension of the lateral malleolus.[297] The most common mechanism of injury, plantar flexion and inversion, may cause injury to the anterior talofibular, calcaneofibular, posterior talofibular (with increasing rotational component), and tibiofibular ligaments (with severe injuries).[298] The differential diagnosis of an acute inversion injury to the ankle include acute rupture of the peroneal retinaculum with subluxation of the peroneal tendons, fracture of the distal fibula, osteochondral fracture of the dome of the talus, or fracture of the anterior process of the os calcis.

The method of presentation is as an acute traumatic injury, and the *tissue injury complex* will involve the anterior talofibular, calcaneofibular, and posterior talofibular and tibiofibular ligaments.

The clinical symptom complex will include pain with active or passive inversion, and localized lateral ankle pain.

The functional biomechanical deficit will present as decreased inversion stability of the ankle, especially in plantar flexion.

The functional adaptation complex will be functional pronation of the forefoot, and the ***tissue overload complex*** will be to the lateral ligamentous complex of the ankle.

Rehabilitation is outlined in Table 8-23. Immediate use of ice and compression will greatly decrease edema and hemorrhage and accelerate the rehabilitation process. Use of cryotherapy for 20 minutes every 2 to 3 hours during the first 48 hours

TABLE 8-23

TREATMENT FOR INVERSION SPRAIN

Time (d)	Treatment
0–1	Early ROM exercises, writing alphabet with foot in the air
0–3	Ice, compression, bracing
3–7	Elastic tubing, resistance band exercises
7–14	Proprioception, closed chain kinetic exercises

ROM: range-of-motion

after injury is advised. After the acute swelling subsides, electric stimulation, Jobst-type pump, or contrast baths can decrease residual and recalcitrant edema. Early ankle motion can be started on the first day of rehabilitation with active motion (nonweight bearing) in the form of 2 or 3 sets of writing the alphabet with the ankle 3 to 4 times a day. This will decrease edema, provide motion to prevent contracture, and start strengthening all planes of the ankle. Use of elastic ankle supports or inflatable air stirrup splints can give excellent support to an injured ankle during the rehabilitation process. As edema subsides, strengthening with resistance tubing is begun in the directions of inversion, eversion, dorsiflexion, and plantar flexion. Weight bearing should progress as tolerated, and once comfortable, weight bearing closed chain kinetic exercises can begin (partial squats and toe raises), as well as proprioceptive retraining with a balance board (Figure 8-28). Proximal limb strengthening is also important because of the significant degree of weakness in the linkage system noted with distal limb problems. Strengthening needs to be done in both concentric and eccentric modes to more accurately simulate physiological stresses.[299] These include exercises for the hip abductors and adductors, and for the knee flexors and extensors. Stretching exercises, such as with a slant board for

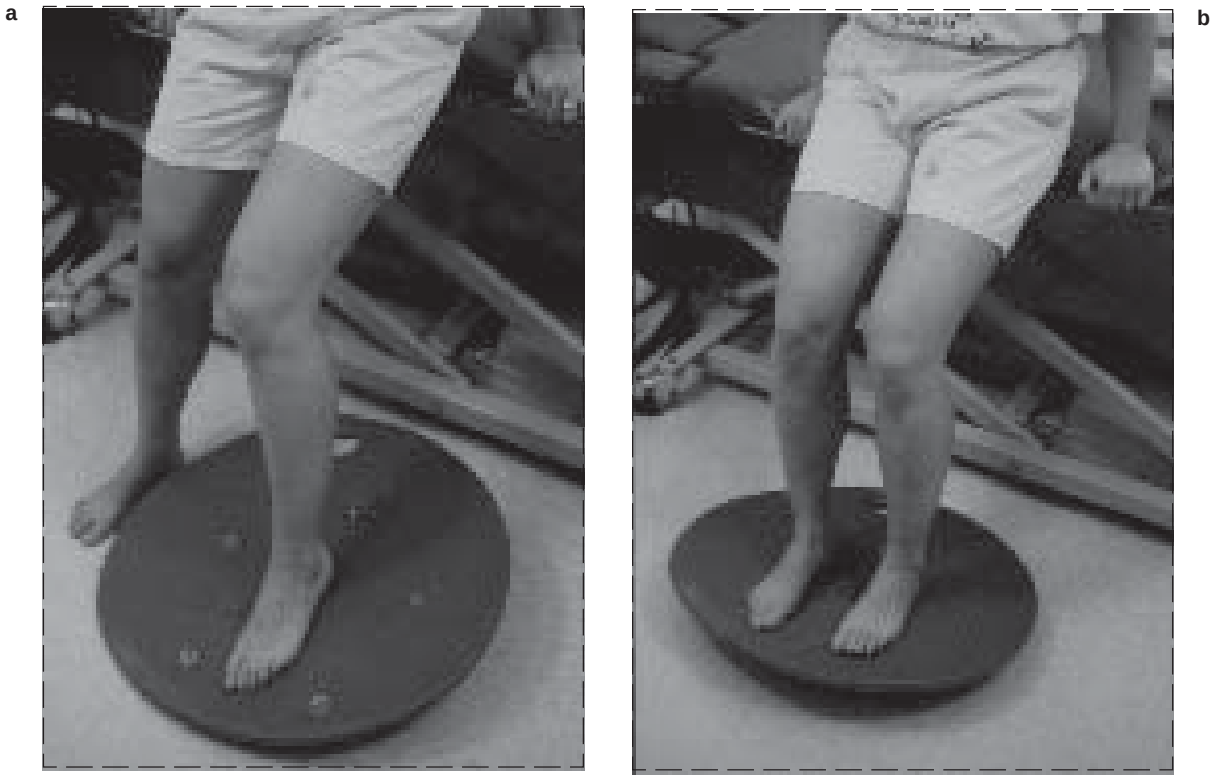


Fig. 8-28. (**a**) Balance board training for proprioception and ankle strengthening with one leg. The patient is instructed to slowly press the outer rim of the board against the ground, first in a clockwise rotation and then in a counterclockwise rotation. (**b**) Balance board training for proprioception and ankle strengthening with two legs. The instructions are the same as those for one-legged balance board exercises. These exercises can be done initially while supporting the upper extremities and later advanced to unsupported for more proprioceptive and balance training.

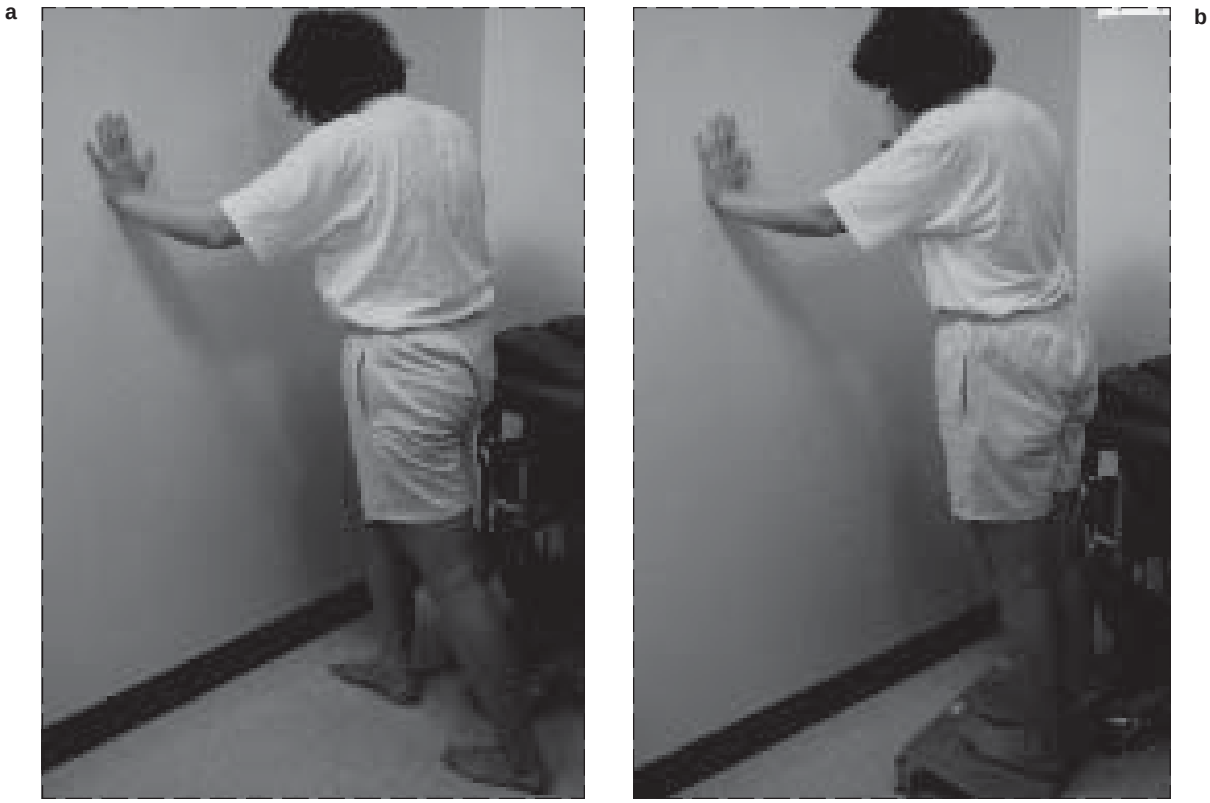


Fig. 8-29. Wall stretches for the gastrocnemius-soleus muscles. (**a**) The front leg is maintained in a flexed position to stretch the soleus muscle, the back leg is extended to stretch the gastrocnemius. The heels are to remain in contact with the ground. (**b**) A slant board is used in conjunction with the wall stretches.

the gastrocnemius-soleus group are important (Figures 8-29). Hamstring, quadriceps, ITB, and hip flexor flexibilities are also stressed.

