RULE 17 EXHIBIT 10

Traumatic Brain Injury

Medical Treatment Guidelines

Revised: November 26, 2012

Effective: January 14, 2013

Revised: September 29, 2005 Effective: January 1, 2006

Revised: January 8, 1998 Effective: March 15, 1998

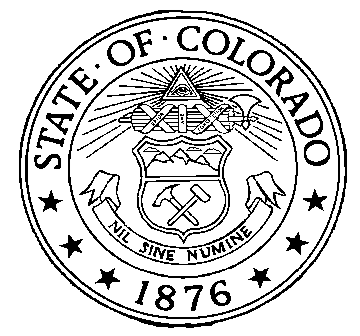
Revised: March 1, 2005 Effective: May 1, 2005

*Presented by:*

State of Colorado

Department of Labor and Employment

DIVISION OF WORKERS’ COMPENSATION



**tABLE OF CONTENTS**

|  |  |  |
| --- | --- | --- |
| **sECTION** | **DESCRIPTION** | **PAGE** |

[a. INTRODUCTION 1](#_Toc394643161)

[b. GENERAL GUIDELINE PRINCIPLES 2](#_Toc394643162)

[1. APPLICATION OF GUIDELINES 2](#_Toc394643163)

[2. EDUCATION 2](#_Toc394643164)

[3. TREATMENT PARAMETER DURATION 2](#_Toc394643165)

[4. ACTIVE INTERVENTIONS 2](#_Toc394643166)

[5. ACTIVE THERAPEUTIC EXERCISE PROGRAM 2](#_Toc394643167)

[6. POSITIVE PATIENT RESPONSE 2](#_Toc394643168)

[7. RE-EVALUATE TREATMENT EVERY THREE TO FOUR WEEKS 2](#_Toc394643169)

[8. SURGICAL INTERVENTIONS 3](#_Toc394643170)

[9. RETURN TO WORK: 3](#_Toc394643171)

[10. DELAYED RECOVERY 3](#_Toc394643172)

[11. GUIDELINE RECOMMENDATIONS AND INCLUSION OF MEDICAL EVIDENCE 3](#_Toc394643173)

[12. PoST MAXIMUM MEDICAL IMPROVEMENT (MMI) CARE 4](#_Toc394643174)

[c. INTRODUCTION TO TRAUMATIC BRAIN INJURY AND PHILOSOPHY OF CARE 5](#_Toc394643175)

[1. DEFINITIONS AND DIAGNOSIS OF TRAUMATIC BRAIN INJURY 5](#_Toc394643176)

[a. Mild TBI (MTBI) 5](#_Toc394643177)

[b. Moderate/Severe TBI 5](#_Toc394643178)

[c. Other Terminology 6](#_Toc394643179)

[2. INTERVENTION 6](#_Toc394643180)

[3. EDUCATION 6](#_Toc394643181)

[4. RETURN TO WORK 7](#_Toc394643182)

[5. DISABILITY 7](#_Toc394643183)

[6. COURSE OF RECOVERY 8](#_Toc394643184)

[a. MTBI 8](#_Toc394643185)

[b. Moderate/Severe TBI 8](#_Toc394643186)

[7. GUARDIANSHIP AND CONSERVATORSHIP 8](#_Toc394643187)

[8. SYSTEMS OF CARE 9](#_Toc394643188)

[a. Acute Care 10](#_Toc394643189)

[b. Comprehensive Integrated Inpatient Rehabilitation Hospital or “Acute Rehabilitation”: 11](#_Toc394643190)

[c. Long-Term Acute Care (LTAC) Programs 11](#_Toc394643191)

[d. Sub-Acute Skilled Nursing Facility (SNF) Rehabilitation Programs 11](#_Toc394643192)

[e. Post-Acute Rehabilitation 12](#_Toc394643193)

[f. Long-Term Support Care 12](#_Toc394643194)

[9. INTERDISCIPLINARY TREATMENT TEAM 12](#_Toc394643195)

[a. Behavioral Psychologist 13](#_Toc394643196)

[b. Behavioral Analyst 13](#_Toc394643197)

[c. Case Manager 13](#_Toc394643198)

[d. Chiropractor 13](#_Toc394643199)

[e. Clinical Pharmacist 14](#_Toc394643200)

[f. Clinical Psychologist 14](#_Toc394643201)

[g. Driver Rehabilitation Specialist 14](#_Toc394643202)

[h. Independent Life Skills Trainer 14](#_Toc394643203)

[i. Music Therapist 14](#_Toc394643204)

[j. Neurologist 14](#_Toc394643205)

[k. Neuro-ophthalmologist 14](#_Toc394643206)

[l. Neuro-otologist 14](#_Toc394643207)

[m. Neuropsychologist 14](#_Toc394643208)

[n. Neuroscience Nurse 14](#_Toc394643209)

[o. Neurosurgeon (Neurological Surgeon) 14](#_Toc394643210)

[p. Nurse 15](#_Toc394643211)

[q. Occupational Therapist 15](#_Toc394643212)

[r. Occupational Medicine Physician 15](#_Toc394643213)

[s. Optometrist 15](#_Toc394643214)

[t. Ophthalmologist 15](#_Toc394643215)

[u. Otolaryngologist 15](#_Toc394643216)

[v. Physical Therapist 15](#_Toc394643217)

[w. Physiatrist 15](#_Toc394643218)

[x. Psychiatrist/Neuropsychiatrist 15](#_Toc394643219)

[y. Rehabilitation Counselor 15](#_Toc394643220)

[z. Rehabilitation Nurse 16](#_Toc394643221)

[aa. Social Worker 16](#_Toc394643222)

[bb. Speech-Language Pathologist 16](#_Toc394643223)

[cc. Therapeutic Recreation Specialist 16](#_Toc394643224)

[10. PREVENTION 16](#_Toc394643225)

[a. Primary Prevention 16](#_Toc394643226)

[b. Secondary Prevention 17](#_Toc394643227)

[c. Tertiary Prevention 18](#_Toc394643228)

[d. INITIAL DIAGNOSTIC PROCEDURES 19](#_Toc394643229)

[1. HISTORY OF INJURY 19](#_Toc394643230)

[a. Identification Data 19](#_Toc394643231)

[b. Precipitating Event 19](#_Toc394643232)

[c. Neurological History 19](#_Toc394643233)

[d. Review of Medical Records 20](#_Toc394643234)

[e. Medical/Health History 20](#_Toc394643235)

[f. Activities of Daily Living (ADLs): 20](#_Toc394643236)

[g. Family History 21](#_Toc394643237)

[h. Social History 21](#_Toc394643238)

[i. Review of Systems 21](#_Toc394643239)

[j. Pain Diagnosis 21](#_Toc394643240)

[k. Psychiatric History 21](#_Toc394643241)

[2. PHYSICAL EXAMINATION 21](#_Toc394643242)

[3. NEUROLOGICAL EXAMINATION 21](#_Toc394643243)

[4. INITIAL NEUROPSYCHOLOGICAL ASSESSMENT 22](#_Toc394643244)

[a. Initial Neuropsychological Assessment – MTBI 22](#_Toc394643245)

[b. Initial Neuropsychological Assessment – Moderate/Severe Traumatic Brain Injury 23](#_Toc394643246)

[c. Post-Acute Testing 24](#_Toc394643247)

[5. Initial IMAGING PROCEDURES 25](#_Toc394643248)

[a. Skull X-Rays 25](#_Toc394643249)

[b. Computed Axial Tomography (CT) 25](#_Toc394643250)

[c. Magnetic Resonance Imaging (MRI) 26](#_Toc394643251)

[6. VASCULAR IMAGING TESTS 26](#_Toc394643252)

[a. CT Angiography (CTA): 26](#_Toc394643253)

[b. Arteriography 27](#_Toc394643254)

[c. Venography 27](#_Toc394643255)

[d. Noninvasive Vascular Assessment (NIVA) 27](#_Toc394643256)

[e. Magnetic Resonance Angiography (Magnetic Resonance Arteriography (MRA)/Magnetic Resonance Venography (MVA)) 27](#_Toc394643257)

[f. Brain Acoustic Monitor 27](#_Toc394643258)

[7. LUMBAR PUNCTURE 27](#_Toc394643259)

[e. FOLLOW-UP DIAGNOSTIC PROCEDURES 28](#_Toc394643260)

[1. IMAGING 28](#_Toc394643261)

[a. Structural Imaging 28](#_Toc394643262)

[b. Dynamic Imaging 28](#_Toc394643263)

[2. ADVANCED MRI TECHNIQUES 29](#_Toc394643264)

[a. Magnetic Resonance (MR) Spectroscopy 29](#_Toc394643265)

[b. Functional MRI (fMRI) 29](#_Toc394643266)

[c. Diffusion Tensor Imaging, Susceptibility—Weighted Imaging and Magnetic Transfer Imaging 30](#_Toc394643267)

[3. NEUROPSYCHOLOGICAL ASSESSMENT 30](#_Toc394643268)

[a. Mild Traumatic Brain Injury 30](#_Toc394643269)

[b. Moderate/Severe TBI 31](#_Toc394643270)

[4. PERSONALITY/PSYCHOLOGICAL/PSYCHOSOCIAL EVALUATIONS 31](#_Toc394643271)

[a. Qualifications 32](#_Toc394643272)

[b. Indications 32](#_Toc394643273)

[c. Clinical Evaluation 32](#_Toc394643274)

[5. ELECTROENCEPHALOGRAPHY 34](#_Toc394643275)

[a. Electroencephalography (EEG) 34](#_Toc394643276)

[b. Quantified Electroencephalography (QEEG) (Computerized EEG) 35](#_Toc394643277)

[6. ELECTRODIAGNOSTIC STUDIES 35](#_Toc394643278)

[a. EMG and Nerve Conduction Studies 35](#_Toc394643279)

[b. Electroneuronography (EnoG) 35](#_Toc394643280)

[c. Dynamic Electromyographies 35](#_Toc394643281)

[d. Evoked Potential Responses (EP) 35](#_Toc394643282)

[7. LABORATORY TESTING 36](#_Toc394643283)

[8. NERVE BLOCKS – Diagnostic 37](#_Toc394643284)

[9. VISION EVALUATION 37](#_Toc394643285)

[a. Visual Field Testing 38](#_Toc394643286)

[b. Ultrasonography 38](#_Toc394643287)

[c. Fluorescein Angiography 38](#_Toc394643288)

[d. Visual Perceptual Testing 38](#_Toc394643289)

[e. Low Vision Evaluation 38](#_Toc394643290)

[f. Electrodiagnostic Studies 38](#_Toc394643291)

[g. Optical Coherence Tomography 39](#_Toc394643292)

[10. OTOLOGY and AUDIOMETRY 39](#_Toc394643293)

[a. Audiometry 39](#_Toc394643294)

[b. Tympanometry 39](#_Toc394643295)

[c. Vestibular Function Tests 39](#_Toc394643296)

[11. SWALLOWING EVALUATION 41](#_Toc394643297)

[a. Clinical Assessment 41](#_Toc394643298)

[b. Instrumental Evaluation 41](#_Toc394643299)

[12. SPECIAL TESTS for RETURN-TO-WORK ASSESSMENT 42](#_Toc394643300)

[a. Job Site Evaluations and Alterations 43](#_Toc394643301)

[b. Functional Capacity Evaluation (FCE) 44](#_Toc394643302)

[f. ACUTE THERAPEUTIC PROCEDURES – NONOPERATIVE 46](#_Toc394643303)

[1. RESUSCITATION 46](#_Toc394643304)

[2. INTRACRANIAL PRESSURE (ICP) AND CEREBRAL PERFUSION PRESSURE (CPP) 46](#_Toc394643305)

[3. HYPERVENTILATION 46](#_Toc394643306)

[4. MEDICATIONS 46](#_Toc394643307)

[5. HYPOTHERMIA 47](#_Toc394643308)

[6. Surgery 47](#_Toc394643309)

[7. Hyperbaric Oxygen 47](#_Toc394643310)

[g. NONOPERATIVE THERAPEUTIC PROCEDURES – INITIAL TREATMENT CONSIDERATIONS 49](#_Toc394643311)

[1. PATIENT/FAMILY/SUPPORT SYSTEM EDUCATION 49](#_Toc394643312)

[a. MTBI 49](#_Toc394643313)

[b. Moderate/Severe TBI 49](#_Toc394643314)

[2. BEHAVIOR 50](#_Toc394643315)

[3. COGNITION 51](#_Toc394643316)

[a. MTBI 52](#_Toc394643317)

[b. Moderate/Severe TBI 52](#_Toc394643318)

[c. Computer-Based Treatment 54](#_Toc394643319)

[d. Assistive Technology 54](#_Toc394643320)

[4. PSYCHOLOGICAL/EDUCATIONAL INTERVENTIONS: 54](#_Toc394643321)

[a. Acute Psychological/Educational Interventions in MTBI 54](#_Toc394643322)

[b. Problem-Specific Referrals During the First Three Months Following MTBI 55](#_Toc394643323)

[c. Referrals Three or More Months Post-MTBI 55](#_Toc394643324)

[d. Functional Gains 56](#_Toc394643325)

[5. PSYCHOLOGICAL INTERVENTIONS – MODERATE/SEVERE TBI 56](#_Toc394643326)

[a. Acutely Symptomatic Phase 56](#_Toc394643327)

[b. Early Recovery Phase 56](#_Toc394643328)

[c. Stabilization Phase 57](#_Toc394643329)

[d. Consultation in Regard to Usage of Medications 57](#_Toc394643330)

[6. MEDICATION/Pharmacological Rehabilitation 57](#_Toc394643331)

[a. Affective Disorders Medications 59](#_Toc394643332)

[b. Behavior/ Aggression Medications 60](#_Toc394643333)

[c. Cognitive Enhancers 60](#_Toc394643334)

[7. HEADACHE: 67](#_Toc394643335)

[a. Headache Treatment Algorithm 69](#_Toc394643336)

[b. Botulinum Injections: 70](#_Toc394643337)

[8. THERAPEUTIC EXERCISE 70](#_Toc394643338)

[9. DISTURBANCES OF SLEEP: 70](#_Toc394643339)

[h. NONOPERATIVE THERAPEUTIC PROCEDURES – NEUROMEDICAL CONDITIONS in MODERATE/SEVERE BRAIN INJURY 72](#_Toc394643340)

[1. Neurological Complications 72](#_Toc394643341)

[2. Post-Traumatic Seizures/Post-Traumatic Epilepsy (PTE) 72](#_Toc394643342)

[3. Cardiopulmonary Complications 72](#_Toc394643343)

[a. Cardiac System 72](#_Toc394643344)

[b. Pulmonary System 72](#_Toc394643345)

[4. Sleep Complications 73](#_Toc394643346)

[5. Musculoskeletal Complications 73](#_Toc394643347)

[a. Long-Bone Fractures 73](#_Toc394643348)

[b. Heterotopic Ossification (HO) 73](#_Toc394643349)

[6. Gastrointestinal Complications 74](#_Toc394643350)

[7. Genitourinary Complications 74](#_Toc394643351)

[8. Neuroendocrine Complications 74](#_Toc394643352)

[9. Fluid and Electrolyte Complications 74](#_Toc394643353)

[10. Immobilization and Disuse Complications 75](#_Toc394643354)

[11. Vascular Complications 75](#_Toc394643355)

[i. NONOPERATIVE THERAPEUTIC PROCEDURES – REHABILITATION 76](#_Toc394643356)

[1. INTERDISCIPLINARY REHABILITATION PROGRAMS 76](#_Toc394643357)

[a. Behavioral Programs 78](#_Toc394643358)

[b. Comprehensive Integrated Inpatient Interdisciplinary Rehabilitation Programs 78](#_Toc394643359)

[c. Home and Community-Based Rehabilitation 79](#_Toc394643360)

[d. Nursing Care Facilities 80](#_Toc394643361)

[e. Occupational Rehabilitation 80](#_Toc394643362)

[f. Opioid/Chemical Treatment Programs 80](#_Toc394643363)

[g. Outpatient Rehabilitation Services 80](#_Toc394643364)

[h. Residential Rehabilitation 81](#_Toc394643365)

[i. Supported Living Programs (SLP) or Long-Term Care Residential Services 81](#_Toc394643366)

[2. ACTIVITES OF DAILY LIVING (ADLs) 82](#_Toc394643367)

[a. Basic ADLs 82](#_Toc394643368)

[b. Instrumental ADLs (IADLs) 82](#_Toc394643369)

[3. MOBILITY 83](#_Toc394643370)

[a. Therapy 83](#_Toc394643371)

[b. Adaptive Devices 84](#_Toc394643372)

[4. Ataxia 85](#_Toc394643373)

[5. NEUROMUSCULAR re-education 86](#_Toc394643374)

[a. Motor Control 86](#_Toc394643375)

[b. Motor Learning 86](#_Toc394643376)

[6. Work Conditioning 87](#_Toc394643377)

[7. Work Simulation 87](#_Toc394643378)

[8. MUSCLE TONE AND JOINT RESTRICTION MANAGEMENT, Including spasticity 87](#_Toc394643379)

[a. Orthotics and Casting 88](#_Toc394643380)

[b. Postural Control 88](#_Toc394643381)

[c. Functional and Therapeutic Activities 88](#_Toc394643382)

[d. Therapeutic Nerve and Motor Point Blocks 88](#_Toc394643383)

[e. Botulinum Toxin (Botox) Injections 88](#_Toc394643384)

[f. Pharmaceutical Agents 89](#_Toc394643385)

[g. Intrathecal Baclofen Drug Delivery 90](#_Toc394643386)

[j. NONOPERATIVE THERAPEUTIC PROCEDURES – VISION, SPEECH, SWALLOWING, BALANCE, & HEARING 92](#_Toc394643387)

[1. VISUAL TREATMENT 92](#_Toc394643388)

[a. Visual Acuity and Visual Field Function 92](#_Toc394643389)

[b. Disorders Involving Ocular Motor Control and Ocular Alignment: 93](#_Toc394643390)

[c. Visual Perception 93](#_Toc394643391)

[d. Visual Inattention 93](#_Toc394643392)

[e. Total Time Frames for all Vision Therapy (Orthoptic Therapy) 94](#_Toc394643393)

[2. NEURO-OTOLOGIC TREATMENTS 94](#_Toc394643394)

[a. Treatment of Fixed Lesions 94](#_Toc394643395)

[b. Treatment of Recurrent, Non-Progressive Otologic Disorders 95](#_Toc394643396)

[c. Treatment of Progressive Otologic Disorders 95](#_Toc394643397)

[d. In-Office Treatment Procedures 96](#_Toc394643398)

[e. Tympanostomy 96](#_Toc394643399)

[f. Vestibular Rehabilitation 96](#_Toc394643400)

[3. SWALLOWING IMPAIRMENTS (DYSPHAGIA) 99](#_Toc394643401)

[a. Compensatory Treatment 100](#_Toc394643402)

[b. Therapy Techniques 100](#_Toc394643403)

[4. COMMUNICATION 101](#_Toc394643404)

[a. Motor Speech Disorders 101](#_Toc394643405)

[b. Voice Disorders 101](#_Toc394643406)

[c. Language Disorders 101](#_Toc394643407)

[d. Cognitive-Communicative Disorders 101](#_Toc394643408)

[k. NONOPERATIVE THERAPEUTIC PROCEDURES – RETURN TO WORK, DRIVING, & OTHER 105](#_Toc394643409)

[1. DRIVING 105](#_Toc394643410)

[2. RETURN TO WORK 106](#_Toc394643411)

[a. Return to Work – MTBI 106](#_Toc394643412)

[b. Return to Work – Moderate/Severe TBI 108](#_Toc394643413)

[c. The Following Should be Considered when Attempting to Return an Injured Worker with Moderate/Severe TBI to Work 108](#_Toc394643414)

[3. VOCATIONAL REHABILITATION 109](#_Toc394643415)

[4. COMPLEMENTARY AND ALTERNATIVE MEDICINE (CAM) 109](#_Toc394643416)

[5. OTHER TREATMENTS 110](#_Toc394643417)

[a. Hyperbaric Oxygen 110](#_Toc394643418)

[b. Deep Thalamic Stimulation 110](#_Toc394643419)

[c. Transcranial Magnetic Stimulation 110](#_Toc394643420)

[l. OPERATIVE THERAPEUTIC PROCEDURES 111](#_Toc394643421)

[1. EXTRACRANIAL SOFT TISSUE 111](#_Toc394643422)

[2. MAXILLOFACIAL 111](#_Toc394643423)

[3. SKULL 111](#_Toc394643424)

[4. BRAIN 111](#_Toc394643425)

[5. CEREBRAL SPINAL FLUID (CSF) 112](#_Toc394643426)

[a. CSF Leak or Fistula 112](#_Toc394643427)

[b. Ventricular Shunting 112](#_Toc394643428)

[c. Ventriculostomy 112](#_Toc394643429)

[6. OPHTHALMOLOGIC 112](#_Toc394643430)

[7. OTOLOGIC 113](#_Toc394643431)

[a. Direct Trauma Or Barotrauma 113](#_Toc394643432)

[b. Tympanostomy 113](#_Toc394643433)

[c. Middle Ear Exploration 113](#_Toc394643434)

[d. Vestibular Nerve Section 114](#_Toc394643435)

[8. DECOMPRESSION OF FACIAL NERVE 114](#_Toc394643436)

[9. OTHER CRANIAL NERVE REPAIR OR DECOMPRESSION 114](#_Toc394643437)

[10. VASCULAR INJURY 114](#_Toc394643438)

[11. PERIPHERAL NERVE INJURY 114](#_Toc394643439)

[12. ORTHOPEDIC 114](#_Toc394643440)

[13. SPASTICITY 115](#_Toc394643441)

[m. MAINTENANCE MANAGEMENT 116](#_Toc394643442)

[1. GENERAL PRINCIPLES 116](#_Toc394643443)

[2. COGNITIVE/BEHAVIORAL/PSYCHOLOGICAL MANAGEMENT 117](#_Toc394643444)

[3. EXERCISE PROGRAMS REQUIRING SPECIAL FACILITIES 118](#_Toc394643445)

[4. HOME EXERCISE PROGRAMS AND EXERCISE EQUIPMENT 118](#_Toc394643446)

[5. LONG-TERM RESIDENTIAL CARE 118](#_Toc394643447)

[6. MAINTENANCE HOME CARE 119](#_Toc394643448)

[7. MEDICATION MANAGEMENT 119](#_Toc394643449)

[8. NEUROMEDICAL MANAGEMENT 120](#_Toc394643450)

[9. PATIENT EDUCATION MANAGEMENT 120](#_Toc394643451)

[10. PHYSICAL, OCCUPATIONAL, and Speech-Language THERAPY 120](#_Toc394643452)

[11. PURCHASE, RENTAL, AND MAINTENANCE OF DURABLE MEDICAL EQUIPMENT 120](#_Toc394643453)

DEPARTMENT OF LABOR AND EMPLOYMENT

Division of Workers’ Compensation

CCR 1101-3

**RULE 17 EXHIBIT 10**

TRAUMATIC BRAIN INJURY MEDICAL TREATMENT GUIDELINES

1. INTRODUCTION

This document has been prepared by the Colorado Department of Labor and Employment, Division of Workers’ Compensation (Division) and should be interpreted within the context of guidelines for physicians/providers treating individuals who qualify as injured workers with traumatic brain injury (TBI) under the Colorado Workers’ Compensation Act.   
  
Although the primary purposes of this document for practitioners are advisory and educational, this guideline is enforceable under the Workers’ Compensation Rules of Procedure, 7 CCR 1101-3. The Division recognizes that acceptable medical practice may include deviations from this guideline, as individual cases dictate. Therefore, this guideline is not relevant as evidence of a provider’s legal standard of professional care.  
  
To properly utilize this document, the reader should not skip or overlook any sections.

1. GENERAL GUIDELINE PRINCIPLES

The principles summarized in this section are key to the intended implementation of this guideline and are critical to the reader's application of the guidelines in this document.

* 1. APPLICATION OF GUIDELINES: The Division provides procedures to implement medical treatment guidelines and to foster communication to resolve disputes among the provider, payer, and patient through the Workers' Compensation Rules of Procedure. In lieu of more costly litigation, parties may wish to seek administrative dispute resolution services through the Division or the Office of Administrative Courts.
  2. EDUCATION: Education of the individual and family and/or support system, as well as the employer, insurer, policy makers, and the community should be the primary emphasis in the treatment of TBI and disability. Practitioners often think of education last, after medications, manual therapy, and surgery. Practitioners should develop and implement an effective strategy and skills to educate individuals with TBI, employers, insurance systems, policy makers, and the community as a whole. An education-based paradigm should always start with inexpensive communication providing reassuring information to the individual with TBI. More in-depth education currently exists within a treatment regimen employing functional restoration and rehabilitation. No treatment plan is complete without addressing issues of individual and family and/or support system education as a means of facilitating self-management of symptoms and prevention.
  3. TREATMENT PARAMETER DURATION: Time frames for specific interventions commence once treatments have been initiated, not on the date of injury. Obviously, duration will be impacted by the individual’s compliance, as well as availability of services. Clinical judgment may substantiate the need to accelerate or decelerate the time frames discussed in this document.
  4. ACTIVE INTERVENTIONS: Emphasizing personal responsibility, such as therapeutic exercise and/or functional treatment, are used predominantly over passive modalities, especially as treatment progresses. Generally, passive and palliative interventions are viewed as a means to facilitate progress in an active rehabilitation program with concomitant attainment of objective functional gains.
  5. ACTIVE THERAPEUTIC EXERCISE PROGRAM: Goals should incorporate strength, endurance, flexibility, coordination, and education. This includes functional application in vocational or community settings.
  6. POSITIVE PATIENT RESPONSE: Results are defined primarily as functional gains which may be objectively measured. Objective functional gains include, but are not limited to, positional tolerances, range of motion (ROM), strength and endurance, activities of daily living (ADLs), cognition, psychological behavior, and efficiency/velocity measures that may be quantified. Subjective reports of pain and function should be considered and given relative weight when the pain has anatomic and physiologic correlation. Anatomic correlation should be based upon objective findings.
  7. RE-EVALUATE TREATMENT EVERY THREE TO FOUR WEEKS: If a given treatment or modality is not producing positive results within three to four weeks, the treatment should be either modified or discontinued. Reconsideration of diagnosis should also occur in the event of poor response to a seemingly rational intervention.
  8. SURGICAL INTERVENTIONS: Should be considered within the context of expected functional outcome and not solely for the purpose of pain relief. The concept of "cure" with respect to surgical treatment by itself is generally a misnomer. Clinical findings, clinical course, and diagnostic tests must be consistent to justify operative interventions. A comprehensive assimilation of these factors must lead to a specific diagnosis with positive identification of pathologic condition(s).
  9. RETURN TO WORK: Following TBI involves a skillful match between the individual’s abilities (physical, cognitive, emotional, and behavioral) and the work requirements.   
       
     The practitioner must write detailed restrictions when returning an individual with TBI to limited duty. An individual with TBI should never be released to "sedentary or light duty" without specific physical or cognitive limitations. The practitioner must understand all of the physical, visual, cognitive, emotional and behavioral demands of the individual's job position before returning him/her to full duty and should request clarification of job duties. Clarification should be obtained from the employer or others if necessary, including but not limited to: an occupational health nurse, occupational therapist, physical therapist, speech therapist, vocational rehabilitation specialist, case manager, industrial hygienist, or other appropriately trained professional.
  10. DELAYED RECOVERY: All individuals with moderate/severe TBI will require an integrated system of care. For individuals with mild TBI (MTBI), strongly consider requesting a neuropsychological evaluation, if not previously provided. Interdisciplinary rehabilitation treatment and vocational goal setting may need to be initiated for those who are failing to make expected progress 6 to 12 weeks after an injury. In individuals with MTBI, neurological recovery is generally achieved within a range of weeks/months up to one year post-injury ([McCrea, 2009](#McCrea2009)), but functional improvements may be made beyond one year. Neurological recovery following moderate/severe TBI is greatest in the first 12 months post-injury, but may occur for up to two years post-injury, with further functional improvements beyond two years. The Division recognizes that 3–10% of all industrially injured individuals will not recover within the timelines outlined in this document despite optimal care. Such individuals may require treatment beyond the limits discussed within this document, but such treatment will require clear documentation by the authorized treating practitioner focusing on objective functional gains afforded by further treatment. Moderate/severe TBI may have a prolonged recovery and frequently requires continuing treatment as addressed in the post-MMI care section.
  11. GUIDELINE RECOMMENDATIONS AND INCLUSION OF MEDICAL EVIDENCE: Guideline recommendations are based on available evidence and/or consensus recommendations. When possible, guideline recommendations will note the level of evidence supporting the treatment recommendation. When interpreting medical evidence statements in the guideline, the following apply:

● “Some” means the recommendation considered at least one adequate scientific study, which reported that a treatment was effective.

● “Good” means the recommendation considered the availability of multiple adequate scientific studies or at least one relevant high-quality scientific study, which reported that a treatment was effective.

● “Strong” means the recommendation considered the availability of multiple relevant and high quality scientific studies, which arrived at similar conclusions about the effectiveness of a treatment.

● Consensus means the opinion of experienced professionals based on general medical principles. Consensus recommendations are designated in the guideline as “generally well-accepted,” “generally accepted,” “acceptable,” or “well-established.”

There is limited and varied literature on TBI. Therefore, many of the studies cited focus on athletes, the military or treatment for strokes.

All recommendations in this guideline are considered to represent reasonable care in appropriately selected cases, regardless of the level of evidence attached to them. Those procedures considered inappropriate, unreasonable, or unnecessary, are designated in the guideline as “not recommended.”

The remainder of this document should be interpreted within the parameters of this guideline principles that may lead to more optimal medical and functional outcomes for injured workers.

* 1. PoST MAXIMUM MEDICAL IMPROVEMENT (MMI) CARE: This document includes recommendations for post-MMI care in appropriate cases. (refer to Section [M. Maintenance Management](#TBIMMaintenanceManagement)).

1. INTRODUCTION TO TRAUMATIC BRAIN INJURY AND PHILOSOPHY OF CARE
   1. DEFINITIONS AND DIAGNOSIS OF TRAUMATIC BRAIN INJURY: Before a diagnosis of TBI is made, the physician should assess the level of trauma to which the individual was exposed using available objective evidence. According to the Institute of Medicine of the National Academies, TBI is an injury to the head or brain caused by externally inflicted trauma. The Department of Defense defines TBI as a “traumatically induced structural injury and/or physiological disruption of brain functions as a result of an external force.” TBI may be caused by a bump, blow, or jolt to the head, by acceleration or deceleration forces without impact, or by blast injury or penetration to the head that disrupts the normal function of the brain ([Veteran’s Affairs Department of Defense [VADoD], 2009](#VetsDOD2009)). A diagnosis of TBI is based on acute injury parameters and should be determined by the criteria listed below. Severity of initial impairment following TBI is subdivided into two major categories, mild TBI and moderate/severe TBI. These definitions apply to the initial severity of impairment, and do not necessarily define or describe the degree of subsequent impairment or disability.
      1. Mild TBI (MTBI): A traumatically induced physiological disruption of brain function, as manifested by at least one of the following, documented within 24 to 72 hours of an injury ([American Congress of Rehabilitation Medicine, 1993](#AmConRehabMed1993)):
         1. Any loss of consciousness.
         2. Any loss of memory for events immediately before or after the injury.
         3. Any alteration of mental status at the time of the injury (e.g. feeling dazed, disoriented, or confused).
         4. Focal neurological deficit(s) that may or may not be transient; but where the severity of the injury does not exceed the following:
            1. Loss of consciousness for approximately 30 minutes or less,
            2. At 30 minutes, a Glasgow Coma Scale (GCS) of 13–15, and
            3. Post-traumatic amnesia (PTA) not greater than 24 hours.
      2. Moderate/Severe TBI: A traumatically induced physiological disruption of brain function as manifested by at least one of the following ([American Congress of Rehabilitation Medicine, 1993](#AmConRehabMed1993)):
         1. Loss of consciousness for greater than 30 minutes
         2. After 30 minutes, an initial GCS of 12 or less, and
         3. PTA greater than 24 hours.
      3. Other Terminology: Once a patient has met the definitions above, the treatment patterns and diagnostic tools of this guideline apply.
         1. Concussion: There is some disagreement in the literature regarding definitions and terminology. Concussion is used synonymously with MTBI in many papers and is only referenced in this guideline when describing studies using the terminology.
         2. Post-Concussive Syndrome (PCS): An accepted diagnosis which generally is determined by the number of symptoms present after a TBI. Unfortunately the symptoms used to determine the presence of PCS are frequently present in those without MTBI ([Dean, 2012](#Dean2012)). In this guideline, once a person has been diagnosed with MTBI, any of the treatments for continuing symptoms may be used. Thus, the diagnostic category of PCS is not necessary and should not be used in isolation to access the treatments in this guideline.
         3. Complicated Mild Traumatic Brain Injury: A MTBI accompanied by structural brain damage visualized on acute neuroimaging. More patients in this group have slow or incomplete recovery as compared to patients without this finding; however, the finding does not fully predict the clinical course of an individual with MTBI ([Iverson, 2006](#Iverson2006)). The term is not used further in this guideline, but it should be understood that complicated MTBI cases will frequently require more extensive treatment than that described under MTBI and may be given access to care listed under moderate/severe as appropriate for the individual.
   2. INTERVENTION: Early identification and early intervention by providers with specialty training and experience is critical in the diagnosis, treatment, and management of individuals with moderate/severe TBI. Brain injury treatment may also require immediate interdisciplinary evaluation and treatment. The treatment and ultimate functional outcome of individuals with TBI depends upon a complex, interacting set of pre-injury, injury, and post-injury factors. Treatment programs should: be specialized; based on a comprehensive data set; include both functional goals and outcome-oriented goals; and be delivered in the least restrictive setting(s) possible. Treatment settings may include acute care settings, hospitals, rehabilitation hospitals, outpatient settings, residential and behavioral settings, home, and community settings. Treatment should be well- managed and time-appropriate, based on progress.  
        
