Physical Medicine and Rehabilitation Board Review

Third Edition

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I dedicate this book to two of the most important people in my life who have passed on:

My wonderful father, Pasquale Cuccurullo; his love, support, and encouragement are deeply missed since he passed away from lung cancer in 2004.

Also, my dear friend, Kathy Wong, MD. The spirit, integrity, and grace she brought to her patients and the field of Physical Medicine and Rehabilitation is greatly missed since she died of breast cancer at the young age of 36.

This book is also dedicated to:

My husband Alec, my loving partner in life;

My four children Michelle, Alexander, Amanda, and Nicholas, who are the joys of my life;

My mother, Connie, my support system throughout my entire life;

My mentors and teachers, especially Dr. Thomas E. Strax, my inspiration in all aspects of medicine both clinical and academic, and Dr. Ernest W. Johnson, my encouragement to take on a challenge;

And the residents of the Rutgers Robert Wood Johnson Medical School, and JFK Johnson Rehabilitation Institute Residency Program, whose hunger for knowledge inspired the concept of this review book.

It is only because of the support and encouragement of these people that this project could be completed.
Production Staff

We gratefully acknowledge the contributions made by the artists involved in this project. We sincerely thank them for their dedication, expertise, creativity, and professionalism. Special thanks also to Bob Silvestri and the JFK Johnson Rehabilitation Institute Prosthetic and Orthotic Laboratory.

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4.11 6.8 6.15 6.38 7.16 9.10 10.7 10.13 12.13
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4.33 4.47 4.64 4.77 4.97 4.112 4.125 4.133 4.155 4.175 7.5 7.17 7.34 Tables
4.34 4.48 4.71 4.89 4.98 4.113 4.126 4.135 4.158 4.181 7.6 7.18 8.1 10.29

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11.5

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## Contents

*Foreword*  xiii  
*Preface*  xv  
*Acknowledgments*  xvii  
*Contributors*  xix  
*Introduction: Board Certification*  xxiii  

1. **STROKE**  1  
   **Richard D. Zorowitz, MD, Edgardo Baerga, MD, and Sara J. Cuccurullo, MD**  
   - Introduction  1  
   - Basic Neuroanatomical Review of the Major Vessels Involved in Stroke  4  
   - Types of Stroke  7  
   - Diagnostic Studies  18  
   - Medical Treatment  20  
   - Stroke Rehabilitation  26  

2. **TRAUMATIC BRAIN INJURY**  53  
   **Elie Elovic, MD, Edgardo Baerga, MD, Gary F. Galang, MD, Sara J. Cuccurullo, MD, Michael Reyna, MD, and Richard J. Malone, DO**  
   - Introduction  53  
   - Pathophysiology of TBI  55  
   - Disorders of Consciousness  59  
   - Posturing Secondary to Head Injury  62  
   - Prognosis After TBI: An Evidence-Based Approach  62  
   - Medical Management of TBI  70  
   - Surgical Management in TBI  73  
   - Medical and Neurologic Complications After TBI  74  
   - Mild TBI (Concussion) and Postconcussive Syndrome  94  

3. **RHEUMATOLOGY**  101  
   **Thomas R. Nucatola, MD, Eric D. Freeman, DO, and David P. Brown, DO**  
   - Rheumatoid Arthritis  101  
   - Osteoarthritis  113  
   - Juvenile Rheumatoid Arthritis  116  
   - Juvenile Spondyloarthropathies  118  
   - Crystal-Induced Synovitis  120  
   - Seronegative Spondyloarthropathies  121  
   - Other Rheumatoid Diseases  128  
   - Vasculitides  133  
   - Sjögren’s Syndrome  134  
   - Infectious Arthritides  135  
   - Deposition/Storage Disease-Related Arthritis  137  
   - Other Systemic Diseases with Arthritis  138  
   - Charcot Joint  139  

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Atraumatic Arthritis 140
Fibromyalgia Syndrome 142
Complex Regional Pain Syndrome 143
Tendon Disorders 146

4. MUSCULOSKELETAL MEDICINE 149

Upper Extremities
David P. Brown, DO, Eric D. Freeman, DO, Sara J. Cuccurullo, MD, Urania Ng, MD, and Ian B. Maitin, MD, MBA

Lower Extremities
David P. Brown, DO, Eric D. Freeman, DO, Sara J. Cuccurullo, MD, Urania Ng, MD, and Ian B. Maitin, MD, MBA

Spine
Ted L. Freeman, DO, and Eric D. Freeman, DO

Introduction 341
BasicPeripheral Nervous System Anatomy 341
Pathophysiology 351
Clinical Instrumentation 356
Nerve Conduction Studies (NCS) 363
Somatosensory Evoked Potentials (SSEPs) 376
Basic Needle EMG 378
6. PROSTHETICS AND ORTHOTICS 471
Heikki Uustal, MD, Edgardo Baerga, MD, and Jaclyn Joki, MD
Gait Analysis 471
Amputation and Prosthetics 477
Assistive Devices 520
Shoes and Lower Limb Orthoses 522
Orthotics 528
Lower Extremity Orthoses for Pressure Redistribution 534
Upper-Limb Orthoses 536
Spinal Orthoses 542

7. SPINAL CORD INJURIES (SCI) 551
Steven Kirshblum, MD, Jeremiah Nieves, MD, Dana Clark, MD, Priscila Gonzalez, MD, Sara J. Cuccurullo, MD, and Lisa Luciano, DO
Epidemiology 551
Anatomy of the Spine 553
Spinal Pathology 557
SCI Classification 562
Medical Complications of SCI 575
Pain in the SCI Patient 608
Pressure Ulcers 614

8. PHYSICAL MODALITIES, THERAPEUTIC EXERCISE, EXTENDED BEDREST, AND AGING EFFECTS 621
Thomas E. Strax, MD, Martin Grabois, MD, Priscila Gonzalez, MD, Steven V. Escaldi, DO, Michael Reyna, MD, and Sara J. Cuccurullo, MD
Physical Modalities 621
Therapeutic Exercise 640
Effects of Extended Bedrest: Immobilization and Inactivity 647
Evaluation of Functional Independence 648
Physiologic Effects of Aging 649

9. PULMONARY, CARDIAC, AND CANCER REHABILITATION 657
Pulmonary Rehabilitation
Priscila Gonzalez, MD, Nicholas G. Melillo, MD, Daphne Karen MacBruce, MD and Sara J. Cuccurullo, MD
Cardiac Rehabilitation
Iqbal Jafri, MD, and Troy Wood, MD
Cancer Rehabilitation
Priscila Gonzalez, MD, Lisa Luciano, DO, and Richard M. Schuman, MD, FACP
Palliative Care
Anna Maria Dunn, MD
Pulmonary Rehabilitation 657
Cardiac Rehabilitation 684
Cancer Rehabilitation 708
Palliative Care 727

10. PEDIATRIC REHABILITATION 733
*Roger Rossi, DO, Michael Alexander, MD, Kathryn Eckert, BS, and Sara J. Cuccurullo, MD*

- Genetics and Chromosomal Abnormalities 733
- Development and Growth 735
- Pediatric Limb Deficiencies 741
- Diseases of the Bones and Joints 747
- Connective Tissue and Joint Disease 758
- Pediatric Burns 769
- Pediatric Cancers 773
- Pediatric Traumatic Brain Injury (TBI) 776
- Cerebral Palsy (CP) 782
- Spina Bifida (Myelodysplasia) 799
- Neuromuscular Diseases in Children 808

11. PAIN MEDICINE 831
*Jing Liang, MD, Krystle Williams, MD, Joseph Lee, MD, Janet J. Lee, MD, Michael Ra, DO, MPT, and Didier Demesmin, MD*

- Introduction 831
- Pharmacology 833
- Pain Syndromes 838
- Pain Intervention 847

12. ASSOCIATED TOPICS IN PHYSICAL MEDICINE AND REHABILITATION 861

- Spasticity *Elie Elovic, MD, Edgardo Baerga, MD, Steven V. Escaldi, DO, and Michael Reyna, MD*
- Movement Disorders *Elie Elovic, MD, Edgardo Baerga, MD, and Michael Reyna, MD*
- Wheelchairs *Steven Kirshblum, MD, Lisa Luciano, DO, Mary T. Shea, MA, OTR, ATP, and Sean McCarthy, MS, OTR, ATP*
- Osteoporosis *Barbara Hoffer, DO, Sara J. Cuccurullo, MD, Krishna J. Urs, MD, and Chun Ho, MD*
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- Biostatistics *Joseph Lee, MD, Kathy Kalmar, PhD, Bart K. Holland, PhD, and Heather Platt, MD*
- Ethics *Jegy Tennison, MD, and Tejal Patel, MD*
- Multiple Sclerosis *David S. Rosenblum, MD*
- Ultrasound *Steven V. Escaldi, DO*

- Spasticity 861
- Movement Disorders 874
- Wheelchairs 883
- Osteoporosis 902
- Rehabilitation of Burn Injuries 918
- Biostatistics 928
- Basic Principles of Biomedical Ethics 933
- Multiple Sclerosis (MS) 936
- Diagnostic Musculoskeletal Ultrasound 946

Epilogue 955

*Thomas E. Strax, MD*

Index 957
Every book needs, indeed, should have a third edition, and Dr. Sara Cucurullo’s Physical Medicine and Rehabilitation Board Review is one they must have!

Why?

A lot of residents are completing their 4 years of training and education and are now ready for the Board Certification exam challenge.

Over the years many residents have confronted the problem of “what to study for the Boards.” This is a serious problem that had a few, generally unsatisfactory, solutions:

1. Attend one of the review courses. Can’t cover everything but makes a 5-day effort. Costs! Cost is only for the Board Review course—no plane fare to and from, living expenses for 4 to 6 days.
2. Home perusal of the two major PM&R texts and electrodiagnostic medicine. This is a stupendous (one could say—outrageous) task, hardly possible for a busy 4th-year resident to manage.
3. Reviewing the last year of both journals (American Journal of Physical Medicine and Rehab, and PM&R journal)
4. Review all of the literature. One of our former residents made the mistake of trying to review the last 10 years of the Archives of PM&R, and the American Journal of PM&R, Muscle & Nerve. He obviously believed he had learned everything. He only later discovered this time-consuming preparation interfered with his performance rating in the residency program.
5. Get an individual tutor.
6. Option: Instead of reading these books, attend the Journal Club in your local area, approximately one time per month.

In the old days when we had only one skinny journal, one of my resident colleagues decided to review three textbooks—neurology, orthopedics, and internal medicine, with pediatrics in reserve (if time was available). He discovered too late he could not cover the key subjects—even if he only read the boldface type. (N.B. Textbooks are 5 years old when published.)

However, this elegant volume finally fulfills this critical void and will supply reasonable and current PM&R diagnostic and management facts for the prospective Board candidate.

It can be studied in a reasonable time without speed reading and it is up-to-date with valuable and relevant information. The PM&R Board Review Third Edition has over 950 pages of nuggets. In addition, many physiatrists are coming up for re-certification—certainly a major need for a PM&R comprehensive study and the solution is: Dr. Cucurullo’s convenient and relatively inexpensive volume!

My prediction—a third edition is the optimal solution for studying for the board exams and recertification!

Thank you, Dr. Sara Cucurullo!

Ernest W. Johnson, MD
Preface

*Physical Medicine and Rehabilitation Board Review*, Third Edition, will appeal to medical students, residents, and practicing physiatrists. The book concentrates on board-related concepts in the field of Rehabilitation Medicine. Residents will find the book essential in preparing for Part I and Part II of the Physical Medicine and Rehabilitation Board Certification because it is one of the only books of its kind with major focus on board-related material giving a synopsis of up-to-date PM&R orthopedic, neurologic, and general medical information all in one place. Over 500 diagrams simplify material that is Board pertinent. In this way, important concepts are clarified and reinforced through illustration. All of the major texts of this specialty have been referenced to give the board examinee the most timely and relevant information and recommended reading.

The Third Edition differs from previous editions with the addition of the following chapters and subsections: Pain Medicine chapter, and Ethics, Palliative Care, and Ultrasound subsections. In addition, all relevant epidemiology, treatments, and medications have been updated by the authors throughout the book. This book is clearly different from most texts. It is written in outline form and is about one-third the size of most textbooks. The topics are divided into major subspecialty areas and are authored by physicians with special interests and clinical expertise in the respective subjects. Board pearls are highlighted with an open-book icon throughout the text. These pearls are aimed at stressing the clinical and board-eligible aspects of the topics. This format was used to assist with last-minute preparation for the board examination and was inspired by the *Mayo Clinic Internal Medicine Board Review*. The contents are modeled after the topic selection of the *American Academy of Physical Medicine and Rehabilitation (AAPMR) Self-Directed Medical Knowledge Program* (which is used by residents nationwide to prepare for the Self-Assessment Examination [SAE]). This was done specifically to help all residents, Post Graduate Year 2, 3, 4, in yearly preparation and carryover from the SAE preparation to board exam preparation.

Two key points need to be addressed prior to using this text. This book is not a comprehensive textbook of PM&R. All chapters are prepared under the assumption that readers will have studied at length one or more of the standard textbooks of PM&R before studying this review.

My hope is that this text is a valuable tool to all physicians preparing for both the written and oral board exams, and also in managing issues of patient care. Practicing physiatrists should also find this book helpful in preparation for the recertifying exam. Because this is one of the first textbooks designed specifically for PM&R board preparation, the authors welcome any ideas for improvement from any of the readers. We wish you all the best in your studies.

*Sara J. Cuccurullo, MD*
I’ve had the pleasure of helping residents learn what they need to know for their Physical Medicine and Rehabilitation (PM&R) Boards at JFK Johnson Rehabilitation Institute, Robert Wood Johnson Medical School for more than 20 years. Over these years, I have had many requests for my yearly revised notes from former residents and from residents outside our program. For this reason, I gathered together an expert group of knowledgeable physicians to put together a comprehensive PM&R Board Review text. After the first edition was published it was realized that improvements would make this text better with additions, updates, and needed alterations to the existing text. The second edition was an improved version of Physical Medicine and Rehabilitation Board Review. The third edition has been further improved, updated, and expanded, to include new, highly board relevant topics such as Pain Management, Ethics, Ultrasound, and Palliative Care. I want to thank all those individuals who reached out to me to point out edits and subject matter inclusion that would improve this third edition textbook.

Physical Medicine and Rehabilitation Board Review, Third Edition, reflects the commitment of the authors and the faculty at Rutgers Robert Wood Johnson Medical School in the Department of Physical Medicine and Rehabilitation based at JFK Johnson Rehabilitation Institute to produce a text that would be used as a guide containing selected topics considered important for physicians preparing for either the certifying or the recertifying examination offered by the American Board of Physical Medicine and Rehabilitation. This text hopefully presents clear practical information for both residents studying for the boards of PM&R and for practicing physicians. This text should be of great value in not only preparing for the ABPMR board exam but also caring for patients. Thanks for this textbook coming to print is given to Thomas Strax, MD. His encouragement and willingness to support this project from the start has been an inspiration in seeing this textbook come to realization. Special thanks have to be given to the administration of JFK Johnson Rehabilitation Institute and JFK Medical Center for their encouragement and financial support, without which this book would not have been possible. Specifically, I would like to sincerely thank J. Scott Gebhard, Anthony Cuzzola, Marci Gietter, and Ray Fredericks. I would also like to thank Vicente H. Gracias, MD, Interim Dean of Rutgers Robert Wood Johnson Medical School, who supports each and every academic endeavor put forth from our Department of Physical Medicine and Rehabilitation, and Peter Amenta, MD, PhD, Rutgers Robert Wood Johnson Medical School, who has been a support to me personally over the many years I have known him.

I will also be eternally grateful to four of my former students, Joseph Lee, MD (my dedicated and tireless assistant editor), Edgardo Baerga, MD, Eric Freeman, DO, and Priscila Gonzalez, MD. It was their stamina and perseverance that enabled the first edition of this text to come to fruition. Their energy and enthusiasm were truly inspirational. I am grateful to all the authors for their completion of the manuscripts. I greatly acknowledge the support of Demos Medical Publishing, and specifically Beth Barry, Joanne Jay, and Lee Oglesby.

In addition, I would like to thank Beverly Bolger, my program coordinator, for all of her support throughout the production of this third edition.

Special thanks must also be given to Leslie Bagay, MD (also one of my students), project manager of the book, who has been a tremendous support throughout this project. In addition I would like to thank Kathryn Eckert for her help with the front matter.

I would also like to thank Dr. Ernie W. Johnson who has been very inspirational in any educational project I have undertaken. He is truly one of the giants in the field of PM&R. His interest in
writing the foreword, and giving his input prior to its publication is greatly appreciated. I would like to acknowledge the enormous support and understanding I have received from my husband, four children, and mother during the formulation of this new edition. It is my sincere hope that Physical Medicine and Rehabilitation Board Review, Third Edition will receive a warm reception. My co-authors and I look forward to receiving comments and suggestions from the readers.

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Introduction: Board Certification

The discussion in this section is aimed primarily at candidates preparing for the American Board of Physical Medicine and Rehabilitation (ABPMR) certification or maintenance examinations in Physical Medicine and Rehabilitation.

The following information was collected from and calculated by the ABPMR and is available on the ABPMR website at www.abpmr.org.

THE PURPOSE OF CERTIFICATION

The intent of the certification process as defined by Member Boards of the ABMS (American Board of Medical Specialties) is to provide assurance to the public that a certified medical specialist has successfully completed an accredited residency training program and an evaluation, including an examination process, designed to assess the knowledge, experience and skills requisite to the provision of high quality patient care in that specialty. Diplomates of the ABPMR possess particular qualifications in this specialty.

THE EXAMINATION

As part of the requirements for certification by the ABPMR, candidates must demonstrate satisfactory performance in an examination conducted by the ABPMR covering the field of PM&R. The examination for certification is given in two parts, Part I (computer-based) and Part II (oral).

Parts I and II of the Board examination are given once each year at times and places as the Board designates. While Part I of the examination is administered simultaneously at Pearson Professional centers nationwide, Part II is administered only in Rochester, Minnesota.

EXAMINATION ADMISSIBILITY REQUIREMENTS

PART I ADMISSIBILITY REQUIREMENTS

In order to have the application considered for examination, the applicant must be scheduled to complete the graduate medical education requirements on or before August 31 immediately following the scheduled examination date for which he or she has applied. Satisfactory completion of the educational and training requirements in force at the beginning of the resident’s training in an accredited program will be considered acceptable for application for admissibility to the certification examinations. Candidates who are engaged in the Clinical Research Pathway or who are pursuing Dual Specialty Certification should refer to the ABPMR website for more details.

A form included in the application materials is a professional reference form, to be completed and submitted to the Board office by the applicant’s residency program director.

The applicant should supply this form to the program director, who then should promptly send it to the ABPMR office. Final admissibility is contingent upon receipt of the final-year evaluation by the program director, due July 1 in the examination year. If a resident is placed on probationary status during the final year of the residency program, this status must be rescinded by the program director before July 1 for the resident to be admissible.
PART II ADMISSIBILITY REQUIREMENTS

Part II of the ABPMR certification examination is an oral examination. To be admissible for the examination, applicants must have passed Part I prior to applying for Part II. The application and related forms for Part II are available on the ABPMR website.

An applicant applying for Part II must complete a form provided by the Board that describes the professional time spent after completion of residency training. The applicant is required to submit copies of all current, valid, and unrestricted licenses (including expiration date) to practice medicine or osteopathy in a United States or Puerto Rico licensing jurisdiction, or licensure in Canada. Evidence of unrestricted licensure in all states where a license is held will be required prior to issuance of the certificate. Candidates are required to take and pass the Part I (computer-based) certification examination before applying for the Part II examination.

RE-APPLICATION

Physicians who have initially applied for and failed or did not take either Part I or Part II can apply for admissibility for re-examination or examination during any subsequent examination administration during the board eligibility period. The same requirements will be in effect for reapplication as for initial admissibility. Currently, there is no limit to the number of times a physician may reapply for examinations though a physician must successfully complete the initial certification process within seven years of completing residency training.