Achilles Tendinitis

Achilles tendinitis results from repeated episodes of microtrauma to the Achilles tendon, resulting from uneven or inconsistent loading of the gastrocnemius-soleus system, which is not able to undergo an efficient eccentric contraction.[300] These episodes can occur during marching or running on uneven terrain. Microtears in the Achilles tendon result. These tears occur in the area of least vascularity, 2 to 6 cm above the insertion of the tendon, where the tendon is susceptible to chronic inflammation and rupture.[301,302] Anatomic factors that can contribute to Achilles tendon problems include excessive pronation, hindfoot varus, forefoot varus, tight heel cords, and tibia vara.[298] Training errors can include increase in training mileage; a single severe running session; increase in training intensity; running on hills, uneven, or slippery terrain; and resumption after an extended period of inactivity.[298] The differential diagnosis of Achilles tendinitis includes retrocalcaneal bursitis, os trigonum fracture, or plantaris or medial gastrocnemius muscle rupture.

The method of presentation is as an acute exacerbation of a chronic problem, and the ***tissue injury complex*** is a myotendinous junction of the Achilles tendon.

The clinical symptom complex will present as pain 6 to 8 cm proximal to the insertion of the Achilles tendon into the calcaneus, and will be worse with resisted plantar flexion.

The functional biomechanical deficit will include weak ankle dorsiflexors and inflexible plantar flexors, with the ***functional adaptation complex*** being the functional pronation of the foot and increased knee flexion.

The tissue overload complex will include the gastrocnemius-soleus muscle groups and common Achilles tendon.

Rehabilitation for Achilles tendinitis is outlined in Table 8-24. Initial treatment consists of antiin-

TABLE 8-24

TREATMENT FOR ACHILLES TENDINITIS

Time (d)	Treatment
0–3	NSAIDs, cryotherapy, heel lift
3–7	Stretching on incline board
7–14	Strengthening ankle dorsiflexors, plantarflexors, proximal limb muscles

NSAID: nonsteroidal antiinflammatory drug

flammatory medications, ice baths, and gentle stretching on an incline board. Often, one-half inch heel lifts placed in the soldier's shoes can give temporary relief of pain. To prevent an abnormal gait, heel lifts need to be placed in both shoes, even if only one ankle is symptomatic. Placing a rubber Neoprene pad over the Achilles tendon can keep the affected area warm and give symptomatic relief. In chronic conditions, ultrasound and other heating modalities are helpful prior to stretching to help loosen up the scarred, chronically inflamed tissue. Soldiers in whom Achilles tendinitis reaches a chronic stage require a long time to heal; generally, a 4 to 6 week reduction in active duty is mandatory.[303] When in a situation that requires maximum functioning, despite significant symptoms from Achilles tendinitis, taping of the tendon can be done. Techniques are described elsewhere[303] and should be reserved for situations where activity is necessary and taping the Achilles is necessary to prevent rupture. Aerobic, noninjured leg, and upper body conditioning is mandatory.

Plantar Fasciitis

Plantar fasciitis results from repeated traction on the plantar fascia at its insertion into the calcaneus.[304] Microtears and inflammation of the plantar fascia at the calcaneus can result from limited ankle dorsiflexion owing to a tight gastrocnemius-soleus complex.[305] The normal biomechanics of running dictate that the foot strike the ground in the supinated position with subsequent forefoot pronation in foot-flat and return to supination at toe-off. In individuals with pronated feet, stress to the medial aspect of the plantar fascia during running is increased, thereby increasing the chance of plantar fasciitis.[298] The differential diagnosis of plantar fasciitis includes entrapment of the medial calcaneal branch of the tibial nerve, plantar arch strain, tarsal tunnel syndrome, and calcaneal stress fracture.

The method of presentation is as a chronic injury, with the ***tissue injury complex*** being the tensile overload of plantar fascial insertion into the calcaneus.

The clinical symptom complex presents as a point tender pain located along the medial plantar fascia just distal to the calcaneus, which is worse on arising in the morning or after sitting for awhile and then standing, running, or walking.

The functional biomechanical deficit will include plantar flexor inflexibility, plantar flexor peak torque weakness, and a flexor endurance weakness, leading to functional pronation.[30]

The functional adaptation complex will manifest as running on metatarsal heads, decreased stride length, decreased mileage, and foot inversion.

The tissue overload complex will be eccentric overload of plantar flexors due to continued running.

Rehabilitation of plantar fasciitis is outlined in Table 8-25. Initial treatment focuses on the use of cryotherapy and early relative rest of the injured foot. An ice massage or slush bath for 20 minutes, several times a day can temporarily alleviate discomfort, although it may not be successful for long periods of time.[306–308] Antiinflammatory medications are also useful. The injection of steroidal medication into the calcaneal attachment can help control inflammation.[307] The injection is given at the site of maximal tenderness, and it is rarely necessary to give more than 3 weekly injections. Heel pads and arch supports control excessive forefoot pronation and decrease symptoms. Biomechanical factors, such as increased pronation of the foot, can be corrected with an in-shoe, shock-absorbing, medially posted orthosis, or by Low Dye taping.[298,309] Stretching the Achilles tendon, hamstrings, and the plan-

TABLE 8-25

TREATMENT FOR PLANTAR FASCIITIS

Time (d)	Treatment
0–3	Cryotherapy, address biomechanics
0–7	Steroid injection, heel pads
3–7	Achilles, hamstring, plantar fascia stretching, ankle dorsiflexion strengthening

tar fascia are essential components of rehabilitation.[304,310] Aerobic, noninjured leg, and upper body conditioning should be maintained throughout the rehabilitation program. Plantar fasciitis may take 6 to 10 weeks to resolve. Running type activities are usually resumed when tenderness over the plantar fascia, morning stiffness, and pain with weight bearing have abated.[298] Patients who remain symptomatic 6 to 9 months after initiation of care are candidates for surgical intervention.

CONCLUSION

Musculoskeletal injuries are among the most common injuries encountered in soldiers. A rational approach to their rehabilitation is essential. Understanding the pathophysiology of musculoskeletal injuries lays the groundwork for a focused rehabilitation program. Rehabilitation requires (*a*) establishing an accurate diagnosis, (*b*) minimizing the deleterious effects of the acute injury, (*c*) allowing for proper injury tissue healing while maintaining other components of fitness, and ultimately, (*d*) return to normal function while preventing reinjury.

Principles of rehabilitation of specific disorders have been discussed and outlined. Understanding the method of presentation of specific injuries, the tissues injured and overloaded, the clinical symptoms present, and the biomechanical adaptations of an injury are important ingredients in the rehabilitation program. Approximate time frames for treatment options have been addressed. Rehabilitation of musculoskeletal injuries must go beyond the resolution of symptoms to prevent recurrent problems.

REFERENCES

1. Mullins WS, ed. *Surgery in World War II—Orthopedic Surgery in the Zone of Interior.* Washington, DC: Office of The Surgeon General, US Department of the Army; 1970.

2. Woo SLY, Buckwalter JA, eds. *Injury and Repair of Musculoskeletal Soft Tissues.* Park Ridge, Ill: American Academy of Orthopedic Surgeons Symposium; 1987.

3. Herring SA, Nilson KL. Introduction to overuse injuries. *Clin Sports Med.* 1987;6(2):225.

4. Barfred T. Experimental rupture of the Achilles tendon: Comparison of various types of experimental rupture in rats. *Acta Orthop Scand.* 1971;42:528–543.

5. Rotman GP, Schumaker HR, eds. *Primer on the Rheumatic Disease.* Atlanta, Ga: Arthritis Foundation; 1983.

6. Zarro V. Mechanisms of inflammation and repair. In: Michlovitz SL, ed. *Thermal Agents in Rehabilitation.* Philadelphia, Pa: FA Davis; 1986.

7. Kellet J. Acute soft tissue injuries—A review of the literature. *Med Sci Sports Exerc.* 1986;18:489.

8. Oakes B. Acute soft tissue injuries: Nature and management. *Austr Fam Physician.* 1981;10(Suppl 7):3–16.

9. Van DerMeulin JHC. Present state of knowledge on processes of healing in collagen structures. *Int J Sports Med.* 1982;3(Suppl 1):4.

10. Carlstedt CA, Madsen K, Wredmark T. The influence of indomethacin on tendon healing: A biomechanical and biochemical study. *Arch Orthop Trauma Surg.* 1986;105:332.

11. Carlstedt CA, Madsen K, Wredmark T. The influence of indomethacin on collagen synthesis during tendon healing in the rabbit. *Prostaglandins*. 1986;32:353.

12. Akeson WH. An experimental study of joint stiffness. *J Bone Joint Surg Am.* 1961;43:1022–1034.

13. Akeson WH, Amiel D, LaViolette D. The connective tissue response to immobility—a study of chondroitin-4 and 6-sulfate and dermatan sulfate changes in periarticular connective tissue of control and immobilized knees of dogs. *Clinical Orthop.* 1967;51:183–197.

14. Saal JA. General principles and guidelines for rehabilitation of the injured athlete. *Phys Med Rehabil: State of the Art Reviews.* 1987;1(4):523–536.

15. Leadbetter W. *Physiology of Tissue Repair: Athletic Training and Sports*. Rosemont, Ill: American Academy of Orthopedic Surgery; 1991.