      The provision of on-site case managers familiar with TBI rehabilitation treatment protocols is well-accepted and recommended for all moderate/severe TBI cases, and for select MTBI cases, based on complexity and need.
   3. EDUCATION: Outcome following TBI is often dependent on the health, education, and resources of the individual’s family and/or support system. Therefore, education of the individual and family and/or support system, insurer, case manager, and employer should be a primary emphasis in the treatment and management of individuals with TBI. Providers should develop and implement effective strategies and forums to include family and/or support system members with the interdisciplinary treatment team. Education for individuals and their family and/or support system should include, but is not limited to: communication of basic information about the brain and the effects of TBI on behavior, cognition, communication, physical function, and emotional function; appropriate family and/or support system interventions; and possible short-term and long-term outcomes. Written information and material and referral to credible internet resources may be helpful as the individual and their family and or support system may not be able to remember the often vast amount of information provided to them. For similar reasons, they may need to be provided repeated or ongoing information. Insurance carriers, case managers, and treatment providers are highly encouraged to provide hands-on personal consultations, education (written, verbal, internet), and support services to families in order to maximize treatment outcomes and the durability of those outcomes. For moderate/severe cases, long-term life planning may be discussed. Further in-depth education may be required to maximize the individual’s potential for functional living. Treatment plans should include individual and group education as a means of facilitating self-awareness, self-management and prevention of secondary disability (refer to Section [G.1 Patient/Family/Support System Education](#TBIG1PatientFamilySupport) for further details).
   4. RETURN TO WORK: This involves a skillful match between the individual’s physical, cognitive, emotional, and behavioral abilities; the physical, cognitive, emotional, and behavioral requirements of the work; and the ability of the work environment to meet this match. Successful return-to-work activities often include vocational evaluation, job analysis, supervisor and coworker education, on-the-job trials, monitored and skillful increase of job duties and demands, job coaching, and follow-up maintenance support services.   
        
      Caution should be used in returning an individual to work and other activities during the first 3–14 days after MTBI. Both physical and cognitive duties should generally be non-stressful initially, with a gradual increase in activity based on improvement and/or resolution of symptoms. The individual should be competent in most basic ADLs before return to work is considered. Return to full duty depends on the rate of decrease of symptoms. Generally, if symptoms recur during increasing job duties or exertion, duties should be decreased accordingly ([Defense and Veterans Brain Injury Center, 2008](#DefandVetsTBIctr2008)) (refer to Section [K.2 Return to Work](#TBIK2ReturnToWork)).
   5. DISABILITY: The World Health Organization (WHO) conceives of disability as the interaction among health conditions and environmental factors, such as social and legal structures, personal factors, including age, education and coping styles.  
        
      For the purposes of this guideline, we are adopting the International Classification of Functioning, Disability and Health (ICF) of disablement.  
        
      The model recognizes the interaction between the health condition and three major components: body functions and structures, activity, and participation. These in turn are influenced by environmental and personal issues. The following definitions are used:

● Body Functions: Physiological functions of body systems, including psychological functions.

● Activity Limitations: Difficulties an individual may have in executing activities.

● Participation Restrictions: Problems an individual may experience in involvement in life situations.

● Disability: Activity limitations and/or participation restrictions in an individual with a health condition, disorder, or disease.

Because of the nature of TBI and the nature of learning and memory, functional skills often cannot be generalized across working environments. Therefore, the assessment of function, evaluation, and treatment should not only consider the injured worker, but also include evaluations of the individual’s “real world” environment, conducted by qualified practitioners.

* 1. COURSE OF RECOVERY:
     1. MTBI: In general, 80–90% of MTBI fully recover in less than 90 days. Another 10–20% of persons with MTBI do not recover within 90 days and may have post-concussive symptoms. This group may continue to report symptoms for several months or years ([Weightman, 2010](#Weightman2010); [Carroll, 2004](#Carroll2004); [Hou, 2012](#Hou2012)).  
          
        A number of acute and chronic symptoms are associated with mild TBI. Headache and confusion or disorientation are the most common followed by visual disturbances, dizziness or feeling unsteady, light sensitivity, amnesia, fatigue or feeling “foggy,” alteration of consciousness, sleep disturbance, and nausea. In individuals with MTBI, neurological recovery is generally achieved at one year post-injury or sooner, but functional changes may be made beyond one year. In the absence of secondary or tertiary complications like hydrocephalus, seizures, or extra-axial fluid collections (e.g., subdural or epidural fluid collections), ongoing improvement with eventual stability of symptoms is the general expectation after mild TBI. Deterioration over time after mild TBI is uncommon, and in situations where patients have worsening complaints after mild TBI, other issues such as psychological or social stressors should be considered in the differential or other unidentified diagnosis.
     2. Moderate/Severe TBI: Neurological recovery following moderate/severe TBI is greatest in the first 12 months post-injury, but may occur for up to two years post-injury, with further functional improvements beyond two years. Due to the variable and dynamic nature of disability secondary to TBI, individuals with moderate/severe TBI may either improve or deteriorate over time. In most cases of moderate/severe TBI, and in some unusual circumstances of MTBI, impairment will be life-long, and will require a life-long maintenance plan of services. Complications may warrant periods of active treatment in addition to the maintenance plan.   
          
        In at least 40% of cases, TBI is accompanied by other substantial trauma (e.g. internal, endocrine, orthopedic injuries) which may involve dysfunction in other bodily systems. Psychological issues also occur frequently and are discussed in this guideline. Users of these TBI Guidelines are encouraged to employ appropriate guidelines for other disorders and dysfunction as the need arises.
  2. GUARDIANSHIP AND CONSERVATORSHIP: Individuals with TBI, usually moderate/severe TBI, may clinically be determined to lack capacity to make competent informed decisions concerning their medical care, housing, and/or finances. Health care providers, insurance carriers, and case managers should become familiar with Colorado laws regarding incompetency, guardianship, conservatorship, medical and durable power of attorney, advanced directives, living wills, etc., in order to provide family and/or support system members with the appropriate education and/or resources concerning these issues when clinically indicated.
  3. SYSTEMS OF CARE: Integration of systems of care has the goal of assisting individuals with TBI in progressing along a continuum of care toward achieving optimal clinical outcomes as efficiently and as cost-effectively as possible (Figure 1).Long-term outcome and “value” are recognized as superior to short-term, price-driven management. (Please go to the next page).Model Systems Continuum of Care for Individuals with Moderate/Severe TBI

Figure 1: Continuum of Care (adapted from the Rocky Mountain Regional Brain Injury System, 1991)

Home with Family

With Outpatient / Day

Treatment or

Home/Community

Based Services

Independent Living

Supported Living

Program: Group Home

Supported Living Program: Apartment

Home with Family and

Home Service

Skilled Nursing Facility

Intensive

Care Unit

Emergency Evaluation

Emergency Department

Acute Medical Care

Hospital

Unit

Post Acute

Residential

Transitional

Rehabilitation

Comprehensive Integrated

Inpatient

Brain Injury

Rehabilitation

Hospital

Skilled Nursing Facility

Long-Term

Acute Care

Figure 1 shows a schematic depicting an organized continuum of care for individuals with moderate/severe TBI. The system is not a lock-step progression, but a spectrum of TBI programs and services based on the individual’s unique condition and needs.

“The term rehabilitative and habilitative services includes items and services used to restore functional capacity, minimize limitations on physical and cognitive function, and maintain or prevent deterioration of functioning as a result of an illness, injury, disorder or other health condition. Such services also include training of individuals with mental and physical disabilities to enhance functional development.” ([Congressional Record, E462 [March 23, 2010] [Affordable Care Act](#CongressRec2010)]).The type, amount, frequency and duration of medical, rehabilitation, and long-term services are determined by the individual’s condition and needs, degree of functional improvement within specific time frames, as well as the individual’s potential to achieve additional, measurable functional improvements with continued provision of services. Decisions concerning treatment within the continuum of care should be made by specialists in TBI in conjunction with the individual with TBI and family and/or support system. The following paragraphs describe care programs commonly used by individuals with moderate/severe TBIs. Individuals with MTBI usually do not require the acute care inpatient or residential services described in this continuum.

* + 1. Acute Care: Established Emergency Medical Services (EMS) triage guidelines and organized pre-hospital trauma systems improve the delivery of trauma care and should be utilized. Trauma systems with identified regionally-designated neuro-trauma centers (preferable Level I or Level II Trauma Centers) should be utilized for the acute care of individuals with TBI. Neuro-trauma centers should have a multidisciplinary trauma team, an in-house trauma surgeon, a promptly available neurosurgeon, a continuously staffed Operating Room, Neuroscience nurses, a Neuro-Intensive Care Unit, a laboratory, and a CAT scanner immediately available at all times. Other team members should include orthopedists, radiologists, anesthesiologists, occupational therapists, physical therapists, and speech pathologists. Moderate/severe patients are usually admitted to the Intensive Care Unit initially and then progress to acute care units, which are frequently termed transitional or step-down units. Insurance carriers should develop programs to respond quickly to individuals with TBI and their families and/or support systems once moderate/severe TBI is identified. In these instances, insurance carriers are encouraged to deploy on-site certified case managers (CCM) to assist treatment providers, individuals, and family and/or support system.
    2. Comprehensive Integrated Inpatient Rehabilitation Hospital or “Acute Rehabilitation”: Following medical stability, individuals with moderate/severe TBI should be transferred from acute hospital care to a comprehensive integrated inpatient brain injury rehabilitation program unless they are unable to participate in the program. Acute brain injury rehabilitation hospitals should have a designated specialty program, with designated beds for patients with brain injuries, designated staff, treatment areas, therapy programs, equipment, and a sufficient number of individuals with TBI to constitute a peer and family milieu. Acute rehabilitation hospitals should be accredited by the Joint Commission on Accreditation of Healthcare Organizations (Joint Commission), and have components consistent with the Commission on Accreditation of Rehabilitation Facilities (CARF). CARF eligibility implies that programs meet specific care standards of design and efficacy (refer to Section [I.1.b. Comprehensive Integrated Inpatient Interdisciplinary Rehabilitation Programs](#TBII1bComprehensiveIntegratedInpatient)).
    3. Long-Term Acute Care (LTAC) Programs: Some individuals will be unable to participate in a full inpatient program immediately following acute care and may need LTAC for a period of time prior to entering a Comprehensive Integrated Inpatient Rehabilitation Hospital. LTAC is a recognized designation by the Centers for Medicare and Medicaid Services for LTAC and rehabilitation hospitals whose average length of stay is at least 25 days. LTAC hospitals provide specialized care services, including skilled nursing care to manage medical conditions, so that individuals with catastrophic or acute illnesses/injuries may progress toward entry into full rehabilitation programs. LTAC programs should be accredited by the Joint Commission. LTAC rehabilitation is generally accepted, but should not be used in lieu of Comprehensive Integrated Inpatient Rehabilitation Hospitals.
    4. Sub-Acute Skilled Nursing Facility (SNF) Rehabilitation Programs: These programs are located on separate and specially licensed units of hospitals or free-standing SNFs. Individuals appropriate for SNF require skilled nursing care, and they have either completed comprehensive inpatient rehabilitation or are judged to not be able to benefit from inpatient rehabilitation. SNFs are generally accepted, but they should not be used in lieu of Comprehensive Integrated Inpatient Rehabilitation Hospital for individuals who may benefit from a comprehensive inpatient rehabilitation program. SNFs should be accredited by the Joint Commission.
    5. Post-Acute Rehabilitation: This describes programs following an individual’s stay at a Comprehensive Integrated Inpatient Rehabilitation Hospital, including outpatient or day treatment rehabilitation, residential transitional rehabilitation, behavioral treatment programs, neurobehavioral rehabilitation or home/community-based programs. The most appropriate post-acute rehabilitation program is dependent on the individual’s needs following inpatient hospital rehabilitation, as well as proximity and availability of services, family and/or support system dynamics, and projected long-term outcomes. Individuals with significant deficits or who require behavioral treatment or supervision for safety may require neurobehavioral residential rehabilitation. Post-acute rehabilitation should not be used in lieu of Comprehensive Integrated Inpatient Rehabilitation Hospitals. (refer to Section [I.1.a. Behavioral Programs](#TBII1aBehavioralPrograms) and [h. Residential Rehabilitation](#TBIG11hResidentialRehab)). Other individuals may be able to use a combination of home and community-based rehabilitation and outpatient or day treatment rehabilitation. (refer to Sections [I.1.c. Home and Community-Based Rehabilitation](#TBII1cHomeAndCommunity) and [g. Outpatient Rehabilitation Services](#TBII1gOutpatientRehabServies)).
    6. Long-Term Support Care: The range of long-term outcomes following TBI is diverse, ranging from virtually complete independence and function to severe and permanent disability. Therefore, the range of necessary services is complex and individualized. Some individuals with moderate/severe TBI will require significant care and supervision in order to perform ADLs safely, either at home by family and/or support system members with appropriate training or attendant care, in a skilled nursing care facility, or in a long-term supported living residential program. (refer to Sections [I.1.i Supported Living Programs or Long-Term Care Residential Services](#TBIG11iSLPorLongTermCare) or [d. Nursing Care Facilities](#TBII1dNursingCareFacilities)). Individuals may also benefit from periodic re-evaluations based on condition and needs (refer to Section [M, Maintenance Management](#TBIMMaintenanceManagement)). Long-term care programs should have components consistent with certification by CARF. CARF eligibility or certification implies that programs meet specific care standards of design and efficacy.
  1. INTERDISCIPLINARY TREATMENT TEAM: (also known as multidisciplinary treatment team) An alliance of professionals from different medical or therapeutic disciplines (as described below) that provides a coordinated treatment program. The disciplines, which make up the team, will be determined by the particular treatment needs of the individual with TBI. The team establishes treatment priorities and goals and provides treatment. Team members contribute their respective skills, competencies, insight, and perspectives to the rehabilitation process. This includes education, communication, and alignment of expectations for the purpose of optimizing treatment outcomes. It is highly recommended that the individual with TBI, along with his or her family and/or support system, insurance carrier, and case manager, participate in team planning.  
       
     The most common disciplines, in alphabetical order, involved in the medical and rehabilitation treatment of TBI include, but are not limited to:
     1. Behavioral Psychologist: A psychologist with special training, credentials, and licensing, who specializes in the area of behavior analysis and treatment.
     2. Behavioral Analyst – Masters Level: An individual certified as a behavior analyst who designs and supervises behavior interventions. (Behavioral assessments by an analyst do not substitute for neuropsychological assessments.).
     3. Case Manager: Case managers are initially trained under a variety of disciplines such as nursing, social work, and other health and human services fields and should be certified through the Commission for Case Manager Certification ([CCMC, n.d](#CCMCnd).). In order to achieve the best possible outcome for everyone involved, it is best to provide case management services in an environment in which the case manager, the client, and the appropriate service personnel are able to communicate directly. ([Case Management Society of America, [CMSA, 2012], ‘Philosophy of Case Management’ para. 2](#CMSA2012)). It is crucial that the case manager be thoroughly educated in the complexities of treating individuals with TBI.  
          
        The primary functions of TBI case management are:

● To obtain information through a comprehensive assessment of the injured individual and his/her family and/or support system.

* To work with the health care team, the injured worker, and family and/or support system in development, monitoring, and implementation of a comprehensive case management plan. Plan reassessment should be completed on a regular basis.
* To optimize access to appropriate health care services and maintain cost effectiveness.

● To integrate and coordinate service delivery among all providers and to prevent fragmentation of services by facilitation of communication and by involving the injured worker and family and/or support system in the decision-making process.

● To educate and collaborate with the injured worker, family and/or support system, and the health care team when necessary about treatment options, compliance issues, and community resources.

● To predict and avoid potential complications.

Case managers may perform Utilization Review (UR) as a part of case management duties, but UR alone is not case management.

* + 1. Chiropractor: A credentialed and licensed doctor of chiropractic who assesses and treats human illness and injury, including, but not limited to: musculoskeletal injuries; movement dysfunction; impairments in strength; muscle tone; motor control; posture coordination; endurance; functional mobility; neurological injuries and loss of function. Chiropractic utilizes joint manipulation and spinal and joint rehabilitation, along with various therapies and modalities.
    2. Clinical Pharmacist: A pharmacist with expertise in medication management who may be an important part of the multidisciplinary team and might be useful for patients with multiple medication regimens.
    3. Clinical Psychologist: A licensed psychologist with special training, credentials, and licensing, who specializes in: the assessment and treatment of personality and psychological disorders; education and adjustment counseling; psychotherapy; and management of behavior.
    4. Driver Rehabilitation Specialist: An individual with training in the health care field and certified by the Association for Driver Rehabilitation and the American Occupational Therapy Association.
    5. Independent Life Skills Trainer: An individual with documented training to develop and maintain an individual’s ability to independently sustain herself physically, emotionally and economically.Services may include assessment, training, and supervision or assistance to an individual with self care, medication supervision, task completion, communication skill building, interpersonal skill development, socialization, therapeutic recreation, sensory motor skills, mobility or community transportation training, reduction or elimination of maladaptive behaviors, problem solving skill development, benefits coordination, resource coordination, financial management, and household management.
    6. Music Therapist: An individual who is board certified and trained to use music within a therapeutic relationship to improve cognitive, sensory, motor, communication, and behavioral functions that have been affected by neurologic disease of the human nervous system.
    7. Neurologist: A physician with special training and credentials in the area of the nervous system, who has successfully completed an approved residency in neurology.
    8. Neuro-ophthalmologist: An ophthalmologist or neurologist who has completed an approved residency in ophthalmology or neurology as well as a fellowship in neuro-ophthalmology, and who specializes in the treatment of visual disorders related to the nervous system.
    9. Neuro-otologist: A physician who has completed a fellowship in Neurotology or Oto-neurology.
    10. Neuropsychologist: A licensed psychologist with knowledge of and special training in brain-behavior relationships including neuropsychological assessment, causality of neurobehavioral changes, and treatment and management of neurobehavioral disorders.
    11. Neuroscience Nurse: A registered nurse (RN) who has certification in the treatment of individual and family and/or support system responses to nervous system function and dysfunction across the healthcare continuum.
    12. Neurosurgeon (Neurological Surgeon): A physician who has special training and credentialing in the surgery of nervous system disorders and who has successfully completed an approved residency in neurosurgical medicine.
    13. Nurse: An RN with specialty training, credentialing, and licensing, who specializes in the collection and assessment of health data, health teaching, and the provision of treatment supportive and restorative to life and well-being.
    14. Occupational Therapist: A registered therapist who specializes in participation in ADLs. They assess and treat the physical, perceptual, behavioral and cognitive skills needed to perform self-care, home maintenance, and community skills and provide patient and family and/or support system education.
    15. Occupational Medicine Physician: A physician who has education and training in occupational medicine.
    16. Optometrist: A specialist with training, credentials, and licensing who examines, assesses, diagnoses, and treats abnormal conditions of the eye and its appendages. Optometrists cannot treat posterior uveitis, interpret x-rays, or perform invasive laser surgery. Pharmaceutical treatment is limited by statute.
    17. Ophthalmologist: A medical doctor with special training, credentials, and licensing in the diagnosis and treatment of visual disorders and disorders of the visual system, as well as diagnosis related to systemic conditions, who has successfully completed an internship and an approved residency in ophthalmology. The scope of treatment may include surgical procedures on the eye, orbit and adnexa.
    18. Otolaryngologist: A physician who specializes in ear, nose, and throat medical treatment and has completed a residency in otolaryngology.
    19. Physical Therapist: A licensed therapist with expertise in managing movement dysfunction, which specializes in the assessment and treatment of individuals with impairment deficits and functional limitations in the areas of strength, muscle tone, motor control, posture, coordination, balance, endurance, and general functional mobility, and who works to improve functional independence, as well as providing family and/or support system and patient education.
    20. Physiatrist: A physician with special training, credentials, and licensing in the field of physical medicine and rehabilitation, and who has successfully completed an approved residency in physiatry.
    21. Psychiatrist/Neuropsychiatrist: A physician with special training, credentials, and licensing, who specializes in the field of mental health and psychological disorders, and who has successfully completed an approved residency in psychiatry. A neuropsychiatrist is a psychiatrist who has specialized training, credentials, and licensing in neurologically-based behavioral, cognitive, and emotional disturbances, including specialized training in TBI.
    22. Rehabilitation Counselor: A bachelor’s or master’s level counselor, who specializes in assisting individuals in the process of independent living, productive activity, and vocational pursuits. This includes assistance with financial resources, housing, community resources, social skills, vocational evaluation and treatment, integration back into the workforce, and patient and family and/or support system counseling.
    23. Rehabilitation Nurse: An RN who has certification in rehabilitation nursing. Rehabilitation nursing is a specialty practice area within the field of nursing. It involves the recognition, reporting, and treatment of human responses of individuals and groups to present or future health problems resulting from changes in functional ability and lifestyle ([Association of Rehabilitation Nurses, 2012](#AssocRehabNurses2012)).
    24. Social Worker: A bachelor’s or master’s level licensed social worker who specializes in patient and family relationships, as well as housing, financial resources, and society reintegration.
    25. Speech-Language Pathologist: A certified master’s or doctoral level therapist who specializes in the assessment and treatment of individuals in the areas of communication (speech, language, social skills, voice, cognition, swallowing) and family and/or support system and patient education.
    26. Therapeutic Recreation Specialist: A bachelor’s or master’s level therapist who specializes in the assessment and treatment of individuals in the areas of planning and management of leisure activities, time management, mental health through recreation, and community access.
  1. PREVENTION: Prevention of injuries such as TBI is an essential component of any medical treatment guideline or injury management program. TBI is a dynamic condition, and patients may deteriorate over time in the areas of physical and mental health, cognition, employment, and ADLs. The following guideline-specific definitions of the various types and levels of prevention are necessary to prevent the deterioration from a healthy state to pathology and to successfully intervene at the levels of disablement described in the disability section.
     1. Primary Prevention: The prevention of disease in a susceptible, or potentially susceptible, population through specific measures, including general health promotion efforts. All health providers should remind individuals and supervisors of the primary measures for preventing recurring TBIs.  
          
        Always use appropriate protective equipment on jobs that require protection, including following all of the employment policy and procedures related to the safety of the individual, co-workers, or external customers. Examples include the following:

● Protective helmets, complying with American National Standards Institute (ANSI), on jobs requiring protection from falling objects or electrical hazards.

● Safety goggles or glasses on jobs that require protection from flying objects or debris.

● Protective helmets and headwear when involved in contact, collision, and other sports such as biking, horseback riding, skating, skiing, and snowboarding.

● Avoid walking on wet, slippery floors on the worksite.

● Ensure that scaffolding is in good working order.

● Use ladders in accordance with Occupational Safety and Health Administration (OSHA) recommendations—for example, making sure that ladders over 20 feet tall have cages.

● The use and provision of airbags/safety belts, etc. in motor vehicles.

● Avoid alcohol and other drug use, including marijuana, during recreational activities such as boating, hunting, skiing, snowboarding, etc, while driving or operating equipment, when working from elevated surfaces, and at work.

● Practicing fatigue management techniques to maintain optimal energy levels for the required work tasks.

* + 1. Secondary Prevention: Includes efforts to decrease duration of illness, severity of disease, and sequelae through early diagnosis and prompt intervention.   
         
       MTBI is one of the most common neurologic disorders. Health care providers may play a key role in improving outcomes following MTBI. Early diagnosis of individuals with mild and moderate/severe TBI is critical in helping to avoid secondary symptoms and problems in living. Individuals with a previous history of TBI, co-morbid, psychiatric disorders, cognitive disorders, and substance abuse are also at greater risk for poor outcome and represent an opportunity to reduce the effects of TBI. Such individuals should receive appropriate referrals for the co-morbid conditions, and treatment of these co-morbid conditions should be integrated into the individual’s rehabilitation program. For MTBI, providing education about symptoms, their management, and their probable positive outcome is an essential component of treatment. Using the available diagnostic information as the basis for providing education and providing written instructions on the discharge sheet regarding timing for return to regular activities, and high-risk activities, may help to improve outcomes and prevent further injury. Written materials and internet references that provide appropriate education for individuals with TBI and family and/or support system about TBI care and prevention are available in English and Spanish from the Centers for Disease Control and Prevention.  
         
       Workers who have sustained a recent TBI should be especially cautious about returning to work activities that may lead to a second TBI since second injuries occurring prior to a full recovery from initial MTBIs have more serious consequences. Providers should practice secondary prevention by setting appropriate restrictions for these workers and workers who are suffering from impairment, such as dizziness, that could lead to falls in some work environments (refer to Section [K.2 Return to Work](#TBIK2ReturnToWork)) and by providing information to the individual and family and/or support system about subsequent TBIs and the need for follow-up before return to activities which carry a risk for repeat TBI.
    2. Tertiary Prevention: The effort to decrease the degree of disability and promote rehabilitation and restoration of function in individuals with chronic and irreversible diseases and to prevent disease and disability. Life-long management and follow-up services may be required for select moderate/severe TBI individuals with persistent medical, cognitive, psychological, and functional skill deficits.

The majority of this guideline addresses tertiary prevention of disability for workers with TBI.

1. INITIAL DIAGNOSTIC PROCEDURES

The purpose of these procedures is to establish the type and severity of TBI as a diagnosis, and to establish initial treatment goals. If the individual with TBI regains consciousness and is fully oriented in the field or emergency department, and has normal neurological findings on examination and neuroradiological studies when appropriate, he/she may be discharged home with close supervision for the initial twenty-four hours. If the individual does not regain consciousness, is disoriented or has focal neurological findings, persistent altered mental status, or persistent cognitive impairment, then further neurological evaluation, treatment, management and follow-up are indicated. This may include acute hospitalization or outpatient interdisciplinary team treatment, depending on the severity of the TBI.

* 1. HISTORY OF INJURY: In order to establish the TBI diagnosis and treatment plans/goals, it is a generally accepted and widely used practice for a qualified practitioner to obtain a thorough history of the injury. Recommended data obtained in the history-taking generally should include:
     1. Identification Data: Should include name, address, age, gender, and marital/relationship status.
     2. Precipitating Event: Information regarding the detailed circumstances of the TBI should include where and when the injury occurred, how the injury occurred, what the individual was doing at the time of the injury, and what happened. If the injury occurred as a result of a motor vehicle crash, information should be obtained as to: the speed of the vehicle; position or location of the injured worker; use of restraints or helmet; degree of damage to the vehicle; all other involved vehicles, if known; involvement of EMS system, if any; and acute or sub-acute accident-related physical complaints or injuries, including other people involved, if known. The crash outcome regarding non-TBI complaints/injuries may enhance an understanding of the forces involved in the accident and will minimize the possibility of unrecognized physical injury. The accident report and any police records should be obtained and reviewed if available. If the injury occurred as a result of a fall, information should be obtained regarding the type of fall, distance of the fall, type of surface, etc. The goal is to provide a review of the biomechanical forces involved in the event. Reports from first responders should be obtained. If possible, collaborative information (e.g. witnesses, paramedic report, etc.) should be obtained to seek details of the event and the injured person’s behavioral and cognitive responses immediately following the injury. The presence of alcohol and/or drug use at or prior to the time of the injury should be noted. All of this history should be used when establishing the presence of a TBI caused by a work-related event.
     3. Neurological History: Should include a review of chief complaints, presenting problems, and symptoms. Generally accepted data should include information about duration of alteration of mental status, including consciousness, degree and length of retrograde and PTA, as well as cognitive, behavioral, and physical impairments, with collateral sources of information when possible. Information should be collected regarding various time intervals for the following:
        1. Current Neurological Status: A report of the individual’s current the individual’s neurological condition, symptoms, complaints, functional problems, etc.
        2. Initial Neurological Status: A report of the individual’s neurological condition at the time of the injury, symptoms, complaints, functional problems, etc. The GCS, when performed in the field and the emergency department, may aid in grading the severity of TBI. Individuals with MTBIs may have a normal score on the GCS. Serial GCS scores may be helpful when intoxication may be a factor. It may be helpful to ask the patient to describe in detail the first event they remember after the injury in order to assess post-traumatic amnesia or loss of time sequence ([Ruff, 2009](#Ruff2009)). When evaluating alteration in mental state at the time of the injury, it is also important to consider the individual’s emotional reaction to the distressing event. For instance, whether the feeling of “being dazed” could be a manifestation of emotional numbing should be considered. It is possible to have dazing due to TBI and emotional reactions to the event (for example, numbing and/or detachment). The diagnosis of acute stress disorder should be considered in evaluating individuals with possible MTBI.
        3. Evolution of Neurological Status: A report of change in the individual’s neurological condition between the time of the injury and the present, including symptoms, complaints, and functional problems. The individual’s report of when he/she was able to return to independent activity is relevant to understanding the course of the injury. A family and/or support system member’s history of the patients ability to perform their usual duties is often helpful. Other measures of functional activity that are standardized and can be repeated during treatment may also be helpful.
     4. Review of Medical Records: In addition to the individual’s self-report, practitioners should attempt to obtain and review any external sources of data, including police reports, ambulance reports, emergency department records, eyewitness reports, etc. The practitioner should utilize this information to establish or verify the probable degree of trauma involved in the incident and the consistency between these reports and current symptoms.
     5. Medical/Health History: Taking a history is a generally accepted practice and should include a history of past and current illnesses, injuries, previous brain injuries or other disabilities, seizures/epilepsy, stroke, cerebrovascular disease, developmental/intellectual disabilities, neurodegenerative disorders, any previous intracranial pathology (such as infections, tumors, congenital malformations), pain, previous surgeries of any kind, mental health and medication history, sleep disorders, educational history, and other medical/health data. A report from family and/or support system members or other persons knowledgeable about the individual with TBI relevant to pre-injury as compared to post-injury function should be obtained.
     6. Activities of Daily Living (ADLs): A thorough history should be taken of daily activities. Basic ADLs include: self-care and personal hygiene, communication, ambulation, attaining all normal living postures, travel, non-specialized hand activities, sexual function, sleep, and social and recreational activities. Instrumental Activities of Daily Living (IADLs) are complex self-care activities that may be delegated to others (e.g., financial management, medications, meal preparation). This assessment should delineate the changes in the individual’s ability to perform ADLs prior to and after the injury and any assistance needed from family members or others.
     7. Family History: Should include neurological, psychiatric, and medical history of illness or disability within the family that is relevant to the individual’s condition.
     8. Social History:
        1. Living Situation: Should include marital history, family and/or support system members, household makeup, significant others, etc.
        2. Occupational History: Should include the name of the individual’s current company, job title, primary job duties, special licenses or certifications, length of employment, prior places and dates of employment, previous work-related injuries and their outcomes
        3. Developmental History: Should include educational history, highest level of education obtained, learning disabilities or disorders, any developmental delay, abuse, or neglect, etc.
        4. Avocations: Should include common non-occupational activities, including leisure activities such as sports, hobbies, and personal interests.
        5. Substance Use History: Should be obtained (particularly if there is data to suggest substance abuse was involved in the injury) along with information related to the amount and duration of alcohol, drug, and marijuana use, licit and illicit, including prescription drug use and/or abuse
        6. Legal History: DUIs, violence, speeding/reckless driving violations
     9. Review of Systems: A generally accepted practice and should include a complete review of body systems and functions.
     10. Pain Diagnosis: Recommended, especially during the first visit to document all body parts involved.
     11. Psychiatric History: Should be assessed at the initial visit and at follow-up visits. Depression and anxiety are common conditions following TBI, and symptoms may be subtle or unapparent unless directly assessed. Individuals may not always present with complaints of sadness or anxiety, but instead may endorse other symptoms that are commonly seen in clinical depression or anxiety, particularly disturbances of sleep and energy. Many individuals also tend to focus on somatic complaints that do not always correlate with objective findings. Therefore, it is crucial to question the individual and their family and/or support system about significant changes in appetite, sleep disturbances, decreased interest in pleasurable activities, loss of energy, diminished ability to think or concentrate, irritability, and suicidal ideation, as well as feelings of emptiness, worthlessness, and excessive guilt.
  2. PHYSICAL EXAMINATION: A well-accepted practice and should be performed by a qualified practitioner. A thorough trauma exam should be done during the initial exam and the first follow-up visit to assure all complaints are addressed.
  3. NEUROLOGICAL EXAMINATION: Should be performed by a qualified practitioner, and should include a mental status examination. A comprehensive neurological examination includes, but is not limited to, mental status, cranial nerves, motor status, sensory status, balance and coordination, gait and station. The mental status examination involves both formal and informal observations. It includes observations about the individual’s presentation, social/behavioral decorum, personal hygiene, ability to provide a history, and ability to follow directions. A formal (structured) cognitive examination should be performed to the extent indicated by the situation. It includes an assessment of the individual’s alertness, orientation, attention, concentration, memory, affect, mood, thought process and content, language, ability to perform simple calculations, and higher order assessments of reasoning, judgment, and insight ([Guskiewicz, 1996](#Guskiewicz1996), [2011](#Guskiewicz2011)). Using a standard approach for all visits assists serial functional assessment.
  4. INITIAL NEUROPSYCHOLOGICAL ASSESSMENT: The evaluation of cognitive processes and behavior, using psychological and neuropsychological testing to assess central nervous system function and to diagnose specific behavioral or cognitive deficits or disorders. Neuropsychological assessments are generally accepted and widely used as a valuable component of the diagnosis and management of individuals with TBI. They include sensitive tests that are used to detect cognitive deficits, severity of impairment, and improvement over time. Neuropsychological assessment assists in the differential diagnosis of neurobehavioral disorders, and the cumulative effect of multiple TBIs.  
       