THE EXAMINATION: Part I

The computer-based examination consists of 325 multiple-choice questions, divided with 165 questions administered in the morning session and 160 questions administered in the afternoon session, each allowing three hours. An on-screen tutorial is available at the beginning of the first session, allowing the examinee to become familiar with both the computer and the format of the examination. The examination questions are designed to test the candidate’s knowledge of basic sciences and clinical management as related to PM&R and will be in the form of objective testing. Two forms of state- or government-issued identification (non-expired, including a photo and a signature) will be required of candidates presenting for the examination. No notes, textbooks, other reference materials, scratch paper, or electronic devices may be taken into the examination room. Please refer to the ABPMR website for more detailed information about how to prepare for the Part I computer-based examination.

Part I of the certification exam outline consists of two independent dimensions or content domains, and all test questions are classified into each of these domains. The major content domains appear below along with their approximate target weights.

PART I EXAMINATION OUTLINE

Class 1: Type of Problem/Organ System

A. Neurologic Disorders (30%)
   1. Stroke
   2. Spinal Cord Injury
   3. Traumatic Brain Injury
   4. Neuropathies
      a) Mononeuropathies
      b) Polyneuropathies
      c) Carpal Tunnel Syndrome
      d) Other Entrapment Neuropathies
   5. Other Neurologic Disorders
      a) Multiple Sclerosis
      b) Motor Neuron Disease
c) Poliomyelitis
d) Guillain Barré Syndrome
e) Cerebral Palsy
f) Spina Bifida
g) Duchenne Muscular Dystrophy
h) Myotonic Muscular Dystrophy
i) Inflammatory Myopathies
j) Other Myopathies
k) Thoracic Outlet Syndrome
l) Plexopathy
m) Radiculopathy
n) Parkinson Disease
o) Other Neuromuscular Disorders

B. Musculoskeletal Medicine (32%)
1. Arthritis
   a) Rheumatoid Arthritis
   b) Osteoarthritis
   c) Collagen Disease
d) Spondyloarthropathy
e) Other Arthritis
2. Soft Tissue & Orthopedic Problems
   a) Acute Trauma
   b) Chronic Trauma/Overuse
c) Complex Regional Pain Syndrome type I (Reflex Sympathetic Dystrophy)
d) Fibromyalgia/Myofascial Pain
e) Burns
f) Fractures
g) Osteoporosis
h) Spinal Disorders
   i) Strains/Sprains
   j) Tendinitis/Bursitis
   k) Orthopedic/Rheumatology
   l) Other Soft Tissue Disease

C. Amputation (5%)
1. Upper Extremity
2. Lower Extremity

D. Cardiovascular & Other Systems (8%)
1. Cardiovascular
   a) Ischemic Heart Disease
   b) Other Heart Disease
c) Peripheral Arterial Disease
d) Venous Disease
e) Vascular Disorders
   f) Lymphedema
g) Hypertension
   h) Other Cardiovascular
2. Pulmonary Disease
   a) Asthma
   b) COPD
c) Pneumonia
d) Impaired Ventilation
e) Other Pulmonary Problems
3. GU/GI Disorders
   a) Neurogenic Bladder
   b) Renal Impairment/Failure
   c) Neurogenic Bowel
   d) Sexuality and Reproductive Issues
   e) Other GU/GI Disorders
4. Cancer
5. Infectious Disease
6. Endocrine/Metabolic [incl. Diabetes]

E. Rehabilitation Problems & Outcomes (15%)
1. Physical Complications
   a) Spasticity
   b) Contracture
   c) Hydrocephalus
   d) Seizures
   e) Pressure Ulcer
   f) Posture/Balance Disorders
   g) Abnormal Gait
   h) Dysphagia/Aspiration
   i) Bed Rest/Deconditioning
   j) Paralysis/Weakness
   k) Heterotopic Ossification
   l) Other Physical Complications
2. Cognitive/Sensory Dysfunction
   a) Speech and Language Disorders
   b) Hearing Impairment
   c) Visual Dysfunction
   d) Cognitive Disorders
   e) Sleep Disorders
   f) Other Cognitive/Sensory Dysfunction
3. Psychiatric/Psychological Problems
   a) Depression
   b) Substance Abuse
   c) Dementia/Pseudodementia
   d) Vegetative State
   e) Other Psych. Problems
4. Pain
5. Other
F. Basic Sciences (10%)

Class 2: Focus of Question/Patient Management

A. Patient Evaluation & Diagnosis (31%)
   1. Physical Exam, Signs & Symptoms
   2. Diagnosis & Etiology
   3. Diagnostic Procedures
      a) Cardiopulmonary Assess/Stress Test
      b) Gait Analysis
      c) Urodynamics
      d) Lab Studies
      e) Synovial Fluid Analysis
f) Medical Imaging  

g) Neuropsychological Evaluation  

h) Other Diagnostic Procedures  

4. Functional Evaluation  

5. Prognosis [Incl. Outcome Measures]  

B. Electrodiagnosis (15%)  

1. General Electrodiagnosis  

2. Instrumentation  

3. Nerve Conduction  

4. Electromyography  

5. Neuromuscular Transmission  

6. H Reflex/F Wave  

7. Special Studies  

C. Patient Management (32%)  

1. Clinical Decision-Making (incl. Ethics)  

2. Physical Agents  

   a) Heat/Cryotherapy  

   b) Hydrotherapy  

   c) Electrostimulation  

   d) Ultrasound  

3. Therapeutic Exercise and Manipulation  

   a) Motor control  

   b) Mobility and Range of Motion  

   c) Strength and Endurance  

   d) Manipulation and Massage  

   e) Traction/Immobilization  

4. Pharmacologic Interventions  

   a) Analgesics  

   b) Antiseizure and Antispasmodics  

   c) Antibiotics  

   d) Psychopharmacologics  

   e) Anti-inflammatory  

   f) Other medications  

5. Procedural/Interventional  

   a) Nerve Blocks  

   b) Anesthetic Injections  

   c) Surgery  

   d) Other Procedural/Interventional  

6. Behavioral/Psychological Modalities  

   a) Relaxation Therapy  

   b) Behavior Modification  

   c) Psychotherapy/Counseling  

   d) Education  

   e) Biofeedback  

D. Equipment & Assistive Technology (10%)  

1. Prosthetics  

2. Orthotics  

3. Other Rehabilitation Technology  

   a) Shoes  

   b) Functional Electrical Stimulation  

   c) Transcutaneous Electrical Nerve Stimulation  

   d) Augmentative Communication
e) Ventilation
f) Wheelchair/Seating
g) Other Devices

E. Applied Sciences (12%)

1. Anatomy
   a) Central Nervous System
   b) Peripheral Nerves
   c) Head/Neck
   d) Shoulder
   e) Arm
   f) Wrist
   g) Hand
   h) Hip
   i) Knee
   j) Leg
   k) Ankle
   l) Foot
   m) Muscle
   n) Bone
   o) Back/Spine: General
   p) Spine: Cervical
   q) Spine: Thoracic
   r) Spine: Lumbosacral
   s) Other Anatomy

2. Physiology
   a) Neurophysiology
   b) Neuromuscular
   c) Cardiovascular
   d) Pulmonary
   e) Genitourinary
   f) Gastrointestinal
   g) Skin and Connective Tissue
   h) Bone and Joints
   i) Autonomic Nervous System
   j) Endocrine

3. Pathology/Pathophysiology
   a) Neurophysiology
   b) Neuromuscular
   c) Cardiovascular
   d) Pulmonary
   e) Genitourinary
   f) Gastrointestinal
   g) Skin and Connective Tissue
   h) Bone and Joints
   i) Autonomic Nervous System
   j) Endocrine

4. Kinesiology/Biomechanics

5. Histology

6. Epidemiology/Risk Factors

7. Nutrition

8. Biochemistry

9. Pharmacology
INTRODUCTION: BOARD CERTIFICATION

10. Research and Statistics
11. Growth and Development
12. Other Basic Science (e.g., physics)

QUESTION FORMAT

The 1998 ABPMR Booklet gave an idea of how the exam looks. These items are not from previous ABPMR exams, nor will they appear on future tests. They are given by ABPMR as a sample for your use. All items are of the “best single choice answer multiple choice” type.

1. Post-acute recovery and community reintegration of the traumatically brain-injured patient are most often hampered by:
   A. Language impairment
   B. Memory impairment
   C. Physical impairment
   D. Financial disincentives
   E. Personality and behavioral impairment

2. Which best describes a feature of short-wave diathermy?
   A. It is used to heat the hip joint.
   B. It produces both direct and reflex blood flow increase.
   C. It is used around the thigh to improve circulation in an ischemic limb.
   D. The dose is regulated by measuring the flow of the high-frequency current through the patient.
   E. Commercially available machines operate at a frequency of 950 MHz.

3. The single most reliable clinical sign for the detection of inflammatory arthritis is
   A. Local tenderness
   B. Painful, limited range of motion
   C. Synovial swelling
   D. Joint effusion
   E. Skin color change

4. Which condition is most likely a contraindication for intra-articular corticosteroid injection therapy?
   A. Crystal-induced synovitis
   B. Diabetes mellitus
   C. Peptic ulcer
   D. Bacteremia
   E. Osteoarthritis

Answers for the above examples are as follows; 1. E, 2. B, 3. C, 4. D. Attempts have been made to avoid ambiguity and typographical or spelling errors, but occasionally they occur. They are not intended to “trip you up” or confuse you.

THE EXAMINATION: Part II

As currently structured, the oral examinations consist of three examiners individually examining the candidate, each conducting a 40-minute segment of the 120-minute examination. Two five-minute breaks divide the three portions of the oral examination.

The Part II Examination is an interactive process between the candidate and the examiner. The examiner will present a vignette comprised of a clinical case description and will subsequently ask questions about diagnostic procedures, therapeutic procedures, and patient management. Candidates will be expected to present, in a concise, orderly fashion, evidence of their proficiency in the management of various clinical conditions within the field of PM&R. Performance on each
vignette is evaluated using performance criteria within the following domains: data acquisition, problem solving, patient management, systems-based practice, and interpersonal and communication skills. The examination content is classified according to both the Class 1 and Class 2 material in the exam outline that follows. A demonstration video of a Part II examination is available on the ABPMR website.

PART II EXAMINATION OUTLINE

Class 1: Patient Diagnosis

A. Cerebral Vascular Disease
   1. Embolic/Thrombotic
   2. Hemorrhagic
   3. Vascular Malformation
   20. Other

B. Central Nervous System (CNS)
   1. Brain Tumor
   2. Cerebral Palsy
   3. Hypoxic Ischemic Encephalopathy
   4. Movement Disorder and Parkinson Disease
   5. Infectious or Inflammatory Disease
   6. Multiple Sclerosis
   20. Other

C. Medical Conditions Resulting in Impairment or Disability
   1. Cancer
   2. Cardiac Rehabilitation
   3. Chronic Obstructive Pulmonary Disease (COPD)
   4. Other Pulmonary Problems
   5. Deconditioning
   6. Immunosuppressive (HIV)
   7. Organ Transplantation
   8. Peripheral Vascular Disease
   20. Other

D. Musculoskeletal—Occupational and Sports Injuries
   1. Acute Trauma
   2. Fractures
   3. Overuse Syndromes/Tendinitis
   4. Strains/Sprains
   20. Others

E. Musculoskeletal Disorders
   1. Amputation and Limb Deficiencies
   2. Burns
   3. Complex Regional Pain Syndrome
   4. Fibromyalgia
   5. Inflammatory Arthritis
   6. Joint Replacement/Arthroplasty
   7. Osteoarthritis
   8. Osteoporosis
   20. Others

F. Neuromuscular Disorders
   1. Hereditary Myopathies and Dystrophies
   2. Inflammatory Myopathies
   3. Focal and Entrapment Neuropathies
4. Hereditary Neuropathy
5. Infectious or Inflammatory Neuropathy
6. Metabolic Neuropathy
7. Plexus Lesions
8. Polyneuropathies
9. Motor Neuron Disorders
10. Neuromuscular Transmission Disorders
20. Other

G. Spinal Cord Injury
1. Infectious and Inflammatory Disease
2. Meningomyelocele and Neural Tube Defects
3. Spondylotic Myelopathy
4. Toxic/Metabolic Conditions
5. Traumatic
6. Vascular Disorders
20. Other

H. Spine Disorders and Radiculopathy
1. Cervical Radiculopathy
2. Thoracic Radiculopathy
3. Lumbosacral Radiculopathy
4. Degenerative Disk Disease
5. Low Back Pain
6. Spondylosis and Spondylolisthesis
20. Other

I. Traumatic Brain Injury
1. Mild
2. Moderate/Severe
20. Other

Class 2: Focus of Patient Evaluation and Management

A. Acute Pain Management
B. Chronic Pain Management
C. Cardiovascular Impairments
D. Complications of Primary Diagnosis
E. Electrodiagnostic Evaluation
F. Gastrointestinal Impairments
G. Genitourinary Impairments
H. Geriatric Rehabilitation
I. Metabolic Nutrition Conditions
J. Musculoskeletal Impairments
K. Neurological Impairments
L. Pediatric Rehabilitation
M. Pressure Ulcers and Other Skin Conditions
N. Prevention of Impairments and Disabilities
O. Psychological and Neurobehavioral Impairments
P. Pulmonary Impairments
Q. Rehabilitative Management
1. Vocational Rehabilitation (Return to Work, etc.)
2. Prosthetics/Orthotics (Prescription, etc.)
3. Durable Medical Equipment
4. Treatment Planning (PT, OT, Modalities, ADLs, etc.)
EXAMINATION RESULTS

Official notification of examination results are sent in writing 6–8 weeks after an examination is administered. Pass/fail results also will be available on the individual candidate’s “Physician Home Page” on the ABPMR website. In the interest of maintaining confidentiality of candidate information, examination results are not given over the telephone, via fax, or e-mail.

Requests to have results mailed to a temporary or new address must be submitted to the ABPMR office in writing, either by mail, fax, or e-mail.

THE CERTIFICATE

Upon approval of the application and the candidate’s successful completion of the examinations, the ABPMR will grant a time-limited certificate to the effect that the candidate has met the requirements of the ABPMR. The recipient of a certificate will be known as a diplomate, or a certificant, of the American Board of Physical Medicine and Rehabilitation.

The Board began issuing 10-year, time-limited diplomate certificates in 1993. The expiration date for these certificates is transitioning to December 31 of the given year. Maintenance of Certification procedures and requirements are described briefly in the following section, and in-depth in a separate Maintenance of Certification Booklet of Information, available at the ABPMR website. Certificates issued prior to 1993 have no time-limited stipulations; however, holders of these pre-1993 certificates may voluntarily participate in the Maintenance of Certification program.

Residents entering a training program must be aware that time-limited certification for PM&R began in 1993 for all diplomates certified thereafter.

PREPARATION FOR THE TEST

The ABPMR has prepared a document that describes the computer testing process. The brochure, titled Preparing for the ABPMR’s Computer-Based Certification Exam, is available on the ABPMR website (https://www.abpmr.org/documents/Preparing_for_CBT.pdf). All candidates should read and understand the testing process including ABPMR policies, as well as testing policies of the computer-based testing center.

Training during medical school forms the foundation on which advanced clinical knowledge is accumulated during residency training. However, the serious preparation for the examination actually starts at the beginning of the residency training in PM&R. Most candidates will require a minimum of 6–8 months of intense preparation for the examination. Cramming just before the examination is counterproductive. Some of the methods for preparation for the Board examination are described below. Additionally, each candidate may develop his or her own system.

It is essential that each candidate study a standard textbook of PM&R from beginning to end. Any of the standard textbooks of PM&R should provide a good basic knowledge base in all areas of PM&R. Ideally, the candidate should read one good textbook and not jump from one to another, except for reading certain chapters that are outstanding in a particular textbook. This book and similar board review syllabi are excellent tools for brushing up on important Board-relevant information several weeks to months before the examination. They, however, cannot take the place of comprehensive textbooks of PM&R. This book is designed as a study guide rather than a comprehensive textbook of PM&R. Therefore, it should not be used as the sole source of medical information for the examination.
HELPFUL RESOURCES

Use past Self-Assessment Examinations for Residents (SAE-R). These are extremely valuable for obtaining practice in answering multiple choice questions. These annual exam questions are available in print format from the American Academy of Physical Medicine and Rehabilitation (AAPMR). These questions are not used on the Board exams, but serve as a means to assess your knowledge on a range of PM&R topics. These study guides are available on the AAPMR website: www.aapmr.org.

Formation of study groups, three to five candidates per group, permits study of different textbooks and review articles in journals. It is important that the group meet regularly and that each candidate be assigned reading materials. Selected review papers and state-of-the-art articles on common and important topics in PM&R should be included in the study materials.

Indiscriminate reading of articles from many journals should be avoided. In any case, most candidates who begin preparation 6 to 8 months before the examination will not find time for extensive study of journal materials. Notes and other materials the candidates have gathered during their residency training are also good sources of information. These clinical “pearls” gathered from mentors will be of help in remembering certain important points.

Certain diseases, many peculiar and uncommon, are eminently “Board-eligible,” meaning that they may appear in the Board examinations more frequently than in clinical practice. Most of these are covered in this book. Several formulas and points should be memorized (such as Target Heart Rate). Most significantly, the clinical training obtained and the regular study habits formed during residency training are the most important aspects of preparation for the examination. Review courses are also available if desired.

DAY OF THE EXAMINATION

Adequate time is allowed to read and answer all the questions; therefore, there is no need to rush or become anxious. You should watch the time to ensure that you are at least halfway through the examination when half of the time has elapsed. Start by answering the first question and continue sequentially (do not skip too many). Do not be alarmed by lengthy questions; look for the question’s salient points. When faced with a confusing question, do not become distracted by that question. Mark it so you can find it later, then go to the next question and come back to the unanswered ones at the end. Extremely lengthy stem statements or case presentations are apparently intended to test the candidate’s ability to separate the essential from the unnecessary or unimportant information.

Some candidates may fail the examination despite the possession of an immense amount of knowledge and the clinical competence necessary to pass the examination. Their failure to pass the examination may be caused by the lack of ability to understand or interpret the questions properly. The ability to understand the nuances of the question format is sometimes referred to as “boardsmanship.” Intelligent interpretation of the questions is very important for candidates who are not well versed in the format of multiple-choice questions. It is very important to read the final sentence (that appears just before the multiple answers) several times to understand how an answer should be selected. For example, the question may ask you to select the correct or incorrect answer. Nevertheless, it is advisable to recheck the question format before selecting the correct answer. It is important to read each answer option thoroughly through to the end. Occasionally a response may be only partially correct. Watch for qualifiers such as “next,” “immediately,” or “initially.” Another hint for selecting the correct answer is to avoid answers that contain absolute or very restrictive words such as “always,” “never,” or “must.” Another means to ensure that you know the correct answer is to cover the answers before tackling the question; read each question and then try to think of the answer before looking at the list of potential answers. Assume you have been given all the necessary information to answer the question. If the answer you had formulated is not among the list of answers provided, you may have interpreted the question incorrectly. When a patient’s case is presented, write down the diagnosis before looking at the list of answers. It will be reassuring to
realize (particularly if your diagnosis is supported by the answers) that you are on the “right track.” If you do not know the answer to the question, very often you are able to rule out one or several answer options and improve your odds at guessing.

Candidates are well advised to use the basic fund of knowledge accumulated from clinical experience and reading to solve the questions. Approaching the questions as “real-life” encounters with patients is far better than trying to second-guess the examiners or trying to analyze whether the question is tricky. There is no reason for the ABPMR to trick the candidates into choosing the wrong answers.

It is better not to discuss the questions or answers (after the examination) with other candidates. Such discussions usually cause more consternation, although some candidates may derive a false sense of having performed well in the examination. In any case the candidates are bound by their oath to the ABPMR not to discuss or disseminate the questions.

**MAINTENANCE OF CERTIFICATION**

Please note: This information is taken directly from the ABPMR Maintenance of Certification Informational Booklet 2013–2014. It is the applicant’s responsibility to seek information concerning the current requirements of recertification in PM&R. The most current requirements supersede any prior requirements and are applicable to each candidate for recertification.