16. Puddu G. A classification of Achilles tendon disease. *Am J Sports Med*. 1976;4:145–150.

17. Clancy W. Tendinitis and plantar fasciitis in runners. In: Drez D, D'Ambrosia R, eds. *Prevention and Treatment in Running Injuries.* Thorofare, NJ: Slack Publishers; 1989: 77–87.

18. Leach R. Running injuries of the knee. In: Drez D, D'Ambrosia R, eds. *Prevention and Treatment in Running Injuries.* Thorofare, NJ: Slack Publishers; 1989: 55–74.

19. Kibler WB, Chandler TJ, Pace BK. Principles for rehabilitation after chronic tendon injuries. In: Renström PAFH, Leadbetter WB, eds. Tendinitis I: Basic concepts. *Clin Sports Med.* 1992;11(3):661–671.

20. Dehaven KE. Acute ligament injuries and dislocations. In: Evarts CM, ed. *Surgery of the Musculoskeletal System.* New York: Churchill Livingston; 1983: 7.5–7.30.

21. Akeson WH, Woo SLY, Amid D, et al. The biology of ligaments. In: Funk FJ, Hunter LY, eds. *Rehabilitation of the Injured Knee.* St. Louis, Mo: CV Mosby; 1984: 220–238.

22. Akeson W, Woo S, Amiel D, et al. The connective tissue response to immobility: Biomechanical changes in periarticular connective tissue of the immobilized rabbit knee. *Clin Orthop.* 1973;93:356–362.

23. Noyes FR. Functional properties of knee ligaments and alterations induced by immobilization: A correlative biomechanical and histological study in primates. *Clin Orthop.* 1977;123:210–242.

24. Dehaven KE, Linter DM. Athletic injuries: Comparison by age, sport and gender. *Am J Sports Med.* 1986;14:218–224.

25. Powell J. 636,000 injuries annually in high school football. *Athletic Training.* 1987;22:19–22.

26. Lavos GS, Tipton CM, Cooper RR. Influence of physical activity on ligament insertions in the knees of dogs. *J Bone Joint Surg Am.* 1971;53:275–286.

27. Woo SLY, Matthews JV, Akeson WH, et al. Connective tissue response to immobility: Correlative study of biomechanical and biochemical measurements of normal and immobilized rabbit knees. *Arthritis Rheum*. 1975;18:257–264.

28. Garrett WE. Muscle strain injuries: Clinical and basic aspects. *Med Sci Sports Exerc.* 1990;22(4):436–443.

29. Herring SA. Rehabilitation of muscle injuries. *Med Sci Sports Exerc.* 1990;22(4):453–456.

30. Kibler WB. Concepts in exercise rehabilitation. In: Leadbetter W, Buckwalter JA, Gordon SL, eds. *Sports Induced Inflammation.* Chicago, Ill: American Academy of Orthopedic Surgery; 1990.

31. McMaster PE. Tendon and muscle ruptures: Clinical and experimental studies on the causes and location of subcutaneous ruptures. *J Bone Joint Surg.* 1933;15:705–722.

32. Almekinders LC, Garrett WE Jr, Seaber AV. Pathophysiologic response to muscle tears in stretching injuries. *Trans Orthop Res Soc.* 1984;9:307.

33. Stone MH. Muscle conditioning and muscle injuries. *Med Sci Sports Exerc.* 1990;22(4):457–462.

34. Booth FW. Time course of muscular atrophy during immobilization of hind limbs in rats. *J Appl Physiol.* 1977;43:656–661.

35. Muller EA. Influence of training and of inactivity on muscle strength. *Arch Phys Med Rehab.* 1970;51:449–462.

36. Jones VT, Garrett WE Jr, Seaber AV. Biomechanical changes in muscle after immobilization at different lengths. *Trans Orthop Res Soc.* 1985;10:6.

37. Satran MR, Garrett WE Jr, Seabor AV, et al. The role of warm up in muscular injury prevention. *Am J Sports Med.* 1989;16(2):123–129.

38. Hilyer JC, Brown KC, Sirles AT, Peoples L. A flexibility intervention to reduce the incidence and severity of joint injuries among municipal firefighters. *J Occup Med.* 1990;32(7):631.

39. Kibler WB. Clinical aspects of muscle injury. *Med Sci Sports Exerc.* 1990;22(4):450–452.

40. Chambers R. Clinical uses of cryotherapy. *Phys Ther.* 1969;49:245–249.

41. Drez D, Faust DC, Evans JP. Cryotherapy and nerve palsy. *Am J Sports Med.* 1981;9:256–257.

42. Grana WA, Curl WL, Reider B. Cold modalities. In: Drez D, ed. *Therapeutic Modalities for Sports Injuries.* Chicago, Ill: Year Book Medical Publishers; 1986: 25–31.

43. Halvorson GA. Principles of rehabilitating sports injuries. In: Teitz CC, ed. *Scientific Foundations of Sports Medicine.* Toronto, Ont, Can: BC Decker; 1989: 345–371.

44. Lehman JF, DeLateur BJ. Cryotherapy. In: Lehmann JF, ed. *Therapeutic Heat and Cold.* Baltimore, Md: Williams & Wilkins; 1982: 563–602.

45. McMaster WC. Cryotherapy. *Phys Sports Med.* 1982;10:112–119.

46. McMaster WC, Liddle S, Waugh TR. Laboratory evaluation of various cold therapy modalities. *Am J Sports Med.* 1978;6:291–294.

47. Ork H. Uses of cold. In: Kuprian W, ed. *Physical Therapy for Sports.* Philadelphia, Pa: WB Saunders; 1982: 62–68.

48. Quillen WS, Rouiller LH. Initial management of acute ankle sprains with rapid pulsed pneumatic compression and cold. *J Orthop Sports Phys Ther.* 1982;4:39–43.

49. Sloan JP, Giddings P, Hain R. Effects of cold and compression on edema. *Sports Med*. 1988;16:116–120.

50. Grant AE. Massage with ice (cryokinetics) in the treatment of painful conditions of the musculoskeletal system. *Arch Phys Med Rehab.* 1964;45:233–238.

51. Lehman JF, DeLateur BJ. Diathermy and superficial heat and cold therapy. In: Kottke FJ, Stillwell GK, Lehmann JF, eds. *Krusen's Handbook of Physical Medicine and Rehabilitation.* Philadelphia, Pa: WB Saunders; 1982: 275–350.

52. Roy S, Irvin R, eds. *Sports Medicine: Prevention, Evaluation, Management, and Rehabilitation.* Englewood Cliffs, NJ: Prentice-Hall; 1983.

53. Akeson WH. An experimental study of joint stiffness. *J Bone Joint Surg Am.* 1961;43:1022–1034.

54. Lehman J. Therapeutic heat and cold. *Clin Orthop.* 1974;99:207.

55. Lehman J, DeLateur B. Diathermy and superficial heat and cold therapy. In: Kottke EJ, Stillwell GK, Lehman JF, eds. *Krusen's Handbook of Physical Medicine and Rehabilitation.* Philadelphia, Pa: WB Saunders; 1982: 275–350.

56. McMasters W, Liddle S, Waugh T. Laboratory evaluation of various cold therapy modalities. *Am J Sports Med.* 1978;6:291.

57. Marino M. Principles of therapeutic modalities: Implications for sports injuries. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 195–244.

58. Huskisson E. Non-narcotic analgesics. In: Wall PD, Melzack, R. eds. *Text Book of Pain.* New York: Churchill-Livingston; 1984: 505–513.

59. Goodman AC, Gilman LS, Gilman A. *The Pharmacological Basis of Therapeutics.* New York: Macmillan; 1980:686.

60. Woo SLY, Matthews JV, Akeson WH, et al. Connective tissue response to immobility: A correlative study of biochemical and biomechanical measurements of normal and immobilized rabbit knees. *Arthritis Rheum.* 1975;18:257–264.

61. Halpern AA, Horowitz BG, Nagel DA. Tendon ruptures associated with corticosteroid therapy. *West J Med.* 1977;127:378–432.

62. Kennedy JL, Baxter-Willis R. The effects of local steroid injections on tendons: A biochemical and microscopic correlative study. *Am J Sports Med.* 1976;4:11–18.

63. Sweetham R. Corticosteroid arthropathy and tendon rupture. *J Bone Joint Surg Br.* 1969;51:397–398.

64. Dehn E, Torp RR. Treatment of joint injuries by immediate rehabilitation. *Clin Orthop.* 1971;77:218–231.

65. Jarvinen M. Healing of crush injury in rat striated muscle: A histological study of the effect of early mobilization and immobilization on the repair process. *Acta Pathol Microbiol Scand.* 1975;A83:269–282.

66. Jarvinen M. Healing of a crush injury in rat striated muscle: A micro angiographical study of the effect of early mobilization and immobilization on capillary ingrowth. *Acta Pathol Microbiol Scand.* 1976;A84:85–94.

67. Jarvinen M. Healing of a crush injury in rat striated muscle: Effect of early mobilization and immobilization on the tensile properties of gastrocnemius muscle. *Acta Chir Scand.* 1976;142:47–56.

68. Leach R. The prevention and rehabilitation of soft tissue injuries. *Int J Sports Med.* 1982;3(Suppl 1):18.

69. Frank G, Woo S, Amiel D, Harwood F, Gomez M, Akeson W. Medial collateral ligament healing: A multidisciplinary assessment in rabbits. *Am J Sports Med.* 1983;11:379.