     Neuropsychological assessments may be utilized to formulate how the individual's underlying TBI impacts behavior and the ability to function effectively in daily life. Neuropsychological assessments are also used as a basis for formulating rehabilitation strategies, and may provide information related to prognosis and outcome.   
       
     Neuropsychological assessments utilize standardized testing procedures. Test reliability and validity are important considerations. Examiners should be aware that abnormal cognitive function could occur in the setting of chronic pain, psychological disorders, fatigue, medication use, malingering, developmental/intellectual disabilities, acute or chronic substance abuse, and co-morbid or pre-existing cognitive or neurologic disorders. In cases where co-morbid diagnoses are suspected, formal psychological evaluation should accompany the neuropsychological battery in order to assist in characterization and differentiation of diagnoses. Multiple sources of data (self-report information, medical history, psychosocial history, family report, etc.) are integrated with test performance factors to draw inferences about brain-behavior relationships. The individual’s cultural background, race, age, and developmental and educational history including primary language should be considered. When practical, educational records including history of learning disability should be obtained and reviewed.  
       
     The specific neuropsychological tests used may vary according to the neurologic intactness of the individual and the purpose of the evaluation. Tests usually assess the following cognitive domains: level of orientation, attention, language, memory, praxis, executive function, speed of processing, visual-spatial ability, recognition, personality, and function. All reports should include a clinical interview that notes the patient and family medical/psychiatric/substance abuse history, developmental milestones, educational history, psychosocial issues, and current medical conditions and treatment. Interpretation of these tests should always discuss the impact of information from the clinical interview that might affect test results, such as medications causing confusion or drowsiness, fatigue from lack of sleep, anxiety, depression and similar issues.
     1. Initial Neuropsychological Assessment – MTBI: The referral for neuropsychological assessment during the first month post-MTBI is advantageous for those patients meeting the indications below, in that it documents the attentional, memory, emotional status, and other cognitive deficits as well as cognitive strengths and preserved cognitive capabilities. This provides a baseline for following the injury and permits the adequate documentation of the severity of the injury and improvements over time.  
          
        Neuropsychological consultation is indicated in the acute setting for:

● Determining emergence from PTA.

● Documenting a post-injury baseline and the time course of improvements in attentional functioning, memory, and executive functions in order to contribute to treatment planning.

● Providing relevant information regarding the individual’s current functioning in domains such as speed of information processing, memory, and executive functions. A test battery that permits serial testing focused on attention/concentration skills, memory, speed of processing, executive functions, and emotional/personality status may be indicated.

Individuals with MTBI should be considered for testing, in the following circumstances:

● Glasgow Coma Scale less than 15 at two hours post-injury ([National Institute for Health and Clinical Excellence [NICE], 2007a](#NICE2007a),[b](#NICE2007b),[c](#NICE2007c)).

● Retrograde amnesia for events more than 30 minutes before injury.

● Injuries at the upper end of the mild continuum [duration of coma greater than ten minutes, duration of post-traumatic amnesia (PTA) greater than four to six hours].

● Other risk factors, such as very demanding or stressful vocations, or being employed in the current job for a short period of time.

● Age above 40 years.

● Injury complicated by the presence of intracranial lesions, current or previous.

● History of prior brain injury, cognitive impairment, or developmental delay.

● Associated orthopedic, soft tissue, or organ injuries.

● The patient is not recovering from MTBI within the expected time frame.

During the first three months after sustaining a MTBI, assessment with a full neuropsychological test battery may be relevant when issues include return to highly demanding and/or safety-sensitive positions or when there are complex questions related to differential diagnosis (brain injury versus other diagnosis). There should be a clear rationale for undertaking testing on any occasion, and the influence of practice effects should be considered in serial testing.

* + 1. Initial Neuropsychological Assessment – Moderate/Severe Traumatic Brain Injury:
       1. In the acute setting, neuropsychological consultation and assessment in moderate/severe TBI is indicated for:
          1. Determining emergence from PTA.
          2. Documenting the early course of improvements in attentional functioning, memory, visual-perceptual abilities, and language and executive functions. This information may be utilized in:

● Treatment planning and team consultation.

● Family and/or support system education/support and use of community services.

● Education and/or psychotherapy.

* + - * 1. Education and counseling patients with pre-existing psychological issues or other history predisposing to delayed recovery.
      1. During the sub-acute phase, when cognitive/physical stamina is reduced, availability for testing may be limited due to medical priorities and other rehabilitation commitments.  
           
         Selective neuropsychological testing may be indicated to:

● Identify cognitive strengths and weaknesses.

● Design treatment plans such as psychotherapy.

● Educate the individual and family and/or support system about TBI.

● Assess or recommend behavioral management interventions.

During this time period, test selection will be dependent on the individual’s neurobehavioral status and other aspects of his/her medical condition.  
  
Neuropsychological testing is often undertaken to identify treatment goals and to monitor progress over time. During this phase, descriptive psycho-educational testing is commonly performed in rehabilitation by speech-language pathologists and occupational therapists.   
  
Administration of a full neuropsychological test battery is not indicated in moderate/severe cases until the individual with TBI has clearly emerged from PTA. In most cases, administration of a full battery of neuropsychological tests should not be undertaken until attentional functioning has improved to the point where such extensive testing may be meaningfully undertaken and will contribute to long-term treatment planning and rehabilitation.

* + 1. Post-Acute Testing: Once the individual’s behavior has improved in attentional disturbance, fatigue, pain from other injuries, and neurobehavioral disinhibition to the point where valid test data may be obtained, testing with a full neuropsychological test battery may be helpful.
  1. Initial IMAGING PROCEDURES:
     1. Skull X-Rays: A well-established diagnostic tools used to detect a fracture of the cranial vault. CT scanning is preferred if fractures are suspected because of its much higher sensitivity and accuracy compared to skull radiographs and CT scanning’s ability to identify clinically significant fractures as well as potentially co-existent contusions or hemorrhages. Skull x-rays are generally accepted only if CT scans are not available or in cases where there is only a low suspicion of intracranial injury.
     2. Computed Axial Tomography (CT): A well-established brain imaging x-ray study comprised of a mathematical reconstruction of the tissue densities of the brain, skull, and surrounding tissues. CT scans require the use of computer-based scanning equipment. For acute brain trauma, iodine contrast enhancement is not necessary. CT scans are noninvasive and will reveal the presence of blood, skull fracture, and/or structural changes in the brain. They do, however, expose the patient to higher doses of ionizing radiation than skull radiographs. CT scans provide somewhat limited information compared to MRI about intrinsic cerebral damage involving deep brain structures, although many types of intrinsic damages can be seen on CT scans.  
          
        CT scans are widely accepted for acute diagnostic purposes and for planning acute treatment. They are the screening image of choice in acute brain injury and are used to assess the need for neurosurgical intervention. CT scans are recommended for abnormal mental status [GCS less than 13 on admission] ([American College of Emergency Physicians [ACEP], Centers for Disease Control and Prevention [CDC], Jagoda et al., 2008](#ACEP2008); [NICE 2007a](#NICE2007a)), focal neurologic deficits, or acute seizure, and they should also be considered in the following situations:

● Severe and persistent headache .

● More than one episode of vomiting.

● Coagulopathy.

● Dangerous mechanism of injury (e.g., fall from a height of one meter or five steps, ejection from vehicle, pedestrian hit by car).

● Signs of basilar skull fracture, or open or depressed fractures.

● Physical evidence of trauma above the clavicles and /or multiple trauma and/or basilar skull fracture.

● Acute traumatic seizure.

● Age greater than 60.

● Deficits in short-term memory.

● Drug or alcohol intoxication.

● Any recent history of TBI, including MTBI.

● Use of anticoagulant medication.

* + 1. Magnetic Resonance Imaging (MRI): A well-established brain imaging study for patients with moderate/severe TBI, in which the individual is positioned in a magnetic field and a radio-frequency pulse is applied. Hydrogen proton energy emission is translated into visualized structures. Normal tissues give off one signal, while abnormal structures give off a different signal. Due to their high contrast resolution, MRI scans are superior to CT scans for the detection of some intracranial pathology (e.g. axonal injury, subtle cortical contusions, small extra-axial fluid collections, etc.), except for bone injuries such as fractures. CT is superior to MRI in detecting acute intracranial bleeds and remains the preferred initial imaging study in the first 24 hours following head injury. MRI may reveal an increased amount of pathology when compared with CT. Specific MRI sequences and techniques are very sensitive for detecting acute traumatic cerebral injury; they may include, but are not limited to, diffusion weighted imaging (DWI), susceptibility weighted imaging, gradient echo weighted imaging, and fluid attenuated inversion recovery (FLAIR). Some of these techniques are not available on an emergency basis. MRI scans are useful to assess transient or permanent changes, to determine the etiology of subsequent clinical problems, and to plan treatment. MRI is more sensitive than CT for detecting traumatic cerebral injury. MRI should not be used to diagnose MTBI. Initially, MRI scans are clinically useful in the following situations to:

● Determine neurological deficits in moderate/severe TBI not explained by CT.

● Evaluate prolonged intervals of disturbed consciousness or other prolonged alteration in mental status.

● Define evidence of acute changes super-imposed on previous trauma or disease.

* 1. VASCULAR IMAGING TESTS: Reveal arterial or venous abnormalities in the chest, neck, head, or extremities (e.g., thrombosis, dissection, spasm, emboli, or tearing). These tests are generally used if more standard CT/MRI scans fail to demonstrate suspected vascular abnormalities. They may be useful in moderate/severe TBI as an adjunct to aforementioned studies (refer to Sections [D.5.a. Skull X-Rays](#TBID5aSkullXrays), [b. Computed Axial Tomography (CT)](#TBID5bComputedAxialTomography) and [c. Magnetic Resonance Imaging (MRI)](#TBID5cMRI)), but only rarely are they useful in MTBI. Often, patients with clinical signs of blunt trauma to the neck or with a significant mechanism of injury require imaging to detect injuries to the carotid or vertebral arteries that cannot be diagnosed on physical exam. Procedures that are generally accepted include:
     1. CT Angiography (CTA): At the time of this guideline, this is the most common and accepted test for screening patients for injuries to the carotid or vertebral arteries in the setting of trauma. CTAs are noninvasive tests that are readily available in essentially all emergency rooms that treat patients with traumatic injuries. They can be obtained rapidly, often just subsequent to the screening CT head exam. They provide excellent 2D and 3D imaging of the vessels from the aortic arch to the skull vertex and also show the relationship of those vessels to surrounding bones and soft tissues. Some limitations include poor vessel opacification if the timing of the study is incorrect, artifact from dental hardware and the skull base, and patient motion. CTAs should generally only be performed on scanners with at least 16 detectors, with 64 being the preferable number of detectors. CT venography (CTV) is also the most commonly utilized technique to evaluate the dural venous sinuses for injury in the setting of trauma.
     2. Arteriography: Generally accepted when the above noted traumatic vascular abnormalities are suspected but unproven with the techniques discussed so far, or when further investigation of the vascular lesion is necessary. This is particularly true with arteriovenous fistulous change.
     3. Venography: Generally accepted if increased venous flow and pressure are suspected and still undemonstrated. This is done by either the jugular or orbital system.
     4. Noninvasive Vascular Assessment (NIVA): The least invasive and may demonstrate direction of blood flow and general patency of the carotid and vertebral arterial systems in the neck, but not in the head.
     5. Magnetic Resonance Angiography (Magnetic Resonance Arteriography (MRA)/Magnetic Resonance Venography (MVA)): Indicated when vessel changes are suspected but not demonstrated by other simpler tests. Internal obstruction of an artery (e.g., thrombosis, spasm, dissection, neck injury, or emboli from concomitant injuries) may be demonstrated. Arterial compression due to external pressure (e.g., bony fracture or mass effect from a large intra-axial hemorrhage or cerebral edema) may be demonstrated. Dissection or arteriovenous fistula formation may be seen, but as with other vascular abnormalities, may need conventional contrast arteriography/venography to confirm or refute the MRA or MRV findings. The source for intra- or extra-axial bleeding may be seen. Intracerebral dural venous sinus thrombosis, as well as poor venous return may be demonstrated by MRA or MRV.
     6. Brain Acoustic Monitor: This device identifies turbulent blood flow in the brain. It is considered investigational for the purpose of detecting deficits requiring CT scanning in the emergency room. There is some evidence that it cannot reliably predict the development of post-concussive symptoms, and therefore it is not recommended at the time of this guideline ([Dutton, 2011](#Dutton2011)).
  2. LUMBAR PUNCTURE: A well-established diagnostic procedure for examining cerebrospinal fluid (CSF) in neurological disease and injury. The procedure should be performed by qualified and trained physicians under sterile conditions. Lumbar puncture is contraindicated in acute trauma to the spinal column, certain infections, increased intracranial pressure due to space occupying lesions, and in some coagulation disorders or defects. Additionally, it should be avoided if there are cutaneous infections in the region of the puncture site. In individuals with suspected or known increased intracranial pressure, lumbar puncture should be preceded by fundoscopic examination and by a CT scan or MRI.

1. FOLLOW-UP DIAGNOSTIC PROCEDURES
   1. IMAGING: Practitioners should be aware of the radiation doses associated with various procedures. Coloradoans have a background exposure to radiation, and unnecessary CT scans or x-rays increase the lifetime risk of cancer death ([Hendrick, 2011](#Hendrick2011)).
      1. Structural Imaging:
         1. Computed Axial Tomography (CT): May be used to follow identified pathology or to screen for late pathology. Subsequently, CT scans are generally accepted when there is suspected intracranial blood, extra-axial blood, hydrocephalus, altered mental state, or a change in clinical condition, including development of new neurological symptoms or post-traumatic seizure (within the first days following trauma). MRI scans are generally recommended as opposed to CT once the initial acute stage has passed.
         2. Magnetic Resonance Imaging (MRI): The image of choice to detect the late, sub-acute, and chronic structural changes in the brain which underlie abnormal functioning and is a well-accepted technique for follow-up imaging. Complications of TBI that may be explained by MRI include, but are not limited to, post-traumatic epilepsy, post-traumatic movement disorder, post-traumatic cranial neuropathy, post-traumatic infection, or failure to recover within the expected time frame (refer to Section [E.2. Advanced MRI Techniques](#TBIE2AdvancedMRITech) for more advanced imaging).
      2. Dynamic Imaging: In contrast to anatomical imaging procedures, the following procedures are designated to detect physiologic activity of the brain, including cerebral blood flow and cerebral metabolism.
         1. Single Photon Emission Computed Tomography (SPECT): Not generally accepted as a diagnostic test for TBI of any severity and is considered investigational for diagnostic purposes. It is a functional image of the brain created by a flow tracer or a receptor-binding substance tagged with a radionuclide and injected intravenously into the individual. The radiotracer is assumed to accumulate in different areas of the brain proportionately to the rate of delivery of nutrients to that volume of brain tissue. Using a gamma camera and the techniques of CT, a 3-D image of the distribution of a radionuclide in the brain is obtained. SPECT may identify areas of decreased perfusion and provide a qualitative estimate of regional cerebral blood flow (CBF), which correlates with metabolism in many neurologic disorders. There is a variable correlation of SPECT with other measures, such as neuropsychological test findings. Its interpretation should take into account its low specificity, making the predictive value of SPECT no better than CT ([Gowda, 2006](#Gowda2006)).

Although it should not be used to diagnose MTBI, there is some evidence ([Jacobs, 1996](#Jacobs1996)) that SPECT may provide useful information in some cases in which the prognosis is in question, particularly if structural neuroimaging is normal. Given its high sensitivity, SPECT may be useful when expected recovery from MTBI is not occurring within several months from the time of injury. A normal SPECT scan in this setting indicates a likelihood of resolution of symptoms within twelve months. However, due to its lack of specificity, an abnormal SPECT scan does not mean that symptoms will persist. Symptoms may resolve even when areas of abnormal perfusion continue to be seen on the SPECT scan ([Belanger, 2007](#Belanger2007); [Kou, 2010](#Kou2010)).  
  
For severe TBI, SPECT may be useful for individuals with prolonged low levels of responsiveness (i.e., persistent vegetative state) in cases of anoxia, or when additional data is needed.  
  
In all severities of TBI, it is recommended that medical necessity and clinical usefulness for this study be justified.

* + - 1. Positron Emission Testing (PET): A functional brain imaging procedure. A tracer molecule tagged with a positron-emitting radioisotope is injected into the body. Biodistribution of the tracer is imaged, producing information about local cerebral glucose utilization and cerebral perfusion. This procedure requires on-site access to a cyclotron.   
           
         PET can reveal areas of decreased metabolism in the brain. In individuals with moderate/severe TBI, PET findings are closely correlated with the site and the extent of cerebral dysfunction derived from neurological and neurobehavioral examinations. Little information is available about its use and results in MTBI. In all severities of TBI, it is recommended that medical necessity and clinical usefulness for this diagnostic study be justified. It is not generally accepted as a diagnostic study and should not be used solely to diagnose the presence of TBI ([Belanger, 2007](#Belanger2007)).
  1. ADVANCED MRI TECHNIQUES:
     1. Magnetic Resonance (MR) Spectroscopy: A noninvasive test that applies a burst of radio frequency energy to tissue inside an applied magnetic field. The resulting excitation and relaxation of nuclei generates a signal which carries information about the chemical environment of those nuclei. MR spectroscopy may detect changes in levels of n-acetyl-aspartate, an intermediate in neurotransmitter synthesis which is present in large amounts in normal functioning neurons but is decreased in damaged brain tissue. Its spectral signal may correlate with neuronal integrity and function and may show loss of function in tissue which appears normal on conventional CT or MRI studies. MR spectroscopy may increase the sensitivity of MR imaging for traumatic lesions. This sensitivity may allow for increased correlation to more specific neuro-cognitive deficits, guide treatment planning, and be useful information in determining long-term outcome. MR spectroscopy remains predominantly a research tool at this time and should not be used solely to diagnose the presence of TBI ([Belanger, 2007](#Belanger2007)). It may be considered with adequate documentation of its medical necessity in unusual cases. such as in patients with a minimally conscious state, when the information will assist in clarifying the pathology to direct a therapeutic approach to the individual with TBI.
     2. Functional MRI (fMRI): Uses MRI to detect physiologic responses of brain tissue to various tasks. Blood oxygenation level dependent (BOLD) contrast, the most popular fMRI technique, derives an image from differences in the magnetic properties and therefore differences in MR decay parameters, of oxygenated and deoxygenated hemoglobin. A typical fMRI study compares images under two or more behavioral conditions, which may involve motor, cognitive, or visual tasks. Functional MRI studies have shown functional reorganization as a general response to TBI ([Belanger, 2007](#Belanger2007)). Alterations in patterns of cerebral activity seen on fMRIs may correlate with cognitive deficits in individuals with TBI, but the specificity of the test is not sufficient to make fMRI a diagnostic tool. It is, as of the time of this guideline, a research tool and not recommended for clinical use. Recent publications report problems with the mathematical formulas used, relating false positives and false negatives ([Sanders, 2009](#Sanders2009)).
     3. Diffusion Tensor Imaging, Susceptibility—Weighted Imaging and Magnetic Transfer Imaging: Have been used to explore the effects of MTBI. They remain research tools because, as of the time of this guideline, there are no studies validating their use clinically to differentiate MTBI patients with cognitive deficits from those without. They are not recommended to diagnose MTBI ([Belanger, 2007](#Belanger2007); [McCrea, 2009](#McCrea2009); [Kou, 2010](#Kou2010)). Diffusion Tensor Imaging may be useful for identifying pathology and guiding treatment in patients with documented physiological deficits, such as hemianopsia ([Yeo, 2012](#Yeo2012)).
  2. NEUROPSYCHOLOGICAL ASSESSMENT: Neuropsychological assessment past the acute period is appropriate in the following situations when:

● Input is needed to plan treatment to maximize long-term cognitive and overall functional outcomes.

● It is useful to define how strengths may be utilized in cognitive rehabilitative therapy to compensate for weakness.

● There is a question of the individual’s ability to perform work-related duties and/or there are safety issues (i.e., possible harm to self or others), or when the person’s vocation necessitates more extensive testing prior to vocational re-entry or return to school/training.

● Assistance is needed with differential diagnosis including the diagnosis of TBI past the acute/sub-acute period.

● It is deemed necessary to evaluate and/or monitor effectiveness of treatment approaches (i.e., cognitive rehabilitation therapy, somatic therapies, or medication trials) in specific individuals.

● The individual with TBI fails to show improvement in cognitive abilities, and symptom magnification, emotional functioning, or personality factors are suspected to be interfering with treatment progress.

● Subjective complaints are disproportionate to the clinical history or objective findings, as observed by provider(s).

● The degree of disability is disproportionate to the clinical history, and objective findings as observed by provider(s).

Neuropsychology testing should not be used in isolation to diagnose malingering, although it may provide information suggesting poor effort or intentional manipulation of symptoms.

The following information may aid in delineating when a full neuropsychological battery is necessary versus more limited testing:

* + 1. Mild Traumatic Brain Injury: Between one and six months post-injury, serial testing with specialized tests that are sensitive to speed of processing, memory, and executive functions will usually be appropriate for treatment planning and monitoring progress. Some neuropsychological assessment should be done at about the three-month interval for those patients who are experiencing and/or manifesting impaired cognitive function (e.g. not making appropriate progress at work or demonstrating significant organizational issues). The administration of a full neuropsychological test battery (typically including assessment of effort) may become necessary in this time period when:

● There is no known or documented medical history of TBI, and there is the question of whether a TBI occurred.

● It is necessary to address issues on the initial indicators list.

* + 1. Moderate/Severe TBI: The administration of a full neuropsychological test battery after the acute period is appropriate in a number of situations when:

● Developmental issues are interacting with a history of TBI (e.g. determining if age related memory or cognitive changes are impairing functioning in a person with a history of moderate/severe TBI).

● Late complications develop that affect cognition and overall function (e.g. seizures, depression, anxiety disorders).

● There are questions of competency, guardianship, or conservatorship.

● It is necessary to address any of the issues on the initial indications tests.

* 1. PERSONALITY/PSYCHOLOGICAL/PSYCHOSOCIAL EVALUATIONS: Generally accepted and well-established diagnostic procedures with selective use in the TBI population. They have more widespread use in the sub-acute and chronic populations. Diagnostic testing may be indicated for individuals with symptoms of post-traumatic disturbances of sleep, mood, anxiety, psychosis, substance use, aggression/agitation, and pain, as well as depression, delayed recovery, chronic pain, recurrent painful conditions, and disability problems. An individual with a PhD, PsyD, or psychiatric MD/DO credentials may perform these evaluations.   
       
     Psychosocial evaluations can help to determine if further psychosocial or behavioral interventions are indicated for patients diagnosed with TBI. The interpretations of the evaluation can provide clinicians with a better understanding of the patient in his or her social environment, thus allowing for more effective rehabilitation. Psychosocial assessment requires consideration of variations in experience and expression resulting from affective, cognitive, motivational, and coping processes, as well as other influences such as gender, age, race, ethnicity, national origin, religion, sexual orientation, disability, language, or socioeconomic status.  
       
     A comprehensive psychological evaluation should attempt to identify both primary psychiatric risk factors, or “red flags” (e.g. psychosis, active suicidality), as well as secondary risk factors, or “yellow flags” (e.g. moderate depression, job dissatisfaction, symptom magnification) ([Bruns, 2009](#Bruns2009)). Significant personality disorders should also be taken into account in treatment planning.   
       
     Psychometric testing is a valuable component of a consultation to assist the physician and other members of the treatment team in making a more effective treatment plan. There is good evidence that psychometric testing can predict medical treatment outcome ([Block, 2001](#Block2001); [Sinikallio, 2009](#Sinikalio2009), [2010](#Sinikalio2010)). Psychometric testing can assist in predicting a patient’s likely adherence to and cooperation with medical treatment plans and in enhancing general medical outcomes.  
       
     Even in cases where no diagnosable psychological condition is present, these evaluations can identify social, cultural, coping, and other variables that may be influencing the patient’s recovery process and may be amenable to various treatments, including behavioral therapy.
     1. Qualifications:
        1. A psychologist with a PhD, PsyD, EdD credentials, or a physician with Psychiatric MD/DO credentials may perform the initial comprehensive evaluations. It is preferable that these professionals have experience in diagnosing and treating MTBI in injured workers.
        2. Psychometric tests should be administered by psychologists with a PhD, PsyD, or EdD credentials, or health professionals working under the supervision of a doctorate level psychologist. Physicians with appropriate training may also administer such testing, but interpretation of the tests should be done by trained psychologists.
     2. Indications:
        1. A psychological assessment may be necessary if symptoms do not correlate with a diagnosis of TBI. Complaints of cognitive dysfunction may also be associated with a variety of conditions that do not involve neurological disease, TBI, or concussion. This includes conditions that may have been pre-existing or are concurrent, such as depression, anxiety, chronic pain, somatoform disorders, and factitious orders. At times, a set of symptoms may not coincide with expected objective findings for those with a diagnosis of TBI. To identify non-neurological contributions to cognitive or other functional complaints, a psychological evaluation focusing on mental disorder diagnoses is appropriate when:

● Delayed recovery is present,

● There is a question of whether a brain injury has occurred,

● Neuropsychological testing yields a pattern of test results that is not consistent with the clinical history,

● Neurologically improbable symptoms are present, or

● It is necessary to assess for accompanying psychological components.

* + 1. Clinical Evaluation:   
         
       Special note to health care providers: most providers are required to adhere to the federal regulations under the Health Insurance Portability and Accountability Act (HIPAA). Unlike general health insurers, workers compensation insurers are not required to adhere to HIPAA standards thus, providers should assume that sensitive information included in a report sent to the insurer could be forwarded to the employer. The Colorado statute provides a limited waiver of medical information regarding the work-related injury or disease to the extent necessary to resolve the claim. It is recommended that the health care provider either (1) obtain a full release from the patient regarding information that may go to the employer or (2) not include sensitive health information that is not directly related to the work-related conditions in reports sent to the insurer.  
         
       The clinical evaluation should address the following areas:
       1. History of Injury: The history of the injury should be reported in the patient’s words or using similar terminology. Caution must be exercised when using translators.

● Nature of injury.

● Psychosocial circumstances of the injury.

● Current symptomatic complaints.

● Extent of medical corroboration.

● Treatment received and results.

● Compliance with treatment.

● Coping strategies used, including perceived locus of control, catastrophizing, and risk aversion.

● Perception of medical system and employer.

● History of response to prescription medications.

● Medication history related to this injury.

* + - 1. Health History:

● Nature of injury.

● Medical history.

● Psychiatric history.

● History of alcohol or substance abuse, including abuse of prescription medication.

● ADLs.

● Previous injuries, including disability, impairment, and compensation.

● Complete medication history, including prescription and over-the-counter medications.

* + - 1. Psychosocial History:

● Childhood history, including abuse/neglect and developmental/intellectual disability or delay.

● Educational history.

● Family history, including disability.

● Marital history and other significant adulthood activities and events.

● Legal history, including criminal and civil litigation.

● Employment history.

● Military duty—because post-traumatic stress disorder (PTSD) might be an unacceptable condition for many military personnel to acknowledge, it may be prudent to screen initially for signs of depression or anxiety—both of which may be present in PTSD.

● Signs of pre-injury psychological dysfunction.

● Current and past interpersonal relations, support, and living situation.

● Financial history.

* + - 1. Mental status exam including cognition, affect, mood, orientation, thinking, and perception. May include mini mental status exam or frontal assessment battery, if appropriate, and detailed neuropsychological testing.
      2. Assessment of any danger posed to self or others.
      3. Psychological test results, if performed.
      4. Current psychiatric/psychological diagnosis consistent with the standards of the American Psychiatric Association’s most recent Diagnostic and Statistical Manual of Mental Disorders.
      5. Pre-existing psychiatric conditions. Treatment of these conditions is appropriate when the pre-existing condition affects recovery.
      6. Causality (to address medically probable cause and effect, distinguishing pre-existing psychological symptoms, traits, and vulnerabilities from current symptoms).
      7. Treatment recommendations with respect to specific goals, frequency, timeframes, and expected outcomes.
  1. ELECTROENCEPHALOGRAPHY:
     1. Electroencephalography (EEG): A well-established diagnostic procedure that monitors brain wave activity using scalp electrodes and provocative maneuvers such as hyperventilation and photic strobe. Information generated includes alterations in brain wave activity such as frequency changes (non-specific) or morphologic (seizures). EEG is not generally indicated in the immediate period of emergency response, or during acute evaluation and treatment. Following initial assessment and stabilization, the individual’s course should be monitored. If during this period there is failure to improve, or the medical condition deteriorates, an EEG may be indicated to assess seizures, focal encephalopathy due to persistent effects of hemorrhage, diffuse encephalopathy due to the injury, or other complicating factors such as hydrocephalus or medications. A normal EEG does not definitively rule out a seizure disorder. If there is sufficient clinical concern that a seizure disorder may exist despite a normal EEG, then a 72 hour ambulatory EEG or inpatient video-EEG monitoring may be appropriate.
     2. Quantified Electroencephalography (QEEG) (Computerized EEG): A modification of standard EEG using computerized analysis of statistical relationships between power, frequency, timing, and distribution of scalp recorded brain electrical activity. These statistically generated values are then compared to those recorded from selected control and specific populations, generally using multiple regression analysis of multiple measurements and calculated parameters.

Recent studies suggest that in the future, QEEG may become a useful tool in the retrospective diagnosis of TBI and its severity, but this application remains investigational ([Arciniegas, 2011](#Arciniegas2011); [Coburn, 2006](#Coburn2006)). In moderate/severe TBI, the results of QEEG are almost always redundant when traditional electroencephalographic, neurologic, and radiologic evaluations have been obtained. QEEG is not recommended for diagnosing MTBI or moderate/severe TBI.

* 1. ELECTRODIAGNOSTIC STUDIES: Limited to EMG, nerve conduction studies, and multisensory evoked potentials including visual evoked potentials (VEP), somatosensory evoked potentials (SSEP), and brain stem auditory evoked responses (BSAER).
     1. EMG and Nerve Conduction Studies: Generally accepted, well-established diagnostic procedures. These studies may be useful for individuals with brain injury and EMG associated suspected peripheral nervous system involvement. They are often used to differentiate peripheral versus central spinal cord or brain deficits. These electrodiagnostic studies are possibly complementary to other imaging procedures such as CT, MRI, and/or myelography. These studies provide useful correlative neuropathophysiologic information that is unattainable from standard radiologic studies.
     2. Electroneuronography (EnoG): A well-established and generally accepted test that measures facial nerve function. This test measures the action potential of different branches of a facial nerve. It is used in individuals with TBI resulting in a facial paralysis and is key in determining the need for surgical intervention. This test is most useful within the first three weeks of facial nerve dysfunction. If the action potentials on the affected side are 90–100% less than those on the normal side, it suggests significant injury to the nerve and calls for surgical exploration. Individuals with TBI whose nerve is less than 90% decreased in function have a reasonably good outcome with observation alone.
     3. Dynamic Electromyographies: Electrodiagnostic studies utilized to distinguish the voluntary capacity of a muscle from a spastic reaction. This aids the clinician in better planning specific rehabilitative treatment. This study is helpful in the differential diagnosis and diagnostic work-up of disordered muscle tone. This is a generally accepted procedure.
     4. Evoked Potential Responses (EP): Generally accepted, well-established diagnostic procedures. EPs are stimulus-based central nervous system electrophysiologic responses to a stimulus, either externally generated via one or more sensory modalities, or internally generated via the processing of information. Multisensory EP studies are limited to visually evoked potentials, brain stem auditory evoked potentials, somatosensory evoked potentials and cognitive evoked potentials. In moderate/severe TBI, including the “minimal responsive or vegetative state,” there is some utility in the use of these studies for differential diagnosis, prognosis, and to determine an individual’s more specific level of neurologic functioning.
        1. Brainstem Auditory Evoked Response (BAER): A generally accepted diagnostic procedure useful in assessing damage to the brain stem, midbrain and other neural structures that govern hearing and/or balance. It may be more useful than the Somatosensory Evoked Potential (SSEP) in MTBI. A normal test does not rule out structural damage, and the test may be abnormal in middle ear and non-traumatic disease affecting the auditory pathway. Waves one and three but particularly five, are most useful in assessing injury. While amplitude and the presence of wave are important, the latency and interwave latency are equally important. This test is often sensitive but non-specific.
        2. Electroretinogram (ERG): A generally accepted diagnostic procedure for occult retinal trauma accompanying TBI. Most traumatic retinal pathology presents as a field deficit detected by direct examination.
        3. Cognitive Event-Related Potential: An acceptable diagnostic procedure for moderate/severe TBI. Event-related potential provides no diagnostic information in MTBI that cannot be obtained through other diagnostic procedures and is not recommended in MTBI. It may be used when other diagnoses are suspected.
        4. Somatosensory Evoked Potential (SSEP): A generally accepted diagnostic procedure for moderate/severe TBI. SSEP provides no information in MTBI that cannot be obtained through other diagnostic means. SSEP is not recommended in MTBI. It may be used when other diagnoses are suspected.
        5. Visual Evoked Potential (VEP): A generally accepted diagnostic procedure. Pattern reversal monocular VEP recording may detect pathology in the anterior-posterior visual pathway from the retina to the occipital cortex. It may be indicated in the event of compromised acuity or visual field defect. The VEP may occasionally be normal in cases of severe structural damage if there is enough preserved central visual field.
        6. Vestibular Evoked Myogenic Potentials (VEMP): Refer to Section 10.c.v.
  2. LABORATORY TESTING: A generally accepted, well-established procedure. In MTBI, laboratory tests are rarely indicated at the time of initial evaluation unless there is suspicion of systemic illness, infection, neoplasm, drug or alcohol intoxication, endocrine dysfunction, or underlying disease. Hypopituitarism occurs in approximately 17% of MTBI cases; thus, endocrine testing is frequently appropriate ([Schneider, 2007](#Schneider2007)). In moderate/severe TBI, extensive lab testing will be necessary to monitor electrolyte status, organ and endocrine functions, and other physiologic processes, depending on the medications used and the severity of the injury. Any individual with TBI on medication will require laboratory testing to monitor the effects on organ function and therapeutic drug levels.
  3. NERVE BLOCKS – Diagnostic: Generally accepted procedures involving percutaneous needle injection techniques to a specific nerve. These diagnostic blocks are typically performed with quick-acting, short duration local anesthetics such as lidocaine or bupivacaine. Temporary diagnostic nerve blocks evaluate limb ROM, dystonia, or spasticity and assist in planning subsequent more specific therapy.
  4. VISION EVALUATION: A well-established series and combination of examination techniques and diagnostic tests To establish the diagnosis of visual disorders, it is a generally accepted practice for a qualified practitioner to provide a thorough vision evaluation. The visual evaluation measures a wide range of visual processes that involve the functional status of the eyes, the visual pathways of the brain, and the systemic health conditions that affect the eyes and visual pathways. It generates information regarding the presence or absence of refractive error, vision loss, oculomotor dysfunction, binocular vision disorder, ocular injury, and pathology. Visual evaluation may be necessary to evaluate central and peripheral nervous system disorders including central visual acuity loss, visual field loss, nystagmus, ocular motility impairment, cranial nerve palsy, ophthalmoplegia, pupillary reflex disorders, and visual perceptual disorders.  
       