Beginning in 1993, the ABPMR issued time-limited certificates that are valid for 10 years. To maintain certification beyond the 10-year period, diplomates certified in 1993 and thereafter must participate in the Maintenance of Certification (MOC) program. The intent of the initial certification and subsequent Maintenance of Certification© (MOC) processes is to provide assurance to the public that a certified medical specialist has successfully completed an approved educational program and an evaluation, including an examination process, designed to assess the knowledge, experience, and skills requisite to the provision of high quality patient care in that specialty.

**MAINTENANCE OF CERTIFICATION COMPONENTS**

The Maintenance of Certification© (MOC) program is based on documentation of individual participation in the four components of MOC: 1) professional standing, 2) lifelong learning and self-assessment, 3) cognitive expertise, and 4) practice performance. Within these components, MOC addresses six competencies—medical knowledge, patient care, interpersonal and communication skills, professionalism, practice-based learning and improvement, and systems-based practice.

**MAINTENANCE OF CERTIFICATION MODEL**

The ABPMR MOC program has four components: professional standing, lifelong learning and self-assessment, cognitive expertise, and practice performance. In the MOC process, six competencies (below) are evaluated through these four components.

**Medical Knowledge**

Demonstrate knowledge about established and evolving biomedical, clinical, and cognate (e.g., epidemiological and social-behavioral) sciences, as well as the application of this knowledge to patient care.

**Patient Care**

Provide patient care that is compassionate, appropriate, and effective for the treatment of health problems and the promotion of health.
Interpersonal and Communication Skills
Demonstrate interpersonal and communication skills that result in the effective exchange of information and collaboration with patients, their families, and health professionals.

Professionalism
Demonstrate a commitment to carrying out professional responsibilities, adherence to ethical principles, and sensitivity to diverse patient populations.

Practice-Based Learning and Improvement
Demonstrate the ability to investigate and evaluate the care of patients, to appraise and assimilate scientific evidence, and to continuously improve patient care based on constant self-evaluation and lifelong learning.

Systems-Based Practice
Demonstrate an awareness of and responsiveness to the larger context and system of health care. Be able to call on system resources to provide optimal care (e.g., coordinating care across sites or serving as the primary case manager when care involves multiple specialties, professions, or sites).

The overriding principle of the ABPMR MOC program is to evaluate the six basic competencies through implementation of the four components. This practice will evolve into a continuous process of lifelong learning and self-assessment, which stresses the adult learning concepts of self-direction, knowledge-into-action, practical content, self-discovery, and incorporation of knowledge and skills into the practice.

MAINTENANCE OF CERTIFICATION REQUIREMENTS

COMPONENT I: PROFESSIONAL STANDING
In order to maintain ABPMR certification, diplomates must hold a current, valid, and unrestricted license to practice medicine. Failure to retain a valid, unrestricted license will result in the loss of ABPMR certification.

COMPONENT II: LIFELONG LEARNING AND SELF-ASSESSMENT
Lifelong Learning: CME Requirement
Diplomates are encouraged to complete and report Category 1 CME credits annually. Diplomates with time-limited certificates issued before 2012 must complete and report a minimum of 300 Category 1 CME credits during the 10-year MOC cycle. Diplomates with time-limited certificates issued in 2012 and beyond must complete and report 150 Category 1 CME credits in years 1-5 and in years 6-10 of their MOC cycle.

Certificates for Category 1 CME activities should be retained by the diplomate in the event that the Board requests verification.

Specialty-specific CME: A minimum of 50% of the 300 total CME credits must be related to the specialty of physical medicine and rehabilitation and/or its subspecialties.

Category 1 credit involves activities designated by an accredited provider. A minimum of 300 credits must be met by the following types of CME experiences:

- CME programs of universities, hospitals, organizations, and institutions accredited by the Accreditation Council for Continuing Medical Education (ACCME).
- CME activities offered by other accrediting organizations such as the American Medical Association (AMA), the American Academy of Physical Medicine and Rehabilitation (AAPMR),
the Association of Academic Physiatrists (AAP), or the American Association of Neuromuscular and Electrodagnostic Medicine (AANEM).

- Category 1A and 2A credits from the American Osteopathic Association (AOA).

**Category 2** credit may be reported for tracking purposes only and do not count toward the 300 credit minimum.

### Lifelong Learning: Self-Assessment Requirement

Diplomates with time-limited certificates issued before 2012 are required to complete 4 ABPMR—approved self-assessment activities during the 10-year MOC cycle. Diplomates with time-limited certificates issued in 2012 and beyond must complete an average of 8 CME credits per year involving ABPMR—approved self-assessments for a total of 40 CME credits in years 1-5 and in years 6-10 of their MOC cycle.

### COMPONENT III: COGNITIVE EXPERTISE (EXAMINATION)

This component consists of a cognitive examination covering all aspects of the specialty. The ABPMR Maintenance of Certification (MOC) Examination is a computer-based, closed-book examination. The examination consists of multiple-choice questions related to clinical practice.

**Number of multiple-choice questions on the MOC examinations:**
- Primary MOC: 160
- Pain Medicine MOC: 200
- Pediatric Rehabilitation Medicine MOC: 280
- Spinal Cord Injury Medicine MOC: 280

The table below contains the primary MOC examination outline:

<table>
<thead>
<tr>
<th>CONTENT SPECIFICATION</th>
<th>APPROXIMATE TARGET WEIGHT (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CV, Pulmonary, and Cancer Rehabilitation</td>
<td>4</td>
</tr>
<tr>
<td>Electrodiagnosis</td>
<td>9</td>
</tr>
<tr>
<td>Geriatric Rehabilitation</td>
<td>5</td>
</tr>
<tr>
<td>Industrial Rehabilitation</td>
<td>6</td>
</tr>
<tr>
<td>Joint and Connective Tissue Disorders</td>
<td>4</td>
</tr>
<tr>
<td>Musculoskeletal and Soft Tissue Disorders</td>
<td>8</td>
</tr>
<tr>
<td>Nerve and Muscle Disorders</td>
<td>6</td>
</tr>
<tr>
<td>Pain Management</td>
<td>13</td>
</tr>
<tr>
<td>Pediatric Rehabilitation</td>
<td>4</td>
</tr>
<tr>
<td>Physiatric Therapeutics</td>
<td>4</td>
</tr>
<tr>
<td>Prosthetics, Orthotics, and Assistive Devices</td>
<td>5</td>
</tr>
<tr>
<td>Spinal Cord Injury Medicine</td>
<td>6</td>
</tr>
</tbody>
</table>
COMPONENT IV: PRACTICE PERFORMANCE

The fourth component contains various assessments designed to address quality improvement in practice.

Diplomates with time-limited certificates issued before 2012 must complete a minimum of 1 practice performance project during the 10-year MOC cycle. Diplomates with time-limited certificates issued in 2012 and beyond must complete 2 ABPMR-approved practice performance projects (1 in years 1-5 and 1 in years 6-10) during the 10-year MOC cycle. A list of ABPMR-approved practice performance options can be found on the ABPMR website: https://www.abpmr.org/diplomates/pp_options_table.html

Please refer to the ABPMR website (www.abpmr.org) for further detail about how to submit practice performance projects.

MOC REQUIREMENTS SUMMARY

<table>
<thead>
<tr>
<th>CERTIFICATE ISSUE DATE</th>
<th>ACTIVITIES REQUIRED TO RECERTIFY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before 2012</td>
<td>• Licensure&lt;br&gt;• Complete and report a minimum of 300 Category 1 CME credits&lt;br&gt;• Complete at least 4 ABPMR—approved self-assessment activities&lt;br&gt;• Examination&lt;br&gt;• Complete at least 1 ABPMR—approved practice performance project</td>
</tr>
<tr>
<td>2012 and Beyond</td>
<td>• Licensure&lt;br&gt;• Complete and report a minimum of 300 Category 1 CME credits&lt;br&gt;• Complete an average of 8 CME credits per year (averaged over 5 years) involving ABPMR—approved self-assessment activities&lt;br&gt;• Examination&lt;br&gt;• Complete 2 ABPMR—approved practice performance projects (1 in years 1–5 and 1 in years 6–10)</td>
</tr>
</tbody>
</table>

CERTIFICATE ISSUANCE

The Board will issue a 10-year time-limited certificate to each diplomate who successfully completes the Maintenance of Certification process. Prior to receiving a certificate, diplomates must complete all MOC components and pay all annual fees that are due.

Diplomates who have not completed all MOC program requirements prior to the expiration date of their certificate may reinstate their diplomate status pursuant to the ABPMR MOC Reinstatement Policy.
EXAMINATION STATISTICS

TOTAL PM&R DIPLOMATES CERTIFIED AS OF 2013: 11,047

<table>
<thead>
<tr>
<th>PART I: COMPUTER-BASED EXAMINATION</th>
<th>AUGUST 2013</th>
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<td>Total taking exam</td>
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<td>Total first-time (pass)</td>
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<tr>
<td>Total first-time (fail)</td>
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<td>Total first-time (fail)</td>
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MAINTENANCE OF CERTIFICATION STATISTICS FOR 2013

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<thead>
<tr>
<th>COMPUTER-BASED EXAMINATION</th>
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<tr>
<td>Total taking exam</td>
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<td>14/2%</td>
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</table>

Further details and current information for the certification and recertification programs can be obtained by writing to the ABPMR or by visiting their website:
The American Board of Physical Medicine and Rehabilitation
3015 Allegro Park Lane SW
Rochester MN 55902-4139
Phone: 507-282-1776
Fax: 507-282-9242
Website: www.abpmr.org
E-mail: office@abpmr.org
INTRODUCTION

DEFINITION OF STROKE

• A cerebrovascular event with rapidly developing clinical signs of focal or global disturbances of cerebral function with signs lasting 24 hours or longer or leading to death, with no apparent cause other than of vascular origin (World Health Organization).
• Symptoms <24 hours = transient ischemic attack (TIA).
• Reversible ischemic neurologic deficit (RIND): This term is no longer used.

EPIDEMIOLOGY

• After heart disease and cancer, stroke is the third leading cause of death in the United States.
• The American Heart Association (AHA) estimates 795,000 strokes annually: 610,000 new cases and 185,000 recurrent cases (AHA, 2013).
• Nearly 6.8 million (about 3.0 million males and 3.8 million females) stroke survivors in the United States in 2010.
• 46% decline in cerebral infarcts and hemorrhages from 1950 to 1954 period to 1975 to 1979 period (Broderick, 1993).
  – Decline was attributed to better management of hypertension (HTN) and heart disease, decrease in cigarette smoking, etc.
• Incidence increased 17% from the 1975 to 1979 period to the 1980 to 1984 period (attributed to increased use of CT scan).
• There has been no change in the incidence of aneurysmal rupture.
• Mortality from strokes has declined since the 1950s.
  – A sharp decline was noted in the 1970s, possibly related to improved diagnosis and treatment of hypertension.
  – From 1999 to 2009, the annual stroke death rate decreased 36.9%, and the actual number of stroke deaths declined 22.9%.
  – Approximately 56% of stroke deaths in 2009 occurred outside the hospital.
  – In the first 30 days after stroke, 90% of deaths were due to direct effects of the brain lesion or complications of immobility resulting from the stroke (Dennis et al., 1993).
Between 30 days and 6 months after stroke, 44% of deaths were secondary to complications or direct effects of the first stroke (Dennis et al., 1993).

After 6 months, the most likely cause of death is cardiovascular (non-stroke) or sudden death (Dennis et al., 1993).

- **2013 Heart Disease and Stroke Statistics**
  - Approximately 15% of all strokes are heralded by a TIA (AHA, 2013). The prevalence of TIA in men 65 to 69 years is 2.7%, and 3.6% in men 75 to 79 years (Kleindorfer et al., 2005; Lisabeth et al., 2004).
  - The short-term risk of stroke after TIA is approximately 3% to 10% at 2 days and 9% to 17% at 90 days (AHA, 2013).
  - Individuals who have a TIA and survive the initial high-risk period have a 10-year stroke risk of roughly 19% and a combined 10-year stroke, MI, or vascular death risk of 43% (4% per year) (AHA, 2013).

## RISK FACTORS (Stewart, 1999)

### Nonmodifiable Risk Factors

- **Age**—the single most important risk factor for stroke worldwide. After age 55, incidence increases for both males and females.
  - Risk more than doubles each decade after age 55.
- **Sex** (male > female).
- **Family history of stroke.**

### Modifiable (Treatable) Risk Factors

- **Hypertension**—probably the most important modifiable risk factor for both ischemic and hemorrhagic stroke (sevenfold increased risk). Subjects with BP lower than 120/80 mmHg have about half the lifetime risk of stroke compared to subjects with high BP (Seshadri et al., 1997).
- **History of TIA/prior stroke**: Approximately 5% of patients with TIA will develop a stroke within 1 month and approximately 14% within 1 year if untreated. After a TIA, the 90-day risk of stroke is 3% to 17.3% and is highest within the first 30 days (Coull et al., 2004; Johnston et al., 2003).
- **Heart disease**:
  - Congestive heart failure (CHF) and coronary artery disease (CAD) increase risk by twofold.
  - Valvular heart disease and arrhythmias increase risk of embolic stroke.
- **Atrial fibrillation (AF)**: Fivefold increased risk (Wolf et al., 1991).
- **Diabetes**: Twofold increase in risk. Unfortunately, good blood sugar control has not been shown to alter the risk of stroke.
- **Cigarette smoking**: Risk of ischemic stroke in smokers is about double that of nonsmokers.
- **Carotid stenosis (and carotid bruit)**: Carotid endarterectomy is of some benefit to prevent stroke in patients with 50% to 69% symptomatic stenosis (absolute risk reduction 4.6%) and highly beneficial in patients with 70% to 99% stenosis (absolute risk reduction 16.0%).
  - **ETOH abuse/cocaine use**: <2 drinks/day relative risk 0.51; >7 drinks/day relative risk 2.96 (Sacco et al., 1999).
- **High-dose estrogens (birth control pills)**: Considerable increased risk when linked with cigarette smoking.
1. STROKE

- Systemic diseases associated with hypercoagulable states:
  - Elevated RBC count, hematocrit, fibrinogen
  - Protein S and C deficiencies
  - Sickle-cell anemia
  - Cancer
• Hyperlipidemia: Several clinical trials have shown a reduction in stroke with use of cholesterol-reducing agents (approximately 30% reduction in stroke with use of HMG-CoA reductase inhibitors).
• Migraine headaches.
• Sleep apnea.
• Patent foramen ovale (PFO).
• [Obesity/sedentary lifestyle (no clear relationship with increased risk of stroke)]

Other Risk Factors (American Heart Association, 2008)
• Geographical location: Higher risk of stroke in the southeastern United States than in other areas—the so-called “stroke belt” states.
• Socioeconomic factors: Some evidence that strokes are more common in people with low-income status than among more affluent people.

BASIC NEUROANATOMICAL REVIEW OF THE MAJOR VESSELS INVOLVED IN STROKE

FIGURE 1–3 Major vascular supply to the brain and functional diagram of the motor strip. It is evident that the lower limb extremity motor strip is in anterior cerebral artery (ACA) distribution, while the upper-extremity motor strip is supplied by the middle cerebral artery (MCA). (From Rosen, 1992, with permission.)
1. Most of the lateral aspect of the hemisphere is supplied mainly by the middle cerebral artery (MCA).

2. The anterior cerebral artery (ACA) supplies the medial aspect of the hemisphere from the lamina terminalis to the cuneus.

3. The posterior cerebral artery (PCA) supplies the posterior inferior surface of the temporal lobe and the visual cortex.

**FIGURE 1-4** The 3 cerebral arteries’ cortical territories. (A) Lateral aspect of the hemisphere. (B) Medial and inferior aspects of the hemisphere.

**FIGURE 1-5** Major vascular territories are shown in this schematic drawing of a coronal section through the cerebral hemisphere at the level of the thalamus and the internal capsule.
1. CSF is produced in the brain largely by modified ependymal cells in the choroid plexus in the lateral, third, and fourth ventricles, with the remainder formed around blood vessels and along ventricular walls.

2. CSF circulates from the lateral ventricles to the foramina of Monro (interventricular foramina), third ventricle, aqueduct of Sylvius (cerebral aqueduct), fourth ventricle, foramen of Magendie (Median aperture) and foramina of Luschka (lateral apertures), and subarachnoid space over brain and spinal cord.

3. Hemorrhage can cause occlusion of the foramina, resulting in hydrocephalus.
# TYPES OF STROKE

<table>
<thead>
<tr>
<th></th>
<th>ISCHEMIC (87%)</th>
<th>HEMORRHAGIC (13%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Thrombotic</td>
<td>Embolic</td>
</tr>
<tr>
<td>Frequency (%)</td>
<td>48</td>
<td>26</td>
</tr>
<tr>
<td>Factors associated with onset</td>
<td>Occurs during sleep</td>
<td>Occurs while awake</td>
</tr>
<tr>
<td>Major causes/etiology</td>
<td>Perfusion failure distal to site of severe stenosis or occlusion of major vessels</td>
<td>Due mainly to cardiac source</td>
</tr>
<tr>
<td>Presentation</td>
<td>Slow (gradual), progressive deficit</td>
<td>Sudden, immediate deficit (seizures may occur)</td>
</tr>
<tr>
<td>Association with TIA</td>
<td>50% with preceding TIA (50% occurring in same vascular territory of preceding TIA)</td>
<td>TIA less common than in thrombotic; 11% with preceding TIA</td>
</tr>
</tbody>
</table>

**ISCHEMIC STROKES**

**THROMBOTIC STROKES (LARGE ARTERY THROMBOSIS): 48% OF ALL STROKES**

- Usually occurs during sleep (patient often awakens unaware of deficits).
- May have “stuttering,” intermittent progression of neurologic deficits, or be slowly progressive (over 24 to 48 hours).
- Profound loss of consciousness (LOC) is rare except when area of infarction is large or when brainstem is involved.
- Neurologic deficit varies according to cerebral territory affected.
• Perfusion failure distal to site of severe stenosis or occlusion of major vessels.
• Emboli from incompletely thrombosed artery may precipitate an abrupt deficit. May have embolism from extracranial arteries affected by stenosis or ulcer.

EMBOLECT STROKES: 26% OF ALL STROKES
• Immediate onset of neurologic deficits.
• Usually occurs during waking hours.
• Seizures may occur at onset of stroke.
• Most commonly due to cardiac source: mural thrombi and platelet aggregates.
• Emboli most commonly originate from cardiac thrombus caused by atrial fibrillation. Also occur in rheumatic heart disease (e.g., mitral stenosis), post-MI, and vegetations on heart valves in bacterial or marantic endocarditis or prosthetic heart valves.
  – From clots that develop after open-heart surgery or atheromas in neck arteries or in the aortic arch.
  – Emboli may dislodge spontaneously or after invasive cardiovascular procedures (e.g., cardiac catheterization).
  – 75% of cardiogenic emboli go to the brain.
• Occasionally, embolus may consist of fat (from fractured long bones), air (in decompression sickness), or venous clot that passes through a patent foramen ovale (PFO) with shunt (paradoxical embolus).
• Rarely, a subclavian artery thrombosis may embolize to the vertebral artery or its branches.

LACUNAR STROKES: 13% OF ALL STROKES
• Onset may be abrupt or gradual: up to 30% develop slowly over or up to 36 hours.
• Lacunes are small infarcts (less than 15 mm) seen in the putamen, pons, thalamus, caudate, and internal capsule.
• Are due to occlusive arteriolar or small artery disease (occlusion of deep penetrating branches of large vessels).
• Occlusion occurs in small arteries of 50 to 200 mm in diameter.
• Strong correlation with hypertension (up to 81%); also associated with microatheroma, microembolism, or rarely arteritis.
• CT shows lesion in about 2/3 of cases (MRI may be more sensitive).
• Often relatively pure syndromes (motor, sensory); discussed below.
• Absence of higher cortical function involvement (language, praxis, nondominant hemisphere syndrome, vision).