70. Knight KL. Guidelines for rehabilitation of sports injuries. *Clin Sports Med.* 1985;4(3):405.

71. Houglum P. Techniques of PNF in athletic training. *Athletic Training.* 1975;10:44–45.

72. Knott M, Voss DE. *Proprioceptive Neuromuscular Facilitation, Patterns and Techniques.* New York: Harper and Row; 1965.

73. Saal JS. Flexibility training. *Phys Med Rehabil: State of the Art Reviews*. 1987;1(4):537

74. Moore MA, Hutton RS. Electromyographic investigation of muscle stretching techniques. *Med Sci Sports.* 1980;12:332–329.

75. Tanigawa MC. Comparison of the hold-relax procedure and passive mobilization on increasing muscle length. *Phys Ther.* 1972;52:725–735.

76. Muller EA. Influence of training and of inactivity on muscle strength. *Arch Phys Med Rehabil.* 1970;51:449–462.

77. Booth FW. Time course of muscular atrophy during immobilization of hind limbs in rats. *J Appl Physiol.* 1977;43:656–661.

78. Booth FW, Seider MJ. Effects of disuse by limb immobilization on different muscle fiber types. In: Pette D, ed. *Plasticity of Muscle.* New York: de Gruyter; 1980.

79. Hettinger T, Mueller EA. Muskelleistung and muskeltraining. *Arbeitsphysiologie.* 1953;15:111.

80. Paulos LE, Payne FC, Rosenberg TD. Rehabilitation after anterior cruciate ligament surgery. In: Jackson D, Drew D, eds. *The Anterior Cruciate Deficient Knee.* St. Louis, Mo: CV Mosby; 1987:291–314.

81. Eriksson E. Sports injuries of the knee ligaments: Their diagnosis, treatment, rehabilitation and prevention. *Med Sci Sports Exerc.* 1976;8:133.

82. Ericksson E, Haggmark T. Comparison of isometric muscle training and electrical stimulation supplementing isometric muscle training in the recovery after major knee ligament surgery. *Am J Sports Med.* 1979;7:169.

83. Haggmark T, Ericksson E. Cylinder or mobile cast brace after knee ligament surgery: A clinical analysis and morphologic and enzymatic studies of changes in the quadriceps muscle. *Am J Sports Med.* 1979;7:48.

84. Zarins B. Soft tissue injury and repair-biomechanical aspects. *Int J Sports Med.* 1982;3(Suppl II):9.

85. Finsterbush A, Friedman B. Reversibility of joint changes produced by immobilization in rabbits. *Clin Orthop.* 1975;111:290.

86. Dillingham MF. Strength Training. *Phys Med Rehabil: State-of-the-Art Reviews.* 1987;1(4):555.

87. Dickinson A, Bennett KM. Therapeutic Exercise. *Clin Sports Med.* 1985;4(3):417.

88. Allman FL. *Exercise in sports medicine*. In: Basmajian JV, ed. *Therapeutic Exercise.* Baltimore, Md: Williams & Wilkins; 1984.

89. Knapik JJ, Wright JE, Mawdsley RH, Braun JM. Isokinetic, isometric and isotonic strength relationships. *Arch Phys Med Rehabil*. 1983;64:77–80.

90. Knapik JJ, Wright JE, Mawdsley RH, Braun JM. Isometric, isotonic, and isokinetic torque variations in four muscle groups through a range of joint motion. *Phys Ther*. 1983;63:939–947.

91. Jones, McCartney, McComas, eds. *Human Muscle Power.* Champaigne, Ill: Human Kinetics; 1986.

92. Brooks, Fahey, eds. *Fundamentals of Human Performance.* New York: Macmillan; 1987.

93. Asterand, Rodahl, eds. *Work Physiology: Physiologic Basis of Exercise.* New York: McGraw Hill; 1986.

94. Saltin B, Nagar K, Costill DL, et al. The nature of the training response; peripheral and central adaptions to one-legged exercise. *Acta Physiol Scand.* 1976;96:289–305.

95. Lindh M. Increase of muscle strength from isometric quadriceps exercise at different knee angles. *Scand J Rehab Med.* 1979;11:33–36.

96. Lesmes GR, Costill DL, Coyle EF, Fink WJ. Muscle strength and power changes during maximal isokinetic training. *Med Sci Sports.* 1978;10:266–269.

97. Moffroid M, Whipple R, Hotkoch J, et al. A study of isokinetic exercise. *J Am Phys Ther Assoc.* 1969;49:735–747.

98. DeLateur BJ. Exercise for strength and endurance. In: Basmajian JV, ed. *Therapeutic Exercise.* Baltimore, Md: Williams & Wilkins; 1984.

99. Fahey TD. Physiological adaptation to conditioning. In: Fahey TD, ed. *Athletic Training: Principles and Practice.* Mayfield Publishing Company; 1986.

100. Harris FA. Facilitation Techniques and Technological Adjuncts in Therapeutics Exercise. In: Basmajian JV, ed. *Therapeutic Exercise.* Baltimore, Md: William & Wilkins; 1984:110–178.

101. Wiens JJ, Saal JA. Rehabilitation of cervical spine and brachial plexus injuries. *Phys Med and Rehab: State of the Art Reviews.* 1982;1(4):583–595.

102. Shelokov AP. Evaluation, diagnosis and initial treatment of general disc disease. *Spine: State-of-the-Art Reviews.* 1991;5(2):167–176.

103. Reynolds M. Myofascial trigger point syndromes in the practice of rheumatology. *Arch Phys Med Rehabil.* 1981;6(2):111–113.

104. Simons D. Myofascial trigger points: A need for understanding. *Arch Phys Med Rehabil.* 1981;62:97–99.

105. Paris SV. Manipulation of the lumbar spine. In: Weinstein JM, Weisel SW, eds. *The Lumbar Spine.* Philadelphia, Pa: WB Saunders; 1990: 805–811.

106. Haldeman S. Spinal manipulative therapy in sports medicine. *Clin Sports Med.* 1986;5(2):277–293.

107. Geiringer SR, Kincaid CB, Rechtein. Traction, manipulation and massage. In: Delisa JA, ed. *Rehabilitation Medicine: Principles and Practice.* Philadelphia, Pa: JB Lippincott; 1988:272.

108. Watkins RG. Neck injuries in football players. *Clin Sports Med.* 1986;5(2):215–246.

109. LaBan MM. Whiplash: Its evaluation and treatment. *Phys Med Rehabil: State-of-the-Art Reviews*. 1990;4(2):293.

110. Colachis SC Jr, Strohm BR. Effect of duration of intermittent cervical traction on vertebral separation. *Arch Phys Med Rehabil.* 1966;47:353–359.

111. Sweeney D, Prentice S, Saal JA, Salle JS. Cervicothoracic muscular stabilization techniques. *Phys Med Rehabil: State-of-the-Art Reviews.* 1990;4(2):335–359.

112. Cousins MJ, Bromage PR. Epidural neural blockage. In: Cousins MJ, Bridenbaugh PO, eds. *Neural Blockage in Clinical Anesthesia and Management of Pain.* New York: JB Lippincott; 1988.

113. Clancy WG, Jr, Brand RL, Bergfeld JA. Upper trunk brachial plexus injuries in contact sports. *Amer J Sports Med.* 1977;5:209.

114. Stanwood J, Kraft G. Diagnosis and management of brachial plexus injuries. *Arch Phys Med Rehabil.* 1971;52–60.

115. Bateman J. Nerve lesions about the shoulder. *Orthop Clin North Am.* 1980;11:307–326.

116. Herring SA, Weinstein SM. Electrodiagnosis in sports medicine. *Arch Phys Med Rehabil: State of the Art Reviews.* 1989;3(4):809–822.

117. Adson AW. Surgical treatment for symptoms produced by cervical ribs and the scalenus anticus muscle. *Surg Gynecol Obstet.* 1947;85:687.

118. Lindgren KA, Leino E. Subluxation of the first rib: A possible thoracic outlet syndrome mechanism. *Arch Phys Med Rehabil.* 1988;68:692–695.

119. Roos DB. New concepts of thoracic outlet syndrome that explain etiology, symptoms, diagnosis and treatment. *Vasc Surg.* 1979;5:313.

120. Cuetter AC, Bartoszek DM. The thoracic outlet syndrome: Controversies, overdiagnosis, overtreatment, and recommendations for management. *Muscle Nerve.* 1989;12:410–419.

121. Urschel HC, Paulsen DL, McNamara JJ. Thoracic outlet syndrome. *Ann Thorac Surg.* 1968;1:1.

122. Cailliet R. *Neck and Arm Pain.* Philadelphia, Pa: FA Davis; 1981.

123. Smith KF. The thoracic outlet syndrome: A protocol of treatment. *J Orthop Sports Phys Ther.* 1979;Fall:89–99.

124. Dale AW, Lewis MR. Management of thoracic outlet syndrome. *Ann Surg.* 1975;181:575.

125. Britt LP. Nonoperative treatment of thoracic outlet syndrome symptoms. *Clin Orthop.* 1967;51:45.

126. Watson K. Impingement and rotator cuff lesions. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 213–220.

127. Hawkins RJ, Kennedy JC. Impingement syndrome in athletes. *Am J Sports Med.* 1980;8:151.

128. Purdam CR, Fricker PA, Cooper B. Principles of treatment and rehabilitation. In: Bloomfield J, Findar PA, Fileh KD, eds. *Textbook of Science and Medicine in Sports.* Chicago, Ill: Human Kinetics; 1992: 218.

129. Skyhar MJ, Warren RF, Altchek DW. Instability of the shoulder. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 181–212.