     Signs and symptoms of visual dysfunction commonly include, but are not limited to, the following:
* Signs:
* Blurred vision.
* Eye pain.
* Foreign body sensation.
* Red or irritate eyes.
* Symptoms:
  + Loss of vision in one or both eyes.
  + Focusing problems.
  + Double vision.
  + Eye turn (strabismus).
  + Eye closure or eye cover to improve function.
  + Impaired depth perception.
  + Impaired peripheral vision

Other symptoms that may involve central nervous processing include:

* Headache or eye strain with use of eyes.
* Head tilt.
* Dizziness with use of eyes.
* Visual distortions (e.g., objects, floor, or walls appear bowed, slanted, or tipped).
* Reduced visual attention or concentration for visual tasks.

Visual evaluation is indicated when signs or symptoms consistent with a visual problem are reported by the individual, or observed by others. Significant signs and symptoms not directly and solely attributable to other causes (e.g., cognitive, vestibular, medication, psychological) indicate the need for vision evaluation as soon as is reasonably possible post-injury. Mild signs and symptoms may be monitored for several weeks to allow for resolution or improvement.   
  
In MTBI, self-reported photosensitivity, blurred vision, double vision and saccadic deficits are relatively common, but there is usually resolution by three months post-injury. Moderate/severe TBI patients are more likely to have binocular, pursuit and /or saccadic deficits, and visual spatial deficits, and they should have a comprehensive evaluation if these are reported ([Veterans Affairs Technology Assessment Program, 2009](#VATechAssess2009)).   
  
A vision examination may be intermediate, extended, or comprehensive, depending on the nature of the deficits. The vision examination includes, but is not limited to the following: case history, visual acuity, refraction, color vision testing, pupillary examination, visual field by confrontation, tangent screen, automated perimetry Amsler grid testing, ocular motility examination, binocularity examination, accommodation testing, intraocular pressure testing, and anterior and posterior segment examinations. Internal examination may be needed for diagnoses such as Tersenn syndrome and retinal detachment and to rule out other related diagnoses.   
  
Diagnostic tests include, but are not limited to, visual field testing, ultrasonography, fluorescein angiography, anterior segment and fundus photography, electrodiagnostic studies, low vision evaluation, and visual perceptual testing.

* + 1. Visual Field Testing: A well-established technique to evaluate central and peripheral vision. It is indicated when a field defect is suspected by the practitioner or noted by the patient. Visual field testing beyond the basic examination should be performed using a procedure and tool that is well-established and standardized. Examples include automated perimetry and Goldmann perimetry.
    2. Ultrasonography: A well-established diagnostic test that is indicated for evaluation of ocular or orbital pathology. It is indicated for ocular lesions that are suspected but poorly visualized due to opaque ocular media or for further evaluation of ocular or orbital pathology.
    3. Fluorescein Angiography: A well-established diagnostic test to evaluate the retinal and choroidal circulation. It is indicated when lesions of one or both of these circulations are suspected.
    4. Visual Perceptual Testing: Consists of functional assessments to evaluate an individual’s recognition and interpretation of visual sensory information. Visual perceptual testing is indicated for determination of the level of visual perceptual impairment and/or confirmation of suspected impairment. Perceptual areas assessed include visual memory, judgment of visual spatial relationships, visual discrimination, visual motor integration, visual figure-ground discrimination, and visual attention. Numerous tests are used for the evaluation of visual perception. Some of these tests are well-established. It is suggested that only tests with established norms be used. Visual perception testing should not be used in isolation to diagnose MTBI. Interpretation should occur in the context of assessment of other cognitive functions, including attention, memory, language and executive function. Neuropsychological assessment must be performed by a neuropsychologist, neurologist, psychiatrist, occupational therapist, or speech pathologist. In complex presentations, the full battery may be required (refer to Section [E.3. Neuropsychological Assessment](#TBIE3NeuropsychologicalAssessment)).
    5. Low Vision Evaluation: Well-established and indicated in the presence of subnormal bilateral visual acuity or visual field. The goal is to provide low vision aids for distance or near vision that improves visual functioning.
    6. Electrodiagnostic Studies: Well-established and indicated in the presence of reduced visual acuity or visual fields, ocular pathology, or suspected optic nerve or visual pathway deficit (refer to Section [E.6. Electrodiagnostic Studies](#TBIE6ElectrodiagnosticStudies) for further description).
    7. Optical Coherence Tomography: An interferometric technique, usually with near-infrared wavelengths, used to evaluate the optic nerve pathway, cortical visual deficits, or retina, and may be done serially.
  1. OTOLOGY and AUDIOMETRY: Neurotologic evaluation is a widely used and generally accepted practice in cases of hearing loss, dizziness, balance problems, facial nerve injury, and cerebrospinal fluid leak. An individual with TBI may experience these symptoms.
     1. Audiometry: A generally accepted and well-established procedure that measures hearing. An audiologist or skilled trained technician administers the test using an audiometer. The machine presents individual frequencies to the person with TBI (typically ranging from 125–8000 Hz) at different levels of loudness (in dBHL). The individual is asked to respond to the sound at its lowest detectable intensity (threshold). Normal thresholds are from 0–25dBHL and are depicted on an audiogram. The audiologist or physician should determine the presence and type (non-organic, conductive, sensorineural, presbycusis, or mixed) of hearing loss based on the audiogram and other tests reasonably deemed necessary.  
          
        If available, obtain pre-injury baseline audiograms/audiometry studies to include a summary of past audiometric history, if known (e.g., prior hearing loss, prior tinnitus, prior vestibular problems, prior injury, etc.).  
          
        Baseline audiometry following TBI is indicated when the individual with TBI presents with hearing loss, dizziness, tinnitus, or facial nerve dysfunction.  
          
        Audiograms may be obtained in serial fashion to monitor inner ear function in response to time and treatment.
     2. Tympanometry: A generally accepted and well-established procedure that measures middle ear air pressures. It is used to help identify the presence of tympanic membrane perforations, ossicular abnormalities, and the presence of fluid in the middle ear.
     3. Vestibular Function Tests: The most common type of vertigo is benign positional vertigo, which usually does not require additional testing because it is diagnosed with the clinical Dix-Hallpike maneuver and treated with a variety of canalith repositioning maneuvers (CRM), such as Epley and Semont maneuvers ([Fife, 2000](#Fife2000)) (refer to Section [J.2.c.iv. Benign Positional Vertigo (BPV)](#TBIJ2fivBenignPositionalVertigo)). The following tests are used to verify the presence of vertigo and specify the origin when possible.
        1. Electro- or Video-Nystagmography (ENG/VNG): A generally accepted and well-established procedure that measures inner ear/central balance function. The test measures eye movement responses to inner ear balance stimulation making use of the vestibulo-ocular reflex. There are several components to the ENG/VNG. They include oculomotor testing, positional and positioning nystagmus testing and caloric testing. This series of tests may identify peripheral and central abnormalities, abnormalities in oculomotor function, positional nystagmus, and unilateral and bilateral vestibular dysfunction. The ENG/VNG can be helpful in identifying the affected ear. This test is often used in individuals with TBI complaining of dizziness or dysequilibrium and may help diagnose conditions such as labyrinthine concussion, vestibular hypofunction, and central vertigo. It is often used in conjunction with other tests such as the audiogram and clinical history to help arrive at a diagnosis.
        2. Rotary Chair Testing: A generally accepted and well-established test that evaluates the ocular responses of the inner ear to rotation. It is used to identify the extent of bilateral vestibular loss and is more accurate than VNG caloric tests for this purpose. It is also useful in assessing the ability of vision to compensate for vestibular impairments and so provides prognostic information regarding recovery.
        3. Computerized Dynamic Platform Posturography: A generally accepted and well-established test that assesses the contributions of vision, somatosensation, and the inner ear to balance control. It separately evaluates the role of lower extremity motor control to balance. It can be used to determine whether a vestibular lesion is present, but it does not localize the lesion. The purpose of this procedure is to identify the integral components of a functional balance deficit that may help in treatment planning. This technique also may be useful in monitoring neurologic recovery in individuals with TBI and balance deficits. These functional methods of evaluation are considered generally accepted practices in the evaluation of persistent vestibular and balance deficits that may require specific treatment and remediation strategies. Non-physiologic findings on this exam can result from either symptom exaggeration, anxiety, psychiatric disorders, atypical results, or malingering and should not be interpreted as malingering without other evidence. One study demonstrated positive VNG testing in a number of cases where dynamic posturography was non-physiologic ([Larrosa, 2012](#Larrosa2012)).
        4. Electrocochleography (ECoG): A well-established and generally accepted procedure that tests endolymphatic fluid pressures indirectly. It identifies the affected ear in cases of post-traumatic endolymphatic hydrops and post-traumatic perilymphatic fistula.  
             
           The inner ear has two fluid chambers—the perilymphatic and the endolymphatic. After TBI, it is not uncommon for patients to develop an increase in the endolymphatic fluid pressure; this condition is called hydrops. When the endolymphatic pressures are abnormally high, the inner ear membranes distend, and the ear malfunctions. Symptoms include hearing loss, dizziness that is sporadic, tinnitus, aural fullness, and sensitivity to sound.  
             
           The ECoG is a test that uses evoked potentials. The patient listens to a series of clicks. Monitors, including one sitting on the tympanic membrane, measure three potentials: the cochlear microphonic, the summating potential (SP), and the action potential (AP). An increase in the ratio of the summating potential of the action potential (SP/AP) suggests the presence of hydrops or perilymphatic fistula. The test varies in sensitivity and specificity. Diagnosis of endolymphatic hydrops requires a characteristic clinical picture with progressive hearing loss, fluctuating hearing, and recurrent vertigo episodes lasting for hours. See [J.2.c.i Progressive Vestibulopathy with or without Hearing Loss](#TBIJ2ciProgressiveVestibulopathy). Diagnosis should not be based solely on an abnormal ECoG test result in the absence of these clinical features
        5. Vestibular Evoked Myogenic Potentials (VEMP): A generally accepted test that evaluates the function of the saccule, one of the gravity-sensing organs of the inner ear. It is the only objective test of these organs. It is a form of auditory evoked response and is measured using the ABR and EMG equipment. A loud sound stimulus is introduced into the ear, and a vestibulo-colic reflex response from the saccule is recorded as a brief relaxation of the ipsilateral sternocleidomastoid muscle by EMG. A characteristic wave form is recorded for each ear that is analyzed for presence or absence, threshold, amplitude, and latency. Absence of a response in persons under age 60 suggests saccular damage. Reduced thresholds are indicative of semicircular canal dehiscence
        6. Other Clinical Referrals: The treating physician may often refer individuals with TBI who have balance problems to other clinicians with appropriate training in balance to assist in their assessment. The referrals may include, but are not restricted to, neuro-ophthamology, optometry, physical therapy, occupational therapy, and chiropractic therapy. There should be a coordinated approach between these disciplines and the physician specialist in the individual’s treatment.
  2. SWALLOWING EVALUATION: Swallowing impairment or dysphagia may be due to neurologic, structural, or cognitive deficits and may result from head trauma. Dysphagia may result in aspiration, airway obstruction, pneumonia, inadequate nutrition, dehydration, weight loss, failure to thrive, and death.
     1. Clinical Assessment:
        1. Clinical Bedside Assessment: This generally accepted clinical examination of oral-pharyngeal swallow function consists of pertinent medical history, examination of function of the jaw, lip, tongue, soft palate, oral sensitivity, function of pharynx and larynx, observation of dry swallow(s) and, if appropriate, with various food/liquid consistencies, ability to follow directions and discipline his/her own behaviors. If pharyngeal dysfunction or aspiration is suspected, an instrumental assessment may be indicated.
        2. Modified Evans Blue-Dye Test (MEBDT): A variation of the clinical bedside assessment used to detect the presence or absence of aspiration in an individual with tracheostomy. This procedure uses blue dye (FC&C Blue No. 1), or methylene blue placed on the tongue, or into liquids, ice chips, or food items. Aspiration is assumed if tracheal suctioning reveals blue-tinged secretions. The MEBDT has not been found reliable in identifying individuals who aspirated trace amounts (less than 10% of the bolus). Recognizing the limitations and risks of MEBDT, it is a common and practical means of screening individuals to determine readiness for cuff deflation or further swallowing evaluation.
     2. Instrumental Evaluation: Instrumental evaluations of swallow function are generally accepted diagnostic tests. They are conducted by a speech-language pathologist and physician in collaboration (radiologist, ENT or other physician familiar with the procedure as appropriate), or by the speech-language pathologist under the supervision of a physician.
        1. Modified Barium Swallow Studies (MBS) or Videofluoroscopic Study: Well-established and the most common instrumental procedure used to study swallow function. The individual’s swallowing function involving the oral cavity, pharynx, and upper esophagus is visualized while swallowing various quantities and textures of food and/or liquid containing barium contrast material.  
             
           The MBS is useful in visualizing, identifying, and documenting the presence of risk of penetration and/or aspiration and the swallowing disorder responsible for it. Specific factors assessed during the MBS may include the anatomy and physiology of the swallow, clearance of material through the mouth and pharynx, the timing of the swallow, the percentage of penetration/aspiration, effectiveness of treatment techniques and strategies to improve swallow safety and efficiency. Recommendations are made concerning the safety of oral intake, optimal delivery method for diet and hydration, diet texture/sensation modifications, therapy techniques, compensatory postures, and strategies to ensure optimum swallow safety and efficiency. Repeated studies may be needed to determine change in swallow function over time.
        2. Fiberoptic Endoscopic Evaluation of Swallowing (FEES): Used to evaluate the pharyngeal phase of the swallow with a flexible endoscope that is placed transnasally into the hypopharynx. It may be completed at bedside and may be useful in those who may not tolerate the radiographic procedure, or when such procedures are not readily available. FEES permits direct visualization of anatomy as well as vocal fold motor activity and morphology. It allows an assessment of briskness of swallow initiation, timing of bolus flow, and swallow initiation, adequacy of bolus driving/clearing forces, adequacy of velar and laryngeal valving forces, penetration or aspiration and presence of hypopharyngeal reflux.
        3. Fiberoptic Endoscopic Evaluation of Swallowing with Sensory Testing (FEEST): A modification of the FEES procedure, which adds quantification of the sensory threshold in the larynx. The sensory evaluation involves the delivery of pulses of air at sequential pressures to elicit the laryngeal adductor reflex, thus establishing a sensory threshold. Sensory testing is a quantifiable indicator of those persons at risk for aspiration. It provides better understanding of laryngeal sensory deficits, which may be useful in dietary and behavioral management of individuals with dysphagia.
        4. Manofluorographic Swallowing Evaluation (MSE): A videofluoroscopic swallowing study with the addition of an oropharyngeal pressure assessment. Solid state pressure transducer sensors are typically placed in the esophagus, upper esophageal sphincter (UES), hypopharynx, and tongue base. Manometry provides quantitative information at rest and during swallowing on pharyngeal, UES, and esophageal pressures, completeness of UES relaxation, and coordination of timing between pharyngeal contraction and UES relaxation.
  3. SPECIAL TESTS for RETURN-TO-WORK ASSESSMENT: A return-to-work format should be part of a company’s health plan, knowing that return to work can decrease anxiety, reduce the possibility of depression, and reconnect the worker with society. Evaluations used to define these abilities, such as the functional capacity evaluation (FCE) and the worksite analysis, should be objective.The professional performing the FCE and worksite analysis should be specifically trained and familiar with the unique presentation of the individual who has suffered a TBI. The ability to tolerate these evaluations and follow commands may be limited due to TBI and should not be construed as non-cooperative or suggestion of malingering.   
       
     Caution should be used in returning an individual to work and other activities during the first 3–14 days after MTBI. Both physical and cognitive duties should generally be non-stressful initially, with a gradual increase in activity based on improvement and/or resolution of symptoms. The individual should be competent in most basic ADLs before return to work is considered. Return to full duty depends on the rate of decrease of symptoms. Generally, if symptoms recur during increasing job duties or exertion, duties should be decreased accordingly. Because a prolonged period of time off work will decrease the likelihood of return to work, the first weeks of treatment are crucial in preventing and/or reversing chronicity and disability mindset. In complex cases, experienced nurse case managers or occupational therapists may be required to assist in return to work. Other services, including psychological evaluation and/or treatment and vocational assistance should be employed. Two evaluations that may be used are:
     1. Job Site Evaluations and Alterations: For many patients with TBI, job alterations may be needed. These may be in the form of: (1) instructing the worker how specific duties might be performed to avoid excessive mental stress; (2) actual job worksite or duty changes; and /or (3) a formal job site evaluation and alterations at the worksite.  
          
        Job site evaluation and alteration should include input from a health care professional with experience with TBI cases, the employee, and the employer. The employee should be observed performing all job functions in order for the job site evaluation to be a valid representation of a typical workday.  
          
        A formal job site evaluation is a comprehensive analysis of the physical, mental and sensory components of a specific job and may be important initially to determine causation. These components may include, but are not limited to: (a) postural tolerance (static and dynamic); (b) aerobic requirements; (c) ROM; (d) torque/force; (e) lifting/carrying; (f) cognitive demands; (g) social interactions; (h) interpersonal skills management; (i) visual perceptual challenges; (j) environmental requirements of a job; (k) repetitiveness; and (l) essential functions of a job.  
          
        Changes that provide a therapeutic benefit or relieve the patient’s ongoing symptoms are part of the required medical treatment for TBI and therefore, it is assumed that the insurer will be responsible for paying for reasonably necessary job site alterations.  
          
        Job descriptions provided by the employer are helpful but should not be used as a substitute for direct observation.  
          
        A job site evaluation may include observation and instruction of how work is done, what material changes should be made, and determination of readiness to return to work. Refer to the Chronic Pain Guidelines.  
          
        Requests for a job site evaluation should describe the expected goals for the evaluation. Goals may include, but are not limited to, the following:

● Provide a detailed description of the physical and cognitive job requirements,

● Make recommendations for and assess the potential for job site changes,

● Assist the patient in their return to work by educating them on how they may be able to do their job more safely, and/or

● Give detailed work/activity restrictions.

Frequency: One time with additional visits as needed for follow-up.

* + 1. Functional Capacity Evaluation (FCE): May be indicated to identify residual physical limitations. FCE is a comprehensive assessment of the various aspects of physical and cognitive function as they relate to the individual’s ability to perform functional activities necessary for return to work. When cognitive, emotional and/or behavioral sequelae are also present, a comprehensive FCE may provide indications of return-to-work readiness.  
         
       Components of the physical portion of the FCE may include, but are not limited to: musculoskeletal screen, cardiovascular profile/aerobic capacity, coordination, lift/carrying analysis, job specific activity tolerance, maximum voluntary effort, pain assessment, non-material and material handling activities, balance/dizziness, climbing, physical fatigue, endurance, and visual skills. The physical portion of any FCE should include all of the physical skills required for specific job placement.   
         
       Components of the cognitive portion of the FCE may include, but are not limited to: memory, executive skill function, attention and concentration, communication, speed of information processing, multi-tasking, new learning, and cognitive fatigue and endurance.  
         
       Components of the emotional portion of the FCE may include, but are not limited to: temperament, ability to manage stress, adaptation to change, mood changes, toleration of feedback, and anger control.  
         
       Components of the behavioral portion of the FCE may include, but are not limited to: appropriate social and behavioral interactions. This may present as inability to complete or cooperate with the tests, inconsistent or erratic behavior, or the inability to get along with coworkers and supervisors.  
         
       FCEs include tools that are an extension of the basic medical examination and may be useful for the determination of impairments, functional/cognitive restrictions, determination of progress, and planning and monitoring of the rehabilitation program. Whenever possible, FCEs should be supplemented with information from neuropsychology, speech/language pathology, occupational therapy, and physical therapy to determine physical, cognitive, and psychological abilities in order for the patient to function safely and productively in a work setting. FCEs are typically conducted in four to six hours, but for individuals who have suffered a TBI, additional time may be required, or it may be necessary to conduct the evaluation in two or three separate sessions to allow for the potential variability of cognitive and physical fatigue. Total time for an FCE would rarely exceed eight to ten hours.  
         
       When an FCE is being used to determine return to a specific jobsite, the provider is responsible for fully understanding the job duties. A jobsite evaluation is frequently necessary. FCEs cannot be used in isolation to determine work restrictions. The authorized treating physician must interpret the FCE in light of the individual patient’s presentation and medical and personal perceptions. FCEs should not be used as the sole criteria to diagnose malingering.  
         
       Frequency: Can be used: (1) initially to determine baseline status; and (2) for case closure when the patient is unable to return to the pre-injury position and further information is desired to determine permanent work restrictions. Prior authorization is required for FCEs performed during treatment.

1. ACUTE THERAPEUTIC PROCEDURES – NONOPERATIVE
   1. RESUSCITATION:
      1. The first priority in TBI is complete and rapid physiologic resuscitation.
      2. Special considerations for isolated communities without neurosurgical support:
         1. Trauma surgeons and emergency physicians may perform the initial resuscitation and neurologic treatment in the deteriorating individual.
         2. Once the individual is stable, transport to a designated neuro-trauma center for further evaluation and management should occur expeditiously.
   2. INTRACRANIAL PRESSURE (ICP) AND CEREBRAL PERFUSION PRESSURE (CPP):
      1. ICP Monitoring is indicated in individuals with low GCS (less than 9) or when the individual cannot have continual neurologic evaluation (e.g., use of anesthesia, pain medicine for other injuries that preclude a neurologic exam), and it should also be considered when the individual’s age is over 40 or systolic blood pressure is less than 90 mmHz ([Bratton, 2007a](#Bratton2007a)). ICP monitoring may be done by a variety of technologies, but a ventriculostomy is the most accurate. Other options include parenchymal monitors placed in the supratentorial cranial vault.
      2. Cerebral oxygen saturation monitoring may be used, usually in conjunction with ICP monitoring, to assess the effects of treatment interventions on oxygen delivery to the injured brain, and to optimize the management of brain swelling and intracranial pressure in the setting of severe TBI.
   3. HYPERVENTILATION:  
        
      Hyperventilation is generally not recommended in the setting of acute TBI.  
      In rare cases, controlled hyperventilation may be necessary for brief periods in acute neurological deterioration not attributable to systemic pathology (i.e., hypotension), but it is not recommended for prolonged periods of time ([[Cochrane] Roberts, 2009](#Roberts2009)).
   4. MEDICATIONS:
      1. Hyperosmolar agents may be used prior to ICP monitoring if there is neurologic deterioration not attributable to systemic pathology (i.e. hypotension) and/or signs of transtentorial herniation ([Bratton, 2007b](#Bratton2007b)).
      2. Glucocorticoids (steroids) are not useful or generally accepted to improve outcome or decrease ICP, and in some instances may be harmful. There is good evidence that they do not decrease mortality, and there is some evidence that they may even increase the mortality rate in individuals with TBIs ([CRASH Trial Collaborators, 2004](#CRASH2005)).
      3. **Anticonvulsants**:
         1. Anticonvulsant treatment may be used to prevent early post-traumatic seizures in the high-risk individual and are usually administered for one week in those with intracranial hemorrhage. Prevention of early seizures has no statistically significant impact on long-term outcome or the development of late seizures or chronic epilepsy ([Chang, 2003](#Chang2003)). Prevention of early seizures is reasonable to reduce seizure-associated complications during acute management ([Chang, 2003](#Chang2003)).
         2. Prophylactic Anticonvulsants: Should not be used routinely after the first week unless other clinical indicators warrant their use, such as brain penetration, excessive intraparenchymal bleeding, or others ([Chang, 2003](#Chang2003)).
      4. **Progesterone**:
         1. There is good evidence that progesterone in the setting of acute TBI can reduce mortality and disability, although most patients with severe TBI may not avoid residual disability,andthe studies do not yet support routine use ([[Cochrane] Junpeng, 2011](#Junpeng2011)). Ongoing studies may change this recommendation.
   5. HYPOTHERMIA: Therapeutic hypothermia involves the lowering of core body temperature by such techniques as surface heat exchange devices, intravascular infusion of cold crystalloid, and body cavity lavage ([Seder, 2009](#Seder2009)). This is done in order to decrease some metabolic and physiologic processes that result in neural damage after TBI, including increased intracranial pressure. Studies of its effectiveness have varied widely regarding the timing, depth, and duration of hypothermia and the rate of rewarming, and studies have differed significantly in estimates of its therapeutic value. Study quality has also influenced reporting of outcomes, with higher quality studies reporting less favorable effects on mortality and function than lower quality studies ([[Cochrane] Sydenham, 2009](#Sydenham2009)). A recent trial of early hypothermia induction sponsored by the National Institutes of Health ([Clifton, 2011](#Clifton2011)) was terminated early due to concerns of the monitoring board over issues of slow accrual and patient safety. The interim analysis suggested an increased risk of a poor outcome in patients with diffuse brain injury but a reduced risk of a poor outcome in patients with surgically removed hematomas, but the number of outcomes were too few to exclude chance as an explanation of the results.  
        
      Because of the complexities of the determinants of outcome of hypothermia, recommendations cannot be made regarding its routine use. Decisions for or against its use must be made on a case-by-case basis according to factors of severity of injury, time since injury, level of intracranial pressure, the presence of other injuries, and other circumstances. Side effects include immunosuppression, cold diuresis with hypovolemia, electrolyte disturbances, impaired drug clearance, and mild coagulopathy ([Polderman, 2009](#Polderman2009)).  
        
      In contrast to the appropriateness of induced hypothermia, there is general agreement that fever with the TBI patient is associated with poor long-term outcomes and should be monitored and managed ([Badjatia, 2009](#Badjatia2009)).
   6. Surgery:   
        
      In many cases, surgery is appropriate (refer to section [L. Operative Therapeutic Procedures](#TBILOperativeTherProcedures) for details).
   7. Hyperbaric Oxygen: Despite evidence of limited physiological changes with hyperbaric oxygen, there is insufficient evidence to suggest that hyperbaric oxygen would functionally benefit stroke or TBI patients ([[Cochrane] Bennett, 2004](#Bennett2004); [[Cochrane] Bennett, 2005](#Bennett2005); [Rockswold, 2010](#Rockswald2010)). Complications can occur, including tension pneumothrorax (Lee, 2012). Hyperbaric oxygen is not recommended acutely or chronically. Ongoing studies could affect this recommendation.
2. NONOPERATIVE THERAPEUTIC PROCEDURES – INITIAL TREATMENT CONSIDERATIONS

Due to the complex nature of the brain, individuals with TBI require coordinated interdisciplinary treatment. Usually, the impairment(s) and functional limitations are appropriately treated by more than one therapeutic discipline. Treatment should emphasize functional, outcome-oriented, and community reintegration goals. Treatment session duration and frequency will vary depending on the individual’s tolerance and may evolve over time. The location of treatment sessions may be in a clinical setting initially, but eventually may be more effective in the home, workplace, or community, based on functional goals. Moderate/severe TBI may result in lifetime deficits, so a long-term disability management model is appropriate. Frequency and duration of specific, non-acute treatments should be included in every treatment plan and should be re-evaluated approximately every four weeks (refer to Section [B. General Guideline Principles](#TBIBGeneralGuidelinePrinciples)). Experienced practitioners should not use all of the therapies and modalities listed in this guideline. Periodic modification or consultation may be necessary throughout an individual’s lifetime following TBI. Therapy for specific impairments and functional limitations may be reinitiated for goal-specific, time-limited treatment as new goals are identified and developed. Treatment should be based on medical diagnosis and associated impairment, cognitive ability, clinical evaluations, anticipated functional gains, and progress demonstrated by documented functional outcomes.

* 1. PATIENT/FAMILY/SUPPORT SYSTEM EDUCATION: Education for individuals with TBI and their family and/or support system is appropriate, generally accepted, and widely used in TBI rehabilitation ([Veterans Affairs, Department of Defense [VADoD], 2009](#VetsDOD2009)).
     1. MTBI: Most cases will progress to recovery with sufficient education and not require interdisciplinary treatment ([Zafonte, 2006](#Zafonte2006)).

❖ Frequency: Weekly one-hour sessions initially during the first month as part of the primary treatment and return-to-work evaluations. Once the patient has returned to normal function without impairing symptoms, visit frequency should decrease or treatment should be terminated.

❖ Optimum Duration: 1 to 3 months.

❖ Additional sessions may be required as justified.

* + 1. Moderate/Severe TBI: Formal treatment team conferences involving the individual with TBI, family and/or support system individuals and case managers, including insurance case manager, should be held regularly during the inpatient, residential, neurobehavioral, and outpatient phases of rehabilitation and periodically during the home and community-based phases of community reintegration. Education may include, but may not be limited to, brain-behavior relationships, health issues related to TBI and co-morbid illnesses or injury, family and/or support system interventions, emotional adjustments, and family and/or support system roles changes. Families and/or support systems and individuals with TBI require education, support, and caregiver training as part of the long-term maintenance plan. Education for the individual and family and/or support system is typically provided by case managers, social workers, rehabilitation counselors, family counselors, licensed mental health professionals, and/or nurses.

❖ Frequency and Duration: May require daily one-hour sessions for the first month.

● Up to twice weekly for 2 to 3 months.

● Up to twice monthly for 6 months.

● Monthly for an additional 6 months.

❖ Additional sessions may be required as justified.

* 1. BEHAVIOR: Moderate/Severe TBI – The neuropathological deficits occurring in TBI often result in behavioral changes and deficits in the skills needed to: (1) monitor and control one’s behavior; (2) interpret the behavior of others; and (3) respond effectively to social situations. Functional limitations and behavioral disabilities include: deficits regarding functional skills, insight judgment, self-monitoring, and behavioral and emotional regulation. These deficits may be compounded by secondary emotional reactions such as depression or anxiety. Behavioral therapy is a well-accepted and widely used therapy for TBI; it acknowledges that behavioral problems are always multi-factorial and therefore should consider medical, neurosurgical, neurological, psychiatric, environmental, and psychosocial issues. Behaviorally-based therapies rely on an interdisciplinary treatment team approach and are frequently implemented in conjunction with cognitive and/or other psychological treatment. A behavioral therapy plan should be approved and monitored by a neuropsychologist, psychologist, behavior analyst, or physician familiar with TBI.   
       
     Post-traumatic neuropathologically-based behavioral problems may be exacerbated by co-morbidities such as personal history, personality or family and/or support system issues, psychiatric illnesses, cognitive impairment, medication side effects, and substance abuse. Successful resolution of behavioral problems will usually require treatment of these associated co-morbidities. Behavioral problems are also influenced by developmental issues. Treatment requires appropriate consideration of developmental and life stage issues (i.e., adolescent, elderly). Treatment may require specialized treatment settings with professionals experienced in the management of these populations. Depending on the severity of the behavior problem, treatment may require focused applied behavioral analysis available only in a specialized rehabilitation or psychiatric setting. In less severe situations, applied behavior analysis can be provided in outpatient and community settings. Behavioral analysis and treatment involves:

● Identification and prioritization of undesirable or negative target behaviors to be managed or extinguished.

● Identification of behavioral strengths and positive/desirable target behaviors (frequently called alternative, competing, or replacement behaviors) to be encouraged and positively reinforced.