NEUROANATOMIC LOCATIONS OF ISCHEMIC STROKE (Ropper & Samuels, 2009)

1. Anterior Circulation
INTERNAL CAROTID ARTERY (ICA)—(FIGURE 1-8)
• The most variable syndrome. Occlusion occurs most frequently in the first part of the ICA immediately beyond the carotid bifurcation. ICA occlusions are often asymptomatic (30% to 40% of cases).
• Ocular infarction: Embolic occlusion of either retinal branch or central retinal artery.
• Transient monocular blindness (amaurosis fugax): The ICA nourishes the optic nerve and retina as well as the brain. Transient monocular blindness occurs prior to onset of stroke in approximately 25% of cases of ICA occlusion. Central retinal artery ischemia is very rare because of collateral supply.
• Cerebral infarction: Variable presentation with complete ICA occlusion; from no symptoms (if good collateral circulation exists) to severe, massive infarction in ACA and MCA distributions.
• Distal ICA occlusion affects part or all of the ipsilateral MCA territory and, when the anterior communicating artery is small, the ipsilateral anterior cerebral artery territory. Patients will present with contralateral motor and/or sensory symptoms.

MIDDLE CEREBRAL ARTERY (MCA)—(FIGURE 1-9)
• Occlusion occurs at the stem of the MCA or at one of the two main divisions (superior or inferior) of the artery in the Sylvian sulcus.
1. STROKE

Superior division of the MCA:
- The superior division of MCA supplies Rolandic and pre-Rolandic areas.
- Most common cause of occlusion of superior division of MCA is an embolus.
- Sensory and motor deficits on contralateral face and arm > leg.
- Head and eyes deviated toward side of infarct.
- With left side lesion (dominant hemisphere)—global aphasia initially, then turns into Broca’s aphasia (motor speech disorder).
- Right side lesion (nondominant hemisphere)—deficits on spatial perception, hemineglect, constructional apraxia, dressing apraxia.
- Muscle tone usually decreased initially and gradually increases over days or weeks to spasticity.
- Transient LOC is uncommon.

Inferior division of the MCA:
- The inferior division of the MCA is the blood supply to the lateral temporal and inferior parietal lobes.
- With lesion on either side—superior quadrantanopia or homonymous hemianopsia.
- Left side lesion → Wernicke’s aphasia.
- Right side lesion → left visual neglect.

ANTERIOR CEREBRAL ARTERY—(FIGURE 1-10)
- If occlusion is at the stem of the ACA proximal to its connection with the anterior communicating artery, it is usually well tolerated because adequate collateral circulation comes from the contralateral ACA.
Figure 1-9: The distribution of the middle cerebral artery (MCA) on the lateral aspect of the cerebral hemisphere. Principal regions of cerebral localization are noted.

Figure 1-10: The distribution of the anterior cerebral artery (ACA) and posterior cerebral artery (PCA) on the medial aspect of the cerebral hemisphere, showing principal regions of cerebral localization.

- Occlusion of one anterior cerebral artery distal to an anterior communicating artery results in:
  - Contra lateral weakness and sensory loss, affecting mainly the distal contralateral leg (foot/leg more affected than thigh).
  - Mild or no involvement of upper extremity.
  - Head and eyes may be deviated toward side of lesion acutely.
  - Urinary incontinence with contralateral grasp reflex and paratonic rigidity (Gegenhalten) may be present.
1. Stroke

- May produce transcortical motor aphasia if left side is affected.
- Disturbances in gait and stance = gait apraxia.
- If both anterior cerebral arteries arise from one stem, major disturbances occur with infarction occurring at the medial aspects of both cerebral hemispheres, resulting in aphasia, paraplegia, incontinence, and frontal lobe/personality dysfunction (e.g., emotional instability, disinhibition, apathy).

2. Posterior Circulation: Vertebrobasilar Arteries and Posterior Cerebral Arteries

**POSTERIOR CEREBRAL ARTERY (PCA)**

- Occlusion of PCA can produce a variety of clinical effects because it supplies the upper brainstem and the inferior parts of the temporal lobe, as well as the medial parts of the occipital lobe.
- The particular area of occlusion varies for PCA because anatomy varies.
  - 70% of the time, both PCAs arise from the basilar artery and are connected to the internal carotids through the posterior communicating artery.
  - 20% to 25%: One PCA comes from basilar; one PCA comes from ICA.
  - 5% to 10%: Both PCAs arise from carotids.

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• Clinical presentation:
  - Visual field cuts (when bilateral, may have denial of cortical blindness = Anton syndrome).
  - May have prosopagnosia (can’t read faces).
  - Palinopsia (abnormal recurring visual imagery).
  - Alexia (can’t read).
  - Transcortical sensory aphasia (loss of power to comprehend written or spoken words; patient can repeat).
  - Structures supplied by the interpeduncular branches of the PCA include the oculomotor cranial nerve (CN3) and trochlear (CN4) nuclei and nerves.
  - Clinical syndromes caused by the occlusion of these branches include Weber syndrome (oculomotor palsy with contralateral hemiplegia), which is discussed below, and trochlear nerve palsy (vertical gaze palsy).

**VERTEBROBASILAR SYSTEM (SEE FIGURES 1–1 AND 1–2)**

• The vertebrobasilar arteries supply the midbrain, pons, medulla, cerebellum, and posterior and ventral aspects of the cerebral hemispheres (via the PCAs).
  - Vertebral arteries originate from the subclavian arteries and are the main arteries of the medulla.
  - At the pontomedullary junction, the two vertebral arteries join to form the basilar artery, which provides the blood supply to the pons and midbrain.
  - The cerebellum is supplied by the posterior–inferior cerebellar arteries (PICA) originating from the vertebral arteries as well as the anterior–inferior cerebellar arteries (AICA) and superior cerebellar arteries, both of which originate from the basilar artery.

• Vertebrobasilar system involvement may present with any combination of the following signs/symptoms:
  - Vertigo
  - Nystagmus
  - Abnormalities of motor function, often bilaterally
  - Ipsilateral cranial nerve dysfunction
  - Crossed signs: motor or sensory deficit on ipsilateral side of face and contralateral side of body; ataxia, dysphagia, dysarthria

• **Important**: There is absence of cortical signs (such as aphasias or cognitive deficits) that would be characteristic of anterior circulation involvement.

• **Important**: While isolated attacks of vertigo can be the initial and only symptom of vertebrobasilar insufficiency, attacks of vertigo in vertebrobasilar insufficiency usually last less than 30 minutes and have no associated hearing loss. If no other symptoms, especially nystagmus, accompany vertigo, a diagnosis of benign paroxysmal positional vertigo (BPPV) should be considered (Bhattacharyya et al., 2008).

**SYNDROMES OF THE VERTEBROBASILAR SYSTEM**

I. **Wallenberg (Lateral Medullary) Syndrome**

• Wallenberg syndrome is also known as lateral medullary syndrome, PICA syndrome, and vertebrobasilar artery syndrome.

• This syndrome is one of the most striking in neurology. It occurs due to occlusion of the following:
  1. Vertebral arteries (involved in 8 out of 10 cases)
  2. Posterior inferior cerebellar artery (PICA)
  3. Superior lateral medullary artery
  4. Middle lateral medullary artery
  5. Inferior lateral medullary artery

• **Signs and symptoms** include the following:
  - Ipsilateral side:
    ■ Horner’s syndrome (ptosis, anhidrosis, and miosis).
    ■ Decrease in pain and temperature sensation on the ipsilateral face.
    ■ Cerebellar signs such as ataxia on ipsilateral extremities (patient falls to side of lesion).
1. STROKE

- Contralateral side:
  - Decreased pain and temperature on contralateral body.
  - Dysphagia, dysarthria, hoarseness, paralysis of vocal cord.
  - Vertigo; nausea and vomiting.
  - Hiccups.
  - Nystagmus, diplopia.

  Note: No facial or extremity muscle weakness seen in this syndrome.

II. Benedikt Syndrome (Red Nucleus/Tegmentum of Midbrain)
- Obstruction of interpeduncular branches of basilar or posterior cerebral artery, or both.
- Ipsilateral CN3 nerve paralysis with mydriasis, contralateral hypesthesia (medial lemniscus)
- Contralateral hyperkinesia (ataxia, tremor, chorea, athetosis) due to damage to red nucleus.

III. Syndromes of the Paramedian (Medial) Brainstem
- The paramedian area of the brainstem contains:
  - Motor nuclei of CNs
  - Corticospinal tract
  - Medial lemniscus
  - Corticobulbar tract

- Signs/symptoms include:
  - Ipsilateral CN paralysis
  - Contralateral hemiparalysis

GROSS LOCATIONS OF CN BRAINSTEM NUCLEI.

NOTE: CN1 and CN2 nuclei are located in forebrain. CN 11 has two divisions, the cranial and the spinal division. The spinal division of CN11 arises from ventral horns of CN1 to CN6 levels.

TABLE 1–2 Syndromes of the Paramedian (Medial) Brainstem

<table>
<thead>
<tr>
<th>WEBER SYNDROME</th>
<th>MILLARD-GUBLER SYNDROME</th>
<th>MEDIAL MEDULLARY SYNDROME “ANOTHER LESION”</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Ipsilateral CN3 palsy</td>
<td>• Ipsilateral CN6 paralysis (often CN7 also involved)</td>
<td>• Ipsilateral CN12 palsy</td>
</tr>
<tr>
<td>• Contralateral hemiplegia</td>
<td>• Contralateral hemiplegia (extension into medial lemniscus is Foville syndrome with gaze palsy to side of lesion)</td>
<td>• Contralateral hemiplegia</td>
</tr>
<tr>
<td></td>
<td>• Contralateral lemniscal (tactile sensation) sensory loss secondary to damage to medial lemniscus</td>
<td>• Contralateral lemniscal sensory loss</td>
</tr>
</tbody>
</table>
1. STROKE

Weber Syndrome (Base of Midbrain)
- Obstruction of interpeduncular branches of PCA or posterior choroidal artery, or both
- Ipsilateral CN3 paralysis
- Contralateral hemiplegia, contralateral Parkinson signs, contralateral dystaxia (mild degree of ataxia)

Millard–Gubler Syndrome (Base of Pons)
- Obstruction of circumferential branches of basilar artery
- Ipsilateral abducens (CN6) and facial (CN7) palsies
- Contralateral hemiplegia, analgesia, hypoesthesia
- Extension to medial lemniscus = Raymond-Foville Syndrome (with gaze palsy to side of lesion)

GROSS DEPICTION OF THE PARAMEDIAN BRAINSTEM AND ASSOCIATED SYNDROMES.

Medial Medullary Syndrome (Medial Medulla)
- Caused by an infarction of the medial medulla due to occlusion (usually atherothrombotic) of penetrating branches of the vertebral arteries (upper medulla) or anterior spinal artery (lower medulla and medullocervical junction).
- Rare; ratio of medial medullary infarct to lateral medullary infarct approximately 1 to 2:10.
- Signs and symptoms:
  - Ipsilateral hypoglossal palsy (with deviation toward the side of the lesion)
  - Contralateral hemiparesis
  - Contralateral lemniscal sensory loss (proprioception and position sense)

<table>
<thead>
<tr>
<th>TABLE 1–3 Brainstem Syndromes</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAIN ARTERIES</td>
</tr>
<tr>
<td>----------------</td>
</tr>
<tr>
<td>Midbrain</td>
</tr>
<tr>
<td>Pons</td>
</tr>
<tr>
<td>Medulla</td>
</tr>
<tr>
<td></td>
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</tbody>
</table>

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IV. Basilar Artery Occlusion Syndrome

- Occlusion may arise in several ways:
  - Atherosclerotic plaque in the basilar artery itself (usually lower third).
  - Occlusion of both vertebral arteries.
  - Occlusion of one vertebral artery when it is the only artery of adequate size.

- Note:
  - Thrombosis usually only obstructs a branch of the basilar artery rather than the trunk.
  - Emboli, if they get through the vertebral arteries, usually lodge in one of the posterior cerebral arteries or at the upper bifurcation of the basilar artery.
  - May cause internuclear ophthalmoplegia, conjugate horizontal gaze palsy, or ocular bobbing.

  Ptosis and nystagmus are common but variable.

- May cause internuclear ophthalmoplegia, conjugate horizontal gaze palsy, or ocular bobbing.

- Locked-in syndrome: Tetraparesis with patients who are only able to move eyes vertically or blink. The patient remains fully conscious secondary to sparing of the reticular activating system (RAS). It is caused by bilateral lesions of the ventral pons (basilar artery occlusion). Some degree of paresis accompanies nearly all cases of basilar artery occlusion (Note: Majority of RAS is located primarily in the midbrain).

<table>
<thead>
<tr>
<th>LACUNAR SYNDROME</th>
<th>ANATOMICAL LOCATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Pure motor hemiplegia</td>
<td></td>
</tr>
<tr>
<td>– Weakness involving face, arm, and leg; no sensory deficits, aphasia, or parietal signs</td>
<td>Posterior limb of internal capsule (supplied by the lenticular striate artery) Corona radiata Pons</td>
</tr>
<tr>
<td>2. Pure sensory stroke</td>
<td>Thalamus (ventro-lateral) Parietal white matter Thalamocortical projections</td>
</tr>
<tr>
<td>3. Dysarthria / “clumsy hand syndrome”</td>
<td>Basis pontis Internal capsule (anterior limb)</td>
</tr>
<tr>
<td>4. Sensorimotor stroke</td>
<td>Junction of internal capsule and thalamus</td>
</tr>
<tr>
<td>5. Ataxia and leg paralysis</td>
<td>Pons Midbrain Internal capsule Cerebellum Parietal white matter Corona radiata</td>
</tr>
<tr>
<td>6. Hemichorea-hemiballismus</td>
<td>Head of the caudate Thalamus Subthalamic nucleus</td>
</tr>
</tbody>
</table>

HEMORRHAGIC STROKES (SEE TABLE 1–1)

13% of all strokes may be secondary to hypertension, ruptured aneurysm, arteriovenous malformation (AVM), blood dyscrasias/bleeding disorders, anticoagulants, bleeding into tumors, or angiopathies.

I. Hypertensive Intracerebral Hemorrhage (ICH)

- Linked to chronic HTN (>1/3 occur in normotensives).
- Preceded by formation of “false” aneurysms (microaneurysms) of Charcot and Bouchard = arterial wall dilations secondary to HTN.
- Frequently extends to ventricular subarachnoid space.

- Symptoms
  - Sudden onset of headache (HA) and/or LOC.
  - Vomiting at onset in 22% to 44%.
- Seizures occur in 10% of cases (first few days after onset).
- Nuchal rigidity is common.

**Locations** include the putamen, thalamus, pons, cerebellum, and cerebrum:

1. **Putamen**: Most common. Hemiplegia secondary to compression of adjacent internal capsule. Vomiting in approximately 50%. HA is frequent but not invariable.
   - Large hemorrhage: Stupor/coma + hemiplegia with deterioration in hours.
   - With smaller hemorrhages: HA leading to aphasia, hemiplegia, eyes deviate away from paretic limbs.
   - These symptoms, occurring over a few minutes to one-half hour, are strongly suggestive of progressive intracerebral bleeding.

2. **Thalamus**: Hemiplegia by compression of adjacent internal capsule; contralateral sensory deficits; aphasia present with lesions of the dominant side; contralateral hemineglect with involvement on the nondominant side. Ocular disturbances with extension of hemorrhage into subthalamus.

3. **Pons**: Deep coma results in a few minutes; total paralysis, small pupils (1 mm) that react to light; decerebrate rigidity → death occurs in a few hours. Patient may survive if hemorrhage is small (smaller than <1 cm).

4. **Cerebellum**: Develops over several hours. Coma/LOC, unusual vomiting, occipital HA, vertigo, inability to sit, stand, or walk, eyes deviate to opposite side (ipsilateral CN6 palsy), dysarthria, dysphagia.

5. **Lobar (cerebral) hemorrhage**: HA and vomiting. A study of 26 patients revealed:
   - 11 occipital: dense homonymous hemianopsia and pain ipsilateral eye
   - 7 left temporal: partial hemianopsia/fluent aphasia/pain ear
   - 4 frontal: contralateral hemiplegia (mainly the arm) and frontal HA
   - 3 parietal: hemisensory deficit (contralateral)/anterior temporal HA
   - 1 right temporal (Ropper and Davis, 1980)

**II. Subarachnoid Hemorrhage (SAH)**

- Typically due to a ruptured saccular arterial aneurysm.
  - Saccular aneurysms = berry aneurysms.
- Arterial dilations found at bifurcations of larger arteries at the base of the brain (Circle of Willis or major branches; see Figure 1–12).
  - 90% to 95% of saccular aneurysms occur on the anterior part of the Circle of Willis.
  - Presumed to result from congenital medial and elastic defects versus hemodynamic forces, causing focal destruction of internal elastic membrane at bifurcations (Ropper, 2009).
- Multiple in 20% of patients (either unilateral or bilateral).
- Other types of aneurysms: Arteriosclerotic, mycotic, dissecting aneurysms, traumatic, neoplastic.
- More likely to rupture if 10 mm or larger (rupture may occur in smaller aneurysms).
- Rupture occurs usually when patient is active rather than during sleep (e.g., straining, coitus).
- Peak age for rupture = fifth and sixth decade.

### TABLE 1–5  
**Hunt and Hess Scale for Nontraumatic Subarachnoid Hemorrhage Patients**

<table>
<thead>
<tr>
<th>DESCRIPTION</th>
<th>GRADE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymptomatic, mild HA, slight nuchal rigidity</td>
<td>1</td>
</tr>
<tr>
<td>Moderate to severe HA, nuchal rigidity, no neurologic deficit other than cranial nerve palsy</td>
<td>2</td>
</tr>
<tr>
<td>Drowsiness/confusion, mild focal neurologic deficit</td>
<td>3</td>
</tr>
<tr>
<td>Stupor, moderate-severe hemiparesis</td>
<td>4</td>
</tr>
<tr>
<td>Coma, decerebrate posturing</td>
<td>5</td>
</tr>
</tbody>
</table>

*Source: www.strokecenter.org/trials/scales/hunt_hess.html*
1. Stroke

Clinical Presentations of Saccular Aneurysms/SAH:

- Saccular aneurysms are usually asymptomatic prior to rupture. Intracranial aneurysms are common—found in 3% to 5% of patients on routine autopsies.

- Compression of adjacent structures such as the oculomotor nerve (CN3) with posterior communicating-internal carotid junction aneurysm or posterior communicating-posterior cerebral artery aneurysm.

  Signs of CN3 involvement:
  - Deviation of ipsilateral eye to lateral side (lateral or divergent strabismus) because of unopposed action of lateral rectus muscle.
  - Ptosis.
  - Mydriasis (dilated pupil) and paralysis of accommodation due to interruption of parasympathetic fibers in CN3.

- Rupture of aneurysm producing SAH with or without intracerebral hematoma.
  - "Sentinel" HA: Sudden, intense, and persistent, preceding SAH by days or weeks in approximately 50% of patients.
  - With a SAH, blood is irritating, causing severe HA that is classically described as “worst HA of my life.”
  - Sudden, transient LOC in 20% to 45% at onset.
  - Focal neurologic deficits include CN3 or CN6 palsy (from direct pressure from the aneurysm versus accumulation of an intracerebral hematoma versus early development of arterial spasm), hemiparesis, aphasia (dominant hemisphere), and memory loss.
  - Seizures: 4% at onset/25% overall.
  - Mortality: 25% during first 24 hours.
  - Risk of rebleeding within 1 month 30%; 2.2% per year during first decade.
  - Mortality from rebleeding in the early weeks after initial event: 50% to 60%.
  - Vasospasm: Common complication occurring in approximately 25% of cases; caused by the presence of blood breakdown products (vasoactive amines) on the subarachnoid space, acting in the adventitia of the arteries. Occurs 3 to 12 days after rupture (frequently approximately 7 days after rupture).
  - Medications: nimodipine (calcium channel blocker) is useful in the treatment of cerebral blood vessel spasm after SAH (see Treatment section below).