130. Turek SL. *Orthopedics: Principles and Their Application*. 4th ed. 1984: 937.

131. Simonet WT, Colfield RH. Prognosis in anterior shoulder dislocation. *Am J Sports Med.* 1984;12:19.

132. Yoneda B, Welsh RP, MacIntosh DL. Conservative treatment of shoulder dislocation. *J Bone Joint Surg Br.* 1982; 64:254.

133. Mendoz FX, Nicholas JA, Sands A. Principles of shoulder rehabilitation in the athlete. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990:251–264.

134. Neer CS, Foster CR. Inferior capsular shift for involuntary inferior and mutlidirectional instability of the shoulder. *J Bone Joint Surg Am.* 1980;62:897.

135. Derscheid G. Rehabilitation of common orthopedic problems. *Nurs Clin North Am.* 1981;16:709.

136. Turkel SJ, et al. Stabilizing mechanisms preventing anterior dislocation of the glenohumeral joint. *J Bone Joint Surg Am.* 1981;63:1208.

137. Allman FL Jr. Fracture and ligamentous injuries of the clavicle and its articulation. *J Bone Joint Surg Am.* 1967;9:774.

138. Bergfeld JA. Acromioclavicular complex. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 169–180.

139. Brems JJ. Degenerative joint disease in the shoulder. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 235–250.

140. Cox JS. The fate of the acromoclavicular joint in athletic injuries. *Am J Sports Med.* 1977;5:264.

141. Leach RE , Miller JK. Lateral and medial epicondylitis of the elbow. *Clinics Sports Med.* 1987;6(2):259.

142. Gunn CC, Milbrandt WE. Tennis elbow and the cervical spine. *Can Med Assoc J.* 1976;114:803–809.

143. Roles NC, Mandsley RH. Radial tunnel syndrome: Resistant tennis elbow as a nerve entrapment. *J Bone Joint Surg Br.* 1972;54:449–508.

144. Nirschl RP. Tennis elbow. *Orthop Clin North Am.* 1973;4:787–800.

145. Nirschl RP. Tennis elbow. *J Bone Joint Surg Am.* 1979;61:832–839.

146. Parkes JC. Overuse injuries of the elbow. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 335–346.

147. Allman F, Carlson CA. Rehabilitation of elbow injuries. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 351.

148. Kiefhaber TR, Stern PJ. Upper extremity tendinitis and overuse syndromes in the athlete. *Clin Sports Med.* 1992;11(1):39–55.

149. Posner MA. Hand injuries. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 495.

150. Shaw Wilgis EF, Yates AY Jr. Wrist pain. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 483.

151. Wood MB, Dobyns JH. Sports related extraarticular wrist syndromes. *Clin Orthop Rel Res.* 1986;202:93.

152. Engkvist O, Balkfors B, Lindsjo U. Thumb injuries in downhill skiing. *Int J Sports Med.* 1982;3:50.

153. Kahler DM, McCue FC III. Metacarpophalangeal and proximal interphalangeal joint injuries at the hand, including the thumb. *Clin Sports Med.* 1992;11(1):57–76.

154. Mayer VA, McCue FC III. Rehabilitation and protection of the hand and wrist. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 619.

155. Neviaser RJ, Wilson JN, Lievario A. Rupture of the ulnar collateral ligament of the thumb (gamekeeper's thumb): Correction by dynamic repair. *J Bone Joint Surg Am.* 1971;53:1357.

156. Helm RH. Hand function after injuries to the collateral ligaments of the metacarpophalangeal joint of the thumb. *J Hand Surg.* 1982;12B:252.

157. Andersson GBJ. Epidemiologic aspects on low-back pain in industry. *Spine.* 1981;6:53–60.

158. Bigos SJ, Spengler DM, Martin NA, et al. Back injuries in industry: A retrospective study II. Injury factors. *Spine.* 1986;11:246–251.

159. Clemmer DI, Mohr DL, Mercer DJ. Low-back injuries in a heavy industry: Worker and workplace factors. *Spine.* 1991;16:824–830.

160. Frymoyer JW, Pope MH, Clements JH, et al. Risk factors in low-back pain: An epidemiological survey. *J Bone Joint Surg Am.* 1983;65:213–218.

161. Frymoyer JW, Pope MH, Costanza MC, et al. Epidemiologic studies of low-back pain. *Spine.* 1980;5:419–423.

162. Kelsey JL, Golden AL. Occupational and workplace factors associated with low back pain. *Spine: State of the Art Reviews.* 1987;2:61–73.

163. Pope MH, Andersson GBJ, Frymoyer JW, Chaffin DB, eds. *Occupational Low Back Pain Assessment, Treatment, and Prevention.* St. Louis, Mo: CV Mosby; 1991.

164. Svensson HO, Andersson GBJ. Low-back pain in 40 to 47 year-old men: Work history and work environment factors. *Spine*. 1983;8:272–276.

165. Troup JDG, Martin JW, Lloyd DCEF. Back pain in industry: A prospective survey. *Spine*. 1981;6:61–69.

166. Wilder DG, Woodsworth BB, Frymoyer JW, Pope MH. Vibration and the human spine. *Spine.* 1982;7:243–254.

167. Weinstein SM, Herring SA. Rehabilitation of the patient with low back pain. In: DeLisa J, ed. *Rehabilitation Medicine*: *Principles and Practice.* Philadelphia, Pa: JB Lippincott; 1993: 996–1017.

168. Kirkaldy-Willis WH, Wedge JH, Yong-Hing K, Reily J. Pathology and pathogenesis of lumbar spondylosis and stenosis. *Spine*. 1978;3:319–328.

169. Boden S, Davis DO, Dina TS, et al. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. *J Bone Joint Surg Am.* 1990;72:403–408.

170. Hitselberger WE, Witten PM. Abnormal myelogram in asymptomatic patients. *J Neurosurg.* 1968;28:204–206.

171. Wiesel SW, Tsourmas N, Feffer HL, et al. A study of computer-assisted tomography: The incidence of positive CAT scans in an asymptomatic group of patients. *Spine*. 1984;9:549–551.

172. Mooney V, Robertson J. The facet syndrome. *Clin Orthop Rel Res.* 1976;115:149–156.

173. Helbig T, Lee CK. The lumbar facet syndrome. *Spine.* 1988;13:61–64.

174. Raymond J, Dumas JM. Intraarticular facet block: Diagnosis test or therapeutic procedure? *Radiology*. 1984;151:333–336.

175. Stokes IAF, Frymoyer JW. Segmental motion and instability. *Spine*. 1987;12:688–691.

176. Farfan HF, Gracovetsky S. The nature of instability. *Spine*. 1984;9:714–719.

177. Hall S, Bartleson JD, Onofrio BM, et al. Lumbar spine stenosis: Clinical features, diagnostic procedures, and results of surgical treatment in 68 patients. *Ann Intern Med.* 1985;103:271–275.

178. Vanharanta H. Etiology, epidemiology and natural history of lumbar disc disease. *Spine: State of the Art Reviews.* 1989;3:1–12.

179. Herzog RJ. Selection and utilization of imaging studies for disorders of the lumbar spine. *Clin Phys Med Rehabil.* 1991;2:7–59.

180. Adams MA, Dolan P, Hutton, WC. The stages of disc degeneration as revealed by discograms. *J Bone Joint Surg Br*. 1986;68:36–41.

181. Bernard TN. Lumbar discography followed by computed tomography: Refining the diagnosis of low-back pain. *Spine*. 1990;15:690–707.

182. Bodner RJ, Heyman S, Drummond DS, et al. The use of single photon emission computed tomography (SPECT) in the diagnosis of low-back pain in young patients. *Spine*. 1988;13:1155–1160.

183. Dvorak J, Panjabi M, Novotny JE, et al. Clinical validation of functional flexion-extension roentgenograms of the lumbar spine. *Spine*. 1991;16:943–950.

184. Forristall RM, Marsh HO, Pay NT. Magnetic resonance imaging and contrast CT of the lumbar spine: Comparison of diagnostic methods and correlation with surgical findings. *Spine*. 1988;13:1049–1050.

185. Gibson MJ, Szypryt EP, Buckley JH, et al. Magnetic resonance imaging of adolescent disc herniation. *J Bone Joint Surg Br.* 1987;69:699–703.

186. Grubb SA, Lipscomb HJ, Bonner-Guilford W. The relative value of lumbar roentgenograms, metrizamide myelography and discography in the assessment of patients with chronic low back syndrome. *Spine*. 1987;12:282–286.

187. Hayes MA, Howard TC, Gruel CR, et al. Roentenographic evaluation of lumbar spine flexion-extension in asymptomatic individuals. *Spine*. 1989;14:327–331.

188. Hoffman RM, Kent DL, Deyo RA. Diagnostic accuracy and clinical utility of thermography for lumbar radiculopathy: A meta-analysis. *Spine*. 1991;16:623–628.

189. Rosenthal H, Rosenthal DI. CT scanning for lumbar disc disease. *Surg Rounds Orthop.* 1988:15–23.

190. Sachs BL, David JF, Olimpio D, et al. Spinal rehabilitation by work tolerance based on objective physical capacity assessment of dysfunction: A prospective study with control subjects and twelve-month review. *Spine*. 1990;15:1325–1332.

191. Schnebel BE, Kingston S, Watkins R, Dillon W. Comparison of MRI to contrast CT in the diagnosis of spinal stenosis. *Spine*. 1989;14:332–337.

192. Schneiderman G, Flannigan B, Kingston S, et al. Magnetic resonance imaging in the diagnosis of disc degeneration: Correlation with discography. *Spine*. 1987;12:276–281.

193. Stockley I, Getty CJM, Dixon AK, et al. Lumbar lateral canal entrapment: Clinical radiculographic and computed tomographic findings. *Clin Radiol.* 1988;39:144–149.