● Analysis and modification of environmental variables to reduce antecedents or precursors of maladaptive behaviors (i.e., loud noise, crowds, requests to do non-preferred activities, changes in daily routines). Analysis and modification of internal precursors of maladaptive behaviors (i.e., pain, sleep-deprivation, anxiety, helplessness, depression, thought disturbance) and environmental issues to reduce antecedents or precipitants of maladaptive behaviors.

● Analysis of the function of maladaptive behavior and developing strategies that replace the need to engage in maladaptive behavior (i.e., teaching and reinforce asking for assistance instead of yelling or aggression).

● Progressive refinement of the strategies of internal and environmental modifications in response to an analysis of changes in behavior.

● Extensive training and monitoring of treatment plan adherence for all treating staff and family and/or support system interacting with the patient during neurobehavioral interventions.

Effective behavioral management and treatment requires individualized approaches. The setting of treatment should consider individual resources and circumstances. Inpatient and outpatient settings may require one-on-one supervision at critical phases of recovery. Coordination of treatment resources and professionals are essential, as well as training the family and/or support system and other caregivers in the behavioral plan, as treatment consistency in all environments is an important variable in the behavioral treatment outcome. Analysis of the environment and personnel during periods of transition between treatment settings is generally essential to minimize the stress of change and to avoid the loss of critical environmental reinforcers and learned behavior-reinforcer relations.  
  
In long-term maintenance programs, treatment may be appropriate on an episodic basis as follows: treatment may be ‘on hold’ for several weeks or months until certain goals are reached or until additional goals emerge. At such times, therapy may be restarted for a time-limited, goal-specific treatment as prescribed and routinely monitored by a neuropsychologist, psychologist, behavior analyst, or physician familiar with TBI. Progress should be re-evaluated and documented every four weeks (refer to Section [B. General Guideline Principles](#TBIBGeneralGuidelinePrinciples)).

* 1. COGNITION: The “process of knowing” by which individuals: (1) make decisions as to the most functional ways of interacting with their environment; (2) execute those decisions; (3) monitor their responses to determine appropriateness and accuracy of their decisions; and (4) adjust their behavior if it is determined to be inappropriate and/or inaccurate. Deficits in cognition are a frequent result of TBI, may persist, and may vary from mild to severe. Cognitive processes that are often impaired after TBI may include, but are not limited to:

● Impaired arousal and attention.

● Inefficient processing of information (rate, amount, and complexity).

● Impaired perception of sensory (auditory, visual, olfactory, and tactile) information.

● Impaired acquisition, retention, and retrieval of verbal and visual information, which impairs new learning and memory skills.

● Impaired executive functioning skills: problem solving, insight, reasoning and judgment, self-awareness and evaluation (including awareness of strengths and weaknesses), goal setting, planning, organizing, initiation , self-inhibiting (or disinhibition and self-monitoring).

● Impaired or inappropriate social awareness and behavior.

There is some evidence that a cognitive program aimed at high order reasoning instruction is likely to improve some aspects of executive function (e.g. working memory, inhibition, switching tasks) for chronic TBI individuals ([Vas, 2011](#Vas2011)). There is some evidence that intensive therapy, 15 hours/week for 16 weeks in a group setting emphasizing integration of cognitive, interpersonal, and functional gains, is superior to the same amount of therapy from multiple individual providers ([Cicerone, 2008](#Cicerone2008)).  
  
There is good evidence that structured, goal-oriented, individualized multidisciplinary cognitive rehabilitation for patients requiring hospitalization improves mobility, personal care, and independence in ADLs for individuals with TBI [([Cochrane] Turner-Stokes, 2005](#TurnerStokes2005)). Improvement in mobility and independence significantly reduces indirect costs over a long period of time, which may not be measured accurately in the relatively short periods during which most clinical studies are conducted. Cognitive rehabilitation is recommended by the VADoD for treatment of individuals with TBI with cognitive deficits ([VADoD, 2009](#VetsDOD2009)).

* + 1. MTBI: In MTBI, acute cognitive deficits are common, and spontaneous cognitive improvement is expected within the first three months, frequently within days or weeks, in the majority of injured individuals ([McCrea, 2009](#McCrea2009)). There is good evidence that MTBI without post-traumatic amnesia does not routinely require multidisciplinary rehabilitation ([[Cochrane] Turner-Stokes, 2005](#TurnerStokes2005)). There is some evidence that routine scheduling for cognitive rehabilitation for uncomplicated MTBI is not likely to improve outcomes and that MTBI cases with a psychiatric history are more likely to benefit from routine assessment for cognitive rehabilitation treatment ([Ghaffar, 2006](#Ghaffar2006)). Compensatory memory strategies are useful in this population ([Sohlberg, 1987](#Sohlberg1987)).  
         
       Rehabilitation of cognitive impairments should only be initiated if:

● The individual is not demonstrating the expected cognitive improvement.

● The individual exhibits more severe cognitive impairments on formal evaluation.

● The individual’s vocation or other life circumstances necessitate the learning of compensatory strategies.

● There are safety issues in question (e.g. possible harm to self or others).

If therapy is required for these patients use the following timelines,

❖ Frequency: Weekly one-hour sessions initially during the first month as part of the primary treatment and return-to-work evaluations. Once the patient has returned to normal function without impairing symptoms, visit frequency should decrease or treatment should be terminated.

❖ Optimum duration: 1 to 3 months.

❖ Maximum duration: Additional sessions may be required as justified., For example in cases with complicated TBI or a number of co-morbid conditions treatment patterns may resemble that for moderate/severe injuries.

* + 1. Moderate/Severe TBI: In individuals with moderate/severe TBI, rehabilitation of cognitive deficits is appropriate, clinically necessary, and based on evidence. Rehabilitation is most beneficial when an individual demonstrates adequate arousal, appropriate responsiveness to stimulation and at least a minimum ability to focus attention (e.g. fully oriented). Prior to demonstration of these skills, rehabilitation efforts should focus on monitoring and attempting to elicit responses, environmental structuring (e.g. maintaining a normal sleep/wake cycle), and staff/family and/or support system education.  
         
       Rehabilitation includes procedures designed to improve cognitive efficiency, develop specific cognitive skills, enhance awareness of impairments and skills, and develop appropriate compensation strategies for residual cognitive deficits. Individuals with MTBI and memory deficits are more likely to improve with compensatory memory strategies training than individuals with moderate/severe TBI who may require memory notebooks or other external aids to improve memory ([Sohlberg,1987](#Sohlberg1987)).  
         
       Rehabilitation treatment for cognitive deficits may be provided by speech-language pathologists, neuropsychologists, occupational therapists, music therapists, physical therapists, or paraprofessionals closely supervised by these professionals. It frequently may be necessary for other disciplines to apply cognitive rehabilitation techniques while addressing non-cognitive goals (i.e., mobility and daily nursing activities). A cognitive therapy plan should be approved and monitored by a speech-language pathologist, neuropsychologist or physician experienced with TBI. Physicians may also be involved in pharmacological treatment and management of cognitive disorders. Family and/or support system members, caregivers, and partners should always be included in the therapy plan. Thus, therapy is routinely multi-disciplinary for patients with moderate/severe TBI.   
         
       There is good evidence that this type of multi-disciplinary rehabilitation of moderate/severe TBI patients is likely to provide functional and symptomatic benefit once the patient is able to participate ([[Cochrane] Turner-Stokes, 2005](#TurnerStokes2005)).  
         
       There is some evidence that a multi-faceted cognitive rehabilitative intervention focused on aspects of executive function can lead to lasting improvement (Spikeman, 2010). In this study, group treatment sessions occurred twice per week for one hour over a period of three months and were focused on self-awareness, self-initiation, goal setting, planning, flexibility, strategic behavior, self-monitoring, and self-inhibition.   
         
       Social communication skills training is also appropriate for moderate/severe TBI. There is some evidence that group instruction, 90 minutes weekly over 12 weeks, by a skilled leader results in improved communication skills ([Dahlberg, 2007](#Dahlberg2007)). Also refer to Section [J.4. Communication](#TBIJ4Communicaton) for further information.  
         
       There was some evidence from an older study of young military patients with moderate/severe TBI who could safely live at home without continual supervision, that psychological treatment in a supported home environment had similar results to inpatient multidisciplinary treatment ([Salazar, 2000](#Salazar2000)). The applicability of this evidence to those with work related injuries is questionable, and this guideline recommends that moderate/severe TBI cases receive interdisciplinary therapy as deemed appropriate for the individual rather than isolated home programs.   
         
       Rehabilitative treatment is indicated following a neuropsychological and/or neurological evaluation that identifies cognitive impairments. The evaluation should include statements of TBI severity and prognosis for improvement, outline recommended goals/objectives and methodologies of treatment, and establish frequency and duration parameters.  
         
       A treatment plan outlining current goals is recommended with each evaluation. If documented improvement is not shown, the treatment goals and program should either be modified or discontinued. Periodic upgrading or consultation may be necessary throughout a lifetime following TBI. Therapy may be re-initiated for time limited, goal-specific treatment as new goals or TBI related problems develop.

❖ Frequency: Acute and post-acute – daily. Sub-acute outpatient and home/community setting – daily to weekly.

❖ Optimum Duration: Typically 8 weeks with evaluation at the 4-week mark.

❖ Maximum Duration: Beyond 8 weeks requires documentation of progress with the exception of periodic consultations and new treatment goals.

* + 1. Computer-Based Treatment: The use of computers as a primary and independent form of treatment in cognitive remediation has limited application because of: (1) limitations in the rationale and specific application of software programs to address the needs of the individual with TBI and (2) difficulty with generalization of learned computer skills into functional environments. Integrated computer-based treatment (i.e., both individualized cognitive and interpersonal therapies) may improve functioning within the context of an interdisciplinary, neuropsychological rehabilitation program. Computer-based interventions that include active therapist involvement to foster insight into cognitive strengths and weaknesses, development of compensatory strategies, and facilitation of transferring skills into real-life situations may be used as part of a multi-modal intervention for cognitive deficits. Sole reliance on repeated exposure and practice on computer-based tasks without extensive involvement and intervention by a therapist is not recommended.
    2. Assistive Technology: A variety of devices are available to assist individuals with language and functional problems. These should be trialed within a rehabilitation therapy program by physical therapists, occupational therapists and speech-language therapists, to determine which tools are most suitable for individual cases.
  1. PSYCHOLOGICAL/EDUCATIONAL INTERVENTIONS: Psychological and educational interventions may include, or be performed in conjunction with, cognitive and behavioral treatment. Cognitive behavioral therapy (CBT) is a specialized goal-oriented systematic process used to problem solve that focuses on changing thought processes and is usually provided by a trained therapist or psychologist. One study noted sustained improvement after six months with either face-to-face or telephone contact. This should always be performed within the construct of a fuller therapy program ([Arundine, 2012](#Arundine2012)).  
       
     The acute symptoms of MTBI (e.g. feeling dazed, disoriented, or confused) overlap with those of emotional trauma and acute stress disorder. Over the course of recovery, the symptoms of MTBI also overlap with a variety of psychological conditions, such as depression, anxiety, insomnia and PTSD. Consequently, when the degree of cognitive symptoms exceeds what would be expected given objective findings, the mechanism of injury, or acute signs of MTBI, or if there is an unexplained, marked worsening of cognitive symptoms over time, the possibility that the symptoms are psychological rather than neuropsychological in origin should be evaluated. A psychological evaluation is especially important if the injury occurred in an emotionally traumatic context, or if there are clinical indications of another mental health disorder.
     1. Acute Psychological/Educational Interventions in MTBI: Early interventions that educate individuals, their family and/or support system, or the employer about the symptoms, natural history, prognosis, and management of MTBI symptoms are very important. Psychological interventions to educate the individual and family and/or support systems regarding coping may occur with the individual and family and/or support system, or alternatively with close friends and co-workers.   
          
        When certain risk factors are present, psychological interventions are appropriate to promote positive coping skills and to manage symptoms. Risk factors include the following: usual recovery does not occur, history of previous TBIs, the desire to return to a highly demanding job, significant injury stress, pre-injury psychiatric disorder, pre-injury learning disability, PTA greater than four to six hours, loss of consciousness greater than ten minutes, chronic pain, substance abuse, poor psychosocial support, depression, or associated orthopedic injuries ([Ghaffar, 2006](#Ghaffar2006)). The presence of other injuries requiring medical attention should not exclude anyone from appropriate psychological treatment. When licensed mental health professionals other than psychologists or psychiatrists are providing treatment, their therapy plan should be overseen by a psychologist, neuropsychologist, or psychiatrist.
     2. Problem-Specific Referrals During the First Three Months Following MTBI: Mental health services are appropriate to address specific problems that are directly caused by the injury (e.g., memory deficits, slowed speed of thinking, difficulties with decision making, irritability and fatigue) or that are secondary to the injury (e.g., anxiety, depression, adjustment disorder, difficulties with self-acceptance, and difficulties in adapting one’s work demands due to diminished cognitive capacity). Post-traumatic stress disorder (PTSD) may be present in a minority of MTBI patients and should be assessed early on and treated ([Hoffman, 2012](#Hoffman2012)). Mental health interventions to address PTSD may include individual psychotherapy, cognitive/behavioral therapy, instructions in specific techniques such as relaxation training or biofeedback, instruction in symptom management, trauma resolution techniques (e.g., EMDR), group therapy, medications, and interventions in the community.  
          
        Therapists or speech-language pathologists may work with the individual with TBI in their own home or other community settings in order to teach individuals adaptive skills, compensatory techniques, or new ways of solving problems that assist them in coping more effectively during recovery. Treatment generally includes cognitive therapy to enhance attention/concentration, reduce distractibility, improve confidence in cognitive abilities, and teach practical decision-making strategies to enhance coping and reduce stress. The interdisciplinary treatment team approach is particularly beneficial in these cases and it is strongly encouraged, especially during the first three months post-injury. Medications may be helpful to address the individual’s symptoms (refer to Section [F.4. Medications](#TBIF4Medication)).
     3. Referrals Three or More Months Post-MTBI: A referral for psychological and/or psychiatric services should strongly be considered at three or more months post-injury when the individual is having difficulty coping with symptoms or stressors, or when secondary psychological symptoms such as intolerance to certain types of environmental stimuli or anxiety or depression are hindering recovery and return to pre-injury level of function. Pre-existing personality traits (e.g. perfectionism, dependency, overachiever or personality disorders), demanding responsibilities, or lack of experience on the current job may also interact with cognitive deficits and other symptoms to necessitate the provision of ongoing psychotherapeutic services. Treatment may include individual psychotherapy, marital/family therapy, group therapy, instruction in relaxation and related techniques, cognitive/behavioral therapy, medication management, social skills training, and interventions/consultation in the community.
     4. Functional Gains: To be documented and achieved with therapy and may include, but are not limited to, improved mood, irritability, frustration tolerance, concentration, memory, sleep quality, and interpersonal skills such as empathy and capacity to effectively interact with family and/or support system members and co-workers.  
          
        Time intervals for suggested treatment

❖ Frequency: Weekly one-hour sessions initially during the first month as part of the primary treatment and return to work evaluations. Once patient has returned to normal function without impairing symptoms, visit frequency should decrease or treatment should be terminated.

❖ Optimum Duration: 1 to 3 months.

❖ Additional sessions may be required as justified.

* 1. PSYCHOLOGICAL INTERVENTIONS – MODERATE/SEVERE TBI: Moderate/severe TBI may result in a variety of cognitive, psychological and/or behavioral symptoms which, if left untreated, can negatively impact each other, recovery from TBI, and/or functional outcomes; therefore, psychological treatment is recommended for patients with any of these symptoms ([Hudak, 2012](#Hudak2012)). Psychological interventions may include, or be performed in conjunction with, cognitive and behavioral treatment. Although the effect of this treatment for the brain injured population is unknown, psychological treatment is recommended for all patients with psychological symptoms.
     1. Acutely Symptomatic Phase: During the period of PTA, self-awareness is often compromised, and behavioral problems such as impulsivity, agitation, uninhibited behaviors, aggression, and confabulation may emerge. At this stage, psychological interventions are typically focused on: (1) development of specific environmental strategies to manage problematic behaviors and increase the safety of the individual and staff; (2) consultation with other team members, support of the nursing staff, and ongoing contacts with the individual’s family and/or support system; and (3) education of the family and/or support system about the TBI and its behavioral manifestations. Cognitive status is monitored during this time period as the level of environmental stimuli is gradually and slowly increased. The psychological interventions described here typically occur throughout the period of PTA. Furthermore, psychological intervention to help manage problematic behaviors, such as perseveration, aggressive behaviors, and disorders of memory, typically continues into the acute rehabilitation phase of treatment as PTA resolves. Behavior treatment, which may include applied behavior analysis and a focused behavioral plan based on the results of a functional analysis, is frequently used in these cases. Psychological interventions may be delivered by licensed mental health clinicians.
     2. Early Recovery Phase: Once PTA has completely resolved and the TBI patient is fully oriented in all spheres, psychological clinical services are typically provided to educate him/her about the injury, increase insight into deficits and support the development of positive coping. Treatment also typically involves psychotherapeutic intervention to assist in dealing with feelings of anxiety, loss, frustration and grief. Psychological treatment is often required to address depression, heightened irritability, sleep disturbance, and anxiety. Psychological interventions including psychotherapy, sleep hygiene, cognitive behavior modification, and environmental restructuring may be required to address social skills, behavioral deficits, and impulsivity excesses. In addition to psychological services provided directly to the individual, consultation by licensed mental health professionals with other team members is appropriate and encouraged in order to train team members and family and/or support system members to support the process of recovery. Family therapy and educational sessions are often indicated.
     3. Stabilization Phase: Once the individual’s condition has stabilized, the goals of psychological treatment center on supporting the transition to, and functioning within, the community. Alterations in cognitive and emotional functioning (e.g., mood disorders, emotional lability, irritability, preservative and disinhibited impulsive behaviors, apathy, memory problems and disorders related to diminished or impaired judgment) may necessitate ongoing psychological treatment. Individuals with TBI typically receive psychological services before discharge from the hospital in order to address specific deficits and abilities that will play an important role in successful functioning in their home and community. Depression may be linked to permanent decrease in functioning after treatment ([Hudak, 2012](#Hudak2012)). These services are typically individualized and may take a variety of forms including individual psychotherapy, skills training (e.g., parenting), marital/family psychotherapy, medication management, and group psychotherapy.  
          
        Treatment should be specially tailored to the needs of the individual and his/her cognitive deficits. For example, an individual with significant memory problems may need to have information from psychotherapy sessions video or audio recorded. Depending on the severity of the behavioral problems, outpatient psychotherapy may be held initially as frequently as once a day for severe problems (e.g., rage reactions, sexually disinhibited behaviors, or other behaviors that constitute safety risks). Sessions may occur several times a week to address adjustment issues in a psychotherapeutic approach.  
          
        In view of the fact that deficits from moderate/severe TBI can persist throughout life, intermittent mental health interventions may be required during the course of the individual’s lifetime in order to address the behavioral problems and emotional distress that may arise secondary to developmental issues, the onset of medical/neurologic/psychiatric co-morbidities, or changes in environmental structure. Periodic upgrading or consultation may also be necessary throughout a person’s lifetime following TBI. Therapy may be reinitiated for time limited, goal-specific treatment as new goals or TBI related problems develop.
     4. Consultation in Regard to Usage of Medications: Medication management for emotional, behavioral, cognitive and physical functioning, for moderate/severe TBI patients is often needed. An interdisciplinary team approach is beneficial and encouraged. Thus, attending physicians will often request consultation from other physicians, including psychiatrists, and non-physician team members, such as psychologists, social workers, and family service counselors, to provide data and input regarding behavioral observations that may assist in assessing how the person is responding to various medications.
  2. MEDICATION/Pharmacological Rehabilitation: The use of medications requires careful monitoring and collaboration between the individual, physician, family and/or support system, and other members of the interdisciplinary team. Common symptom categories targeted for medication treatment may include, but are not limited to:

● Pain (headache, axial, soft tissue, etc.) (refer to Section [G.7. Headache](#TBIG7Headache) of the Chronic Pain Guidelines).

● Sensory alterations (dysesthesias).

● Motor symptoms (motor control, coordination, spasticity, weakness, Parkinsonism, tremor, etc.).

● Emotional conditions (depression, lability, anxiety, etc.).

● Behavioral problems (poor self-monitoring, dyscontrol, irritability, aggression, poor initiation, etc.).

● Cognitive issues (arousal, attention, speed of processing, memory, executive function, fatigue).

● Psychotic symptoms (disturbances of thought content such as hallucinations and delusions, thought process, and thought disorganization, which can contribute to behavioral problems).

● Neurological issues (seizures, paroxsysmal sympathetic hyperactivity, etc.).

● Disturbances of sleep (insomnia, hypersomnia, sleep-wake cycle reversals).

● Endocrine dysfunction.

There is no single formula for pharmacological treatment of patients with acute, sub-acute, or chronic problems due to TBI of any level of severity. A thorough medication history, including use of alternative and over-the-counter medications, should be performed initially and when medication changes are made. The medication history may consist of gathering corroborating information from caregivers and prescribing pharmacies, particularly if the individual has memory or other deficits which may impair their ability to accurately report their medications and adherence to the prescriber. Appropriate application of pharmacological agents depends on the patient’s age, past history (including history of substance abuse), drug allergies, and all medical problems. It is incumbent upon the healthcare providerto thoroughly understand pharmacological principles when dealing with the different drug classes, their respective side effects, drug interactions**,**bioavailability profiles, and the primary reason for each medication’s usage. Patients and their caretakers should be aware that medications alone are unlikely to provide complete symptom relief. A primary goal of drug treatment is to improve the patient’s function as measured behaviorally. Essential elements of post-traumatic deficits require continuing participation in rehabilitative programs appropriate to and consistent with the level of recovery and techniques such as cognitive behavioral therapy and other individualized physical and psychological practices, as described elsewhere in this guideline.  
  
Control of chronic post-traumatic deficits, particularly in moderate/severe TBI, is expected to involve the use of medication. Strategies for pharmacological control of post-traumatic symptoms cannot be precisely specified in advance. Rather, drug treatment requires close monitoring of the patient’s response to therapy, flexibility on the part of the prescriber, and a willingness to change treatment when circumstances change; this includes lowering and/or discontinuing medications when symptoms improve and periodic trials of lowering medications when symptoms are stable. Many of the drugs discussed in the medication section are FDA approved for other indications but may appropriately be used for various aspects of TBI treatment and associated conditions. When prescribing off-label FDA use of a medication, indications and functional goals should be clearly stated as part of a comprehensive, functionally-based treatment plan. It is generally wise tobegin management with lower cost medications whose safety and efficacy equals that of higher cost medications, and medications with a greater safety profile. Decisions to progress to more expensive, non-generic, and/or riskier medications are made based on the drug profile, patient/caregiver feedback, and improvement in function. The provider should carefully balance the untoward side effects of the different drugs with therapeutic benefits,as well as monitoring forany drug interactions.  
  
Prescribed medications should be given an appropriate trial in order to test for therapeutic effect and tolerance to the medication. The length of an appropriate trial varies widely depending on the drug, as well as the individual and their response to the drug. Certain medications may take several weeks to months (e.g. antidepressants) to determine the efficacy, while others require only a few doses (e.g. psychostimulants). It is recommended that patients with chronic post-TBI symptoms who require maintenance medications use those that have the least serious side effects.   
  
Drugs of potential abuse, such as sedative/hypnotics or benzodiazepines, should be used sparingly in properly selected patients, e.g. for refractory insomnia, although total elimination of these medications is desirable whenever clinically feasible. It is strongly recommended that such pharmacological management be monitored or managed by an experienced physician, and referral to a specialist experienced in TBI may be necessary. Non-pharmacologic interventions should be used in combination with pharmacologic treatments to minimize the amount of medication necessary in patients with all levels of severity of TBI. Clinical pharmacologist can provide useful guidance in medication selection.  
  
Problems associated with mild, moderate, and severe TBI can be treated with a variety of medications; however, all have specific side effects and interactions of which clinicians should be mindful. Persons who sustain a TBI, particularly moderate/severe TBI, are particularly sensitive to central nervous system side effects, such as sedation, dizziness, cognitive impairment, and motor impairment. Starting doses and titration of medications will usually need to start lower and go slower, respectively; target doses may also be lower than when using these medications in a person without a TBI. Mild TBI cases are less likely to require medication as the majority of cases do well without prescription medications. When medication is used for MTBI, it is reasonable to consider a trial of tapering at 1–2 years post-injury.  
  
For the clinician to interpret the following material, it should be noted that: (1) drug profiles listed are not complete; (2) dosing of drugs will depend upon the specific drug, especially for off-label use; (3) not all drugs within each class are listed, and (4) other drugs within the class may be appropriate for individual cases. Clinicians should refer to informational texts or consult a pharmacist before prescribing unfamiliar medications or when there is a concern for drug interactions.  
  
The following drug classes are listed in alphabetical order, not in order of suggested use.

* + 1. Affective Disorders Medications: Classified into a number of categories based on their chemical structure and their effects on neurotransmitter systems. Their effects on depression are attributed to their actions on norepinephrine, serotonin, and dopamine at the level of the synapse; although these synaptic actions are immediate, the symptomatic response in depression is delayed by several weeks. Selective Serotonin Reuptake Inhibitors (SSRIs) or Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs) may be used first line, although there is more data to support the use of SSRIs as first line intervention ([Fann, 2009](#Fann2009)). Doses should be started low and slowly increased with attention to any fatigue, headache, insomnia, or drowsiness, which could impede cognitive progress. Several sources recommend citalopram or sertraline as they may also have a dopaminergic effect ([Fann, 2009](#Fann2009); [VADoD, 2009](#VetsDOD2009); [Warden, 2006](#Warden2006)).These drugs have fewer drug interactions and are likely better tolerated. Tricyclic antidepressants (TCAs) may also be used, however some have sedating qualities. (Refer to the Chronic Pain Guidelines for details)*.* A combination of dextromethorphan and quinidine may be used for patients suffering from pseudobulbar affect, which manifests as frequent, involuntary, and sudden episodes of crying or laughing. This can be seen in TBI and should be distinguished from depression and mania/hypomania to assure that the correct medication is used. There is no generic brand of this medication at the time of this guideline.
    2. Behavior/ Aggression Medications: There are no FDA approved drugs for the treatment of aggression in TBI, but many agents have been shown possibly to possibly have efficacy, including antipsychotics, antidepressants, mood stabilizers, anticonvulsants, and beta blockers. Beta blockers are relatively contraindicated in patients with asthma, heat block, or diabetes. Although there is some data suggesting that conventional and atypical antipsychotics can slow recovery from TBI, they may assist in the management of highly agitated or psychotic patients and those patients with co-morbid mood disorders. Use of these drugs should include careful monitoring for the development of tardive dyskinesia, weight gain, impaired cognition or coordination, hyperlipidemia, and glucose intolerance. An attempt to periodically reduce the dose or completely eliminate the drug should be made once the patient has stabilized, and clinicians should have a low threshold for consulting a psychiatrist if prolonged use of the class of medication appears likely. All medication use should consider the effects on cognition and interaction with other medication. Anti-epileptic medications, such as carbamazepine and valproate and other antihypertensive medications, such as clonidine, may also be beneficial.
    3. Cognitive Enhancers: Several areas are addressed by these agents: memory, attention, speed of information processing, executive function, and other general cognitive domains. Many of the medications are off-label use, and all should be carefully followed for side effects that may interfere with recovery. Medications given to improve cognition should be monitored with periodic neuropsychological assessment or cognitive screening to confirm positive response and the need to continue the medication. These medications should also have trial decreases periodically for eventual weaning. Most cognitive enhancers fall into the general categories of stimulants, cholinesterase inhibitors, or dopamine enhancers. The VADoD and other studies support the use of methylphenidate, modafanil, or amantadine in some cases with impaired cognitive function ([VADoD, 2009](#VetsDOD2009)).   
         
       Moderate/severe TBI cases will require individual management due to the number of issues being addressed, cognitive changes over time, and drug interactions. Considering these issues and the limited number of adequate studies in this area, with many published articles having small case sizes or non-randomized controls, medication regimes for moderate/severe TBI patients have wide variation.   
         
       The alphabetical following list is neither exhaustive nor complete regarding side effects, drug interactions, or studies not included in this review. Providers are advised to seek other sources for detailed drug prescribing information.
       1. Amantadine:
          1. Description: A non-competitive antagonist at NMDA receptors that enhances dopamine. Amantadine stimulates dopamine receptors, increases dopamine release, decreases presynaptic dopamine reuptake, and/or enhances post-synaptic dopamine receptor sensitivity. There is some evidence that amantadine in doses of 100–200 mg/ day can improve the performance of severe TBI patients who are in a minimally conscious state during the time period of drug delivery. Long-term usage and benefits were not addressed in this study ([Giacino, 2012a](#Giacino2012a),[b](#Giacino2012b),[c](#Giacino2012c)).
          2. Indications: FDA approved for Parkinson’s. May be used for cognitive fatigue, general cognitive deficits, arousal and attention deficits, initiation, speed of processing, emerging disorders of consciousness, and intractable motor disturbances [(Warden, 2006](#Warden2006)).
          3. Contraindications: Hypertension or hypersensitivity reaction.
          4. Dosing and Time to Therapeutic Effect: Dosage begins at 50 mg twice a day may increase to 100–200mg.
          5. Major Side Effects: Drowsiness, dizziness, insomnia, nausea, abdominal cramps, may lower seizure threshold, may increase psychological problems including suicide attempts and manic behavior, dysrhythmias, and rare neuroleptic malignant syndrome associated with abrupt withdrawal of amantadine*.*
          6. Drug Interactions: Use cautiously with other sympathetic agents.
       2. Bromocriptine:
          1. Description: Augments dopaminergic effects at the pre and post-synaptic level.
          2. Indications: FDA approved for acromegaly, hyperprolactemia, and Parkinson’s. Indicated for longer term moderate/severe TBI cases with arousal, attention, initiative, and executive function problems or motor control dysfunction. Studies are conflicting regarding effectiveness, and the longevity of the effect is unclear ([McDowell, 1998](#McDowell1998); [Whyte, 2008](#Whyte2004)).
          3. Contraindications: Hypertension or hypersensitivity reaction, hypersensitivity to ergot alkaloid drugs, women who are breastfeeding.
          4. Dosing and Time to Therapeutic Effect: Dosage is usually 0.1–0.2 mg per kg twice a day.
          5. Major Side Effects: Headache, dizziness, nausea and other gastrointestinal (GI) side effects, light headedness, syncope, and, at higher doses, hallucinations or psychosis.
          6. Drug Interactions: Multiple, including use with ergot alkaloids, macrolide antibiotics, other dopamine agonists, or ergot alkaloids.
       3. Carbidopa/L-Dopa:
          1. Description: Augments dopaminergic effects.
          2. Indications: FDA approved for use in Parkinson’s. May be used in TBI for hypoarousal, fatigue, disturbances of initiation, or motor control.
          3. Contraindications: Hypertension or hypersensitivity reaction, neuroleptic malignant syndrome history, psychosis.
          4. Dosing and Time to Therapeutic Effect: Dosage begins at 25/100mg twice a day may increase to 50/250 four times a day.
          5. Major Side Effects: Somnolence, increased intraocular pressure, nausea, dyskinesia, hallucinations (usually visual), paranoia, dizziness, abdominal cramps, manic behavior, may lower seizure threshold.
          6. Drug Interactions: MAO inhibitors and others.
       4. Dextroamphetamine and Mixed Amphetamine Salts:
          1. Description: A central nervous system stimulant (also called psychostimulant). Mixed amphetamine salts include both levoamphetamine and dextroamphetamine.
          2. Indications: FDA approved for use in patients with attention deficit disorder and narcolepsy. May improve attention, arousal, speed of processing, and memory ([Warden, 2006](#Warden2006)).
          3. Contraindications: Marked anxiety, tension or agitation, history of drug abuse, closed angle glaucoma, uncontrolled hypertension, cardiovascular disease or arrhythmia, use of MAO inhibitors, hypersensitivity reaction.
          4. Dosing and Time to Therapeutic Effect: Dosage is usually 0.1–0.2 mg per kg twice a day.
          5. Major Side Effects: There is a warning for drug dependence for patients with a history of alcohol addiction or drug dependence. Precautions exist for the possibility of serious psychiatric conditions with pre-existing manic or psychotic behavior. Those with serious cardiovascular disease are at risk for sudden death, stroke, or myocardial infarction. Common minor side effects include insomnia, agitation, dry mouth, anorexia, weight loss, bruxism, and increased sweating with exertion.
          6. Drug Interactions: Multiple, including some anticonvulsants and antidepressants.
       5. Donepezil:
          1. Description: Acetylcholinesterase inhibitor.
          2. Indications: FDA approved for use in patients with Alzheimer’s dementia. Recommended for moderate/severe TBI in the sub-acute phases to improve attention and memory. There is some evidence from a small study of sub-acute patients with moderate/severe TBI of improvement in working memory, retrieval of declarative information, sustained attention, and the rate of cognitive recovery with use of donepezil. The effect was evident at ten weeks and may persist after stopping the medication ([Zhang, 2004](#Zhang2004)).
          3. Contraindications: Hypersensitivity reaction, women who are pregnant or breast feeding, cardiac conduction abnormalities (first-degree A-V block), and symptomatic bradycardia.
          4. Dosing and Time to Therapeutic Effect: Dosage is titrated carefully over weeks from 2.5 mg per day. Continual use of the medication requires documentation of improved function. Duration of treatment for this population is unknown.
          5. Possible Side Effects: Bradycardia, nausea, peptic ulcer disease, GI bleeding, weight loss, difficulty voiding, seizures, exacerbations of pulmonary disease.
          6. Drug Interactions: Anticholinergic medications, concurrent administration of quindine and medications that inhibit hepatic metabolism CYP450 (isoenzymes 3A4,and 2D6), such as ketoconazole, may increase blood levels of donepezil. Inducers of hepatic metabolism (phenobarbital, phenytoin, carbamazepine, dexamethasone, rifampin) may decrease therapeutic blood levels.
       6. Methylphenidate:
          1. Description: A central nervous system stimulant
          2. Indications: FDA approved for use in patients with attention deficit disorder. There is good evidence that it has a short-term effect on improving test performance on standardized measures of attention in patients with moderate/severe TBI ([Whyte, 2004](#Whyte2004); [Willmott, 2009](#Willmott2009)). Also used in TBI for hypoarousal, attention, memory, and speed of processing problems ([Warden, 2006](#Warden2006)).
          3. Contraindications: Marked anxiety, tension or agitation, hypersensitivity reaction, psychosis, anorexia nervosa, hyperthyroidism, glaucoma, MAOI use.
          4. Dosing and Time to Therapeutic Effect: Dosage in studies was 0.3 mg per kg twice a day.
          5. Major Side Effects: There is a warning for drug dependence for patients with a history of alcohol addiction or drug dependence. Precautions exist for the possibility of serious psychiatric conditions in patients with pre-existing manic or psychotic behavior. Those with serious cardiovascular disease or structural cardiac abnormalities are at risk for sudden death, stroke, or myocardial infarction. Common minor side effects include insomnia, agitation, dry mouth, anorexia, and weight loss.
          6. Drug Interactions: Multiple, including some anticonvulsants and antidepressants.
       7. Modafinil & Armodafinil:
          1. Description: Sympathomimetic /stimulant increases dopamine levels, but also has effects on serotonin, glutamate, and GABA
          2. Indications: FDA approved for narcolepsy, obstructive sleep apnea, and shift work disorder. May be used for hypoarousal, daytime sleepiness, or fatigue following TBI.
          3. Contraindications: Hypersensitivity.
          4. Dosing and Time to Therapeutic Effect: Dosage 100mg – 400 mg per day for modafinil or 150–250 mg per day for armodafinil.
          5. Major Side Effects: Headache, nausea, nervousness, dizziness, paranoia. Serious rashes, including Stevens-Johnson syndrome and toxic epidermal necrolysis, have been reported.
          6. Drug Interactions: Potential interactions with drugs using the cytochrome P-450 isoenzymes or hepatic enzymes such as tricyclics, phenobarbital, and carbamazepine.
       8. Rivastigmine:
          1. Description: An acetylcholinesterase inhibitor.
          2. Indications: FDA approved for Alzheimer’s and Parkinson’s dementia. Several studies that are inadequate for evidence purported to show better outcomes for those patients with moderate/severe TBI with adequate safety and tolerance ([Silver, 2006](#Silver2006); [Tenovuo, 2009](#Tenovuo2009)). May be used for cognitive dysfunction in moderate to severe TBI cases. It is possible for patients to have a greater effect from one specific acetylcholinesterase inhibitor than from another one. Thus, rivastigmine may be useful if donepezil or less costly agents have failed.
          3. Contraindications: The same as the contraindications for donepezil.
          4. Dosing and Time to Therapeutic Effect: Generally 1.5 mg twice a day for four weeks, with increases in increments of 1.5 mg twice a day every four weeks up to 12 mg/day. Rivastigmine is available in a transdermal patch if oral administration is not possible.
          5. Major Side Effects: The same as the side effects for donepezil. It may have more side effects than donepezil but allows for a slower titration if that is desirable in a given patient.
          6. Drug Interactions: The same as the drug interactions for donepezil.
       9. Other Medications approved for use in Parkinson’s: The off-label use of dopamine agonists in moderate/severe TBI for conditions including, but not limited to, disorders of consciousness (arousal), attention, initiation, speed of processing, and motor control (bradykinesis, rigidity, tremor). May be appropriate in specific cases.