III. Vascular Malformations/AVMs

- A vascular malformation in the brain consisting of a tangled mass of dilated vessels that forms an abnormal communication between the arterial and venous systems is known as an arteriovenous malformation (AVM).
1. STROKE

- These congenital lesions originate early in fetal life.
- AVMs displace rather than invade normal brain tissue.
- AVMs are usually low-pressure systems: the larger the shunt, the lower the interior pressure. Thus, with these large dilated vessels, there needs to be an occlusion distally to raise luminal pressures to cause hemorrhage.
- Hemorrhage appears to be more common in smaller malformations, likely due to higher resistance and pressure within these lesions.
- Patients are believed to have a 40% to 50% lifetime risk of hemorrhage from AVM.
- Natural history of AVMs: Bleeding rate per year approximately 2% to 4%.
- Rebleeding rate 6% first year post-hemorrhage.
- Annual mortality rate: 1% per year.
- First hemorrhage is fatal in approximately 10% of these patients.
- Bleeding commonly occurs between the ages of 20 to 40 years.

- **Clinical presentation of AVM rupture:**
  - Hemorrhage: First clinical symptom in approximately 50% of cases
  - May be parenchymal (41%), subarachnoid (23%), or intraventricular (17%) hemorrhage (Brown, Whisnant, Sicks, O’Fallon, & Wiebers, 1996).
  - Seizures: 30% of cases.
  - HAs: 20% of cases; 10% overall with migraine-like HA.
  - Neurologic deficit (symptoms vary according to area that is affected).

### DIAGNOSTIC STUDIES

<table>
<thead>
<tr>
<th>TABLE 1–6 Diagnostic Studies</th>
<th>INFARCT</th>
<th>HEMORRHAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>CT</td>
<td>– Focally decreases density (hypodense) = darker than normal.</td>
<td>– Blood</td>
</tr>
<tr>
<td></td>
<td><strong>BLACK</strong> – Not seen immediately (unless there is a mass effect).</td>
<td>– Hyperdense (radio-opaque)</td>
</tr>
<tr>
<td></td>
<td>– May be seen after 24 hr (due to increase in edema); seen best in 3–4 days.</td>
<td><strong>WHITE</strong> – Seen immediately</td>
</tr>
<tr>
<td>MRI</td>
<td>– Edema</td>
<td>– Blood</td>
</tr>
<tr>
<td></td>
<td>– Fluid: high signal density</td>
<td>– Low signal density</td>
</tr>
<tr>
<td></td>
<td><strong>WHITE</strong> – Can be seen immediately as bright area on T2 = edema = water (H₂O)</td>
<td><strong>BLACK</strong> (on either T1 or T2)</td>
</tr>
</tbody>
</table>

### 1. Head CT Scan

- Major role in evaluating presence of blood (cerebral hemorrhage or hemorrhagic infarction), especially when thrombolysis is being considered.
- If an intracranial hemorrhage (ICH) is suspected, a head CT **without contrast** is the study of choice.
  - This avoids confusing blood with contrast, as both appear white on CT scan.

**Ischemic Infarction:**

- Regardless of stroke location or size, head CT studies are often normal during the first few hours after a nonhemorrhagic brain infarction.
- The infarcted area appears as a hypodense (black) lesion usually after 24 to 48 hours after the stroke (occasionally positive scans at 3–6 hours → subtle CT changes may be seen early with large infarcts, such as obscuration of gray-white matter junction, sulcal effacement, or early hypodensity).
- Hypodensity initially mild and poorly defined; edema better seen on third or fourth day as a well-defined hypodense area.
• Head CT with contrast: IV contrast provides no brain enhancement in day 1 or 2, as it must wait for enough damage to the blood–brain barrier; more evident in 1 to 2 weeks. Changes disappear 2 to 3 months later.
• Some studies suggest worse prognosis for patients receiving IV contrast early.
• Hemorrhage can occur within an infarcted area, where it will appear as a hyperdense mass within the hypodense edema of the infarct.

Hemorrhagic Infarct/ICH:
• High density (white) lesion seen immediately in approximately 100% of cases. Proven to be totally reliable in hemorrhages 1 cm or larger in diameter. Demonstration of clot rupture into the ventricular system (32% in one series) not as ominous as once thought.

Subarachnoid hemorrhage (SAH):
• Positive scan in 90% when CT performed within 4 to 5 days (may be demonstrated for only 8–10 days). SAH can really be visualized only in the acute stage, when blood is denser (whiter) than the cerebrospinal fluid (CSF).
• Appears as a hyperdense (or isodense) area on CT scan—look for blood in the basal cisterns or increased density in the region around the brainstem. May sometimes localize aneurysm based upon hematoma or uneven distribution of blood in cisterns.
• Once diagnosis of SAH has been established, angiography is generally performed to localize and define the anatomic details of the aneurysm and determine if other aneurysms exist.

2. Brain MRI
• More sensitive than CT scan in detecting acute ischemic infarcts (including small lacunes) and posterior cranial fossa infarcts (images are not degraded by bone artifacts).
  – Edema due to ischemia detected earlier on MRI than with CT—within a few hours of onset of infarct.
  – Diffusion-weighted imaging (DWI) MRI has emerged as the most sensitive and specific imaging technique for acute infarct, far better than CT or any other type of MRI sequence.

Cerebral Infarction:
• DWI has a high sensitivity and specificity for detecting infarcted regions, even within minutes of symptom onset.
• Early, increased (white) signal intensity on T2-weighted images, more pronounced at 24 hours to 7 days. Tl-weighted images may show mildly decreased signal.
• Chronic (21 days or more)—decreased Tl- and T2-weighted signals

Intracerebral Hemorrhage:
• Acute hemorrhage: decreased (black) signal or isointense on Tl- and T2-weighted images.
• Edema surrounding hemorrhage appears as decreased intensity on Tl-weighted image; increased (white) signal on T2 images.
• As hemorrhage ages, it develops increased signal on Tl and T2 images.

Subarachnoid or Intraventricular Hemorrhage:
• Acutely low signal (black) on Tl and T2 images.

Lacunes:
• CT scan documents most supratentorial lacunar infarctions, while MRI successfully documents both supratentorial and infratentorial infarctions when lacunes are 0.5 cm or greater.

3. Carotid Ultrasound
• Real time B-mode imaging; direct Doppler examination. Screening test for carotid stenosis; identification of ulcerative plaques less certain. Useful in following patients for progression of stenosis.

4. Transcranial Doppler Ultrasound
• Low-frequency Doppler sound wave used to insonate basal cranial vessels through temporal bone, orbit, foramen magnum.
• Velocity and direction of blood flow in all vessels of Circle of Willis may be identified.
• Detects vasospasm and intracranial collateral pathways.
5. Angiography
- Includes conventional angiography, magnetic resonance angiography (MRA), and intra-arterial digital subtraction angiography (DSA). These studies evaluate extracranial and intracranial circulation.
- Valuable tools for diagnosing aneurysms, vascular malformations, arterial dissections, narrowed or occluded vessels, and angiitis.
- Complications occur in 2% to 12%.
  - Include aortic or carotid artery dissection, embolic stroke, vascular spasm, and vascular occlusion.
- Morbidity associated with procedure: 2.5%.
- Carotid and vertebral angiography are the only certain means of demonstrating an aneurysm.
  - Positive in 85% of patients with “clinical” SAH.
- DSA studies safer; may be performed as outpatient.

MRA
- Can reliably detect extracranial carotid artery stenosis.
- May be useful in evaluating patency of large cervical and basal vessels.
- Detects most aneurysms on the basal vessels; insufficient sensitivity to replace conventional angiography.

6. Transthoracic Echocardiography (TTE) / Transesophageal Echocardiography (TEE)
- TTE can quickly assess heart valves and ejection fraction.
- TEE superior for evaluating aorta, pulmonary artery, heart valves, atria, atrial septum, left atrial appendage, and coronary arteries; TEE also used for detection of patent foramen ovale.
- Using cardiac catheterization and/or operation as a gold standard, contrast TEE was found to be more sensitive (100% vs. 63%, p <0.005) and accurate (97% vs. 78%, p <0.05) than contrast TTE in the detection of PFO (Chen et al., 1992).

7. Lumbar Puncture (LP)
- Can detect blood in the CSF.
- Primarily used in diagnosing suspected SAH when head CT is not available or, occasionally, when CT is negative and there is still high clinical suspicion.

MEDICAL TREATMENT

IMMEDIATE MANAGEMENT (Ferri, 2010; Rosen, 1992; Stewart, 1999)
- ABCs of critical care: airway, breathing, circulation:
  - **Airway obstruction** can occur with paralysis of the throat, tongue, or mouth muscles and pooling of saliva. Stroke patients with recurrent seizures are at increased risk of airway obstruction. Aspiration is also a concern in hemorrhagic strokes (increased association of vomiting at onset).
  - **Respiratory support**: Breathing abnormalities (central apnea) occasionally seen in patients with severe strokes.
  - **Control of BP**: See following section.
- **IV fluids**: Normal saline solution (NSS) or Ringer’s lactate. Avoid hypotonic solutions or excessive fluid loading, as it may worsen brain edema.
- **Keep patient NPO (nothing by mouth)** to avoid risk of aspiration.
- **Emergent head CT scan**:
  - Because the clinical picture of hemorrhagic and ischemic stroke may overlap, head CT scan without contrast is needed in most cases to definitively differentiate between the two.
  - One of the criteria that determines whether a patient is a candidate for emergent thrombolytic therapy.
1. STROKE

Impaired level of consciousness/coma: If there is acute deterioration of level of consciousness, evaluate for hematoma/acute hydrocephalus. Treatment would be emergency surgery.

Rule out hemorrhage in the presence of an underlying coagulopathy.

Fever and concern regarding meningitis.

Seizure management: See following section.

Check blood sugar levels:
- Hypoglycemia → bolus 50% IV dextrose.
- Hyperglycemia: Shown to potentiate severity of brain ischemia in animal studies.
- Insulin if blood sugar >300 mg/dL.

Control of intracranial pressure (ICP): See following section.

Fever: Potentially damaging to the ischemic brain.
- Antipyretics (e.g., acetaminophen) should be given early while the source of fever is being ascertained.

BP MANAGEMENT

The management of BP after acute ischemic and hemorrhagic stroke is challenging, and depends on whether or not the patient is a candidate for thrombolysis. Data to guide recommendations for treatment are inconclusive or conflicting. Many patients have spontaneous declines in blood pressure during the first 24 hours after onset of stroke (Jauch et al., 2013).

Antihypertensive treatment can lower cerebral perfusion and lead to worsening of stroke. The response of stroke patients to antihypertensive medications can be exaggerated.

Current treatment recommendations are based on the type of stroke (ischemic vs. hemorrhagic).

Ischemic Stroke BP Management

- IV labetalol and enalapril are favored antihypertensive agents.

<table>
<thead>
<tr>
<th>TABLE 1–7 American Heart Association (AHA) Recommendations for Hypertension (HTN) Management in Ischemic Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nonthrombolytic candidates</strong></td>
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<tr>
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<tr>
<td><strong>Thrombolytic candidates (before thrombolytic treatment given)</strong></td>
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<tr>
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</table>

Source: Adapted from American Heart Association website.

Hemorrhagic Stroke BP Management

- Treatment of increased BP during hemorrhagic strokes is controversial. The usual recommendation is to treat at lower levels of BP than for ischemic strokes because of concerns of rebleeding and extension of bleeding.

- Frequent practice is to treat BP if systolic blood pressure (SBP) >180, diastolic blood pressure (DBP) >105.

Agent of choice: IV labetalol (does not cause cerebral vasodilation, which could worsen increased ICP).

Seizure Management

- Recurrent seizures: Potentially life-threatening complication of stroke (see also Medical Management Problems in Stroke Rehabilitation section).

- Seizures can substantially worsen elevated ICP.

- Benzodiazepines = first-line agents for treating seizures.
  - IV lorazepam or diazepam

© Demos Medical Publishing
If seizures do not respond to IV benzodiazepines, treat with long-acting anticonvulsants:
- Phenytoin—18 mg/kg
- Fosphenytoin—17 mg/kg
- Phenobarbital—1,000 mg or 20 mg/kg

**Intracranial Pressure (ICP) Management**
- Increased ICP reduces cerebral blood perfusion pressure (CPP).
- CPP is calculated by subtracting ICP from mean arterial pressure (MAP).
  - CPP = MAP − ICP.
  - CPP should remain >60 mmHg to ensure cerebral blood flow.
- Fever, hyperglycemia, hyponatremia, and seizures can worsen cerebral edema by increasing ICP.
  - ICP ≤15 mmHg is considered normal.
  - Keep ICP <20 mmHg.

**ICP Management**
- Correction of underlying factors exacerbating increased ICP
  - Hypercarbia
  - Hypoxia
  - Hyperthermia
  - Acidosis
  - Hypotension
  - Hypovolemia
- Positioning
  - Avoid flat, supine position; instead elevate head of bed to 30°.
  - Avoid head and neck positions compressing jugular veins.
- Hyperventilation
  - Intubation and hyperventilation: Reduction of PaCO₂ through hyperventilation is the most rapid means of lowering ICP. Keep ICP <20 mmHg.
  - Hyperventilation should be used with caution because it reduces brain tissue PO₂ (PbrO₂). Hypoxia may lead to ischemia of brain tissue, causing further CNS damage after the stroke.
  - Optimal PaCO₂ approximately 25 to 30 mmHg.
- Medications
  - Hyperosmolar therapy with mannitol improves ischemic brain swelling through diuresis and intravascular fluid shifts.
  - Furosemide/acetazolamide may also be used.
  - High doses of barbiturates (e.g., thiopental) rapidly lower ICP and suppress electrical brain activity.
- Hypothermia.
- Fluid restriction
  - Avoid glucose solutions; use normal saline; maintain euvoolema.
  - Replace urinary losses with normal saline in patients receiving mannitol.
- Surgical therapy:
  - Neurosurgical decompression for lesions with mass effect causing increased ICP.

**THROMBOLYTIC THERAPY**

**Intra-Venous Thrombolytic Therapy**
- Intravenous tissue plasminogen activator (tPA) is the first FDA-approved treatment for acute ischemic strokes in selected patients.
- In the National Institute of Neurologic Disorders (NINDS) trial (1995), patients given tPA within 3 hours of onset of stroke were 30% more likely to have minimal or no disability at 3 months compared to patients treated with placebo.
- There was a tenfold increase in hemorrhage (overall) with tPA compared to placebo (6.4% vs. 0.6%) and in fatal ICH (3% vs. 0.3%).
- However, overall mortality was higher in the placebo group than in tPA groups: 17% in tPA group (including hemorrhage cases) vs. 21% in placebo group.
  - In 2008, ECASS III demonstrated that intravenous rtPA can be given safely to, and can improve outcomes for, carefully selected patients treated 3 to 4.5 hours after stroke (Hacke et al., 2008).
  - European Medicines Agency expanded approval of intravenous rtPA to the 3 to 4.5 hour window, but the FDA has declined to do so.
  - However, American Heart Association/American Stroke Association has recommended tPA in eligible patients who can be treated 3 to 4.5 hours after stroke onset.

**Inclusion Criteria for tPA Use**

- Age 18 years or older.
- Time of symptom onset well established and is <4.5 hours before treatment would begin.
- Patients with measurable neurologic deficits (moderate to severe stroke symptoms).
- Head CT negative for blood.
- Informed consent of the patient.

**Exclusion Criteria for tPA Use**

- Minor stroke symptoms/TIA (symptoms rapidly improving).
- Head CT positive for blood.
- BP >185/100 despite medical treatment.
- Coagulopathy/patient on anticoagulants (warfarin, antiplatelet agents, heparin):
  - PT >15 seconds or INR >1.7.
  - Patient received heparin within 48 hours prior with elevated PTT.
  - Patient taking warfarin.
- Platelet count <100,000.
- Blood sugar <50 or >400.
- History of stroke or severe head injury in past 3 months.
- History of ICH, AVM, or aneurysm.
- History of gastrointestinal (GI) or genitourinary (GU) bleeding within past 21 days.
- No pregnancy or lactation within past 30 days.
- Major surgery within past 14 days.
- Seizure at onset of stroke.
- Acute myocardial infarction (MI).
- Additional exclusion criteria for use of tPA 3 to 4.5 hours post-onset:
  - Age >80 years old
  - Patient on anticoagulants regardless of INR
  - Patient with baseline NIHSS score >25
  - Imaging evidence of ischemic injury involving more than one-third of MCA territory
  - History of both stroke and diabetes mellitus

**Intra-Arterial Thrombolytic Therapy**

- Intra-arterial fibrinolysis is beneficial for treatment of carefully selected patients with major ischemic strokes <6 hours post-onset caused by occlusions of the MCA who are not otherwise candidates for intravenous tPA.
- tPA does not have FDA approval for intra-arterial use.

**Intra-Arterial Mechanical Thrombectomy**

- Thrombectomy devices can be useful in achieving recanalization alone or in combination with pharmacological fibrinolysis in carefully selected patients.
- Stent retrievers (e.g., Solitaire FR, Trevo) are generally preferred to coil retrievers (e.g., Merci).
1. STROKE

The usefulness of mechanical thrombectomy devices other than the Merci retriever, the Penumbra System, Solitaire FR, and Trevo is not well established.

ANTICOAGULANT THERAPY

Heparin, Low Molecular-Weight Heparin (LMWH), Warfarin

(Kay et al., 1995; Sandercock et al., 2008)

- Immediate anticoagulant therapy in patients with acute ischemic stroke is not associated with any significant net short- or long-term benefit.
- There is no evidence to support the routine use of any type of anticoagulant in an acute ischemic stroke.
- Urgent anticoagulation with the goal of preventing early recurrent stroke, halting neurologic worsening, or improving outcomes after acute ischemic stroke is not recommended for treatment of patients with acute ischemic stroke (Jauch et al., 2013).
- In patients with atrial fibrillation or prothrombotic states who have had a recent stroke or TIA, long-term oral anticoagulation is preferred.

Indications for Anticoagulation

- Cardiac emboli: Best reason to anticoagulate.
  - Primarily from nonvalvular atrial fibrillation (AF) and mural thrombus from MI (Ryder & Benjamin, 1999).
    - Among patients with nonvalvular AF, anticoagulation can reduce stroke by 60% (Hart et al., 2007).
  - Timing of anticoagulation in patients with cardiac emboli controversial; probable risk of inducing cerebral hemorrhage or hemorrhagic infarction within large infarcts if anticoagulated in the first 24 to 36 hours.
  - If neurologic deficit is mild (and CT shows no hemorrhage), may begin anticoagulation immediately.
  - If deficit severe (clinically and/or CT), wait 3 to 5 days before starting anticoagulation.
  - 75% of cardiogenic emboli lodge in the brain. The most common cause is chronic atrial fibrillation.
- Transient ischemic attacks:
  - Full anticoagulation in TIA is indicated only with cardioembolic sources and in crescendo TIA, while performing a rapid workup.
- Completed stroke:
  - Anticoagulation is not considered beneficial after major infarction and usually not of great value once stroke is fully developed.
  - Anticoagulation generally not employed for lacunar infarction.
- Anticoagulation agents:
  - Warfarin (Coumadin) inhibits vitamin K-dependent coagulation factors.
  - Dabigatran (Pradaxa) is a direct thrombin inhibitor indicated to reduce the risk of stroke and systemic embolism in patients with nonvalvular AF. (FDA approved in October 2010).
  - Rivaroxaban (Xarelto) is a factor Xa inhibitor indicated to reduce the risk of stroke and systemic embolism in nonvalvular AF (FDA approved November 2011).
  - Apixaban (Elquis) is a factor Xa inhibitor anticoagulant indicated to reduce the risk of stroke and systemic embolism in patients with nonvalvular AF. (FDA approved December 2012.)

ANTIPLATELET THERAPY

Aspirin, Aspirin/Dipyridamole, Clopidogrel (Elkind & Sacco, 2010)

- For patients with noncardioembolic ischemic stroke or TIA, the risk of recurrent stroke and other cardiovascular events is reduced with antiplatelet agents: aspirin (50 to 325 mg/day), aspirin/extended-release dipyridamole (Aggrenox®), or clopidogrel (Plavix®).
• The oral administration of aspirin 325 mg within 24 to 48 hours after an ischemic stroke onset is recommended for secondary stroke prevention for most patients.
• Antiplatelet medications have been shown to be beneficial in the secondary stroke prevention of presumed arterial origin. Aspirin plus extended-release dipyridamole (Aggrenox) has demonstrated statistically significant additive benefit over monotherapy with each agent.
• The combination of clopidogrel and aspirin has not been shown to provide any significant additional protection against recurrent ischemic stroke over each agent individually (Creager, 1998). Moreover, the combination increases the risk of life-threatening or major bleeding side effects (MATCH Study; Diener et al., 2004).