194. Zuckerman J, Derby R, Hsu K, et al. Normal magnetic resonance imaging with abnormal discography. *Spine*. 1988;13:1355–1359.

195. White AA, Panjabi MM, eds. *Clinical Biomechanics of the Spine.* Philadelphia, Pa: JB Lippincott; 1978: 277–344.

196. Epstein NE, Epstein JA, Carras R, Hyman RA. Far lateral lumbar disc herniation and associated structural abnormalities: An evaluation in 60 patients of the comparative value of CT, MRI, and myelo-CT in diagnosis and management. *Spine*. 1990;15:534–539.

197. Derby R. Diagnostic block procedures: Use in pain localization. *Spine: State of the Art Reviews.* 1986;1:47–64.

198. Destouet JM. Lumbar facet syndrome: Diagnosis and treatment. *Surg Rounds Orthop.* 1988;2:2–27.

199. Dooley JF, McBroom RJ, Taguchi T, Macnab I. Nerve root infiltration in the diagnosis of radicular pain. *Spine*. 1988;13:79–83.

200. Jeffries B. Epidural steroid injections. *Spine: State of the Art Reviews.* 1988;2:419–426.

201. Jeffries B. Facet steroid injections. *Spine: State of the Art Reviews.* 1988;2:409–417.

202. Krempen JF, Smith BS, DeFreest LJ. Selective nerve root infiltration for the evaluation of sciatica. *Orthop Clin North Am.* 1975;6:311–314.

203. Didry C, Lopez P, Baixas P, Simon L. Medically treated lumbar disc herniation: Clinical and computed tomographic follow-up. *Presse Med.* 1991;20:299–302.

204. Saal JA, Saal JS, Herzog RJ. The natural history of lumbar intervertebral disc extrusions treated nonoperatively. *Spine*. 1990;15:683–686.

205. Jackson CP. Historic perspectives on patient education and its place in acute spinal disorders. In: Mayer TG, Mooney V, Gatchel RJ, eds. *Contemporary Conservative Care for Painful Spinal Disorders.* Philadelphia, Pa: Lea & Febinger; 1991: 221–234.

206. Deyo RA, Diehl AK, Rosenthal M. How many days of bed rest for acute low back pain: A randomized clinical trial. *New Engl J Med.* 1986;315:1064–1070.

207. McKenzie RA. Prophylaxis in recurrent low back pain. *N Z Med J.* 1979;89:22–23.

208. McKenzie RA. *The Lumbar Spine: Mechanical Diagnosis and Therapy.* Waikance, New Zealand: Spinal Publications; 1981:49–80.

209. Ponte DJ, Jensen GJ, Kent BE. A preliminary report on the use of the McKenzie protocol versus Williams protocol in the treatment of low back pain. *J Orthop Sports Phys Ther.* 1984;6:130–139.

210. Stankovic R, Johnell O. Conservative treatment of acute low-back pain: A prospective randomized trial: McKenzie method of treatment versus patient education in "mini back school." *Spine*. 1990;15:120–123.

211. Melzack R, Wall PD. Pain mechanisms: A new theory. *Science*. 1965;150:971–979.

212. Schnebel BE, Watkins RG, Dillin W. The role of spinal flexion and extension in changing nerve root compression in disc herniations. *Spine*. 1989;14:835–837.

213. Nachemson A, Elfstrom G. Intravital dynamic pressure measurements in lumbar discs: A study of common movements, maneuvers and exercises. *Scand J Rehabil Med.* 1970;1(Suppl):1–40.

214. Williams P. *Low Back and Neck Pain: Causes and Conservative Treatment.* Springfield, Ill: Charles C Thomas; 1974.

215. Borenstein DG, Wiesel SW, eds. *Low Back Pain: Medical Diagnosis and Comprehensive Management.* Philadelphia, Pa: WB Saunders; 1989.

216. Gianakopoulos G, Waylons GW, Grant PA, et al. Inversion devices: Their role in producing lumbar distraction. *Arch Phys Med.* 1985;66:100–102.

217. Jackson DW, Wiltse LL, Dingemann RD, et al. Stress reactions involving the pars interarticularis in young athletes. *Am J Sports Med.* 1981;9:304–312.

218. Porter RW, Hibbert CS. Symptoms associated with lysis of the pars interarticularis. *Spine*. 1984;9:755–758.

219. Libson E, Bloom RA, Dinari G. Symptomatic and asymptomatic spondylolysis and spondylolisthesis in young adults. *Int Orthop.* 1982;6:259–261.

220. Cyron BM, Hutton WC. The failure strength of the lumbar neural arch in spondylolysis. *J Bone Joint Surg Br.* 1978;60:234–238.

221. Dietrich M. Kurowski P. The importance of mechanical factors in the etiology of spondylolysis: A model analysis of loads and stresses in human lumbar spine. *Spine*. 1985;6:532–542.

222. Hutton WC, Stott JRR, Cyron BM. Is spondylolysis a fatigue fracture? *Spine*. 1977;2:202–209.

223. Micheli LJ. Back injuries in gymnastics. *Clin Sports Med.* 1985;4:85–94.

224. Ciullo JV, Jackson DW. Pars interarticularis stress reaction, spondylolysis and spondylolisthesis in gymnasts. *Clin Sports Med.* 1985;4:95–110.

225. Letts M, MacDonald P. Sports injuries to the pediatric spine. *Spine: State of the Art Reviews.* 1990;4:49–83.

226. Anderson SJ. Assessment and management of the pediatric and adolescent patient with low–back pain. *Phys Med Rehab Clin North Am.* 1991;2:157–185.

227. Greenman PE. *Principles of Manual Medicines.* Baltimore, Md: Williams & Wilkins; 1989.

228. Maitland GD, ed. *Vertebral Manipulation.* London, UK: Butterworth; 1986.

229. Paris S. Spinal manipulative therapy. *Clin Orthop.* 1983;79:55–61.

230. Gracovetsky S, Farfan H, Helleur C. The abdominal mechanism. *Spine*. 1985;10:317–324.

231. Van Hoesen L. Mobilization and manipulation techniques for the lumbar spine. In: Grieve GP, ed. *Modern Manual Therapy of the Vertebral Column.* New York: Churchill–Livingston; 1986.

232. Porterfield JA. Dyanmic stabilization of the trunk. *JOSPT.* 1985;6:271–277.

233. Saal JA. Dynamic muscular stabilization in the nonoperative treatment of lumbar pain syndromes. *Orthop Rev.* 1990;19:691–700.

234. Butler D, Trafimow JH, Andersson GBJ, et al. Discs degenerate before facets. *Spine*. 1990;15:111–113.

235. Crisco JJ, Panjabi MM. The intersegmental and multisegmental muscles of the lumbar spine: A biomechanical model comparing lateral stabilizing potential. *Spine*. 1991;16:793–799.

236. Panjabi MM, Abumi K, Duranceay J, Oxland T. Spinal stability and intersegmental muscle forces: A biomechanical model. *Spine*. 1989;14:194–200.

237. Cady LD, Thomas PC, Karwasky RJ. Program for increasing health and fitness of firefighters. *J Occup Med.* 1985;27:110–114.

238. Esposito PW. Pelvis, hip and thigh injuries. In: Mellion MB, Walsh WM, Shelton GL, eds. *The Team Physician's Handbook.* Philadelphia, Pa: Hanley & Belfus; 1990: 401.

239. Brody DM. Running injuries. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 1534.

240. Renne JW. The iliotibilal band friction syndrome. *J Bone Joint Surg Am.* 1975;57:1110–1111.

241. Jones DC, James SL. Overuse injuries of the lower extremity: Shin splints, iliotibial band function syndromes and exertional compartment syndromes. *Clin Sports Med.* 1987;6(2):273.

242. Sim DM, Scott SG. Injuries of the pelvis and hip in athletes: Anatomy and function. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 119.

243. Burkett LN. Causative fractures in hamstring strains. *Med Sci Sports.* 1970;2:39–42.

244. Burkett LN. Investigation into hamstring strains: The case of the hybrid muscle. *Am J Sports Med.* 1975;3:228–231.

245. Cibulka MT, Rose SJ, Delitto A, et al. Hamstring muscle strain treated by mobilizing the sacroiliac joint. *Phys Ther.* 1986;66:1220–1223.

246. Reese RC, Buress JP. Athletic training techniques and protective equipment. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 245.

247. Cibulka MT. Rehabilitation of the pelvis, hip and thigh. *Clin Sports Med.* 1989; 8(4):772.

248. Johnson RJ, Beynnon BD, Nichols CE, Renstrom P. The treatment of injuries of the anterior cruciate ligament. *J Bone Joint Surg Am.* 1992;74:140.

249. Fetto JF, Marshall JL. The natural history and diagnosis of anterior cruciate ligament insufficiency. *Clin Orthop.* 1980;147:29–38.

250. Noyes FR, Mooar PA, Matthews DS, Butler DL. The symptomatic anterior cruciate-deficient knee: The long term functional disability in athletically active individuals. *J Bone Joint Surg Am.* 1983;65:154–162.

251. Walla DJ, Albright JP, McAuley E, et al. Hamstring control and the unstable anterior cruciate ligament-deficient knee. *Am J Sports Med*. 1985;13:34–39.

252. Giove TA, Miller SJ. Non-operative treatment of the torn anterior cruciate ligament. *J Bone Joint Surg Am.* 1983;65:184.