**Indications for use of Cognitive Enhancers**

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
|  | Somnolence | Attention Deficiency | Lack of Motivation/ Initiation | Memory Difficulty | Slowed Speed of Processing | Executive Function Difficulty | Motor Disturbances |
| Amantadine | X | X | X |  | X | X | X |
| Bromocriptine | X | X | X |  |  | X | X |
| Carbidopa/L-Dopa | X |  | X |  |  |  | X |
| Dextroamphetamine | X | X |  | X | X |  |  |
| Donepezil | X |  | X | X |  |  |  |
| Methylphenidate | X | X |  | X | X |  |  |
| Modafinil/Armodafinil | X |  |  |  |  |  |  |
| Rivastigmine | X |  | X | X |  |  |  |

The domain of cognitive function is listed at the top of each column, and the medications are listed alphabetically.

* + 1. **Hypnotics and Sedatives**: Sedative and hypnotic drugs generally decrease activity and induce drowsiness, although some patients may experience agitation with use. Many drugs produce these effects incidental to their usual intended effects, similar to the side effects of many antihistamines and antidepressants. Due to the habit forming potential and adverse cognitive side effects of the benzodiazepines and other drugs found in this class, they are not routinely recommended but may be useful temporarily in some patients with sleep disturbances. Benzodiazepines may be indicated to treat alcohol withdrawal in TBI, but their use in the acute post-injury period after TBI is otherwise discouraged. Most insomnia in TBI patients should be managed primarily through environmental and behavioral interventions, including cognitive behavioral therapy, with medications as secondary measures (refer to Section [G.9. Disturbances of Sleep](#TBIG9DisturbancesOfSleep)).
    2. **Non-Steroidal Anti-Inflammatory Drugs**: Non-steroidal anti-inflammatory drugs (NSAIDs) are useful for pain and inflammation. In mild cases, they may be the only drugs required for analgesia. There are several classes of NSAIDs, and the response of the individual injured worker to a specific medication is unpredictable. For this reason, a range of NSAIDs may be tried in each case with the most effective preparation being continued. Patients should be closely monitored for adverse reactions. The US Food and Drug Administration advises that all NSAIDs may cause an increased risk of serious cardiovascular thrombotic events, myocardial infarction, and stroke, which can be fatal. There is good evidence that naproxen has less risk for cardiovascular events when compared to other NSAIDs ([Trelle, 2011](#Trelle2011)). Administration of proton pump inhibitors, histamine 2 blockers, or prostaglandin analog misoprostol along with these NSAIDs may reduce the risk of duodenal and gastric ulceration but do not impact possible cardiovascular complications. Due to the cross-reactivity between aspirin and NSAIDs, NSAIDs should not be used in aspirin-sensitive patients, and they should be used with caution in all asthma patients. NSAIDs are associated with abnormal renal function, including renal failure, as well as abnormal liver function. Certain NSAIDs may have interactions with various other medications. Individuals may have adverse events not listed above. Intervals for metabolic screening are dependent on the patient’s age and general health status and should be within parameters listed for each specific medication. Complete blood count (CBC), liver function, and renal function should be monitored at least every six months in patients on chronic NSAIDs and initially when indicated.
       1. Non-Selective Non-steroidal Anti-Inflammatory Drugs: Include NSAIDs and acetylsalicylic acid (aspirin). Serious GI toxicity, such as bleeding, perforation, and ulceration can occur at any time, with or without warning symptoms in patients treated with traditional NSAIDs. Physicians should inform patients about the signs and/or symptoms of serious gastrointestinal toxicity and what steps to take if they occur. Anaphylactoid reactions may occur in patients taking NSAIDs. NSAIDs may interfere with platelet function. Fluid retention and edema have been observed in some patients taking NSAIDs.

❖ Optimal Duration: 1 week.

❖ Maximum Duration: 1 year. Use of these substances long-term (3 days per week or greater) is associated with rebound pain upon cessation.

* + - 1. Selective Cyclo-Oxygenase-2 (COX-2) Inhibitors: COX-2 inhibitors differ in adverse side effect profiles from the traditional NSAIDs. The major advantages of selective COX-2 inhibitors over traditional NSAIDs are that they have less gastrointestinal toxicity and no platelet effects. COX-2 inhibitors can worsen renal function in patients with renal insufficiency; thus, renal function may need monitoring.  
           
         COX-2 inhibitors should not be first line for low-risk patients who will be using an NSAID short term but are indicated in select patients for whom traditional NSAIDs are not tolerated. Serious upper GI adverse events can occur even in asymptomatic patients. Patients at high risk for GI bleed include those who use alcohol, smoke, are older than 65, take corticosteroids or anti-coagulants, or have a longer duration of therapy. Celecoxib is contraindicated in sulfonamide allergic patients.

❖ Optimal Duration: 7 to 10 days.

❖ Maximum Duration: Chronic use is appropriate in individual cases. Use of these substances long-term (3 days per week or greater) is associated with rebound pain upon cessation.

* + 1. **Skeletal Muscle Relaxants**: Most useful for acute musculoskeletal injury or exacerbation of injury. Chronic use of benzodiazepines is discouraged due to their habit-forming potential, negative effects on cognition, and seizure risk following abrupt withdrawal. Carisoprodol should not be used because the anxiolytic meprobamate is a metabolite. (For more detailed descriptions, refer to the Chronic Pain Guidelines.).
    2. **Opioids**: Refer to the Chronic Pain Guidelines for appropriate use.
  1. HEADACHE: One of the most common symptoms seen in general medical practices. Following TBI, 50% or more of injured individuals experience headache. The majority of these are self-limited, but headache persisting for more than three months may occur. Brain damage is unlikely to be responsible for post-traumatic headache, which is seen more commonly after MTBI than after moderate/severe TBI. Rather, involvement of extracranial structures, such as the temporomandibular joint (TMJ), the sinuses, and the muscles attaching to the occiput accounts for most headaches following TBI. (Refer to the Division’s Cervical Spine Guidelines when appropriate).  
       
     Headaches may persist longer when associated with other symptoms such as dizziness, memory problems or weakness. Therefore, every effort should be made to identify the cause and treat headaches and other symptoms as early as possible.  
       
     Management of post-traumatic headache should be tailored to the class of non-traumatic (chronic tension, migraine) headache into which it fits. Migraine patients should be provided with the migraine’s diet advisories and restrictions. Both traumatic and non-traumatic headaches may be made worse by overuse of analgesics and cause chronic daily headache. Treatment should be directed toward understanding the cause with re-establishment of activities and away from rumination on the injury. Education, medication adjustment and interdisciplinary team approaches may be called for. Chronic daily headache should be considered as a diagnosis in patients whose daily headache may be in response to iatrogenic complications of other medications or substances. Patients who are prescribed analgesics, or who use caffeine, alcohol or nicotine chronically, may experience chronic daily headaches as serum levels of these substances fluctuate. These patients may require treatment to carefully titrate these substances. Patients with a blow to the side of the head, with chronic neck/shoulder pain, or with bruxism may develop temporal mandibular joint pain that will result in headache. Patients with sinus involvement, sometimes evidenced on early CT imaging, may develop chronic headache pain that requires treatment of the underlying sinus pathology. If depression is present, a sedating antidepressant such as amitriptyline may alleviate the insomnia that often complicates headache. See treatment algorithm (next page). Referral to a specialist may be necessary if initial treatment is not effective.  
       
     Tension type headaches should generally be treated with analgesics initially and/or accompanied by physical therapy modalities for neck and shoulder treatments. Opioid treatment should be avoided ([Bendtsen, 2010](#Bendtsen2010)). There is good evidence that amitriptyline is beneficial for chronic tension headaches ([Bendtsen, 1996](#Bendtsen1996)). The etiology of the headache should be carefully determined prior to initiation of any drug regimens.   
       
     There is strong evidence that aspirin is better than placebo for acute migraine headaches and good evidence that adding an antiemetic improves the outcome([[Cochrane] Kirthi, 2010](#Kirthi2010)). There is insufficient evidence that sumatriptan is better than aspirin plus metoclopramide ([[Cochrane] Kirthi, 2010](#Kirthi2010)). There is good evidence that acetaminophen is effective for acute migraines ([Derry, 2010](#Derry2010))and a single dose of 200–400 mg of ibuprofen is effective for acute migraines ([Rabbie, 2010](#Rabbie2010)). There is also good evidence that acetaminophen with an antiemetic is comparable to sumatriptan ([Derry, 2010](#Derry2010)). There is strong evidence supporting the use of sumatriptan for migraines [([Cochrane] McCrory, 2003](#McCrory2003)). There is insufficient evidence to prefer one triptan over another. There is good evidence that selective serotonin reuptake inhibitors (SSRI’s) and preparations with ergotamine and caffeine are not effective for migraine headaches [([Cochrane] McCrory, 2003](#McCrory2003); [Moja, 2005](#Moja2005)). There is strong evidence that propranolol is superior to placebo for migraine prophylaxis ([[Cochrane] Linde, 2004](#Linde2004)). There is strong evidence that valproate and topiramate are effective in decreasing headache frequency [([Cochrane] Chronicle, 2004](#Chronicle2004)). Prescribers should take into account the significant side effects and contraindications associated with these medications. There is good evidence that Petasites hybridus root (butterbur) is effective for episodic migraine headaches (2–6 per month) ([Lipton, 2004](#Lipton2004); [Grossmann, 2001](#Grossman2001)). It has some hepatic toxicity and should only be purchased from reputable laboratories. It is not a prescription drug and can be purchased over the counter. The following table of recommendations takes into account the American Academy of Neurology and American Headache Society’s latest guideline recommendations ([Silberstein, 2012](#Silberstein2012); [Holland, 2012](#Holland2012)).  
       
     Widely accepted treatments for post-traumatic headache may include, but are not limited to: interdisciplinary treatment, pharmacology, joint manipulation, physical therapy, massage, acupuncture, biofeedback, psychotherapy (i.e., cognitive behavioral therapy), and diet. There is some evidence that spinal manipulation is effective for treatment of cervicogenic headaches ([[Cochrane] Bronfort, 2004](#Bronfort2004)). There is some evidence that exercise is equally efficacious as manipulation and can be used in combination with manipulation([[Cochrane] Bronfort, 2004](#Bronfort2004)). The usual course of treatment was 3–6 weeks and effects were still found at one year ([[Cochrane] Bronfort, 2004](#Bronfort2004)). Refer to the Cervical Spine Guidelines for parameters.  
       
     There is strong evidence that acupuncture and sham acupuncture are prophylactic for migraines([[Cochrane] Linde, 2009](#Linde2009)). There is good evidence that acupuncture has similar results as medication prophylaxis([[Cochrane] Linde, 2009](#Linde2009)). There is some evidence that sham acupuncture is better than no treatment for migraine prophylaxis [([Cochrane] Linde, 2009](#Linde2009)). These procedures should only be continued if functional gains are documented.   
       
     Psychological evaluation is a generally accepted intervention to identify factors for delayed recovery associated with pain and the potential need for cognitive assessment. Refer to the Chronic Pain Guidelines for specific time frames. Special procedures may be useful for specific or intractable head pain syndromes including nerve blocks for neuralgia, trigger point injections for myofascial pain syndromes (refer to Section [I.8 Muscle Tone and Joint Restriction Management Including Spasticity](#TBII6MuscleToneJointRestrictionMngt)), and the use of dental splinting for temporomandibular joint syndrome.  
       
     Inpatient admission is sometimes required when intravenous medications (e.g., dihydroergotamine) and close monitoring are necessary to control migraine or analgesia rebound, especially in individuals with severe depression, suicidal ideation, or complicated medical problems. When greater than two disciplines are necessary, when there is significant dysfunction secondary to headache, when the individual has not returned to work for greater than three months or when treatment is geographically inaccessible, an individualized interdisciplinary outpatient treatment program may be appropriate.  
       
     Long-term maintenance plans are necessary in chronic headache management. Medications may be necessary for an indefinite period; however, a distinction should be made between headache conditions that were pre-existing and those caused by the TBI. In MTBI, most cases will not result in debilitating frequent headaches. If the patient is suffering from debilitating headaches, a full review of the diagnosis, triggering events, and psychosocial issues should take place. All headache treatment modalities should be independent and functional. Even if headaches are permanent, it is expected that the individual will be functional and able to return to work.
     1. Headache Treatment Algorithm:

Initial Evaluation

HEADACHES INTERFERING WITH FUNCTION

**↓**

History and Physical Evaluation

Establish Diagnosis

Lab Studies

Possible Brain Imaging

**↓**

Initiate Treatment

|  |  |  |
| --- | --- | --- |
| **Pharmacological - Preventives**  To be used if 2+ headaches/week, or increased headache severity or duration | **Pharmacological - Abortives**  Limit use to prevent medication overuse headache | **Non-Pharmacological** |
| Education and identification of triggers |
| Medication if terminated rebound present |
| Tricyclic Antidepressants venlafaxine or amitriptyline | Anti-inflammatories or acetaminophen | Physical Therapy |
| Beta Blockers | Sumatriptan and other “Triptans” for migraine type headaches | Biofeedback |
| Tryptans | Acupuncture (Refer to Chronic Pain Guidelines – Section F.2) |
| Topiramate  Divalproex/Valproate | An anti-emetic may accompany either medication metoclopramide | Psychology including cognitive behavioral therapy |
| Complementary medications  Petasites hybridus root (butterbur) Use pharmaceutical grade product  Histamine  Riboflavin  Magnesium | Joint Manipulation Therapy (Refer to Cervical Spine Guidelines for time parameters.) |
| Others with possible effect  ACE inhibitors, Angiotensin receptor blockers, a-agonists, carbamazepine, |  |

**↓**

Headache control

|  |  |  |
| --- | --- | --- |
| Good |  | Sub-optimal |
| Continue treatment 3–6 months, then taper. |  | Reassess diagnosis, optimize treatment, and identify other contributors. |
| Preventive medications as appropriate. |  | Referral to specialist. |

* + 1. Botulinum Injections: No longer generally recommended for cervicogenic or other headaches based on good evidence of lack of effect [([Cochrane] Langevin, 2011](#Langevin2011); [Linde, 2011](#Linde2011), [Aurora, 2011](#Aurora2011); [Naumann, 2008](#Naumann2008)). There is good evidence that botox is not more effective than placebo for reducing the frequency of episodic migraines ([Shuhendler, 2009](#Shuhendler2009)). It may be considered in a very small subset of patients with chronic migraines 12–15 days/month who have failed all other conservative treatment, including trials of at least three drug classes, and who have committed to any life style changes related to headache triggers ([Jackson, 2012a,](#Jackson2012a)[b](#Jackson2012b)).
  1. THERAPEUTIC EXERCISE: With or without mechanical assistance or resistance, may include isoinertial, isotonic, isometric, and isokinetic types of exercises as part of the overall occupational therapy physical therapy program, not to be used in isolation.   
       
     Studies of older adults have consistently shown both cognitive and neuro-physiologic functional gains for those participating in regular aerobic activity ([Barnes, 2003](#Barnes2003); [Colcombe, 2003](#Colcomb2003); [Colcombe, 2004](#Colcomb2004); [Kramer, 1999](#Kramer1999); [Lojovich, 2010](#Lojovich2010); [Weuve, 2004](#Weuve2004)). Apparent physiological benefits from exercise include improved neuroplasticity after the initial period of 3–10 days, improved cognition, decreased emotional stress, and improved hypothalamic-pituitary-adrenal axis function ([Archer, 2011](#Archer2011)). Brain derived neurotrophic factor (BDNF) increases with exercise, although likely transiently ([Ferris, 2007](#Ferris2007); [Knaepen, 2010](#Knaepen2010)). Animals studies have shown a decrease in BDNF when exercise is begun during the first few weeks ([Griesbach, 2004a](#Graesbach2004a),[b](#Graesbach2004b); [Griesbach 2012](#Graesbach2012)). Limited physical and cognitive activity is recommended in the first three days after a concussion with a gradual increase in activity based on decreased symptoms. Return to full duty depends on the rate of decrease of symptoms. Generally if symptoms recur during increasing job duties or exertion, duties should be decreased slightly ([Defense and Veterans Brain Injury Center, 2008](#DefandVetsTBIctr2008)).  
       
     Indications include the need for cardiovascular fitness, improved muscle strength, improved connective tissue strength and integrity, increased bone density, promotion of circulation to enhance soft tissue healing, improvement of muscle recruitment, improved proprioception and coordination, and increased ROM.   
       
     Patients and/or caregivers should be instructed in and receive a home exercise program that is progressed as their functional status improves. Upon discharge, the patient and/or caregiver would be independent in the performance of the home exercise program and would have been educated in the importance of continuing such a program. Educational goals would be to maintain or further improve function and to minimize the risk for aggravation of symptoms in the future.

❖ Time to Produce Effect: 2 to 6 treatments.

❖ Frequency: 1 to 5 times per week.

❖ Optimum Duration: 4 to 8 weeks and concurrent with an active daily home exercise program.

❖ Maximum Duration: 8 to 12 weeks of therapist oversight. Home exercise should continue indefinitely.

* 1. DISTURBANCES OF SLEEP: Common in all types of TBI. Objective sleep studies show reduced sleep efficiency, increased sleep onset latency, and increased time awake after sleep onset ([Ponsford, 2012](#Ponsford2012)). These changes are associated with patient reports of non-restorative sleep. Sleep disorders can aggravate neurologic injury. Sleep apnea may be associated with endocrine disturbance arising from TBI and neuroendocrine assessment is highly recommended. Sleep apneas and other sleep disorders should be treated to optimize recovery from TBI.   
       
     Many patients develop behavioral habits that exacerbate and maintain sleep disturbances. Excessive time in bed, irregular sleep routine, napping, low activity, and worrying in bed are all maladaptive responses that can arise in the absence of any psychopathology. There is some evidence that behavioral modification, such as patient education and group or individual counseling, can be effective in reversing the effects of insomnia (Refer to the Chronic Pain Guidelines) ([Currie, 2000](#Currie2000)). Behavioral modifications are easily implemented and can include:

● Maintaining a regular sleep schedule, including retiring and rising at approximately the same time on weekdays and weekends.

● Avoiding daytime napping.

● Avoiding caffeinated beverages after lunchtime.

● Making the bedroom quiet and comfortable, eliminating disruptive lights, sounds, television sets, and keeping a bedroom temperature of about 65°F.

● Avoiding alcohol or nicotine within two hours of bedtime.

● Avoiding large meals within two hours of bedtime.

● Exercising vigorously during the day, but not within two hours of bedtime, since this may raise core temperature and activate the nervous system.

● Associating the bed with sleep and sexual activity only, using other parts of the home for television, reading, and talking on the telephone.

● Leaving the bedroom when unable to sleep for more than 20 minutes and returning to the bedroom when ready to sleep again.

These modifications should be undertaken before sleeping medication is prescribed for long term use. Modafinal, melatonin, and light therapy may be helpful for some patients ([Ponsford, 2012](#Ponsford2012)).

1. NONOPERATIVE THERAPEUTIC PROCEDURES – NEUROMEDICAL CONDITIONS in MODERATE/SEVERE BRAIN INJURY

There are a number of associated neuromedical problems unique to moderate/severe TBI. These conditions often require specialized evaluation and therapeutic interventions by physicians, nurses and relevant interdisciplinary team disciplines. The resultant problems may be classified as follows:

* 1. Neurological Complications: Ongoing evaluation is often necessary to detect the delayed development of space occupying intraparenchymal lesions, pneumocephalus, hydrocephalus, extra-axial lesions such as subdural and epidural hematomas, and hygromas. If an individual’s neurological status worsens or plateaus, neuroimaging studies may be warranted.
  2. Post-Traumatic Seizures/Post-Traumatic Epilepsy (PTE): Major risk factors for the development of PTE include penetrating head wounds, hematoma, depressed skull fracture, and early seizures. The issue of seizure prophylaxis remains controversial in high-risk individuals. The role of routine seizure prophylaxis utilizing antiepileptic drugs (AEDs) is recommended for seven to ten days post-brain trauma. Prophylactic anticonvulsants should not routinely be used after the first week but at times at times may be appropriate for individual cases ([Chang, 2003](#Chang2003)). The management of late post-traumatic seizures conforms to the treatment of “epilepsy.” This includes principles of mono-therapy, and compliance, considerations of cognitive, behavioral, emotional, and psychosocial functioning. Pseudoseizures should be treated by psychological and psychiatric therapy.
  3. Cardiopulmonary Complications:
     1. Cardiac System: Elevated intracranial pressure and hypoxia may injure the hypothalamus and cardiac regulating centers of the brain, causing pathological changes in autonomic nervous system function. The resulting dysautonomia, paroxysmal sympathetic hyperactivity, or hyperadrenergic syndrome (autonomic storm) includes fever, hypertension, tachycardia, tachypnea, posturing, and hyperhydrosis (increased sweating and flushing). Hypertension in TBI is associated with tachycardia and increased cardiac output with normal or decreased peripheral vascular resistance. This is different from essential hypertension in which there is normal cardiac output with increased peripheral vascular resistance. The preferred treatment for this type of hypertension from hyperadrenergic activity is a beta adrenergic blocking agent or alpha-2 central agonist. However, these approaches should carefully consider the potentially negative cognitive, behavioral, and/or emotional side effects of those medications
     2. Pulmonary System: Moderate/severe TBI and related trauma to the chest wall may adversely affect respiratory function by compromising respiratory drive, swallow reflex, and cough. Brain and brain stem injuries also cause abnormal neurogenic breathing patterns and a dysfunctional swallowing mechanism with the potential for aspiration and a weakened cough with poor mobilization of secretions. These individuals are at increased risk for hypoxemia leading to further central nervous system (CNS) injury, pneumonia, and adult respiratory distress syndrome. The main principle of therapeutic intervention is the avoidance of respiratory failure with appropriate oxygenation, ventilation, and airway control. Treatments may include mechanical ventilation, tracheostomy, routine swallow evaluation to evaluate for aspiration risk, and aggressive pulmonary hygiene.
  4. Sleep Complications: Sleep disturbance is a relatively common complication following moderate/severe TBI. Common sleep disorders for which individuals are at risk include, but are not limited to, post-traumatic hypersomnia, narcolepsy, central sleep apnea, obstructive sleep apnea, nocturnal seizures, periodic limb movement disorder (PLMD), sleep disturbances due to medication and medication side effects, sleep disturbances due to underlying mental health issues and substance abuse and insomnia. Generally accepted subjective measures of post-traumatic sleep disturbance include self-rating scales; however, in this population, it may be preferable to rely on staff when in an inpatient facility and caregivers when at home. Objective measures include techniques that monitor changes in select physiologic processes (heart rate, temperature, cortisol levels, blood/oxygen levels, polysomnography, etc.) up to full polysomnography and sleep lab studies. These subjective and objective measures, combined with serial clinical evaluation, are useful clinical tools in guiding appropriate management. Depending on etiology, management strategies include, but are not limited to, sleep hygiene education, implementation of sleep hygiene, maintaining a normal day/night and wake/sleep cycle, limitation of time in bed and naps, surgery, various medical devices (e.g., oral appliance, continuous positive airway pressure), and medication therapy. Also refer to Section [G.9. Disturbances of Sleep](#TBIG9DisturbancesOfSleep).
  5. Musculoskeletal Complications:
     1. Long-Bone Fractures: When long-bone fractures occur in individuals with a TBI, aggressive, early surgical treatment is recommended within two to twelve hours after injury, provided that hemodynamic stability has been achieved. This would include open reduction and internal fixation, although the specific optimal technique has not been well-documented. Maximal functional use of all extremities should be the goal in this early phase of care. Early stabilization allows the prevention of prolonged immobility that has the subsequent greater risk of infection, venous thrombosis development, pulmonary complications, skin breakdown, and contractures. Fracture healing challenges unique to TBI include the deforming effect spasticity exerts on fracture alignment and an exaggerated healing response. Non-compliance secondary to confusion and agitation often requires reinforced immobilization, strategies, and prolonged time frames of immobilization, and it may preclude the use of common eternal fixation devices. Therefore, it is generally accepted that early, aggressive, surgical management with an emphasis on internal fixation should be instituted to allow for early mobilization when medically indicated in this population.
     2. Heterotopic Ossification (HO): Defined as the development of new bone formation in soft tissue planes surrounding neurologically affected joints, especially the hips, elbows, shoulder and knees, in order of common concurrence. Research puts the incidence at 11–75% following moderate/severe TBI ([Harrington, 2008](#Harrington2008)). If diagnosis and treatment are delayed, ankylosis (bony fusion) may occur with consequent functional limits in mobility. Additional risk factors that often accompany TBI include spinal cord injury, tissue hypoxia, venous stasis, spasticity, and autonomic dysfunction. The greatest risk for development is within the first six months post-injury. Observation by nurses and physical therapists is essential and may include documentation of decreased ROM, joint inflammation, pain, and/or a low-grade fever. Appropriate work-up may include laboratory studies revealing an elevated sedimentation rate and/or alkaline phosphatase with a normal complete blood count (CBC). Plain x-rays are necessary and appropriate; however, the most sensitive radiological study includes the three-phase bone scan and/or gallium scan, MRI, and color Doppler ultrasound. These may be necessary in both the initial diagnostic and follow-up phases to guide treatment. Optimal treatment outcome involves early diagnosis, ROM exercise, and the use of disodium etidronate, which prevents mineralization. Other treatment options include non-steroidal anti-inflammatory drugs (NSAIDs), radiation, and surgery in the chronic state.
  6. Gastrointestinal Complications: Individuals with moderate/severe TBI have demonstrated delays in gastric emptying with frequent regurgitation of nasogastric administered feedings. This, accompanied with dysphagia and/or an inadequate swallow reflex, places the individual at risk for aspiration pneumonia. Dysphagic individuals and those at risk may require total parenteral nutrition (TPN), gastric and/or post-pyloric feeding techniques. Either a endoscopically placed percutaneous (PEG) or surgically placed gastrostomy and/or jejunostomy may be necessary for adequate ongoing nutritional support. Individuals with gastrointestinal hypomotility may require medications. Also, erosive gastritis may be a frequent complication, and the use of H2 blockers, proton pump inhibitors (PPIs), and antacid treatments are usually efficacious. Individuals with TBI may also be at risk for neurogenic bowel, which includes constipation, impactions, bowel obstructions, and/or loose stools. A nursing care regimen on a routine and then consultative basis, may be necessary to establish routine bowel programs.
  7. Genitourinary Complications: Moderate/severe TBI may involve cerebral structures controlling bladder storage and emptying functions. This may result in a neurogenic bladder. Treatment of a neurogenic bladder is aimed at adequate emptying, prevention and treatment of infection, preservation of upper renal tract function, and avoidance of skin soiling from incontinence. An indwelling urethral catheter is often appropriate in the early stages of recovery. Once the urethral catheter is discontinued, either a condom catheter or diaper/adult brief is used for incontinence.  
       
     Following assessment of bladder emptying utilizing ultrasonography for post-void residual checks and urodynamic studies, decisions may be made regarding longer-term management strategies. This may include intermittent catheterization or rehabilitative bladder training utilizing anticholinergic medications and time-interval voiding techniques. Urological consultation and more comprehensive diagnostic studies that may include, but are not limited to, cystoscopy, urodynamics, and renal functions studies may also be necessary. Sexual dysfunction may also occur, secondary to moderate/severe TBI. Examples include disinhibition, arousal disorders, and erectile dysfunction. If present, comprehensive assessment is appropriate in guiding therapeutic management.
  8. Neuroendocrine Complications: Neuroendocrine abnormalities following Moderate/Severe TBI are common. Hypopituitarism occurs in approximately 28% of all TBI and although more common in moderate/severe TBI, may also occur in MTBI with a rate approximating 17% ([Schneider, 2007](#Schneider2007)). The degree of neuroendocrine dysfunction may vary based on differential injuries to the hypothalamus, anterior/posterior pituitary, upper or lower portions of the pituitary stalk, and connections to other brain and brainstem structures. Secondary endocrine effects may include, but are not limited to, the abnormalities of the following: salt and water metabolism including syndrome of inappropriate antidiuretic hormone (SIADH) and temporary or permanent diabetes insipidus (DI), thyroid function, sexual function, hormonal reproductive function, control of body temperature, ACTH-cortisol levels, glucose metabolism, gonadotropin, and growth hormones. These potential complications may require specialized medical evaluation and treatment if correlative symptoms exist and/or persist. Pharmaceutical treatment for other complications may also affect endocrine systems and require treatment.
  9. Fluid and Electrolyte Complications: Abnormalities in individuals with moderate/severe TBI are usually iatrogenic or trauma induced. Specific problems may include, but are not limited to, a resulting water and salt retention with decreased urine output. There may also be problems with hyponatremia from inappropriate antidiuretic hormone, cerebral salt wasting, and increased production of aldosterone. Also, hypernatremia from dehydration or DI may occur. This may require careful evaluation with laboratory studies initially and serially on a follow-up basis.
  10. Immobilization and Disuse Complications: In an comatose individual, skin is at risk for the development of pressure decubitus ulcers that may slowly progress and increase the length of hospital stays. Tissue pressure, shear, and deformation cause the ischemia. Vigilant rehabilitation nursing including accurate staging, specialized beds, wheelchair cushions, padding, positioning, and weight shift management, protects the individual from these complications.
  11. Vascular Complications: Individuals with TBI are at risk for developing deep venous thrombosis (DVT) and pulmonary embolus (PE). Since diagnosis by clinical examination is difficult in this population, a high degree of suspicion is warranted. While in the hospital, daily nursing screening with lower extremity measurements is recommended. Abnormalities requiring confirmation may entail noninvasive studies such as Doppler ultrasonographic flow examination and impedance plethysmography. Also, hematologic conditions, such as, but not limited to, coagulopathies may require comprehensive specialized hematologic evaluation. It is generally accepted that prophylaxis with low molecular weight heparin, intermittent compression devices (ICDs), or sequential compression stockings may reduce the incidence of both complications. If the diagnostic use of noninvasive studies as mentioned are equivocal and/or non-confirmatory, then venography and/or angiography may be necessary. If thrombotic complications occur, standard treatment includes intravenous heparin or subcutaneous low molecular weight heparin followed by oral warfarin sodium. Other newer pharmaceutical agents may also be appropriate. If neuromedical risks of anticoagulation are present and/or complications related to anticoagulation or progressive thrombosis arise, then placement of an inferior vena cava filter may be necessary.