CORTICOSTEROIDS

• No value in ischemic strokes.
• Some studies suggest worsening in prognosis of stroke patients due to hyperglycemia.

CAROTID ENDARTERECTOMY (CEA) (Moore, 1995)
Symptomatic Carotid Stenosis

• CEA for symptomatic lesions with >70% stenosis (70% to 99%) is effective in reducing the incidence of ipsilateral hemisphere stroke (Endarterectomy for moderate symptomatic carotid stenosis: Interim results from the MRC European Carotid Surgery Trial, 1996; Executive Committee for Asymptomatic Carotid Atherosclerosis Study, 1995; Rerkasem K and Rothwell PM, 2011 North American Symptomatic Carotid Endarterectomy Trial Collaborators, 1991).
• CEA reduced risk of disabling stroke, as well as death for patients with stenosis exceeding 70% in the European Carotid Surgery Trial (ECS) or 50% in the North American Symptomatic Carotid Endarterectomy Trial (NASCET).
• Results generalizable only to surgically fit patients operated on by surgeons with complication rates <6%.

Asymptomatic Carotid Stenosis (Chambers & Donnan, 2005)

• Despite about a 3% perioperative stroke or death rate, CEA for asymptomatic carotid stenosis reduces the risk of ipsilateral stroke, and any stroke, by approximately 30% over 3 years.
• However, absolute risk reduction is small (approximately 1% annually over the first few years of follow-up in the two largest and most recent trials) but could be higher with longer follow-up (Young et al., 1996).

TREATMENT OF SAH (SEE ALSO ICP MANAGEMENT SECTION)

• Bedrest in a quiet, dark room with cardiac monitoring (cardiac arrhythmias are common).
• Control of HAs with acetaminophen and codeine.
• Mannitol to reduce cerebral edema.
• BP control. Also have the patient avoid all forms of straining (give stool softeners and mild laxatives).
• Early surgery with clipping or coiling of the aneurysm reduces risk of rebleeding but does not prevent vasospasm or cerebral ischemia.
• Nimodipine (calcium channel blocker) decreases cerebral vasospasms after SAH and has been shown to improve outcome after SAH. It also decreases the incidence of permanent neurologic damage and death.
  • Therapy should be initiated within 96 hours of onset of hemorrhage.
  • Treatment: nimodipine (Nimotop) 60 mg PO every 4 hours × 21 days.
TREATMENT OF ICH
- Management of increased ICP and BP (see earlier sections in this chapter).
- Large intracranial or cerebellar hematomas often require surgical decompression.

TREATMENT OF AVM (Hamilton & Spetzler, 1994; Schaller et al., 1998)
- Treatment advised in both symptomatic and asymptomatic AVMs.
- Surgical excision if size and location feasible (and depending on perioperative risk).
- Embolization.
- Proton beam therapy (via stereotactic procedure).
- Small asymptomatic AVMs: radiosurgery/microsurgical resection recommended.

STROKE REHABILITATION

INTRODUCTION
The primary goal of stroke rehabilitation is functional restoration by maximizing the independence, lifestyle, and dignity of the patient. This approach implies rehabilitative efforts from a physical, behavioral, cognitive, social, vocational, adaptive, and re-educational point of view. The multidimensional nature of stroke and its consequences make coordinated and combined interdisciplinary team care the most appropriate strategy to treat the stroke patient.

Recovery From Impairments
- Hemiparesis and motor recovery have been the most studied of all stroke impairments. Up to 88% of acute stroke patients have some degree of hemiparesis.
- The process of recovery from stroke-induced hemiplegia usually follows a relatively predictable, stereotyped series of events. This sequence of events has been systematically described by several clinical researchers.
- Twitchell (1951) published a highly detailed report describing the pattern of motor recovery following a stroke (Note: Pattern most consistent in patients with cerebral infarction in the MCA distribution). His sample included 121 patients, all except 3 having suffered either thrombosis or embolism of 1 of the cerebral vessels.
  - Immediately following onset of hemiplegia, there is total loss of voluntary movement and loss or decrease of tendon reflexes.
  - Within 48 hours, increased deep tendon reflexes on the involved side develop.
  - This is followed by the return and then increase of muscle tone against passive movement (tone returns → spasticity), especially in the flexors and adductors in the upper extremity (UE) and the extensors and adductors in the lower extremity (LE) on the hemiparetic side.
  - As spasticity increases, clonus of the ankle plantar flexors appears in 1 to 38 days post-onset of hemiplegia.
  - Recovery of movement:
    - 6 to 33 days after the onset of hemiplegia, the first “intentional” movements (shoulder flexion) appear.
    - In the UE, a flexor synergy pattern develops (with shoulder, elbow, wrist, and finger flexion) followed by development of an extensor synergy pattern. Voluntary movement in the lower limb also begins with flexor synergy (also proximal-hip) followed by extensor synergy pattern.
- With increased voluntary movement, there is a decrease in the spasticity of the muscles involved.
- Tendon reflexes remain increased despite complete recovery of movement.
• At onset of hemiplegia, the arm is more involved than the leg. Eventual motor recovery in the leg occurs earlier and is more complete than in the arm.
• Most recovery takes place in the first 3 months and only minor additional recovery occurs after 6 months post-onset.

Predictors of Motor Recovery (Twitchell, 1951)
• Severity of UE weakness at onset:
  – With complete arm paralysis at onset, there is a poor prognosis of recovery of useful hand function (only 9% gain good recovery of hand function).
• Timing of return of hand movement:
  – If the patient shows some motor recovery of the hand by 4 weeks, there is up to a 70% chance of making a full or good recovery.
• Poor prognosis associated also with:
  – No measurable grasp strength by 4 weeks.
  – Severe proximal spasticity.
  – Prolonged “flaccidity” period.
  – Late return of proprioceptive facilitation (tapping) response >9 days.
  – Late return of proximal traction response (shoulder flexors/adductors) >13 days.

Brunnstrom (1966) and Sawner and LaVigne (1992) also described the process of recovery following stroke-induced hemiplegia. The process was divided into a number of stages:

1. **Flaccidity** (immediately after the onset).
   No “voluntary” movements on the affected side can be initiated.
2. **Spasticity appears.**
   Basic synergy patterns appear.
   Minimal voluntary movements may be present.
3. **Patient gains voluntary control over synergies.**
   Increase in spasticity.
4. **Some movement patterns out of synergy are mastered.**
   Synergy patterns still predominate.
   Decrease in spasticity.
5. **If progress continues, more complex movement combinations are learned as the basic synergies lose their dominance over motor acts.**
   Further decrease in spasticity.
6. **Disappearance of spasticity.**
   Individual joint movements become possible and coordination approaches normal.
7. **Normal function is restored.**

## REHABILITATION METHODS FOR MOTOR DEFICITS

### Major Theories of Rehabilitation Training

#### TRADITIONAL THERAPY

A traditional therapeutic exercise program consists of positioning, ROM exercises, strengthening, mobilization, compensatory techniques, and endurance training (e.g., aerobics). Traditional approaches for improving motor control and coordination emphasize the need of repetition of specific movements for learning the importance of sensation to the control of movement, and the need to develop basic movements and postures (Kirsteins et al., 1999).

#### PROPRIOCEPTIVE (PERIPHERAL) NEUROMUSCULAR FACILITATION (PNF) (Knott & Voss, 1968)

• Uses spiral and diagonal components of movement rather than the traditional movements in cardinal planes of motion with the goal of facilitating movement patterns that will have more functional relevance than the traditional technique of strengthening individual group muscles.
• Theory of spiral and diagonal movement patterns arose from observations that the body will use muscle groups synergistically related (e.g., extensors versus flexors) when performing a maximal physical activity.
• Stimulation of nerve/muscle/sensory receptors to evoke responses through manual stimuli to increase ease of movement-promotion function.
• Resistance is used during the spiral and diagonal movement patterns with the goal of facilitating “irradiation” of impulses to other parts of the body associated with the primary movement (through increased membrane potentials of surrounding alpha motoneurons, rendering them more excitable to additional stimuli and thus affecting the weaker components of a given part).
• Mass-movement patterns keep Beevor’s axiom: The brain knows nothing of individual muscle action but only movement.

BOBATH APPROACH / NEURODEVELOPMENTAL TECHNIQUE (NDT) (Bobath, 1978)
• The goal of NDT is to normalize tone, to inhibit primitive patterns of movement, and to facilitate automatic, voluntary reactions as well as subsequent normal movement patterns.
• Probably the most commonly used approach.
• Based on the concept that pathologic movement patterns (limb synergies and primitive reflexes) must not be used for training, because continuous use of the pathologic pathways may make it too readily available to use at the expense of the normal pathways.
• Suppress abnormal muscle patterns before normal patterns are introduced.
• Mass synergies are avoided, although they may strengthen weak, unresponsive muscles, because these reinforce abnormally increased tonic reflexes and spasticity.
• Abnormal patterns are modified at proximal key points of control (e.g., shoulder and pelvic girdle).
• Opposite to the Brunnstrom approach, which encourages the use of abnormal movements; see the following section.

BRUNNSTROM APPROACH / MOVEMENT THERAPY (Sawner & LaVigne, 1992)
• Uses primitive synergistic patterns in training in an attempt to improve motor control through central facilitation.
• Based on the concept that damaged CNS regressed to phylogenetically older patterns of movements (limb synergies and primitive reflexes). Thus, synergies, primitive reflexes, and other abnormal movements are considered normal processes of recovery before normal patterns of movements are attained.
• Patients are taught to use and voluntarily control the motor patterns available to them at a particular point during their recovery process (e.g., limb synergies).
• Enhances specific synergies through use of cutaneous/proprioceptive stimuli, central facilitation using Twitchell’s recovery.
• Opposite to the Bobath approach, in which the goal is to inhibit abnormal patterns of movement

SENSORIMOTOR APPROACH / ROOD APPROACH (Schultz-Krohn, 2013)
• Modification of muscle tone and voluntary motor activity using cutaneous sensorimotor stimulation.
• Facilitatory or inhibitory inputs through the use of sensorimotor stimuli, including quick stretch, icing, fast brushing, slow stroking, tendon tapping, vibration, and joint compression to promote contraction of proximal muscles.

MOTOR RELEARNING PROGRAM / CARR AND SHEPHERD APPROACH (Carr et al., 1985)
• Based on cognitive motor relearning theory and influenced by the Bobath approach.
• Goal is for the patient to relearn how to move functionally and how to problem solve during attempts at new tasks.
• Instead of emphasizing repetitive performance of a specific movement for improving skill, it teaches general strategies for solving motor problems.
• Emphasizes functional training of specific tasks, such as standing and walking, and carryover of those tasks.

OTHER APPROACHES
• **Constraint-induced movement therapy (CIMT)** has been statistically shown to produce clinically significant improvements in arm motor function that persist >1 year (EXCITE Trial, Wolf et al., 2006).
  – CIMT requires that patients be able to extend their wrists and actively move their digits.
  – In the EXCITE trial, participants were required to have at least 10° of active wrist extension, at least 10° of thumb abduction/extension, and at least 10° of extension in at least two additional digits.
• **Body-weight-support treadmill training** was not shown to be superior to progressive exercise at home managed by a physical therapist (LEAPS Trial, Duncan et al., 2011).
  – Subjects who received body-weight-support treadmill training within 2 months after stroke were at higher risk to fall than those in other groups.
• **Functional electrical stimulation (FES)** may improve the ability to voluntarily move the affected limb and/or use the affected limb in everyday activities (Pomeroy et al., 2006).
  – The available evidence suggests there might be a small effect on some aspects of function in favor of electrical stimulation compared to no treatment.
  – Currently, there are insufficient data to support or refute the clinical use of FES for neumuscular retraining.
• **Electromyographic biofeedback (EMG-BF)** makes patient aware of muscle activity or lack of it by using external representation (e.g., auditory or visual cues) of internal activity as a way to assist in the modification of voluntary control.
  – In addition to trying to modify autonomic function, EMG-BF also attempts to modify pain and motor disturbances by using volitional control and auditory, visual, and sensory clues.
  – Electrodes are placed over agonists/antagonists for facilitation/inhibition.
  – Accurate sensory information reaches brain through systems unaffected by brain → via visual and auditory for proprioception.
  – There is insufficient evidence to support or refute use in stroke rehabilitation (Woodford & Price, 2007).
• **Robotic devices** are being developed to improve the rehabilitation of extremities by providing passive and active range of motion and measurement of improvements in mobility and strength.
  – Examples: AUTO ambulator and the treadmill supported orthosis.
  – There is insufficient evidence to support or refute use in stroke rehabilitation.
• **Motor imagery** is a mental process during which an individual rehearses or simulates a given action before it is actually performed.
  – There is insufficient evidence to support or refute use in stroke rehabilitation.
• **Bilateral arm training** hypothesizes that there is a coupling effect that reinforces a possible training benefit to the affected limb when bimanual tasks are performed.
  – There is insufficient evidence to support or refute use in stroke rehabilitation.
• **Mirror therapy** mirror is placed in the patient’s midsagittal plane, thus reflecting movements of the nonparetic side as if it were the affected side.
  – Mirror therapy is effective for improving upper extremity motor function, activities of daily living (ADL), and pain, at least as an adjunct to normal rehabilitation for patients after stroke.
  – Study limitations include small sample sizes, control interventions not used routinely in stroke rehabilitation, and some methodological limitations.
• **Virtual reality** utilizes computer-simulated environment and interactive video gaming to provide patients with engaging activities to improve motor or cognitive function.
  – Limited evidence that the use of virtual reality may be beneficial in improving arm function and ADL function when compared with the same dose of conventional therapy.
  – Unclear which characteristics of virtual reality are most important, and unknown whether effects are sustained.
Noninvasive brain stimulation includes transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) (Sandrini & Cohen, 2013).
- Can be used to modulate cortical excitability during and for several minutes after the end of the stimulation period.
- Cortical excitability can be reduced (inhibition) or enhanced (facilitation) depending upon parameters.
- May induce plastic changes within neural networks active during functional recovery, but still being studied.

**POST-STROKE SHOULDER PAIN (TABLE 1–8) (Lombard et al., 2009)**

- 70% to 84% of stroke patients with hemiplegia have shoulder pain with varying degrees of severity.
- Of the patients with shoulder pain, the majority (85%) will develop it during the spastic phase of recovery.
- It is generally accepted that the most common causes of hemiplegic shoulder pain are CRPS Type I (see below) and soft-tissue lesions (including plexus lesions).

**Complex Regional Pain Syndrome Type I (CRPS Type I)**

(Also see the CRPS section in Chapter 11: Pain Medicine.)
- Also known as reflex sympathetic dystrophy [RSD], shoulder-hand syndrome, or Sudeck atrophy.
- Disorder characterized by sympathetic-maintained pain and related sensory abnormalities, abnormal blood flow, abnormalities in the motor system, and changes in both superficial and deep structures with trophic changes.
- Reported in 12% to 25% of hemiplegic stroke patients.
- CRPS Type I: = RSD.
- CRPS Type II: = causalgia—pain limited to a peripheral nerve distribution.
- Most common subtype of RSD in stroke is shoulder-hand syndrome.

**STAGES (OF RSD)**

- **Stage 1 (acute):** Burning pain, diffuse swelling/edema, exquisite tenderness, hyperpathia and/or allodynia, vasomotor changes in hand/fingers (increased nail and hair growth, hyperthermia or hypothermia, sweating). Lasts 3 to 6 months.
- **Stage 2 (dystrophic):** Pain becomes more intense and spreads proximally, skin/muscle atrophy, brawny edema, cold insensitivity, brittle nails/nail atrophy, decreased ROM, mottled skin, early atrophy, and osteopenia (late). Lasts 3 to 6 months.
- **Stage 3 (atrophic):** Pain decreases; trophic changes occur: hand/skin appear pale and cyanotic with a smooth, shiny appearance, feeling cool and dry; bone demineralization progresses with muscular weakness/atrophy, contractures/flexion deformities of shoulder/hand, tapering digits; no vasomotor changes.

**PATHOGENESIS**

- Multiple theories postulated:
  - Abnormal adrenergic sensitivity develops in injured nociceptors, and circulating or locally secreted sympathetic neurotransmitters trigger the painful afferent activity.
  - Cutaneous injury activates nociceptor fibers → central pain-signaling system → pain.
  - Central sensitization of pain-signaling system.
  - Low-threshold mechanoreceptor input develops capacity to evoke pain.
  - With time, efferent sympathetic fibers develop capacity to activate nociceptor fibers.
DIAGNOSIS
- **X-rays**—normal in initial stages; periarticular osteopenia may be seen in later stages. Use is questionable, given that bone mineral density starts to decrease in the paralytic arm 1 month after stroke.
  - Need 30% to 50% demineralization for detection.
- **Triple phase bone scan**—30 stroke survivors <3 months onset evaluated for CRPS type I using triple phase bone scan (Harbert et al., 1996; Kozin, 1981; Simon & Carlson, 1980).
  - Sensitivity approximately 92%.
  - Specificity approximately 56%.
  - Positive predictive value (PPV) approximately 58%.
  - Negative predictive value (NPV) approximately 91% (Holder & Mackinnon, 1984).
  - Diffusely increased juxta-articular tracer activity on delayed images is the most sensitive indicator for RSD (sensitivity 96%, specificity 97%, and PPV 88%).
- **EMG**—as predictor for CRPS (Cheng & Hong, 1995).
  - Association between spontaneous activity and eventual development of CRPS (vs. no spontaneous activity on EMG).
- **Clinical exam** (Wang et al., 1998):
  - Clinical diagnosis difficult, presentation often incomplete.
  - Most consistent early diagnostic signs: Shoulder pain with ROM (flexion/abduction/external rotation), absence of pain in elbow and with forearm pronation/supination; wrist dorsiflexion pain with dorsal edema; pain metacarpophalangeal (MCP)/proximal interphalangeal (PIP) flexion with fusiform PIP edema.
  - Pain disproportionate to injury and clinical findings.
  - Shoulder/hand pain preceded by rapid ROM loss.
  - 25% had radionuclide evidence for CRPS type I: Positive diagnosis was evident when delayed image showed increased uptake in wrist, MCP, and interphalangeal (IP) joints.
  - In this study, the most valuable clinical sign was MCP tenderness to compression with 100% predictive value, sensitivity 85%, and specificity 100%.
- **Stellate ganglion block:**
  - Successful when patient develops an ipsilateral Horner syndrome.
  - Alleviation of pain following sympathetic blockade of the stellate ganglion using local anesthetic is the gold standard for diagnosis of *sympathetically mediated* CRPS type I.