253. McDaniel WJ Jr, Dameron TB Jr. Untreated ruptures of the anterior cruciate ligament: A follow up study. *J Bone Joint Surg Am.* 1980;62:696–705.

254. Shelbourne KD, Nite P. Accelerated rehabilitation after anterior cruciate ligament reconstruction. *Am J Sports Med.* 1990;18(3):292.

255. Markey KL. Rehabilitation of the anterior cruciate deficient knee. *Clin Sports Med.* 1985;4(3):513.

256. Minkoff J, Shermin OH. Considerations present to the rehabilitation of the anterior cruciate injured knee. *Exerc Sport Sci Rev.* 1987;15:297–349.

257. Dillingham MF, King WD. Treatment and rehabilitation of cruciate ligament injuries in the knee. *J Back Musculoskel Rehabil.* 1992;2(1):42–53.

258. Shields CL, Brewster CE, Morrisse MC. Rehabilitation of the knee in athletes. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 1055.

259. Slocum DB, Larson R. Rotary instability of the knee: Its pathogenesis and a clinical test to demonstrate its presence. *J Bone Joint Surg Am.* 1968;50:211.

260. Warren RF, Marshall JL, Girgis F. The prime static stabilizer of the medial side of the knee. *J Bone Joint Surg Am.* 1974;56:665.

261. Grood ES, Noyes FR, Butler DL, Suntay WJ. Ligamentous and capsular restraints preventing straight medial and lateral laxity in intact human cadaver knees. *J Bone Joint Surg Am.* 1981;63:1257.

262. Zarins B, Boyle J. Knee ligament injuries. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 929.

263. Bergfeld J. Functional rehabilitation of isolated medial collateral sprains: First, second, and third degree sprains. *Am J Sports Med.* 1979;7:207–209.

264. Cox JS. Functional rehabilitation of isolated medial collateral sprains. *Am J Sports Med.* 1979;7:211–213.

265. DeHaven KE. Aftercare of acute ligament injuries. *Knee*. 1982;7:23–30.

266. Ellsasser JC, Reynolds KC, et al. The non-operative treatment of collateral ligament injuries of the knee in professional football players. *J Bone Joint Surg Am.* 1974;56:1185.

267. Hastings DE. The non-operative treatment of collateral ligament injuries of the knee joint. *Clin Orthop.* 1980;147:22.

268. Holden DL, Eggert AW, et al. The non-operative treatment of grade I and II medial collateral ligament injuries to the knee. *Am J Sports Med.* 1983;11:340.

269. Steadman JR. Rehabilitation of first and second degree sprains of the medial collateral ligament. *Am J Sports Med.* 1979;7:300.

270. Simonet WT, Sim FH. Current concepts in the treatment of ligamentous instability of the knee. *Mayo Clin Proc.* 1984;59:67–76.

271. Indelicato PA. Non–operative treatment of complete tears of the medial collateral ligament of the knee. *J Bone Joint Surg Am.* 1983;65:323.

272. Hughston JC, Walsh WM, Puddy G. Patellar subluxation and dislocation. *Saunders Monographs in Clinical Orthopaedics.* Philadelphia, Pa: WB Saunders; 1984.

273. Beckman M, Craig R, Lehman RC. Rehabilitation of patellofemoral dysfunction in the athlete. *Clin Sports Med.* 1989;8(4):841.

274. Radin EL. A rational approach to the treatment of patellofemoral pain. *Clin Orthop.* 1979;144:107–109.

275. Krammer PG. Patella malalignment syndrome: Rationale to reduce excessive lateral pressure. *J Orthop Sports Phys Ther.* 1986;8:306–308.

276. Hungerford DS, Lennox DW. Rehabilitation of the knee and disorders of patellofemoral joint: Relevant biomechanics. *Orthop Clin North Am.* 1983;14:397.

277. O'Neill DB, Micheli LJ, Warner JP. Patellofemoral stress: A prospective analysis of exercise treatment in adolescents and adults. *Am J Sports Med.* 1992;20(2):151–156.

278. MacConnell J. The management of chondromalacia patellae: A long term solution. *Australian J Phys.* 1986;32(4):215.

279. Stanitski CL. Rehabilitation following knee injury. *Clin Sports Med.* 1985;4(3):495.

280. Mubarak SJ, et al. The medial tibial stress syndrome: A cause of shin splints. *Am J Sports Med.* 1982;10:201.

281. Johnell O, Rausing A, Wandeberg B, Westlin N. Morphological bone changes in shin splints. *Clin Orthop.* 1982;167:180.

282. Puranen J. The medial tibial stress syndrome. *J Bone Joint Surg Br.* 1974;56:712.

283. Devas MB. Stress fractures of the tibia in athletes or shin soreness. *J Bone Joint Surg Br.* 1958;40:227.

284. Friedman MJ. Injuries to the leg in athletes. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 601.

285. Andrish JT, Bergfeld JH, Waldheim J. A prospective study on the management of shin splints. *J Bone Joint Surg Am.* 1974;56:1697.

286. Viitasalo JT, Kuist M. Some biomechanical aspects of the foot and ankle in athletes, with and without shin splints. *Am J Sports Med.* 1983;11(3):125–130.

287. McBride AM. Stress fractures in athletes. *J Am Sports Med.* 1976;3:212.

288. Markey KL. Stress fractures. *Clin Sports Med.* 1987;6(2):405.

289. Gilbert RS, Johnson HS. Stress fractures in military recruits—a review of 12 years' experience. *Mil Med.* 1966;131:716.

290. Pester S, Smith PC. Stress fractures in the lower extremities of soldiers in basic training. *Orthop Rev.* 1992;21(3):297–303.

291. Pather JL, Jusynowitz ML, Snowdy HA, et al. Scintigraphic findings in stress fractures. *J Bone Joint Surg Am.* 1977;59:869.

292. Kimball PR, Savastano AA. Fatigue fractures of the proximal tibia. *Clin Orthop.* 1970;70:170

293. Hunter LY. Stress fractures of the tarsal navicular more frequent than we realize. *Am J Sports Med.* 1981;9:217.

294. Vanital ME, Keene JS, Lang TA, Clancy WG Jr. Stress fractures of the great toe sesamoid. *Am J Sports Med.* 1982;10:122.

295. Marymont JH Jr, Mills G, Merritt Q, Davis W. Fractures of the lateral cuneiform bone in the absence of severe direct trauma. *Am J Sports Med.* 1980;8:135.

296. Glick JM, Sampson TG. Ankle and foot fractures in athletics In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990:526.

297. Derscheid GL, Brown WC. Rehabilitation of the ankle. *Clin Sports Med.* 1985;4(3):527.

298. Singer KM, Jones DC. Soft tissue conditions of the ankle and foot. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 498.

299. Nicholas JA, Strizak AM, Veras G. A study of thigh muscle weakness in different pathological states of the lower extremity. *Am J Sports Med.* 1976;4(6):241.

300. Waller JF Jr, Maddalo A. The foot and ankle linkage system. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 412.

301. Gould N, Karson R. Stenosing tenosynovitis of the pseudosheath of the tendo-achilles. *J Foot Ankle Surg.* 1980;1:3.

302. Ljungqvist R. Partial subcutaneous ruptures of the patellar tendon. *Proceedings of the First Scandinavian Sports Medicine Conference.* Syntex Ter. 1977;2:89.

303. Reese RC Jr, Burruss JP. Athletic training techniques and protective equipment. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, Mo: CV Mosby; 1990: 245.

304. Andrews JR. Overuse syndrome of the lower extremity. *Clin Sports Med*. 1983;2(1):137.

305. Krisoff WB, Ferris WB. Runner's injuries. *Phys Sports Med.,* 1979;7(12):53–61.

306. Torg JS, Pavlov H, Torg E. Overuse injuries in sport: The foot. *Clin Sports Med.* 1987;6(2):291.

307. Newell SG, Miller SJ. Conservative treatment of plantar fascia strain. *Phys Sports Med.* 1977;11:68–73.

308. Roy S. How I manage plantar fasciitis. *Phys Sports Med.* 1983;11(10):127–131.

309. Whitesell J, Newell JSG. Modified low dye strapping. *Phys Sports Med.* 1980;8(A):1299–1300.

310. Clancy WG. Runner's injuries: Part 2: Evaluation and treatment of specific injuries. *Am J Sports Med*. 1980;8(4):287.