1. NONOPERATIVE THERAPEUTIC PROCEDURES – REHABILITATION
   1. INTERDISCIPLINARY REHABILITATION PROGRAMS: The recommended treatment for individuals with MTBI who have not responded to less intensive modes of treatment and for moderate/severe TBI. These programs should assess the impact of the injury on the patient’s medical, physical, psychological, social, and/or vocational functioning. The number of professions involved in the team in a TBI program may vary due to the complexity of the needs of the person served. The Division recommends consideration of referral to an interdisciplinary program based on the results of a comprehensive neuropsychological and/or psychiatric assessment, which should be conducted post-injury in MTBI individuals with delayed recovery and as soon as appropriate for more severe cases. The sequencing of treatment is based on the individual’s ability to tolerate and benefit from the specific therapies. For example, a patient with severe balance problems will be unable to participate in physical rehabilitation.   
        
      Patients with addiction and/or substance abuse problems or high dose opioid or other drugs of potential abuse may require inpatient and/or outpatient chemical dependency treatment programs before or in conjunction with other interdisciplinary rehabilitation. Guidelines from the American Society of Addiction Medicine are available and may be consulted relating to the intensity of services required for different classes of patients in order to achieve successful treatment.   
        
      Interdisciplinary programs may be considered for patients who are currently employed, those who cannot attend all day programs, those with language barriers, or those living in areas not offering formal programs. Before treatment has been initiated, the patient, patient’s family and/or support system, physician, and insurer should agree on the treatment approach, methods, and goals. Generally the type of outpatient program needed will depend on the degree of impact the injury has had on the patient’s medical, physical, psychological, social, and/or vocational functioning. There is some evidence that intensive therapy, 15 hours/week for 16 weeks in a group setting emphasizing integration of cognitive, interpersonal, and functional gains, is superior to the same amount of therapy from multiple individual providers ([Cicerone, 2008](#Cicerone2008)).  
        
      When referring a patient for integrated interdisciplinary rehabilitation, the Division recommends the program meets the criteria of the Commission on Accreditation of Rehabilitation Facilities (CARF).   
        
      There is good evidence that MTBI patients without PTA do not require routine multi-disciplinary care ([[Cochrane] Turner-Stokes, 2005](#TurnerStokes2005)). Inpatient rehabilitation programs are rarely needed for MTBI but may be necessary for patients with any of the following conditions: (a) High risk for medical instability; (b) Moderate-to-severe impairment of functional status; (c) Moderate impairment of cognitive and/or emotional status; (d) Dependence on medications from which he or she needs to be withdrawn; and (e) The need for 24-hour supervision.   
        
      Programs should include the following dimensions:

● Communication: To ensure positive functional outcomes, communication between the patient, insurer and all professionals involved must be coordinated and consistent. Any exchange of information should be provided to all professionals, including the patient. Care decisions should be communicated to all and should include the family and/or support system.

● Documentation: All professionals are expected to maintain thorough documentation regarding discussions with the patient/caregivers. It should be clear that functional goals are being actively pursued and measured on a regular basis to determine their achievement or need for modification. All programs should be able to assess activity limitation, participation restrictions, environmental factors, heath status and impairments in a manner consistent with the ICF Guidelines (refer to Section [C.5. Disability](#TBIC5Disability)).

● Patient Education: Patients with TBI need to re-establish a healthy balance in lifestyle. All providers should educate and provide training and resources for patients/caregivers on how to overcome barriers to resuming daily activity, including management of behavioral issues, cognitive losses, decreased energy levels, financial constraints, decreased physical ability, and change in family and/or support system dynamics.

● Neuropsychological Evaluation and Treatment: Initial full neuropsychological evaluation should occur with periodic assessments to document progress and re-evaluate treatment plans. Treatment may include cognitive, behavioral, and psychological aspects.

● Psychosocial Evaluation and Treatment: Psychosocial evaluation should be initiated, if not previously done. Providers of care should have a thorough understanding of the patient’s personality profile, especially if dependency issues are involved. Psychosocial treatment may enhance the patient’s ability to participate in rehabilitation, manage stress, and increase their problem-solving and self-management skills.

● Treatment Modalities: Use of modalities may be necessary early in the process to facilitate compliance with and tolerance to therapeutic exercise, physical conditioning, and increasing functional activities for moderate/severe TBI. Active treatments should be emphasized over passive treatments. Active treatments should encourage self-coping skills and compensatory behavior, which can be continued independently at home or at work. Treatments that can foster a sense of dependency by the patient on the caregiver should be avoided. Treatment length should be decided based on observed functional improvement. A complete list of active and passive therapies is included in Sections [G. Nonoperative Therapeutic Procedures – Initial Treatment Considerations,](#TBIGTherapeuticProceduresNonOp) [H. Nonoperative Therapeutic Procedures – Neuromedical Conditions in Moderate/Severe Brain Injury](#TBIHNonOp), [I. Nonoperative Therapeutic Procedures – Rehabilitation](#TBIINonOp), [J. Nonoperative Therapeutic Procedures – Vision, Speech, Swallowing, Balance & Hearing](#TBIJNonOp), and [K. Nonoperative Therapeutic Procedures – Return to Work, Driving & Other](#TBIKNonOp). All treatment time frames may be extended based on the patient’s positive functional improvement.

● Therapeutic Exercise Programs: A therapeutic exercise program should be initiated at the start of any treatment rehabilitation. Such programs should emphasize education, independence, and the importance of an on-going exercise regime. For MTBI there is no sufficient evidence to support the recommendation of any particular exercise regimen over any other exercise regimen.

● Return to Work: Rehabilitation programs should provide assistance in creating work profiles. For more specific information regarding return to work, refer to Section [K.2 Return to Work](#TBIK2ReturnToWork).

● Vocational Assistance: Vocational assistance can define future employment opportunities or assist patients in obtaining future employment (refer to Section [K.2 Return to Work](#TBIK2ReturnToWork) for detailed information).

Interdisciplinary brain injury programs are characterized by a variety of disciplines that participate in the assessment, planning, and/or implementation of the treatment program. These programs provide outcome focused, coordinated, goal-oriented interdisciplinary team services to measure and improve the functioning of persons and are for patients with greater levels of perceived disability, dysfunction, de-conditioning, and psychological involvement. Programs should have sufficient personnel to work with the individual in the following areas: behavioral, functional, medical, cognitive, pain management, psychological, social, and vocational. All programs for moderate/severe TBI should be able to address all of the associated neuromedical conditions listed in this guideline. Programs should share information about the scope of the services and the outcomes achieved with patients, authorized providers, and insurers.

The following programs are listed in alphabetical order.

* + 1. Behavioral Programs: Generally accepted TBI inpatient or residential rehabilitation programs designed for individuals with TBI who have persistent and significant maladaptive behaviors. While all TBI rehabilitation programs treat behavior, behavioral programs are usually required for individuals who are unsafe, or who have suicidal, homicidal, or violent behavior and individuals who cannot be treated in less restrictive environments. Behavioral programs may be physically located in secured hospital units or in community-based residential programs, which may also be secured.  
         
       Behavioral programs generally use an interdisciplinary approach that may include behavior analysis and modification, medications, socialization skills training, substance abuse treatment, family therapy, and physical management programs, as well as traditional interdisciplinary treatment. Length of stay may greatly vary depending on etiology and severity of the behavioral disorders and may typically range from one to six months or longer. Upon discharge from behavioral programs, disposition is either back to inpatient acute rehabilitation, inpatient programs, supported living programs or home and community-based programs. Use of psychiatric hospitals that are not experienced in TBI rehabilitation is not recommended. Behavioral programs are also appropriate for severe behavioral problems due to other concomitant diagnoses, such as alcohol or substance abuse, and psychiatric disorders, including any personality disorders. Categorical adolescent inpatient hospital and residential programs may be appropriate for adolescent behavioral disorders due to TBI. Programs should be accredited by Joint Commission.
    2. Comprehensive Integrated Inpatient Interdisciplinary Rehabilitation Programs: A generally accepted and widely used treatment. Inpatient brain injury rehabilitation programs should have designated staff for TBI, designated TBI patient rooms, designated TBI treatment facilities and programs, and they should serve at least 25 to 30 TBI individuals per year. One six-week, non-randomized study with blinded outcome evaluators and a neuropsychological focus demonstrated improvement in overall productivity ([Sarajuuri, 2005](#Sarajuri2005)). Another meta-analysis provided good evidence that inpatient care in specialized stroke units resulted in less disability and less need for long term institutional care ([[Cochrane] Stroke Units Trialists’ Collaboration, 2007](#StrokeUnit2007)]). Inpatient programs should be accredited by the Joint Commission on Accreditation of Healthcare Organizations (Joint Commission) and have components consistent with the Commission on the Accreditation of Rehabilitation Facilities (CARF). CARF eligibility or certification implies that programs meet specific care standards of design and efficacy.  
         
       The interdisciplinary team maintains consistent integration and communication to ensure that all interdisciplinary team members are aware of the plan of care for the patient, are exchanging information, and implement the care plan. The team members make interdisciplinary team decisions with the patient and then ensure that decisions are communicated to the entire care team.   
         
       The Medical Director of the program should be board certified in physiatry, or be board certified in his or her specialty area and have completed a one year fellowship in rehabilitation, or have two years experience in an interdisciplinary brain injury rehabilitation program. Individuals who assist in the accomplishment of functional, physical, psychological, social and vocational goal should include a medical director, team physician(s), and a team neuropsychologist. Other members of the team may include, but are not limited to: biofeedback therapist, occupational therapist, physical therapist, chiropractor, registered nurse, case manager, exercise physiologist, therapeutic recreation specialist, psychologist, psychiatrist, speech-language pathologist, music therapist, optometrist, ophthalmologist, and/or nutritionist.  
         
       The length of initial rehabilitation depends on the severity of deficits, complications, and the individual’s medical progress. Continued lengths of stay should be based on documented functional progress, and may typically range from 30 to 90 days for moderate/severe injury. The individual should be re-evaluated every 30 days. On-site insurance case managers are encouraged to be a part of the treatment team, attend team conferences, and assist the individual and his/her family and/or support system members with facility discharge planning in short and long-term management and goal setting.
    3. Home and Community-Based Rehabilitation: Encompasses services provided in an individual’s home and/or community settings and may be delivered as a separate service or in conjunction with outpatient therapy in a treatment facility. These post-acute services are generally accepted and widely used for individuals with TBI who have completed inpatient or residential rehabilitation, or for those who have not required inpatient or residential services.  
         
       Home and community-based services are designed to maximize the transition and generalization of skills and behaviors in those with moderate/severe injuries from facility settings to application and assimilation in the community. In MTBI, community-based services may be the primary type and most appropriate intervention for those who require more assistance.  
         
       One or more therapeutic disciplines are appropriate to deliver home and community-based services, including qualified/credentialed clinicians from physical therapy, occupational therapy, speech-language pathology, music therapy, medicine, neuropsychology, clinical psychology and counseling, therapeutic recreation, nursing, vocational rehabilitation, and chiropractic treatment. Case management should continue during home and community-based treatment. Programs should preferably be accredited by the Joint Commission and have components consistent with CARF certification. CARF eligibility or certification implies that programs meet specific care standards of design and efficacy.

❖ Frequency: 1 to 7 hours per day, 1 to 3 times per week.

❖ Optimum Duration: For moderate/severe TBI, up to 24 months or beyond with monthly re-evaluations.

* + 1. Nursing Care Facilities: Provide care in specialty licensed units of nursing homes. SNF care is generally accepted and widely used for those who are not able to be managed by a home care agency, in a private home, supported living program, group home, or community setting and have completed extensive rehabilitation therapy. Individuals appropriate for this type of care do not generally require skilled nursing care, but require ongoing care that is supervised by RNs (if medications are involved, it is skilled care). Rehabilitation therapies may be necessary to supplement nursing care. Rehabilitation programs are established by appropriately licensed or certified therapists but may be delivered by paraprofessionals. The goal of care is to maintain and improve function, if possible. This usually occurs at a slower rate over an extended period of time. Accreditation by the Joint Commission is recommended.
    2. Occupational Rehabilitation: A generally accepted interdisciplinary program addressing a patient’s employability and return to work. It includes a progressive increase in the number of hours per day that a patient completes work simulation tasks until the patient can tolerate a full workday. A full workday is case specific and is defined by the previous employment of the patient. Safe work place practices and education of the employer and social support system regarding the person’s status should be included. This is accomplished by addressing the medical, psychological, behavioral, physical, functional, cognitive, and vocational components of employability and return to work.  
         
       The interdisciplinary team should, at a minimum, be comprised of a qualified medical director who is board certified with documented training in occupational rehabilitation, as well as team physicians who have experience in occupational rehabilitation, occupational therapy, and physical therapy.   
         
       As appropriate, the team may also include: a chiropractor, an RN, a case manager, a psychologist, a neuropsychologist, and a vocational specialist or certified biofeedback therapist.

❖ Time to Produce Effect: 2 weeks.

❖ Frequency: 2 to 5 visits per week, up to 8 hours/day.

❖ Optimum Duration: 2 to 4 weeks.

❖ Maximum Duration: 6 weeks. Participation in a program beyond six weeks should be documented with respect to need and the ability to facilitate positive symptomatic and functional gains.

* + 1. Opioid/Chemical Treatment Programs: For specifics, refer to Section F.6.c. Opioid/Chemical Treatment Programs in the Chronic Pain Guidelines.
    2. Outpatient Rehabilitation Services: Generally accepted and widely used. These therapeutic interventions may be delivered in a hospital, free-standing outpatient facilities, or community-based, post-acute facilities with focused goals for home and community functioning. In MTBI, community-based services may be the primary type of appropriate intervention. Frequency varies from daily to less than one day per week and from four to six hours per day. Immediately following inpatient rehabilitation, outpatient rehabilitation is usually intensive, followed by a systematic and gradual reduction in therapy as appropriate. Typically, outpatient treatments include one or more of the following disciplines: physical therapy, occupational therapy, speech/language pathology, music therapy, mental health counseling, neuropsychology, therapeutic recreation, family counseling, vocational rehabilitation, and chiropractic treatment. Outpatient rehabilitation should be functionally oriented, goal-specific, time limited, and case managed. There is good evidence that this type of multi-disciplinary rehabilitation for TBI patients who require hospital admission is likely to provide functional and symptomatic benefit once the patient is able to meaningfully participate ([[Cochrane] Turner-Stokes, 2005](#TurnerStokes2005)).  
         
       Formal outpatient rehabilitation programs should be accredited by the Joint Commission and have components consistent with certification by CARF. CARF eligibility or certification implies that programs meet specific care standards of design and efficacy.

❖ Frequency: 2 to 7 hours per day, 1 to 5 days per week more intensive treatment initially; moderate/severe TBI usually require extended sessions FM.

❖ Optimum Duration: For moderate/severe TBI, up to 24 months, or beyond with monthly re-evaluations.

* + 1. Residential Rehabilitation: Also called residential or transitional living, is clinically appropriate and generally accepted for individuals who have completed initial inpatient rehabilitation. This treatment is indicated for individuals who continue to have significant deficits, who are deemed unsafe to be discharged home, who require continued behavioral treatment, or who are deemed to be more effectively treated in a residential setting. Residential rehabilitation typically includes treatment and management by an interdisciplinary treatment team, with an emphasis on safety, independent living skills and functional community re-integration. Residential rehabilitation is also appropriate for those whose condition has changed, such as in caregiver death, disability, or unavailability, as well as for those who may not have had access to appropriate or adequate inpatient or sub-acute rehabilitation treatment, or for those in whom cognitive, communicative, physical, or behavioral status has deteriorated.  
         
       The length of residential rehabilitation treatment depends on the severity of deficits, complications, progress and available discharge options. Residential rehabilitation is a generally adopted and widely used practice, ranging typically from 30 to 120 days, depending on the individual’s condition and discharge needs, with re-evaluations every 30 days.  
         
       Residential programs should be accredited by the Joint Commission and have components consistent with CARF certification. CARF eligibility or certification implies that programs meet specific care standards of design and efficacy.
    2. Supported Living Programs (SLP) or Long-Term Care Residential Services: Include licensed personal care boarding homes (group homes), supported apartment living programs, or supported inpatient programs designed for long-term living at the completion of the rehabilitation continuum. SLPs are designed for those who, due to their TBI, are not able to care for themselves safely and independently in the community and for whom home placement is unavailable or inappropriate. Such programs are appropriate for individuals who are at risk for medical, cognitive, physical, and psychological complications, but who do not require a secured setting. Housing, food, supervision, activity programs, sheltered employment, transportation, and case management are typical components of supported living programs. These programs are becoming more available and are generally accepted services for individuals with chronic brain injury who are moderately to severely disabled, and who require care, supervision, and support services. Specialty supported living programs are available for behaviorally challenged individuals. Long-term residential services should be accredited by the Joint Commission.
  1. ACTIVITES OF DAILY LIVING (ADLs): (also called daily living skills, life skills or living skills) Tasks necessary for an individual’s day-to-day functioning, including both basic and instrumental level tasks. ADL functional limitations and disabilities in ADLs are common following TBI and are often due to changes in physical, cognitive, and emotional/behavioral impairments. Functional limitations and disability in these areas may range from mild to severe, as well as from short-term to life-long.  
       
     Therapeutic intervention for ADLs is generally accepted and widely used. The goal of treatment is to improve one’s ability to perform such tasks, in order to increase functional levels of independence. There is good evidence in the stroke population that occupational therapy provides a modest reduction in disability and risk of death ([[Cochrane] Legg, 2006](#Legg2006)). By including ADLs in treatment, cognitive improvements may occur by applying cognitive rehabilitation principles to the task performance. Likewise, physical deficits may be improved by applying neuromuscular rehabilitation principles to the task performance.
     1. Basic ADLs: Include daily activities that tend to be repetitive, routine, and that may more readily be gained through procedural learning, such as grooming, personal hygiene, bathing/showering, toileting, dressing, feeding/eating, and basic social skills.
     2. Instrumental ADLs (IADLs): Include a wide range of activities that require higher level cognitive skills, including the ability to plan, execute, and monitor performance, as well as the ability to evaluate information and make sound judgments. These abilities are essential to safe, independent functioning. They may include functional communication (e.g., writing, keyboarding, appropriate use of phone), home management, childcare, time management, financial management, food management, management of interpersonal relationships and social skills, avocation, driving, and higher level mobility skills (including navigation and public transportation).

Therapeutic intervention is generally accepted to improve performance of ADLs. Procedures and techniques may include, but are not limited to: (1) task analysis to develop strategies to improve task performance; (2) guided practice and repetition to develop consistent and safe performance; (3) training in safe use of adaptive equipment; and (4) training of caregiver(s).  
  
All treatment should be interdisciplinary. Treatment in sub-acute and acute rehabilitation is provided by one or more therapeutic disciplines, including occupational therapy, physical therapy, speech-language pathology, social work, family counseling, psychology, nursing, and/or vocational rehabilitation as tolerated. In post-acute settings (which may include residential or outpatient), treatment sessions may be provided by more than one discipline. For in-home and community-based treatment, interdisciplinary treatment continues until: (1) functional goals/outcomes are achieved; (2) plateau in progress is reached; (3) the individual is unable to participate in treatment due to medical, psychological, or social factors; or (4) skilled services are no longer needed.

❖ Time to Produce Effect: While rate of progress will depend on the severity and complexity of the injury, effect of treatment should be noted within one month, with ongoing progress noted over a longer period, which may last up to two years or more. Treatment may be provided on an episodic basis to accommodate plateaus in the individual’s progress, with suspension of treatment for periods of time to allow for practice.

❖ Frequency: Daily, depending on the individual’s progress, sessions may vary from one to several hours depending upon individual’s ability to respond to treatment. Periodic upgrading or consultation may be necessary throughout the individual’s lifetime following TBI.

❖ Optimum Duration: 1 to 12 months.

❖ Maximum Duration: 24 months or beyond, requires documentation of progress or the need for maintenance to retain ADLs.

Therapy may be re-initiated for time limited, goal-specific treatment as new goals are developed.  
  
Impaired cognition significantly affects the rate, degree, and manner of progress toward independence in ADLs. In addition, skills learned in one setting or circumstance may facilitate transfer of skills. All treatment to improve performance in this area should include techniques to improve cognition as well.  
  
Standard equipment to alleviate the effects of the injury on the performance of ADLs may vary from simple to complex adaptive devices to enhance independence and safety. Certain equipment related to cognitive impairments may also be required. Equipment needs should be reassessed periodically.  
  
The results of treatment intervention provided throughout the continuum of progress beginning with acute care may be realized in the final stages of integration back into the individual’s community setting. As noted above, treatment is often indicated at this stage to ensure that the individual is able to reintegrate as successfully as possible, given the parameters of the injury.

* 1. MOBILITY:
     1. Therapy: Individuals who have sustained a moderate/severe TBI may experience changes in their mobility control and may require medical, surgical, physical, and functional therapeutic management to improve their movement and function. Impairments may affect functional skills, including a propensity for falls, and may be seen in the following areas: bed mobility, wheelchair mobility, seating and positioning, transfers, and ambulation.  
          
        Therapeutic intervention supervised by a physical or occupational therapist is generally accepted and widely used to improve performance of mobility impairments. Treatment may include, but is not limited to, the areas of bed and mat mobility skills, sensory integration, endurance, balance, coordination, strengthening, stretching, gait training, neuromuscular re-education and postural control. Training is also indicated for individuals and their family and/or support system in the areas of wheelchair mobility, seating and positioning, ROM, functional mobility (bed mobility, and transfers, ambulation), and therapeutic exercise. The use of modalities (functional electrical stimulation, TENS, ultrasound, phonophoresis, biofeedback) may be indicated to improve function. Passive modalities should not be utilized in isolation without a comprehensive therapeutic intervention program. Other indicated therapies may include pool therapy, casting/splinting programs, and facility-based exercise programs. Orthopedic and/or neuromuscular problems may develop along with mobility impairments. These may include, but are not limited to, heterotopic ossification, limb contractures, and abnormal tone, which may interfere with the advancement of independence with mobility skills.  
          
        Therapy to improve gait after moderate/severe TBI or stroke with foot drop or other gait difficulties, is variable and includes treadmill training with body weight support, unsupported treadmill walking, electromyographic biofeedback with therapy, use of gait assistive devices such as a stick or frames and other therapist facilitated therapy. None of these therapies is clearly superior to another ([Williams, 2011](#Williams2011); [[Cochrane] Moseley, 2005](#Moseley2005); [Intiso, 1994](#Intiso1994); [Brown, 2005](#Brown2005)).   
          
        Music therapy is commonly employed for moderate/severe TBI patients. There is good evidence for improving gait speed in acquired TBI patients with music therapy and some evidence that music therapy may improve gait symmetry, cadence, and stride length, although changes in gait endurance are less clear ([[Cochrane] Bradt, 2010](#Bradt2010)).

❖ Time to Produce Effect: While rate of progress will depend on the severity and complexity of the injury, effect of treatment should be noted within one month, with ongoing progress noted over a longer period, which may last up to two years or more. Treatment may be provided on an episodic basis to accommodate plateaus in the individual’s progress, with suspension of treatment for periods of time to allow for practice.

❖ Frequency: Daily for moderate/severe TBI and less frequently for MTBI requiring treatment, usually 1–3 times per week. Depending on the individual’s progress, sessions may vary from one to several hours, based on the individual’s ability to respond to treatment and the setting.

❖ Optimum Duration: 1 to 12 months.

❖ Maximum Duration: 24 months or beyond, requires documentation of progress or the need for maintenance to retain mobility. Periodic upgrading or consultation may be necessary throughout the individual’s lifetime following TBI.

Short-term, goal-directed mobility interventions may be periodically indicated on an ongoing basis as new changes occur in an individual’s functional mobility. Impaired cognition significantly affects mobility as noted by problems with attention, judgment, organization or auditory and/or visual instructions, memory, concentration, problem solving, behavior, and initiation. (Refer to discussion at the beginning of Section [G. Therapeutic Procedures – Nonoperative](#TBIGTherapeuticProceduresNonOp)).

* + 1. Adaptive Devices: Individuals with moderate/severe TBI may be compromised in their mobility and accessibility to their home, work, and community environments. In order to relieve the effects of the injury, certain equipment, adaptive devices, and home modifications may be reasonable and necessary. These items may be necessary to reduce impairment and disability and to enhance functional independence and safety.  
         
       Technology is advancing rapidly in this area, and each year more, adaptive equipment is available. Each case should be considered individually to determine the medical need for the equipment. Possible equipment and devices may include, but are not limited to:

● Hospital bed.

● Transfer devices and lift equipment.

● Standing frames.

● Manual wheelchair (standard or lightweight).

● Manual reclining and tilt wheelchair.

● Power wheelchairs with tilt and/or reclining mechanisms.

● Wheelchair positioning aids (laterals, headrests, seating systems, backs, lapboards).

● Wheelchair cushions.

● Lower extremity bracing.

● Ambulation aids (walkers, crutches, canes).

● Bathroom equipment, accessibility, and safety aids (shower/commode chair, bath seats and benches, tub and wall grab bars, hand held shower attachment, elevated and/or padded toilet seats, etc.).

● Orthotics/prosthetics.

● Vehicle modifications.

* Communication aids and devices including computers.
* Visual adaptive aids.
* Other adaptive equipment for independent ADLs, such as specialized eating utensils.

Environmental modifications may include, but are not limited to: ramping; modifications of the living environment to achieve reasonable levels of independence; and adaptive equipment for mobility and safety. Typically, these evaluations are done by a licensed contractor and occupational or physical therapist with experience in ADA standards. Modifications must be medically necessary. Periodic upgrading of equipment and devices or consultation may be necessary throughout a person’s lifetime following TBI.  
  
Therapy related to equipment and devices may be re-initiated for time limited, goal-specific treatment as new goals are developed.

* 1. Ataxia: A common impairment in coordination resulting from the inability to control muscle timing and the sequencing of agonist and antagonist contraction. This will affect fine motor and gross motor skills of the extremities as well as general mobility, balance, gait, conditioning, endurance, and ADLs. Therapeutic management/intervention includes medication and neuromuscular re-education as well as functional activities, which facilitate normal or inhibit abnormal muscle activity. Specific exercises and activities increase motor learning and control and force production (strength) and endurance. Biofeedback and functional electrical stimulation may assist in treatment. Cognitive impairment may interfere with and prolong the course of therapy. Reasonable and necessary equipment may include splints and braces.
  2. NEUROMUSCULAR re-education: Neurologically-based musculoskeletal impairment may include changes in reflexes, sensory integration, ROM, muscle tone, strength, endurance, postural control, postural alignment, and soft tissue integrity. Functional abilities that are affected may include, but are not limited to, problems in gross and fine motor coordination, motor strength and control, sensory-motor bilateral integration, and praxis. Individuals with neuromuscular impairments may require physical, therapeutic, and medical and/or surgical management to improve their movement and mobility.

There is good evidence that constraint induced motor therapy (CIMT) provides a favorable effect immediately post treatment for stroke victims with paresis of one arm and good cognition ([[Cochrane] Sirtori, 2009](#Sirtori2009)). There is some evidence that the motor function associated with CIMT is maintained at 24 months after treatment ([Wolf, 2008](#Wolf2008)). Therefore, CIMT is a recommended therapy for similarly affected TBI patients.   
  
Medical treatment may be divided into two major areas:

* + 1. Motor Control: Stabilizing the body in space as it applies to postural and balance control, and moving the body in space through motor control as it applies to movement.
    2. Motor Learning: A set of processes leading to relatively permanent changes in the capability for producing skilled action. Motor performance of a skill, task or activity requires learning. Functional motor change requires skilled intervention to insure proper practice schedules, variable type of practice, repetition, and type of timing of feedback. Active problem solving should be part of a rehabilitation program to learn motor skills more appropriately. Continuous, accurate feedback is important in the early stages. Therapists need to provide feedback about muscle contraction and movement that is accurate and immediate.

❖ Time to Produce Effect: While rate of progress will depend on the severity and complexity of the injury, effect of treatment should be noted within one month, with ongoing progress noted over a longer period, which may last up to two years or more. Treatment may be provided on an episodic basis to accommodate plateaus in the individual’s progress, with suspension of treatment for periods of time to allow for practice.

❖ Frequency: Daily for moderate/severe TBI and less frequently for MTBI requiring treatment, usually 1–3 times per week. Depending on the individual’s progress, sessions may vary from one to several hours, based on the individual’s ability to respond to treatment and the setting.

❖ Optimum Duration: 1 to 12 months.

❖ Maximum Duration: 24 months or beyond, requires documentation of progress or the need for maintenance to retain motor skills. Periodic upgrading or consultation may be necessary throughout the individual’s lifetime following TBI.

As the individual progresses, treatment frequency should be decreased. Continued treatment is based on attainment of functional goals as outlined in the treatment plan, which is established during initial interaction with all members of the treatment team.

* 1. Work Conditioning: These well-accepted programs are work related, outcome focused, individualized treatment programs. Objectives of the program include, but are not limited to, improvement of cardiopulmonary and neuromusculoskeletal functions (strength, endurance, movement, flexibility, stability, and motor control functions), patient education, and symptom relief. The goal is for patients to gain full or optimal function and return to work. The service may include the time limited use of modalities, both active and passive, in conjunction with therapeutic exercise, functional activities, general conditioning body mechanics, and re-training of lifting techniques. The patient should be assisted in learning to pace activities to avoid exacerbations.   
       
     These programs are usually initiated once re-conditioning has been completed but may be offered at any time throughout the recovery phase. It should be initiated when imminent return of a patient to modified or full duty is not an option, but the prognosis for returning the patient to work at completion of the program is at least fair to good.

❖ Length of Visit: 1 to 2 hours per day.

❖ Frequency: 2 to 5 visits per week.

❖ Optimum Duration: 2 to 4 weeks.

❖ Maximum Duration: 6 weeks. Participation in a program beyond 6 weeks should be documented with respect to need and the ability to facilitate positive symptomatic and functional gains.

* 1. Work Simulation: A generally accepted program where an individual completes specific work related tasks for a particular job and return to work. Use of this program is appropriate when modified duty can only be partially accommodated in the work place, when modified duty in the work place is unavailable, or when the patient requires more structured supervision. The need for work place simulation should be based on the results of a functional capacity evaluation and/or jobsite analysis.

❖ Length of Visit: 2 to 6 hours per day.

❖ Frequency: 2 to 5 visits per week.

❖ Optimum Duration: 2 to 4 weeks.

❖ Maximum Duration: 6 weeks. Participation in a program beyond 6 weeks should be documented with respect to need and the ability to facilitate positive symptomatic and functional gains.

* 1. MUSCLE TONE AND JOINT RESTRICTION MANAGEMENT, Including spasticity: Defined as velocity dependent hyperactivity of stretch reflexes secondary to the upper motor neuron syndrome. It is characterized by exaggerated deep tendon reflexes, increased muscle tone that results in a range of abnormal reflexes and motor patterns. The Modified Ashworth Scale is a clinical tool for measuring resistance to passive limb movement. If spasticity is interfering with the individual’s general functioning (which may include ROM limitations, limitations in care and/or ADLs, and limitations in mobility), then treatment is often warranted. Individuals with moderate/severe TBI may demonstrate changes in muscle activation based on emotional factors, positional changes, and functional demands. Treatment approaches involve the disciplines of rehabilitation nursing, physical therapy, speech pathology, and occupational therapy. Therapeutic intervention should concentrate on active control, force production, and functional muscle use rather than just tone or spasticity reduction. Specific treatments may include, but are not limited to:
     1. Orthotics and Casting: Serial casting may also be effective to increase ROM by inhibiting tone and increasing passive muscle length. Serial casting should be reapplied every two weeks as appropriate with increasing stretch and may require an overall treatment period of two to three months. An orthosis may be applied across the joint involved as well as at the joints above and below to maintain tone inhibition and muscle length. These orthoses may be removed to allow therapeutic activity, hygiene and modification based on progress in ROM and movement. Functional activity, such as reaching, grasp with the upper extremity, and gait involving the lower extremity, should be performed with the orthosis in place. Functional electrical stimulation may be used as a functional orthosis, and both devices may be required to be long-term, if not permanent. Orthotics are often prescribed to protect affected joints and to prevent contracture. Additionally, special seating positioning devices and techniques may be required above and beyond a standard positioning method.
     2. Postural Control: Trunk control is essential for the body to remain upright and to adjust and control movements against gravity. Postural control, mobility, tone, and stability are evaluated by assessing the basic movement components of the upper and lower body, the coordinated trunk, extremity patterns, and the power production involved in equilibrium and protective reactions. Basic movement components of the trunk are then progressed to the linking of trunk and extremity movements in supine, sitting, and standing positions. The last level involves strength and stability for power production for activities such as walking, stair climbing, jumping, running, and throwing.
     3. Functional and Therapeutic Activities: Provided with instruction for the individual and family and/or support system in the proper positions, sequences, timing, and level of assistance. There is good evidence for the use of mirror therapy to improve motor function of upper or lower limbs after a stroke ([[Cochrane] Thieme, 2012](#Theime2012)).   
          