TREATMENT *Arlet & Mazieres, 1997*
- **ROM exercises of the involved joint:** Pain-free within 3 weeks, most <4 to 6 days with passive stretch of involved joints.
- **Corticosteroids** (systemic): A large majority of patients respond to systemic steroids instituted in the acute phase of the disease. Usually prednisone in doses up to 100 to 200 mg/day or 1 mg/kg, and tapered over 2 weeks.
  - More effective in CRPS type I confirmed by bone scan than in “clinical” CRPS type I with a negative bone scan.
  - Bone scans may be useful not only in establishing the diagnosis of CRPS type I but also in identifying patients likely to respond to oral steroid therapy. In a study, 90% of patients with positive bone scan findings for CRPS type I treated with corticosteroids had a good or excellent response, whereas 64% of patients without bone scan abnormalities had a poor or fair response.
  - In a recent study, 31/34 MCA stroke patients with RSD became pain free by 14 days after starting methylprednisolone 8 mg PO four times a day (patients treated for 2 weeks followed by 2-week taper).
• Medications
  – Nonsteroidal anti-inflammatory drugs (NSAIDs)—analgesia
  – Tricyclic antidepressants (TCAs)
  – Bisphosphonates
  – Calcitonin
  – Anticonvulsants (e.g., gabapentin or carbamazepine)
  – Alpha-adrenergic blockers (e.g., clonidine, prazosin)
  – Beta-blockers (e.g., propranolol, pindolol)
  – Calcium channel blockers (e.g., nifedipine)
  – Topical capsaicin
• Modalities:
  – Edema control measures
  – Transcutaneous nerve stimulation (TENS)
  – Desensitization techniques
  – Contrast baths
  – Ultrasound
• Injections:
  – Intra-articular corticosteroid injections
  – Local injections (procaine, corticosteroid)
  – Sympathetic stellate ganglion blocks may be diagnostic as well as therapeutic
  – Sympathectomy

Shoulder Subluxation
• Clinically presents with a palpable gap between the acromion and humeral head. Characterized by subluxation of the humeral head from the glenoid fossa inferiorly.
• Conflicting evidence as to whether subluxation is correlated with shoulder pain.
• Etiology: Unknown but may be due to changes in the mechanical integrity of the glenohumeral joint.
• Pathogenesis: Factors that are thought to be related to shoulder subluxation include:
  – Angulation of the glenoid fossa.
  – Influence of the supraspinatus muscle on the humeral head sitting.
  – Support of the scapula on the rib cage.
  – Contraction of the deltoid and rotator cuff muscles on the abducted humerus.
• A number of recent studies failed to show any correlation between shoulder subluxation and pain.
• Might be a correlation between shoulder pain and decrease in arm external rotation
• Basmajian principle: Decreased trapezius tone → the scapula rotates and humeral head subluxes from the glenoid fossa.

TREATMENT
• Shoulder sling use is controversial.
• Routine use of sling for the subluxed shoulder (or for shoulder pain) is not indicated.
  – Friedland (1975): Slings do not prevent/correct subluxation and are not necessary to support pain-free shoulder.
  – Hurd et al. (1974): No appreciable difference in shoulder ROM, subluxation, or shoulder pain in patients with or without slings.
• Pros: May be used when patient ambulates to support extremity (may prevent UE trauma, which in turn may cause increase in pain or predispose to development of RSD).
• Cons: May encourage contractures in shoulder adduction/internal rotation or elbow flexion (flexor synergy pattern).
• Other widely used treatments for shoulder subluxation:
  – Functional electrical stimulation (FES).
  – Arm board, arm trough, lapboard—used in poor upper-extremity recovery, primary wheelchair users.
- Arm board may overcorrect subluxation.
- Overhead slings—prevents hand edema (may use foam wedge on arm board).

**PREVENTION**

- Subluxation may be prevented by combining the early reactivation of shoulder musculature (specifically supraspinatus and post- and mid-deltoid) with the provision of FES or a passive support of the soft-tissue structures of the glenohumeral joint (e.g., arm trough).

**Bicipital Tendinitis**

- Chronic pain in anterior shoulder, pain in abduction/external rotation, painful over bicipital groove.
- Positive Yergason test: With the elbow flexed at 90°, and while the forearm is pronated, the patient supinates the forearm, flexes the elbow, and externally rotates the humerus while the examiner resists these movements and pulls downward on the elbow. The test result is positive if the patient experiences pain over the bicipital groove or if the tendon pops out of the groove (see MSK chapter).
- Greatest excursion of long head biceps from flexion/internal rotation, to elevation/abduction, depression/external rotation/extension.
- May progress to adhesive capsulitis (frozen shoulder).
- **Diagnosis:** Positive clinical exam findings and imaging studies. May be confirmed with diagnostic tendon sheath injection of lidocaine, but caution should be used with steroid injection of the tendon sheath due to risk of rupture.

**Rotator Cuff Tear, Shoulder Impingement Syndrome, and Adhesive Capsulitis (Table 1–8)**

- All can be potential causes of post-stroke shoulder pain.
- See also Musculoskeletal chapter for more detail on these topics.

**Brachial Plexus / Peripheral Nerve Injury**

- **Etiology:** “Traction” injury to the plexus/nerve.
- **Diagnosis:**
  - Atypical functional return, segmental muscle atrophy, finger extensor contracture, delayed onset of spasticity.
  - Electrodiagnostic studies (EMG)—lower motor neuron findings.
- **Treatment:**
  - Proper bed positioning to prevent patient from rolling onto paretic arm, trapping it behind the back or through bed rails and causing traction stress.
  - ROM to prevent contracture while traction avoided.
  - 45° shoulder-abduction sling for nighttime positioning.
  - Sling for ambulation to prevent traction by gravity.
  - Armrest in wheelchair as needed.
- **Prognosis:** May require 8 to 12 months for re-innervation.

**Heterotopic Ossification (HO)**

- Infrequent in stroke but may be seen in elbow or shoulder.
- Occurs only on extensor side of elbow.
  - No problems in pronation/supination since proximal radioulnar joint not involved.
- **Treatment:** Joint mobilization/ROM, etidronate disodium.

**Dependent Edema**

- May be treated with compression glove, foam wedge, pneumatic compression, retrograde massage, and arm elevation.
### TABLE 1–8 Post-Stroke Shoulder Pain

<table>
<thead>
<tr>
<th>EXAM</th>
<th>ROTATOR CUFF TEAR</th>
<th>CRPS TYPE I (RSD)</th>
<th>ADHESIVE CAPSULITIS (FROZEN SHOULDER)</th>
<th>IMPINGEMENT SYNDROME</th>
<th>BICEPS TENDINITIS</th>
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</thead>
<tbody>
<tr>
<td>Acromio-humeral</td>
<td>Positive abd. test</td>
<td>MCP compression test</td>
<td>External rotation less than 15°</td>
<td>Pain with abduction of 70°–90°</td>
<td>Positive Speed’s/</td>
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<tr>
<td>separation</td>
<td>Positive drop arm test</td>
<td>Skin changes color</td>
<td>Early decrease in scapular motion</td>
<td>End-range pain with forward flexion</td>
<td>Yergason test</td>
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<tr>
<td>Flaccid</td>
<td>Flaccid or spastic</td>
<td>Flaccid or spastic</td>
<td>Spastic</td>
<td>Usually spastic</td>
<td>Flaccid or spastic</td>
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<tr>
<td>DIAGNOSTIC TEST</td>
<td>X-ray in standing position</td>
<td>X-ray</td>
<td>Arthrogram</td>
<td>Subacromial injection of lidocaine</td>
<td>Tendon sheath injection of lidocaine</td>
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<td></td>
<td>Scapular plane view</td>
<td>Arthrogram</td>
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<td>Triple phase bone scan</td>
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<td>Stellate ganglion block</td>
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<td>TREATMENT</td>
<td>Sling when upright</td>
<td>Steroid injection</td>
<td>Oral corticosteroids</td>
<td>PT/ROM</td>
<td>Tendon sheath injection of steroids</td>
</tr>
<tr>
<td></td>
<td>FES</td>
<td>PT/ROM</td>
<td>Debridement manipulation</td>
<td>Subacromial/GH steroid injections</td>
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<td>Possible surgical repair</td>
<td>Subacromial/GH steroid injections</td>
<td>Intra-articular steroids</td>
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<td>Reduction of internal rotator cuff tone</td>
<td>Oral steroids</td>
<td>Reduction of internal rotator cuff tone</td>
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Note: CRPS I = complex regional pain syndrome type I; FES = functional electrical stimulation; MCP = metacarpophalangeal; MRI = magnetic resonance imaging; RSD = reflex sympathetic dystrophy; PT/ROM = physical therapy/range of motion.

Source: Adapted from Black-Schaffer et al., 1999.
OTHER STROKE REHABILITATION ISSUES

Spasticity
- For a more detailed discussion, see the Spasticity section in the Associated Topics chapter.
- Spasticity is defined as abnormal, velocity-dependent resistance to passive movement of affected muscles at rest and posturing in the patterns previously mentioned during ambulation and with noxious stimuli.
- Usually follows classic upper-extremity flexor and lower-extremity extensor patterns.
- Spasticity usually develops days to weeks after a stroke.

Treatment
- Noninvasive: Stretching, splints/orthosis, serial casting, electrical stimulation, cold modalities.
- Although widely done in clinical practice, the use of medications (e.g., benzodiazepines, baclofen, dantrolene, clonidine, tizanidine) in stroke patients still lacks scientific evidence to support efficacy. These drugs appear to have modest effects on the hypertonicity and posturing associated with stroke; side effects limit their usefulness.
- Chemoneurolysis:
  - Botulinum toxin: May be particularly useful in focal control spasticity (e.g., wrist and finger flexors in the UE; ankle invertors in the LE).
  - Phenol/alcohol (neurolytic agents): May be helpful for treating spasticity of large muscle groups (e.g., hip adductors and extensors, the pectorals, lats, and biceps). Use is limited by adverse effects (e.g., pain during injection, post-injection dysesthesia/chronic pain, vascular injection).
- Intrathecal baclofen (ITB) pump:
  - Limited experience and use in stroke patients; usefulness remains to be determined in this population.
  - Some evidence shows use of ITB therapy with physical therapy may improve walking speed and functional mobility in ambulatory individuals with post-stroke spastic hemiplegia (Francisco, 2003).
- Surgical procedures:
  - Uncommonly used in stroke, probably because of expected decrease in survival and increase in rate of medical comorbidities.
  - May be useful in selected cases when specific goals are pursued (e.g., increase in function, improve hygiene, decrease in pain).

Deep Vein Thrombosis (DVT)
- Common medical complication after stroke, occurring in 20% to 75% of untreated stroke survivors.
- 60% to 75% in affected extremity, 25% proximal DVT; 1% to 2% PE.
- Diagnosis: Usually can be made using noninvasive techniques:
  - Ultrasonography.
  - Impedance plethysmography.
  - Contrast venography reserved for cases with inconclusive results.
  - D-dimer assays (a cross-linked fibrin degradation product): May be useful as screening tool for DVT in stroke patients.
- Recommended prophylaxis regimens:
  - Low-dose subcutaneous (SQ) heparin/low-molecular weight heparin (LMWH).
  - Intermittent pneumatic compression (IPC) of the lower extremities for patients with a contraindication to anticoagulation treatment.
  - Gradient compression stockings in combination with SQ heparin or IPC.
Bladder Dysfunction

- Incidence of urinary incontinence is 50% to 70% during the first month after stroke and 15% after 6 months (similar to general population incidence, approximately 17%).
- Incontinence may be caused by CNS damage itself (i.e., neurogenic bladder), UTI, impaired ability to transfer to toilet, or impaired mobility, confusion, communication disorder/aphasia, and cognitive-perceptual deficits that result in lack of awareness of bladder fullness.
- Types of voiding disorders: Areflexia, uninhibited spastic bladder (with complete/incomplete emptying), outlet obstruction.
- Treatments:
  - Treat possible underlying causes (e.g., UTI)
  - Regulation of fluid intake
  - Transfer and dressing-skill training
  - Patient and family education
  - Medications (if no improvement with conservative measures)
  - Timed bladder-emptying program
  - Remove indwelling catheter and perform post-void residuals (PVRs)
  - Intermittent catheterization (IC)
  - Urodynamics evaluation

Bowel Dysfunction

- Incidence of bowel incontinence in stroke patients 31%.
- Patient unable to inhibit urge to defecate → incontinence.
- Incontinence usually resolves within the first 2 weeks; persistence may reflect severe brain damage.
- Decrease in bowel continence may be associated with infection, inability to transfer to toilet or to manage clothing, and communication impairment/inability to express toileting needs.
- Treatment: Treat underlying causes (e.g., bowel infection, diarrhea), timed-toileting schedule, training in toilet transfer and communication skills.

Impairment of Intestinal Peristalsis—Constipation

- Management: Adequate fluid intake/hydration, diet modification (e.g., increase dietary fiber), bowel management (stool softeners, stool stimulants, suppositories), allow commode/bathroom privileges.

Dysphagia

- Dysphagia (difficulty swallowing) has an overall prevalence of 25% to 65% in stroke patients (Gordon et al., 1987):
  - 67% of brainstem strokes
  - 28% of all left hemispheric strokes
  - 21% of all right hemispheric strokes
- More common in bilateral hemisphere lesions than in unilateral hemisphere lesions.
- More common in large-vessel than in small-vessel strokes.
- Delayed pharyngeal swallow is the most common cause (Veis & Logemann, 1985)

**DIAGNOSIS OF DYSPHAGIA**

- Bedside swallowing evaluation
  - A minimally invasive evaluation procedure that helps to determine whether dysphagia is present.
  - Includes a medical history review for risk.
  - May evaluate gag reflex or pharyngeal sensation.
  - Observes for overt signs of cough or other difficulty during swallowing trials.
Determine whether it is safe to feed a patient orally for purposes of nutrition, hydration, and administering medications.

- Determines whether a patient requires further swallowing assessments.
- Determines whether a patient requires referral for nutritional or hydrational support.

**Videofluorographic swallowing evaluation (VFSS):**
- Also called videofluoroscopic swallowing evaluation, modified barium swallow (MBS).
- “Gold standard” for evaluation and treatment of dysphagia.
- Different amounts and consistencies of solids and liquids mixed with barium are swallowed while visualizing the anatomy of swallowing.
- If abnormal swallowing is identified, the clinician determines the physiologic causes for swallowing breakdowns.
- Compensatory techniques, postures, maneuvers, sensory enhancements, and bolus modifications are attempted to determine methods of safe feeding.
- If no method of safe feeding can be determined, alternative methods of feeding must be considered.

**Fiberoptic endoscopic evaluation of swallowing (FEES):**
- Used as a comprehensive functional evaluation of the pharyngeal stage of swallowing.
- Visualizes anatomic structures that might cause potential bolus obstruction and natural bolus flow and containment.
- Different amounts and consistencies of solids and liquids are swallowed while visualizing the anatomy of swallowing.
- Evaluates swallowing physiology, coordination, and associated events (e.g., obstruction, absence of natural protective anatomy, depth and duration of bolus passage into the pharynx/larynx).
- Evaluates the reaction to the presence of residue, penetration, and/or aspiration (e.g., reduction in percent residue, effectiveness of cough, and expectoration of material from the airway).

**Aspiration**

- Aspiration is the entry of a substance through the vocal folds (true vocal cords) into the trachea.
- Aspiration is missed on bedside swallowing evaluations in 40% to 60% of patients (i.e., silent aspiration).
- It can be reliably diagnosed on a videofluorographic swallowing study (VFSS). Aspiration will show penetration of contrast material below the true vocal cords.
  - Aspiration has been found to occur in 40% to 70% of stroke patients on VFSS.
- Predictors of aspiration on VFSS include:
  - Delayed initiation of the swallow reflex
  - Decreased pharyngeal peristalsis
- Predictors of aspiration on bedside swallowing exam:
  - Abnormal cough
  - Cough after swallow
  - Dysphonia
  - Dysarthria
  - Abnormal gag reflex
  - Voice change after swallow (wet voice) (Aronson, 1990)

**Aspiration Pneumonia**

- Risk factors for development of pneumonia secondary to aspiration include:
  - Decreased level of consciousness
  - Tracheostomy
  - Emesis
  - Reflux
  - Nasogastric tube (NGT) feeding
  - Dysphagia
  - Prolonged pharyngeal transit time
As dysphagia is a frequent and potentially serious (because of aspiration) complication of stroke, careful bedside swallowing evaluation should be performed in all patients before oral feeding is started. If a patient is believed to be at high risk of recurrent aspiration after bedside and/or videofluorographic evaluation, the patient should be kept NPO and enterally fed, initially via NGT, and then via G- or J-tube if long-term enteral feeding is required.

### THE FOUR PHASES OF SWALLOWING (TABLES 1–9 TO 1–12)

1. Oral preparatory phase
2. Oral phase
3. Pharyngeal phase
4. Esophageal phase

#### TABLE 1–9 Oral Preparatory Phase

<table>
<thead>
<tr>
<th>Voluntary vs. reflex</th>
<th>• Voluntary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase duration</td>
<td>• Variable: voluntary phase with duration influenced by consistency of material ingested, number of times person chews, etc.</td>
</tr>
<tr>
<td>Hallmarks of this phase</td>
<td>• Preparation of bolus</td>
</tr>
</tbody>
</table>
| Phase requires       | • Tension in the labial and buccal musculature to close the anterior and lateral sulci.  
• Rotary jaw (circular) motion for mastication/grinding.  
• Lateral tongue movement to position food on the teeth during mastication (tongue moves food back to teeth).  
• Depression and forward movement of the soft palate to seal the oral cavity posteriorly and widen the nasal airway.  
• Saliva. |
| Problems seen in this phase | • Drooling  
• Pocketing |

#### TABLE 1–10 Oral Phase

<table>
<thead>
<tr>
<th>Voluntary vs. reflex</th>
<th>• Voluntary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase duration</td>
<td>• Lasts usually less than 1 second</td>
</tr>
</tbody>
</table>
| Hallmarks of this phase | • Tongue that elevates and occludes the anterior oral cavity and compresses the bolus toward the oropharynx  
• Contraction of the palatopharyngeal folds  
• Elevation of the soft palate |
| Phase requires       | • Tension in the labial and buccal musculature to close the anterior and lateral sulci.  
• Anterior-posterior tongue movement to transport the bolus to the pharynx.  
• Soft palate elevation and velopharyngeal port closure (also seen in the pharyngeal phase)—to close off the nasal cavity and prevent regurgitation into the nasopharynx. |
| Problems seen in this phase | • Drooling  
• Pocketing  
• Head tilt |
TABLE 1–11 Pharyngeal Phase

<table>
<thead>
<tr>
<th>Voluntary vs. reflex</th>
<th>• Reflex</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase duration</td>
<td>• Lasts ~ 0.6 to 1 sec</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hallmarks of this phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Bolus propelled from mouth to esophagus</td>
</tr>
<tr>
<td>• Aspiration most likely to occur during this phase</td>
</tr>
<tr>
<td>• With initiation of pharyngeal phase, inhibition of breathing occurs to prevent aspiration</td>
</tr>
<tr>
<td>• Soft palate elevation and velopharyngeal port closure (also seen in the oral phase)—to close off the nasal cavity and prevent regurgitation into the nasopharynx.</td>
</tr>
<tr>
<td>• Laryngeal elevation, with forward movement of the hyoid bone and folding of the epiglottis to protect the airway.</td>
</tr>
<tr>
<td>• Adduction of the ventricular and true vocal folds to protect the airway.</td>
</tr>
<tr>
<td>• Coordinated pharyngeal constriction and cricopharyngeal (upper esophageal sphincter) relaxation—to facilitate bolus transport into the esophagus.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Problems seen in this phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Food sticking, choking and coughing, aspiration, wet/gurgling voice, nasal regurgitation</td>
</tr>
</tbody>
</table>

TABLE 1–12 Esophageal Phase

<table>
<thead>
<tr>
<th>Voluntary vs. reflex</th>
<th>• Reflex</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase duration</td>
<td>• Longest phase—lasts 6 to 10 sec</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hallmarks of this phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Bolus pass from pharynx → esophagus → stomach</td>
</tr>
<tr>
<td>• Esophageal clearance is assisted by gravity but requires relaxation of the gastroesophageal sphincter</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Phase requires</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Cricopharyngeal muscle contraction</td>
</tr>
<tr>
<td>• Coordinated peristalsis and LES relaxation</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Problems seen in this phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Heartburn, food sticking</td>
</tr>
</tbody>
</table>

Treatment of Dysphagia/Prevention of Aspiration

• Oral feeding:
  – Modifications of diet consistency (thickened fluids, pureed or soft foods in smaller boluses) if the patient is able to tolerate PO without evidence of aspiration.

• Non-oral feeding (NPO):
  – Clear contraindication for oral feeding is pulmonary pathology due to aspiration in the presence of documented airway contamination.
  – NPO indicated in patients at high risk of aspiration because of reduced alertness, reduced responsiveness to stimulation, absent swallow, absent protective cough, and difficulty handling secretions, or when there is significant reduction of oral pharyngeal and laryngeal movements.
  – NPO is disadvantageous in treating dysphagia because swallowing itself is the best treatment.
Changes in posture and head position.
- Elevation of the head of the bed.
- Feeding in the upright position.