ACRONYMS AND ABBREVIATIONS
Volumes 1 and 2

A

AAEM: American Association of Electrodiagnostic Medicine
AAROM: active assistive range-of-motion
ABG: arterial blood gas
ABPM&R: American Board of Physical Medicine and Rehabilitation
ABPM: American Board of Physical Medicine
ABR: auditory brainstem response
AC: acromioclavicular
ACE: angiotensin converting enzyme
ACL: anterior cruciate ligament
ACTH: adrenocorticotropic hormones
ADH: antidisseretic hormone
ADL: activities of daily living
ADP: adenosine diphosphate
AE: above elbow
AFO: ankle/foot orthosis
AFQT: Armed Forces Qualification Test
AGR: Active Guard Reserve
AKA: above-knee amputation
AMC: Army Medical Center
AMEDD: U.S. Army Medical Department
AMSC-CIR: Army Medical Specialist Corps Clinical Investigation and Research
AMSC: Army Medical Specialist Corps
ANG: Army National Guard
AP: anterior–posterior
APDAB: Army Physical Disability Appeals Board
AR: Army Regulation
ARC: American Red Cross
ARG: Army Reservists and National Guard
AROM: active range-of-motion
ARVN: Army of the Republic of Vietnam
ASIA: American Spinal Injury Association
ATH: air transportable hospital
ATN: acute tubular necrosis
ATP: adenosine triphosphate
ATPase: adenosine triphosphatase

B

BAEP: brainstem auditory evoked potential
BAER: brain stem auditory evoked response
BAM: basilar artery migraine
BE: Below elbow
BEAM: brain electrical activity mapping
BEE: basal energy expenditure
BIA: Brain Injury Association
bid: twice daily
BKA: below-knee amputation
BP: blood pressure
BSA: body surface area
BUN: blood urea nitrogen

C

CAD/CAM: computer aided design/computer aided manufacturing
CAPTE: Commission on Accreditation for Physical Therapy Education
CASD: computer aided socket design
CAT/CAM: contoured adducted trochanteric-controlled alignment method
CDP: cytidene 5′-diphophocholine
CED: cognitive enhancing drug
CK: creatine kinase
CMAP: compound motor action potential
CMRR: common mode rejection ratio
CMST: circulation, motion, sensation, and temperature
CMUAP: compound motor unit action potential
CN: cranial nerve
CNS: central nervous system
C.O.: cardiac output
CO_2: carbon dioxide
COMMZ: combat zone
CONUS: continental United States
CP: conventional prosthesis
CP: creatine phosphate
CPAP: continuous positive airway pressure
CPM: continuous passive motion
C-PMN: C polymodal nociceptors
CPR: cardiopulmonary resuscitation
CRPS: Complex Regional Pain Syndrome
CRT: cathode ray tube
CSF: cerebrospinal fluid
CT: computed tomography
CTR: carpal tunnel release
CTS: carpal tunnel syndrome

D

dB: decibel
DCCS: Deputy Commander for Clinical Services
DEXA: dual photon X-ray absorptiometry
DF: dorsiflexion
DIP: distal interphalangeal
diphosphonate EHDP: disodium ethane-1-hydroxy-1, 1-diphosphonate
DoD: Department of Defense
DREZ: dorsal root entry zone
DVA: Department of Veterans Affairs
DVT: deep venous thrombosis

E

ECU: environmental control unit
EEG: electroencephalogram/electroencephalographic/electroencephalography
EF: Epileptiform findings
EMG: electromyographic/electromyography
ENG: electronystagmography
EPB
ERG: electroretinogram
ERMPs: early recovery management programs
ESWL: extracorporeal shock wave lithotripsy

F

FAD: (McMaster) Family Assessment Devise
FAD: flavin adenine dinucleotide
$FADH_2$: FAD gains electrons to become
FAM: Functional Assessment Measure
FDA: Food and Drug Administration
FDP: fibrin degradation product
FES: functional electrical stimulation
FF: full field
FIM: Functional Independence Measure
FNQ: Family Needs Questionnaire

FS: focal slowing
FVC: forced vital capacities
FY: fiscal year

G

GABA: gamma-aminobutyric acid
GAP-43: growth-associated protein-43
GCS: Glasgow Coma Scale
GED: General Education Diploma
GI: gastrointestinal
GMO: general medical officer
GOS: Glasgow Outcome Scale
GPB: glossopharyngeal breathing

H

H_2: Histamine$_2$-receptor
HDL: high density lipoprotein
HF: half field
HIV: human immunodeficiency virus
HO: heterotopic ossification
HR: heart rate
HTL: hearing threshold level
HTM: high threshold mechanoreceptors

I

IASP: International Association for the Study of Pain
IC: intermittent catheterization
ICP: intracranial pressure
ICU: intensive care unit
International 10-20: The International Ten-Twenty System of Electrode Placement
IP: interphalangeal
IPI: interpotential interval
IPOP: immediate postoperative prosthesis
IPORD: immediate postoperative rigid dressing
ISNY: Icelandic-New York
ITB: iliotibial band
IU: international units
IV: intravenous

K

KAFO: knee-ankle-foot orthosis

L

L-dopa: levodopa
LE: lower extremity
LHS: left heelstrike
LLB: long leg brace
LOD: line of duty
LTM: low threshold mechanoreceptors

M

MAOI: monoamine oxidase inhibitor
MCL: medial collateral ligament
MCP: metacarpophalangeal
MCVOST: Medical College of Virginia Olfactory Screening Test
MEB: Medical Evaluation Board
MEDCOM: Medical Command
MEP: motor evoked potential
MESI: Mangled Extremity Syndrome Index
MF2K: Medical Force 2000
ML: medial-lateral
MMPI-2: Minnesota Multiphasic Personality Inventory-2
MMRB: Military Medical Retention Board
MMTN: myelinated mechanothermal nociceptors
MOD: modification
MOS: military occupation specialty
MP: myoelectric prosthesis
MP: metacarpophalangeal
MPA: microstomia prevention appliance
MRI: magnetic resonance imaging
mRNA: messenger ribonucleic acid
MS: multiple sclerosis
MSH: melanocyte stimulating hormone
MTF: medical treatment facility
MTP: metatarsophalangeal
MVC: maximum voluntary isometric contraction

N

NAD^+: nicotinamide adenine-dinucleotide
NADH: NAD+ gains hydrogen to become
NARSUM: narrative summary
NATO: North Atlantic Treaty Organization
NCS: nerve conduction study
NG: National Guard
NGF: nerve growth factor
NHIF: National Head Injury Foundation
NIH: National Institutes of Health
Nm: knee moment
NMDA: *N*-methyl *D*-aspartate
NMS: neuromusculoskeletal
NPO: nothing by mouth
NPT: nocturnal penile tumescence
NSAID: nonsteroidal antiinflammatory drug
NSNA: normal shape, normal alignment

O

OBLA: onset of blood lactate accumulation
OEA: Operations, Evaluations, and Analysis Officer
OH: orthostatic hypotension
OT: occupational therapist/therapy
OTSG: Office of The Surgeon General

P

PA: Physician Assistant
PAFO: plastic ankle-foot orthosis
PCA: patient controlled analgesia
P_{CO_2}: partial pressure of carbon dioxide
PDB: Physical Disability Branch
PEB: Physical Evaluation Board
PEBLO: Physical Evaluation Board Liaison Officer
PEEP: positive end-expiratory pressure
PERSCOM: Personnel Command
PF: plantarflexion
PFT: pulmonary function test
PIP: proximal interphalangeal
PMO: Personnel Management Officer
PMR: physical medicine and rehabilitation
PNF: proprioceptive neuromuscular facilitation
P_{O_2}: partial pressure of oxygen
PROM: passive range-of-motion
PT: physical therapist/therapy
PT: prothrombin time
PTA: posttraumatic amnesia
PTB-TCS: patellar tendon bearing, total contact socket
PTE: posttraumatic epilepsy
PTSD: posttraumatic stress disorder

R

1RM: one repetition maximum test
RDA: recommended dietary allowance
RGO: reciprocating gait orthosis
RH: thyrotropin releasing hormone
RHO: right heeloff
RHS: right heelstrike
RQ: respiratory quotient
RSD: reflex sympathetic dystrophy
RSDS: reflex sympathetic dystrophy syndrome
RTO: right toeoff
RTS: right toestrike

S

3S: silicone suction socket
SACH: solid-ankle cushion-heel
SAFE: stationary attachment, flexible endoskeleton
Sca: Slow component a
Scb: Slow component b
SCI: spinal cord injury
SCL-90: Symptom Checklist-90
SCV: slow component velocity
SEE: standard error of the estimate
SFC: soluble fibrin complex
SIADH: secretion of inappropriate antidiuretic hormone
SIDS: Sudden Infant Death Syndrome
SMP: sympathetically maintained pain
SNAP: sensory nerve action potential
SP: silent period
SPECT: single photon emission computed tomography scans
SPL: sound-pressure level
SSRI: selective serotonin reuptake inhibitors
STEN: STored ENergy foot
STSG: split thickness skin graft
SV: stroke volume
SWOB: separated without benefits
SWSP: separated with severance pay

T

TABPM: American Board of Physical Medicine
TBI: traumatic brain injury
TBSA: total body surface area
TBSAB: total body surface area burned
TCA: tricyclic antidepressants
TCL: tibial collateral ligament
TDRL: Temporary Disability Retired List
TENS: transcutaneous electrical nerve stimulation
TES: total elastic suspension
TD: termal device
THA: tetrahydro-9-aminoacridine
tid: three times per day
TKA: trochanter, knee, ankle (line)
TLE: temperolimbic epilepsy
TLSO: thoracolumbosacral orthosis
TO: Theater of Operations
TOS: thoracic outlet syndrome
TPR: total peripheral resistance
TRH: thyrotropin releasing hormone
TRM: ten repetition maximum
TUN: total urinary nitrogen

U

UCL-BC: University College of London Bioengineering Center
UDS: Utah Dynamic Socket
UE: upper extremity
UN: United Nations
USAPDA: United States Army Physical Disability Agency
USAR: United States Army Reserve
USC: United States Code
UTI: urinary tract infection
UUN: urinary urea nitrogen

V

VA: Veterans Affairs, Department of
VAMC: Veterans Affairs Medical Center
VASRD: Veterans Administration Schedule for Rating Disabilities
VEP: visual evoked potentials
VHIS: Vietnam Head Injury Study
V_{O_2}max: volume of maximum oxygen consumption
V_{O_2}: oxygen consumption

W

WBC: white blood cell
WBGT: wet-bulb globe temperature
WDR: wide dynamic range
WHFO: wrist, hand, finger orthosis
WMSC: Women's Medical Specialist Corps
WRAMC: Walter Reed Army Medical Center

A

B

C

D

E

F

G

H

I

J

K

M

N

O

P

Q

R

S

T

U

V

W

X

Y

Z