**Compensatory strategies:**
- **Chin tuck:** Provides airway protection by preventing entry of liquid into the larynx, probably by facilitating forward motion of the larynx. Chin tuck also decreases the space between the base of the tongue and the posterior pharyngeal wall, and so creates increased pharyngeal pressure to move the bolus through the pharyngeal region.
- **Head rotation:** Closes ipsilateral pharynx, forces bolus into contralateral pharynx, and decreases cricopharyngeal pressures. Turn the head to the paretic side.
- **Head tilt:** Uses gravity to guide bolus into ipsilateral pharynx.
- **Supraglottic swallow:** Concomitant breath holding and swallowing closes the vocal folds to protect the trachea.
- **Super supraglottic swallow:** Adds Valsalva maneuver to maximize vocal fold closing.
- **Mendelsohn maneuver:** Patient voluntarily holds the larynx at its maximal height to lengthen the duration of the cricopharyngeal opening.

Other treatment modalities (inconclusive evidence of long-term efficacy in dysphagia):
- Thermal stimulation (to sensitize the swallowing reflex)
- Oral/motor exercises (to improve tongue and lip strength, ROM, velocity, and precision, and vocal fold adduction)

**Complications of Dysphagia**
- Dehydration.
- Malnutrition found in 49% of patients admitted to rehabilitation in recent study and was associated with a prolonged length of stay and slower rate of functional gains.
- Malnourished patients also found to have higher stress reaction and higher frequency of infection and decubitus ulcers.

**Recovery of Dysphagia in Stroke**
- Few studies available on recovery of dysphagia in stroke.
- **Ickenstein et al. (2012):** Subjects 72 hours post-stroke rated at a level 1 to 3 on the Functional Communication Measure (FCM) of Swallowing and level 5 to 8 on the Penetration-Aspiration Scale (PAS) were 11.8 times less likely to be orally fed 90 days post-stroke.
  - Subjects 7 days post-stroke who tolerated grade 1 fluids or thinner, or who tolerated a modified soft diet or better: none needed a PEG.
  - Subjects needing PEG 14 days post-stroke: 50% could not tolerate grade 3 thickened fluids; 52% could not tolerate a pureed diet.
  - No subject intolerant of grade 2 thickened fluids 7 or 14 days post-stroke could tolerate a normal diet and fluids by day 28.
  - Of subjects tolerating grade 3 thickened fluids at day 7, 36% tolerated a normal diet at day 28.
  - PEG placement should be considered in people unable to tolerate grade 3 thickened fluids or pureed diet 14 days post-stroke. However, even in these groups, half recovered sufficiently to manage oral feeding.
- Gresham (1990) reports findings regarding 53 patients in a swallowing program post-stroke:
  - 85% on full oral nutrition at discharge.
  - 17% could not drink thin liquids safely.
  - 8% could not adequately maintain cohesive bolus of varied texture.
• **Nasal speech:** Hypernasality caused by partial or complete failure of soft palate to close-off the nasal cavity from the oral cavity or by incomplete closure of the hard palate. Uplifting the soft palate prevents nasal speech (speech abnormally resonated in the nasal cavities).

**APHASIA (TABLE 1–13)**

• Aphasia is an impairment of the ability to utilize language due to an injury to the brain. It is characterized by paraphasias, word-finding difficulties, and impaired comprehension. Other common but not obligatory features are disturbances in reading and writing, nonverbal constructional and problem-solving difficulty, and impairment of gesturing.

**TABLE 1–13 Types of Aphasias**

<table>
<thead>
<tr>
<th>FLUENT APHASIA</th>
<th>NONFLUENT APHASIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>+ COMPREHENSION</td>
<td>- COMPREHENSION</td>
</tr>
<tr>
<td>REPETITION</td>
<td>REPETITION</td>
</tr>
<tr>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Conduction</td>
<td>Transcortical Sensory</td>
</tr>
<tr>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Wernicke’s</td>
<td>Transcortical Motor</td>
</tr>
<tr>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Broca’s</td>
<td>Mixed Transcortical</td>
</tr>
<tr>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Normal</td>
<td>Global</td>
</tr>
</tbody>
</table>

---

**Fluent Aphasia**
- Wernicke’s
- Transcortical sensory
- Conduction
- Anomia

**Nonfluent Aphasia**
- Broca’s
- Transcortical motor
- Global
- Mixed transcortical
1. **STROKE**

**ANATOMIC LOCATION OF MAJOR SPEECH AREAS**

- **Location:** posterior part of superior (first) temporal gyrus of the dominant (usually left) hemisphere
  - **Characteristics:**
    - Fluent speech (normal rate/speed)
    - Impaired comprehension
    - Word deafness, difficulty in reading (alexia) and writing (agraphia)
    - Marked paraphasias & neologisms

- **Wernicke’s aphasia**

- **Location:** posterior-inferior frontal lobe (third frontal convolution) of dominant (usually left) hemisphere → anterior to motor cortex areas that supply the tongue, lips and larynx
  - **Characteristics:**
    - Nonfluent speech (telegraphic)
    - Impaired repetition
    - Preserved comprehension
    - Paraphasias & articulatory errors or struggle

- **Broca’s aphasia** (remember “broken” speech)

- **Location:** vary in size and location but usually involve distribution of the left MCA (entire perisylvian region)
  - **Characteristics:**
    - Ranges from mutism (non-fluent) to total repetitive jargon or neologistic output (fluent but incomprehensible speech)
    - Poor comprehension and repetition

- **Global aphasia**
1. STROKE

**Anomic aphasia**

**Location:** temporo-parietal injury, angular gyrus  
**Characteristics:**  
- Fluent, essentially good comprehension and repetition  
- Decreased output of nouns  
- Word-finding difficulties  
- Alexia and agraphia may be present

**Conduction aphasia**

**Location:** lesion of the parietal operculum (arcuate fasciculus) or insula or deep to the supramarginal gyrus (usually left side)  
**Characteristics:**  
- Normal rate of speech (fluent)  
- Preserved comprehension  
- Impaired repetition  
- Literal paraphasias with “targeting” of words (until getting the right one)

**Note:** arcuate fasciculus is a band of white matter running deep to the supramarginal gyrus and insula that joins Broca’s and Wernicke’s areas

**Transcortical motor aphasia**

**Location:** frontal lobe, anterior or superior to Broca’s area or in the subcortical region deep to Broca’s area  
**Characteristics:**  
- Reduced rate of speech, limited language output (with some fluent utterances)  
- Reduced initiation & organization of speech  
- Good comprehension  
- Preserved repetition

**Transcortical sensory aphasia**

**Location:** watershed lesion isolating perisylvian speech structures (Broca’s and Wernicke’s areas) from the posterior brain; angular gyrus or postero-inferior temporal lobe  
**Characteristics:**  
- Poor comprehension  
- Fluent speech (neologisms)  
- Preserved repetition (possibly echolalia)
Transcortical Mixed Aphasia

- Also known as isolation aphasia.
- Lesions in border zone of frontal, parietal, and temporal areas.
- Characteristics:
  - Poor comprehension
  - Nonfluent (decrease rate and initiation of speech)
  - Preserved repetition (echolalia)

Note: Language areas are anatomically clustered around the sylvian fissure of the dominant hemisphere-left hemisphere in 95% of people.

Other Errors in Speech

- Paraphasia: Incorrect substitution of words or parts of words.
  - Literal or phonemic paraphasias: Similar sounds (e.g., “sound” for “found”).
  - Verbal or semantic paraphasias: Word substituted for another from same semantic class (e.g., “fork” for “spoon”).
- Agrammatism: Aphasia in which there is absence of grammatical structure in a sentence.
- Anomia (anomic or nominal aphasia): Difficulty recalling words; word-finding difficulty. Unimpaired comprehension and repetition.
- Echolalia: Repetition (“echoing”) of words or vocalizations made by another person.
- Circumlocution: Roundabout way of describing a word that cannot be recalled. Often seen in conjunction with anomia.
- Neologism: A “new word” that is well articulated but has meaning only to the speaker.
- Jargon: Well articulated but mostly incomprehensible, unintelligible speech. Associated with Wernicke’s aphasia.
- Stereotype: Repetition of nonsensical syllables (e.g., “no, no, no”) during attempts at communication.

Treatment Approaches to Aphasias

- Loss versus interference (most widely accepted approach): Views aphasia as loss of specific linguistic information (compensatory) with concept that brain damage interferes with linguistic operation (facilitory).
- Direct versus indirect: Views aphasia as deficit of linguistic thought processes, which can be used to differentiate direct language-centered therapy from indirect content-centered therapy.
- Behavioral versus psycholinguistic: Views emphasis on content, emphasis on structure of therapy.
- Programmed operant: Measures obtained before and after behavior-modification procedure applied.
- Programmed instruction: Involves many individual steps to reach a desired language behavior.
- Specific interventions:
  - AmerInd: Sign language that uses gestures that represents objects, actions, directions, and descriptions.
  - Am Melodic INTONATION THERAPY (MIT): Recruits the right hemisphere for communication by incorporating melodies or rhythms with simple statements. MIT may be useful in patients with nonfluent (Broca’s) aphasia.

Post-Stroke Aphasia Recovery

- The greatest amount of improvement in patients with aphasia occurs in the first 2 to 3 months after the onset.
- After 6 months, there is a significant drop in the rate of recovery.
- In the majority of cases of patients with aphasia, spontaneous recovery does not seem to occur after 1 year.
• However, there are reports of improvements many years after stroke in patients undergoing therapy.

**MANAGEMENT OF MEDICAL ISSUES**

**Post-Stroke Depression**

• **Etiology:**
  − Organic: May be related to catecholamine depletion through lesion-induced damage to the frontal noradrenergic, dopaminergic, and serotonergic projections (Heilman et al., 2012).
  − Reactive: Grief/psychological responses for physical and personal losses associated with stroke, loss of control that often accompany severe disability, etc.
• Prevalence of depression in stroke patients reported approximately 40% (25% to 79%); occur in similar proportion in their caregivers (Flick, 1999).
• Most prevalent 6 months to 2 years.
• A psychiatric evaluation for DSM-IV criteria and vegetative signs may be a clinically useful diagnostic tool in stroke patients.
• There may be a higher risk for major depression in left frontal lesions (relationship still controversial).
• Risk factors: Prior psychiatric history, significant impairment in ADLs, high severity of deficits, female gender, nonfluent aphasia, cognitive impairment, and lack of social supports.
• Persistent depression correlates with delayed recovery and poorer outcome.
• Treatment: Active treatment should be considered for all patients with significant clinical depression.
• Psychosocial interventional program: Psychotherapy.
• Selective serotonin reuptake inhibitors (SSRIs) preferred because of fewer side effects compared to TCAs; methylphenidate has also been shown to be effective in post-stroke depression.
• SSRIs and TCAs also have been shown to be effective in post-stroke emotional lability.

**Sexual Dysfunction**

• Well documented that the majority of elderly people continue to enjoy active and satisfying sexual relationships.
• No significant changes in sexual interest or desire, but marked decline in behavior in both sexes after a stroke.
• There is a marked decline in sexual activity post-stroke.
• Korpelainen et al. (1999): 192 stroke patients and 94 spouses.
  − 79% of patients and 84% of spouses reported active pre-stroke sexual life, including intercourse at least once a month.
  − After the stroke, however, 45% of patients and 48% of spouses with an active sexual life had markedly decreased.
  − 33% of patients and 27% of spouses reported having ceased sexual intercourse.
• Decreased coital frequency associated with inability to discuss sexuality with spouse (odds ratio [OR] 18.5); general attitude toward sexuality (unimportant: OR 7.7; fairly important: OR 9.2); unwillingness to participate in sexual activity (OR 5.4).
• Other factors related to decrease in sexual activity post-stroke:
  − Emotional factors: Fear, anxiety, and guilt; low self-esteem; and fear of rejection by partner.
  − Medications that worsen sexual function: TCAs, SSRIs, antipsychotics, anticholinergics, histamine (1 and 2) blockers, GABA agonists (e.g., pregabalin), opioid narcotics, saw palmetto.
  − Treatment: Supportive psychotherapy, counseling, medical consultation (e.g., urology, urogynecology) for organic causes.

**Seizures**

• Can be classified as occurring:
  − At stroke onset
  − Early after stroke (1 to 2 weeks)
  − Late after stroke (>2 weeks)
• In prospective study after first-time stroke, 2.5% of patients had seizures within 48 hours post-ictus.
• Seizures associated with older age, confusion, and large parietal or temporal hemorrhages.
• Majority of seizures were generalized, tonic-clonic.
• In-hospital mortality rates are higher in patients with seizures.
• Early seizures tend not to recur. These are associated with acute metabolic derangement associated with ischemia or hemorrhage.
• Stroke patients requiring inpatient rehabilitation have a higher probability of developing seizures than the general stroke population.
• Seizures occurring >2 weeks after stroke have higher probability of recurrence.
• In a study with 77 ischemic stroke victims followed 2 to 4 years:
  – 6% to 9% developed seizures.
  – 26% patients with cortical lesions developed seizures.
  – 2% patients with subcortical lesions developed seizures.
• Risk factors: Cortical lesions, persistent paresis (50%).
• Treatment: Choice of anticonvulsant drugs for patients with cerebral injury discussed in the TBI chapter.

FACTORS THAT PREDICT MORTALITY AND FUNCTIONAL RECOVERY IN STROKE PATIENTS

Mortality Risk Factors
• In persons aged 45 to 64: 8% to 12% of ischemic strokes and 37% to 38% of hemorrhagic strokes die within 30 days (ARIC study, NHLBI).
• In persons age 65 and older: 12.6% of all strokes, 8.1% of ischemic strokes, and 44.6% of hemorrhagic strokes die within 1 month (Medicare Part B random sample).
• Mortality in the first year after stroke: 25% to 40%.
• Risk of another stroke within the first year: 12% to 25%.

RISK FACTORS FOR ACUTE STROKE MORTALITY—30 DAY MORTALITY
• Stroke severity
• Decreased level of consciousness
• Diabetes mellitus
• Cardiac disease
• EKG abnormalities
• Old age
• Delay in medical care
• Elevated blood sugar in nondiabetics
• Brainstem involvement
• Hemorrhagic stroke
• Admission from nursing home

Risk Factors for Disability After Stroke (Kelley-Hayes et al., 2003)
• As stroke mortality has declined in the last few decades, the number of stroke survivors with impairments and disabilities has increased.
• There are 300,000 to 400,000 stroke survivors annually.
• 50% had some hemiparesis.
• 30% were unable to walk without some assistance.
• 26% were dependent in ADLs.
• 19% had aphasia.
• 35% had depressive symptoms.
• 26% were institutionalized in a nursing home.

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RISK FACTORS FOR DISABILITY AFTER STROKE

- Severe stroke (minimal motor recovery at 4 weeks)
- Decreased level of consciousness
- Diabetes mellitus
- Cardiac disease
- EKG abnormalities
- Old age
- Delay in medical care
- Delay in rehabilitation
- Bilateral lesions
- Previous stroke
- Previous functional disability
- Poor sitting balance
- Global aphasia
- Severe neglect
- Sensory and visual deficits
- Impaired cognition
- Incontinence persisting >1 to 2 weeks

NEGATIVE RISK FACTORS FOR RETURN TO WORK POST-STROKE

(Black-Schaffer & Osberg, 1990)

- Low score on Barthel index at time of rehabilitation discharge:
  - Barthel index: Functional assessment tool that measures independence in ADLs on 0 to 100 scale.
- Prolonged rehabilitation length of stay
- Aphasia
- Prior alcohol abuse
# THE BARTHEL INDEX

<table>
<thead>
<tr>
<th>Activity</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FEEDING</strong></td>
<td></td>
</tr>
<tr>
<td>0 = unable</td>
<td></td>
</tr>
<tr>
<td>5 = needs help cutting, spreading butter, etc., or requires modified diet</td>
<td></td>
</tr>
<tr>
<td>10 = independent</td>
<td></td>
</tr>
<tr>
<td><strong>BATHING</strong></td>
<td></td>
</tr>
<tr>
<td>0 = dependent</td>
<td></td>
</tr>
<tr>
<td>5 = independent (or in shower)</td>
<td></td>
</tr>
<tr>
<td><strong>GROOMING</strong></td>
<td></td>
</tr>
<tr>
<td>0 = needs to help with personal care</td>
<td></td>
</tr>
<tr>
<td>5 = independent face/hair/teeth/shaving (implements provided)</td>
<td></td>
</tr>
<tr>
<td><strong>DRESSING</strong></td>
<td></td>
</tr>
<tr>
<td>0 = dependent</td>
<td></td>
</tr>
<tr>
<td>5 = needs help but can do about half unaided</td>
<td></td>
</tr>
<tr>
<td>10 = independent (including buttons, zips, laces, etc.)</td>
<td></td>
</tr>
<tr>
<td><strong>BOWELS</strong></td>
<td></td>
</tr>
<tr>
<td>0 = incontinent (or needs to be given enemas)</td>
<td></td>
</tr>
<tr>
<td>5 = occasional accident</td>
<td></td>
</tr>
<tr>
<td>10 = continent</td>
<td></td>
</tr>
<tr>
<td><strong>BLADDER</strong></td>
<td></td>
</tr>
<tr>
<td>0 = incontinent, or catheterized and unable to manage alone</td>
<td></td>
</tr>
<tr>
<td>5 = occasional accident</td>
<td></td>
</tr>
<tr>
<td>10 = continent</td>
<td></td>
</tr>
<tr>
<td><strong>TOILET USE</strong></td>
<td></td>
</tr>
<tr>
<td>0 = dependent</td>
<td></td>
</tr>
<tr>
<td>5 = needs some help, but can do something alone</td>
<td></td>
</tr>
<tr>
<td>10 = independent (on and off, dressing, wiping)</td>
<td></td>
</tr>
<tr>
<td><strong>TRANSFERS (BED TO CHAIR AND BACK)</strong></td>
<td></td>
</tr>
<tr>
<td>0 = unable, no sitting balance</td>
<td></td>
</tr>
<tr>
<td>5 = major help (one or two people, physical), can sit</td>
<td></td>
</tr>
<tr>
<td>10 = minor help (verbal or physical)</td>
<td></td>
</tr>
<tr>
<td>15 = independent</td>
<td></td>
</tr>
<tr>
<td><strong>MOBILITY (ON LEVEL SURFACES)</strong></td>
<td></td>
</tr>
<tr>
<td>0 = immobile or &lt;50 yards</td>
<td></td>
</tr>
<tr>
<td>5 = wheelchair independent, including corners, &gt;50 yards</td>
<td></td>
</tr>
<tr>
<td>10 = walks with help of one person (verbal or physical) &gt;50 yards</td>
<td></td>
</tr>
<tr>
<td>15 = independent (but may use any aid; for example, stick) &gt;50 yards</td>
<td></td>
</tr>
<tr>
<td><strong>STAIRS</strong></td>
<td></td>
</tr>
<tr>
<td>0 = unable</td>
<td></td>
</tr>
<tr>
<td>5 = needs help (verbal, physical, carrying aid)</td>
<td></td>
</tr>
<tr>
<td>10 = independent</td>
<td></td>
</tr>
<tr>
<td><strong>TOTAL (0–100):</strong></td>
<td></td>
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</table>
The Barthel ADL Index: Guidelines

1. The index should be used as a record of what a patient does, not as a record of what a patient could do.
2. The main aim is to establish degree of independence from any help, physical or verbal, however minor and for whatever reason.
3. The need for supervision renders the patient not independent.
4. A patient’s performance should be established using the best available evidence. Asking the patient, friends/relatives and nurses are the usual sources, but direct observation and common sense are also important. However direct testing is not needed.
5. Usually the patient’s performance over the preceding 24–48 hours is important, but occasionally longer periods will be relevant.
6. Middle categories imply that the patient supplies over 50 per cent of the effort.
7. Use of aids to be independent is allowed.

Source: From Mahoney & Barthel, 1965, with permission.

REFERENCES


RECOMMENDED READING