        Periodic functional upgrading or consultation may be necessary throughout an individual’s lifetime following moderate/severe TBI. Therapy may be re-initiated for time limited, goal-specific treatment as new goals are developed and as new abilities in physical and cognitive function are observed or attained (refer to Section [G.8. Therapeutic Exercise](#TBIG8TherapeuticExercise), for further details).
     4. Therapeutic Nerve and Motor Point Blocks: Useful in targeting specific muscles or muscle groups for diagnostic and therapeutic purposes. The purpose of the nerve or motor point block is to reduce force produced by contracting spastic muscle or muscle group. This reduction in spasticity may lead to improved ROM and enhanced functioning. Therapeutic nerve and motor point blocks are primarily performed with aqueous solutions of phenol. When injected in or near a nerve bundle, phenol denatures protein in the myelin sheath or cell membrane of axons with which it makes contact. Either percutaneous or open neurolytic procedures are considered useful in a variety of spastic disorders related to TBI and are generally accepted procedures. Refer to the appropriate guideline.
     5. Botulinum Toxin (Botox) Injections: Used to temporarily weaken or paralyze muscles. May reduce muscle pain in conditions associated with spasticity, dystonia, or other types of painful muscle spasm. Neutralizing antibodies develop in at least 4% of patients treated with botulinum toxin type A, rendering it ineffective. Several antigenic types of botulinum toxin have been described. Botulinum toxin type B, first approved by the Food and Drug Administration (FDA) in 2001, is similar pharmacologically to botulinum toxin type A. It appears to be effective in patients who have become resistant to the type A toxin. The immune responses to botulinum toxins type A and B are not cross-reactive, allowing type B toxin to be used when type A action is blocked by antibodies. Experimental work with healthy human volunteers suggests that muscle paralysis from type B toxin is not as complete or as long lasting as that resulting from type A. There is strong evidence that botulinum toxin A has objective and symptomatic benefits over placebo for cervical dystonia [([Cochrane] Costa, 2005](#Costa2005)). The duration of treatment effect of botulinum toxin type B for cervical dystonia has been estimated to be 12 to 16 weeks. EMG needle guidance may permit more precise delivery of botulinum toxin to the target area.
        1. Indications: Used to improve ROM and reduce painful muscle spasm, as a temporizing measure when spasticity is evolving, and during the chronic phases to support increased function. Botulinum toxin injections may be useful in musculoskeletal conditions associated with muscle spasm, and in central neurologic conditions that produce spasticity or dystonia (e.g., brain injury, spinal cord injury or stroke). There should be evidence of limited ROM prior to the injection.   
             
           Botulinum injections are no longer generally recommended for cervicogenic or other headaches based on good evidence of lack of effect ([[Cochrane] Langevin, 2011](#Langevin2011); [Linde, 2011](#Linde2011)). For more detailed information regarding headaches, refer to Section [G.7. Headache](#TBIG7Headache). Botulinum injections are not routinely recommended, but they may be used in unusual cases.
        2. Complications: Over-weakening of injected muscles, migraine, and allergic reaction to medications. Rare systemic effects include flu-like syndrome and weakening of distant muscles. There is an increased risk of systemic effects in individuals with motor neuropathy or disorders of neuromuscular junction.

❖ Time to Produce Effect: 24 to 72 hours post-injection with peak effect by 4 to 6 weeks.

❖ Frequency: No less than 3 months between re-administration.

❖ Optimum Duration: 3 to 4 months.

❖ Maximum Duration: Unknown at the time of this guideline. Repeat injections should be based on functional improvement and therefore used sparingly in order to avoid development of antibodies that might render future injections ineffective

* + 1. Pharmaceutical Agents: A variety of oral and transdermal antispasticity medication may also be used.
    2. Intrathecal Baclofen Drug Delivery:
       1. Description: The intrathecal administration of baclofen is indicated for use in the management of severe spasticity. Individuals with moderate/severe TBI should first have a positive response to a diagnostic injection of intrathecal baclofen prior to a consideration of long-term infusion via an implantable pump. An implantable pump should be reserved for those individuals unresponsive to oral baclofen therapy, or those who experience intolerable CNS side effects at effective doses. Individuals with functionally limiting disabling spasticity due to TBI ideally should wait at least one year post-injury, unless there is clear documentation as to plateaued neurological functioning prior to the one-year post-injury mark. Furthermore, there should be clear-cut documentation as to the deleterious effects of their persistent spasticity if not treated effectively, as well as to the specific goals of this invasive therapy. Intrathecal baclofen is intended for use via spinal catheter or lumbar puncture and for chronic use only in implantable pumps approved by the FDA, specifically for the administration of intrathecal baclofen via the intrathecal space.
       2. Diagnostic Injection:
          1. Special Requirements for Diagnostic Injections: Fluoroscopic and/or CT guidance may be used to document technique and needle placement. An experienced physician should perform the procedure. The subspecialty disciplines of the physicians may be varied, including, but not limited to, anesthesiology, radiology, surgery, neurology or physiatry.
          2. Complications: General complications of diagnostic injections may include, but are not limited to, transient neurapraxia, nerve injury, infection, headache, urinary retention, and vasovagal effects, as well as epidural hematoma, permanent neurological damage, dural perforation, CSF leakage, and spinal meningeal abscess. Permanent paresis, anaphylaxis, and arachnoiditis have been rarely reported.
          3. Contraindications: Absolute contraindications to diagnostic injections include, but are not limited to: (a) bacterial infection-systemic or localized to the region of injection, (b) bleeding diathesis, (c) hematological conditions and (d) possible pregnancy. Relative contraindications of diagnostic injections may include: (a) allergy to contrast, (b) aspirin/antiplatelet therapy (drug may be held three days or more prior to injection), and (c) shellfish allergy if contrast is to be used.
       3. Surgical Pump Implantation:
          1. Surgical Indications: Individuals who meet the following criteria should be considered candidates for intraspinal baclofen infusions:

● The individual should have quantifiable relief from the diagnostic baclofen intrathecal injection and have demonstrated clear functional improvement. Functional gains may be evaluated by an occupational therapist and/or physical therapist prior to and before discontinuation of the trial.

● Failure of conservative therapy, including active and/or passive therapy, medication management, or other therapeutic injections.

● The individual and family and/or support system should be motivated for the procedure and should understand the potential for complications and the requirements of treatment maintenance.

* + - * 1. Complications: Intrathecal delivery may be associated with significant complications such as infection, catheter disconnects, CSF leak, arachnoiditis, pump failure, nerve injury and paralysis.
        2. Contraindications: Infection or body size insufficient to support the size and weight of the implanted device. Individuals with other implanted programmable devices should not be given these pumps, since interference between devices may cause unintended changes in infusion rates.
        3. Continuing Use: As with other routes of drug administration, escalation of dose may be required and routine clinical monitoring is warranted. Typically, pump refills are needed every two to three months.

1. NONOPERATIVE THERAPEUTIC PROCEDURES – VISION, SPEECH, SWALLOWING, BALANCE, & HEARING
   1. VISUAL TREATMENT: Visual impairments may occur secondary to TBI. Treatment of visual impairments should be based on a comprehensive evaluation and diagnosis. Treatment should be functionally-based and goal-directed. Individuals should be evaluated at intervals depending on their impairment, and progress should be clearly documented. An ophthalmologist, neuro-ophthalmologist, neurologist, occupational therapist, or optometrist may treat visual impairment resulting from TBI. Treatment should be coordinated with the other interdisciplinary team members with the purpose of achieving the functional goals. Visual impairments may occur in one or more of the following categories:

● Visual acuity and visual field function.

● Ocular motor control and ocular alignment.

● Visual perception.

Note: Visual therapy is also performed for dizziness (refer to Section [J.2.f. Vestibular Rehabilitation](#TBIJ2fVestibularRehab) for details).

* + 1. Visual Acuity and Visual Field Function: Determined by the eye, optic nerve, optic chiasm, optic tracts, optic radiations, and visual cortex. If visual acuity deficits are caused by optic nerve trauma, treatment with high dose intravenous corticosteroids may be useful ([Spoor, 2008](#Spoor2008)). Surgery may be indicated if the trauma results in entrapment, compression of the nerve, or if a hematoma is present within the optic nerve sheath. If visual acuity or visual field deficits are caused by intracranial visual pathway damage, acute treatment should be directed toward the specific injury.  
         
       Low vision aids may be prescribed for those individuals with documented visual acuity or visual field loss after the acute injury. Lenses may be used to improve visual acuity. Tinted lenses may be useful to treat photophobia and glare sensitivity.  
         
       The use of prisms may benefit some individuals with documented visual field loss from visual pathway disorders that affect the visual fields in both eyes.  
         
       Depending on the level of adaptation to the visual field loss, some individuals may need training and education in strategies to improve compensation. Efforts to use visuospatial interventions to improve visual field loss directly without developing compensatory visual scanning are not recommended. The use of computers as a primary and independent form of visual treatment has limited application because of: (1) limitations in the rationale and specific application of software programs to address the needs of the individual with TBI; and (2) difficulty with generalization of learned computer skills into functional environments. Integrated computer-based treatment (i.e., both individualized cognitive and interpersonal therapies) may improve functioning within the context of an interdisciplinary, neuropsychological rehabilitation program. Sole reliance on repeated exposure and practice on computer-based tasks without extensive involvement and intervention by a therapist is not recommended. Virtual reality tools may prove useful for ADL assessment and training; however, they are experimental at the time of these guidelines, as there are no strong studies supporting its success ([[Cochrane] Laver, 2011](#Laver2011); [Schultheis, 2002](#Schultheis2002)). Computerized visual restoration therapy programs or other computerized visual treatment programs, such as virtual reality, are not recommended due to lack of proven clinically meaningful efficacy and cost ([Pelak, 2007](#Pelak2007); [Schreiber, 2006](#Schreiber2006); [McFadzean, 2006](#McFadzean2006); [Reinhard, 2005](#Reinhard2005)).
    2. Disorders Involving Ocular Motor Control and Ocular Alignment: Treated according to the underlying diagnosis. Ocular motor control includes accommodation, versions, vergences, ductions, ability to fixate, pursuits, saccades, vestibulo-ocular responses (VOR), and optokinetic nystagmus (OKN). Multiple deficits may occur together.  
         
       Treatment may include the use of lenses, prisms, rehabilitation vision therapy techniques, and/or surgery. For individuals with disorders of ocular motor and ocular alignment that result in diplopia, monocular eye patching, occlusion of central or peripheral vision, prisms lenses, or strabismus, surgery may be used.  
         
       Lenses may be used to help accommodation. Because of the interaction between accommodation and vergence, lenses may also at times be used to assist in the treatment of a vergence disorder.  
         
       Prisms may be prescribed to provide an immediate improvement in diplopia and other disorders with symptoms. If diplopia is not stable, then appropriate patching (partial selective occlusion) may be more prudent. If deficits are permanent, prisms may be worn indefinitely.  
         
       Individuals may be instructed in orthoptic techniques to address problems related to ocular motility disorders, particularly in cases with cranial nerve palsy or encephalopathy with a correlated shearing injury.  
         
       Strabismus surgery may be useful in certain circumstances if the deficit is stable for six to nine months. An immediate response is usually noted after the first surgery, but additional surgeries may be necessary.
    3. Visual Perception: Problems should be treated with a goal to improve visual processing skills and promote adaptation and compensation to the relevant problem.  
         
       Visual perceptual therapy may be required for some individuals as part of their overall cognitive rehabilitation treatment. The therapy may be provided by specialists with experience in visual perceptual disorders. They may be from various disciplines, including, but not limited to, occupational therapy, speech therapy, neuropsychology, optometry and ophthalmology, neurology, and neuro-ophthalmology. The visual perceptual therapy should be integrated into the complete cognitive rehabilitation program and coordinated with a neuropsychologist or physician experienced in TBI (refer to Section [G.3 Cognition](#TBIG3Cognition)).
    4. Visual Inattention: Inattention of a visual spatial region. Treatment may include the use of prisms and scanning techniques. Visuospatial rehabilitation with scanning is recommended for individuals with visuospatial perceptual deficits associated with visual neglect following TBI and after right parietal stroke. There is some evidence that visuospatial rehabilitation with scanning is effective in those with right hemispheric stroke; therefore, it is recommended in TBI individuals with similar findings ([Weinberg, 1977](#Weinberg1977)). Scanning training is recommended as an important, even critical, intervention element for individuals with severe visual perceptual impairment that includes visual neglect after right hemispheric stroke and TBI. Efforts to use visuospatial interventions to improve visual field loss directly without developing compensatory visual scanning are not recommended.
    5. Total Time Frames for all Vision Therapy (Orthoptic Therapy): Time frames are not meant to be applied to each section separately. The time frames are to be applied to all vision therapy regardless of the type or combination of therapies being provided.

❖ Time to Produce Effect: 4 hours of treatment should result in a measurable functional improvement.

❖ Frequency: 1 to 2 times per week with daily exercise at home. Frequency of treatment is dependent on in-patient versus outpatient, and the medical condition of the individual.

❖ Optimum Duration: 12 hours.

❖ Maximum Duration: 20 hours. Throughout the treatment progress, exams are performed to evaluate status. When progress is no longer occurring, then therapy should be stopped, unless there are mitigating circumstances. If after 20 hours of treatment, there is documented progress, but the individual is not at maximum therapeutic gain, then additional therapy may be indicated. Additional therapy should take into consideration the overall rehabilitation plan for the individual.

* 1. NEURO-OTOLOGIC TREATMENTS: For patients with dizziness causing nausea or affecting balance, treatment of these conditions may be necessary before other rehabilitative therapy can be accomplished.
     1. Treatment of Fixed Lesions:
        1. Post-Traumatic Tinnitus: Individuals with TBI may suffer from debilitating tinnitus (ringing in the ears). They may benefit from anti-depressants, anti-seizure medicines, and anxiolytics. In many situations, devices are recommended and may include hearing aids, maskers, and tinnitus trainers. Tinnitus trainers require a 30 day trial to determine masking. More sophisticated devices that use music as opposed to masking are not recommended due to no proof of their superiority ([Hobson, 2010](#Hobson2010)).
        2. Hyperacusis/Sonophobia: Individuals with TBI may suffer from significant sensitivity to sound. These individuals may benefit from devices such as tinnitus trainers, musician’s plugs, and simple noise plugs.
        3. Sensorineural Hearing Loss: Individuals with TBI may suffer from nerve hearing loss that may be treated with amplification (hearing aids). A full audiometric evaluation may determine if the individual could benefit from such devices.
        4. Vestibular Loss: Individuals with TBI may suffer from loss of inner ear balance function resulting in dizziness and imbalance. This can result from labyrinthine concussion, penetrating injuries, injury to the 8th nerve, and explosive pressure changes. Vestibular rehabilitation is of benefit in speeding compensation for these losses.
     2. Treatment of Recurrent, Non-Progressive Otologic Disorders:
        1. Benign Positional Vertigo (BPV): The most common cause of post-traumatic vertigo (refer to Section [J.2.f.iv. Benign Positional Vertigo (BPV)](#TBIJ2fivBenignPositionalVertigo) for a description). It is an otologic disorder in which particles normally adherent to the gravity sensors of the ear become displaced into the semicircular canals, which cause the sensation of spinning. It is characterized by recurrent, brief spells of vertigo triggered by head movements such as getting in and out of bed, rolling over in bed, tipping the head upward, or bending over. It is diagnosed by the Dix Hallpike maneuver and treated with canalith repositioning maneuvers (CRM) specific to the affected semicircular canals. Patients treated by CRM should be re-evaluated within the first month to ensure resolution of symptoms. Recurrences are common after trauma. These may be treated by repeating the CRM, home exercises, or referral to physical therapy.
        2. Semicircular Canal Dehiscence: An abnormal communication between the CSF space in the skull and perilymph surrounding the inner ear. It can result from blunt head trauma with fracture of the bone separating these spaces. Symptoms include vertigo brought on by loud sounds or straining and autophony, which is the magnification of internal bodily sounds (chewing, eye movement, joint movement, heartbeat) in the affected ear. Vestibular suppressants and avoidance of provoking sounds can be used; surgery is required in severe cases.
        3. Vestibular Migraine: Individuals experiencing an exacerbation of migraine after TBI frequently have an associated dizziness. Treatment includes trigger avoidance, vestibular suppressants, and migraine prophylactic medications such as calcium channel blockers, anti-seizure medication, beta blockers, and SSRI’s (refer to Section [G.7. Headache](#TBIG7Headache)).
        4. Impaired Compensation due to Multisensory Imbalance: A form of persistent dizziness following a fixed vestibular injury characterized by a constant feeling of dysequilibrium that is worsened when walking or making rapid head turns and improved when seated or using a grocery store cart or walker. Compensation to vestibular injury requires normal binocular vision and visual tracking, normal head/neck mobility, normal somatosensation, and strength and mobility in the legs and feet. Injuries to any of these areas can significantly delay recovery from concurrent vestibular injuries. Treatment requires physical therapy directed at these deficits. A rolling walker with handbrakes or other assistive devices may be provided when balance is significantly impaired.
     3. Treatment of Progressive Otologic Disorders:
        1. Progressive Vestibulopathy with or without Hearing Loss: Ears with acute auditory or vestibular injuries occasionally convert to a progressively damaging disorder with recurrent vertigo spells and gradual loss of hearing and/or balance function over time. Cases with discrete vertigo spells of hours in duration associated with tinnitus and fluctuating hearing are called post-traumatic endolymphatic hydrops or Ménière’s disease. Hydrops refers to dilation of the endolymph space of the inner ear at the expense of the surrounding perilymph space, and is highly associated with Ménière’s disease, although the mechanism of the ear dysfunction is not yet known. Treatments include diuretics, calcium channel blockers, steroids, gentamicin perfusion, and surgery.
        2. Perilymphatic Fistula: Ruptures of the round or oval windows of the inner ear or fractures through the ear can result in leakage of perilymph. This can cause progressive hearing loss and recurrent dizziness that is often triggered by straining. Treatment is bed rest with the head elevated and avoidance of straining for mild cases. Surgery is required for severe cases and those not responding to a week of bed rest.
     4. In-Office Treatment Procedures:
        1. Steroid Perfusion: During this procedure, steroids are injected into the middle ear space, allowing absorption into the inner ear via the round window membrane. It can provide a temporary reduction in the frequency of vertigo spells in progressive vestibulopathy and may improve hearing after sudden losses.
        2. Gentamicin Perfusion: This is an in office procedure where gentamicin is injected into the middle ear space. From there it is absorbed into the inner ear via the round window membrane. This procedure may have to be repeated several times to control dizzy spells. The gentamicin is toxic to the cells of the inner ear and therefore destroys the inner ear balance function. There is also a significant risk to the hearing function. This procedure has a 95% success rate, but because of its destructive nature, cannot be used in bilateral disease. Use should be reserved for cases in which a unilateral progressive hearing loss and/or loss of vestibular function has been documented.
        3. The Meniett Device: This is a portable, alternating pressure generator which transmits low-pressure pulses to the middle ear. There is good evidence of short-term symptomatic and functional daily use benefit in individuals with established Ménière’s disease, reduced vestibular function, and severe vertigo, which persist despite adequate medical therapy ([Gates, 2004](#Gates2004); [Gurkov, 2012](#Gurkov2012)). The mechanism of benefit is not yet understood. Individuals must be able to tolerate tympanostomy tubes and practice water precautions and aural hygiene to maintain tube patency. Effectiveness beyond four months of treatment has not been established. \*\*Use of the Meniett device requires a surgical procedure (refer to Section [L.7.b Tympanostomy](#TBIL7bTympanostomy)).\*\*
     5. Tympanostomy: Tube placement may be needed for use of a Meniett device.
     6. Vestibular Rehabilitation: Performed by qualified practitioners, e.g., audiologists, otologists, trained nurses, physical therapists (preferably neurology certified) or occupational therapists. Symptoms of vestibular system dysfunction following TBI may be due to damage of central or peripheral structures and may include vertigo, eye-head dyscoordination affecting the ability to stabilize gaze during head movements, and imbalance affecting stability in standing or walking. Dizziness is commonly associated with TBI. Dizziness and balance disorders may or may not co-exist in the same individual with TBI.
        1. Balance Disorders: Balance disorders occur frequently following TBI. One study of MTBI patients four years after the incident found 30% continuing complaints of balance problems. Balance is a complex motor control task, requiring integration of sensory information, neural processing, and biomechanical factors. It is the ability to control the center of gravity (COG) over the base of support in a given sensory environment. This may be due to a peripheral vestibular lesion or central vestibular lesion secondary to trauma, fracture, hemorrhage or intracranial pressure changes ([Kleffelgaard, 2012](#Kleffelgaard2012)).  
             
           Assessment includes evaluation of the motor system, ROM, and sensory systems that affect the person’s ability to maintain equilibrium. Movement strategies to maintain balance require functional ROM and adequate strength. Sensory information from the vestibular, visual and somatosensory systems are key areas associated with maintenance of balance or posture and are integrated at the central level between the two sides of the body and three sensory systems. Central motor planning is essential for proper strategies that are then transmitted to the peripheral motor system for execution. Deficits at the central level, peripheral motor level, or peripheral sensory level will affect balance and equilibrium.  
             
           The dynamic systems model recognizes that balance and dynamic equilibrium is the result of the interaction between the individual, the functional task, and the environment. Emphasis of treatments performed by a qualified physical or occupational therapist in vestibular and balance dysfunction are head exercises for habituation of vertigo, eye-head coordination exercises for improvement of gaze stabilization, and sensorimotor retraining to remediate postural dyscontrol in all functional movement positions. There is good evidence that vestibular rehabilitation incorporating visual motion performed by the patient alone with brief instruction from a health care provider reduces dizziness and improves function ([[Cochrane] Hillier, 2011](#Hillier2011)).

❖ Time to Produce Effect: 6 to 12 weeks.

❖ Frequency: Initially for training with 2–4 follow-up visits to reinforce treatment; individuals are expected to perform self-directed exercises twice daily at home, but they may require supervision for guidance and safety.

❖ Optimum Duration: 2 to 6 months with re-evaluation.

❖ Maximum Duration: Therapy will be more intense and requires more frequent therapy for patients with moderate to severe dysfunction. If reports document treatment progress, but the individual is not at maximum therapeutic gain, then additional therapy may be indicated.

* + - 1. Postural Control: Treatment involves remediation of stability within the constraints following TBI in the musculoskeletal, neuromuscular, sensory/perceptual, and cognitive areas. Biochemical limitations may limit the individual’s ability to move in ways necessary for compensation. Treatment in this area may include physical modalities to increase ROM, joint mobility, and flexibility. Treatment for muscular incoordination may include therapeutic exercise, electrical stimulation, biofeedback, re-education, and other therapies. A vestibular rehabilitation program needs to be individualized considering cognitive impairments and involves:
         1. Increased need for physical assistance because of movement problems.
         2. Increased need for supervision because of cognitive and behavioral problems.
         3. Slower progression of program..
      2. Dizziness: An abnormal sensation of motion ranging from poorly characterized light-headedness, disequilibrium, rocking and elevator sensations to vertigo. It may be due to vestibular hypofunction, reduced head mobility, and poor gaze stabilization. Physical therapy exercise treatment approaches are based on principles of adaptation, substitution, and habituation, and they require the development of specific individual exercises aimed at the person’s specific area of deficit. Because exercise usually increases dizziness temporarily, the progression, speed and intensity of the exercise program should be tailored to the individual in order to avoid increasing the symptoms and hindering compliance. Programs are developed based on integrating sensory input from the somatosensory, visual, and vestibular systems based on the individual’s function.  
           
         If it is found that the dizziness problem is from a visual disturbance, referral to an ophthalmologist, neuro-ophthalmologist, or optometrist (knowledgeable in TBI) may be necessary, although treatment can be performed by most therapists or trained nurses. The dynamic systems model recognizes that balance and dynamic equilibrium is the result of the interaction between the individual, the functional task, and the environment. Emphasis of treatments performed by a qualified physical or occupational therapist in vestibular and balance dysfunction are head exercises for habituation of vertigo, eye-head coordination exercises for improvement of gaze stabilization, and sensorimotor retraining to remediate postural dyscontrol in all functional movement positions. There is good evidence that vestibular rehabilitation incorporating visual motion performed by the patient alone with brief instruction from a health care provider reduces dizziness and improves function [([Cochrane] Hillier, 2011](#Hillier2011)).  
           
         Special equipment for vestibular treatment may include dynamic platform posturography or a foam/dome apparatus for sensory integration and balance, as well as tilt or rocker boards in the clinic. Other virtual reality devices are not suggested for use with this treatment because therapist intervention and supervisions are important for success. No special equipment is needed at home unless identified by the treating professional and documented as medically necessary.  
           
         Individuals with central traumatic vestibular lesions take longer to improve than those with dizziness from other causes. Studies indicate that at six months, only one-third of individuals with unilateral loss from trauma were symptom-free, as compared with other causes. At 18 months, many individuals continued to show symptoms. Of those with central vestibular loss, 60–70% had persisting symptoms at five years, and half were unable to return to work ([Marzo, 2004](#Marzo2004)).

❖ Frequency: One session per week initially, decreasing to once every 3 weeks; individuals are expected to perform self-directed exercises twice daily at home, but they may require supervision for guidance and safety.

❖ Optimum Duration: 6 months with re-evaluation.

❖ Maximum Duration: May require follow-up for up to 2 years.

* + - 1. Benign Positional Vertigo (BPV): The most common cause of vertigo due to a peripheral vestibular disorder. The most common form is caused by canalithiasis, which is the displacement of microscopic calcium bicarbonate crystals from their normal position in the inner ear. A Dix Hallpike and roll test should be used to determine which canals are involved in this disorder. These tests trigger a nystagmus that is less than 60 seconds in duration and has a paroxysmal quality. A burst of dizziness without nystagmus may also indicate mild BPV. CRM should be applied to the affected semicircular canals based on these tests. The commonly used effective CRM are the Epley and Semont maneuvers for the posterior canal; Gufoni and barbecue roll for the horizontal canal; and deep head hanging maneuvers for the anterior canal. CRM has a success rate exceeding 90%; failure to respond or the presence of a nystagmus beyond 60 seconds suggests that the diagnosis of BPV may be incorrect.  
           
         Some individuals may require an exercise-based approach following, or instead of, the CRM. Home exercises are safe and effective in this disorder.

❖ Frequency: 1 to 3 sessions with repeated CRM at each session and follow-up at 1 month.

❖ Optimum Duration: 1 month with re-evaluation.

❖ Maximum Duration: Reoccurrence can occur randomly for many years following trauma. Home exercises are necessary for those with frequent recurrences. Some patients are unable to perform home exercises, so repeated visits for CRM may be required.

* 1. SWALLOWING IMPAIRMENTS (DYSPHAGIA): The incidence of swallowing disorders in the TBI population is high with presenting dysphagia usually characterized by a combination of oral and pharyngeal stage deficits. Co-existing cognitive and behavioral deficits compromise swallowing safety. Physical damage to the oral, pharyngeal, laryngeal, and esophageal structures complicates neurogenic dysphagia. Prolonged ventilation, endotracheal intubation, and the presence of tracheostomy may also have a negative impact on swallow function.  
       
     The initial goal in oral-pharyngeal dysphagia intervention involves lessening the impact of the dysphagia through prevention of medical complications, such as aspiration pneumonia or malnutrition, and the establishment of alternative nutrition if necessary for the maintenance of adequate nutrition. A stimulation program without presentation of food may be provided early in the course of therapy in preparation for later feeding. In subsequent therapy, there is gradual introduction of oral nutrition using an array of treatment techniques designed to target the physiological impairments underlying the dysphagia while the individual continues to receive alternate nutrition. There is an eventual progression towards total oral nutrition without need for supplementation and independence with any safety precautions or therapy techniques.  
       
     Therapeutic strategies may be divided into two categories:
     1. Compensatory Treatment: Techniques do not involve direct treatment of the swallowing disorder and may not affect the physiological function of the swallow, but they may reduce or eliminate the dysphagic symptoms and risk of aspiration by altering the movement of the bolus through the mouth and pharynx. These include, strategies such as postural adjustments of the head, neck and body to alter the dimensions of the pharynx, the flow of the bolus, altering consistency and viscosity of foods, and varying the volume and rate of presentation of the food or drink.
     2. Therapy Techniques: Designed to change the swallowing physiology. These include, but are not limited to, strategies such as, ROM and bolus control tasks to improve neuromuscular control, swallowing maneuvers which target specific aspects of the pharyngeal phase of the swallow, and swallowing maneuvers to facilitate laryngeal closure during the pharyngeal phase of the swallow. Neuromuscular electrical stimulation has also been used in conjunction with swallowing therapy; however, at the time of this guideline, the evaluation studies available do not meet evidence standards. Thus, it is not routinely recommended but may be used ([Carnaby-Mann, 2007](#CarnabyMann2007); [Permsirivanich, 2009](#Permsirivanich2009)).  
          
        Medical consultation may be necessary to assist with clinical improvement in swallowing function. Medical interventions may include, but are not limited to: medications to reduce production of saliva; elimination of medications associated with reduced saliva production; and vocal fold injection (Teflon, absorbable gelatin sponge) for unilateral vocal fold weakness.  
          
        It is generally accepted that the speech-language pathologist in consultation with the physician establishes the dysphagia treatment plan. Self-feeding and the use of adaptive equipment for this may be coordinated by the occupational therapist. Additional disciplines participate in a team approach to the treatment of dysphagia. These may include, but are not limited to, professionals such as physicians (including otolaryngologist, gastroenterologists, or others), registered dietitians, nurses, and physical therapists.  
          
        Ongoing reassessment and modification of therapy techniques and treatment goals to optimize effectiveness are integral components of therapy. Initial treatment plan and goals should be updated whenever needed, but at least with each re-evaluation. During the earlier phases of recovery, change may occur rapidly, and formal re-evaluation (including instrumental evaluation) may be completed frequently.

❖ Frequency: (1) Acute Care – 1 to 2 times daily; (2) Post-Acute – Once per day; (3) Subacute outpatient/community settings – 1 to 5 sessions weekly.

❖ Optimum Duration: 6 to 8 weeks with 4-week re-evaluations.

❖ Maximum Duration: Beyond 8 weeks, documentation of progress is required.

Therapy is discontinued when goals are met or when it is apparent that the individual is no longer making progress. In the latter case, re-evaluation and further therapy may be appropriate if/when the individual shows new or renewed potential.

* 1. COMMUNICATION: Basic to all daily activity and is necessary for the maintenance of positive quality of life and psychological well-being. Even the most subtle communication impairment may seriously interfere with an individual’s ability to achieve occupational, personal, and interpersonal goals. Speech-language therapy and occupational therapy are well-accepted and widely used. Music therapy may be appropriate for some patients. There is insufficient evidence to recommend specific types of therapy ([[Cochrane] Kelly, 2010](#Kelly2010)).  
       
     Communication (speech-language) impairments are a common result of TBI and may be classified into the following groups: (1) motor speech disorders, which may take the form of dysarthria and/or apraxia of speech; (2) voice disorders; (3) language disorders; (4) communicative/cognitive disorders; and (5) fluency disorders. These may occur together in varying combinations in TBI.
     1. Motor Speech Disorders:
        1. Dysarthria: A reduction in speech intelligibility due to weakness and/or incoordination of the speech musculature secondary to central or peripheral nervous system injury that involves the processes of articulation, resonance, phonation, and respiration. It accounts for approximately one-third of communication impairments following TBI. Any type or level of severity of dysarthria may occur subsequent to TBI, from very minimal slurring or hypernasality in connected speech to the absence of intelligible speech (anarthria).
        2. Apraxia of Speech: A motor impairment that disrupts central motor planning and interferes with voluntary positioning and sequencing of the movements of the speech musculature in the absence of paralysis or muscular weakness. Symptoms may range from very mild articulation errors to inability to produce any functional speech volitionally.
     2. Voice Disorders: Any compromise to airway structures (nasal-pharyngeal cavities, larynx, trachea, lungs, the muscle of respiration) or their function may cause voice disorders. These involve impairment in respiration, phonation, and/or resonance. A present voice symptom may have one or several causes and may range in severity from mild vocal fatigue to the absence of voicing (aphonia).
     3. Language Disorders: Language impairment is often present in the early stages of TBI. In some cases, specific language impairment (aphasia) persists as a result of a focal lesion. Language impairments include those of receptive and expressive language in both spoken and written form, as well as gestural expression and reception. These may be impaired to varying degrees, ranging from very mild difficulty with word finding (anomia) to global impairment involving severe impairment in all language areas. In TBI, language deficits tend to occur against a backdrop of cognitive impairment.
     4. Cognitive-Communicative Disorders: Cognition and language are intrinsically and reciprocally related. An impairment of language may disrupt one or more cognitive processes, and an impairment of one or more cognitive processes may disrupt language. The ability to consciously, efficiently access and manipulate the semantic system requires the complex interplay of language, cognitive, and executive processes. Impairments in linguistic and metalinguistic skill as well as impairments in non-linguistic cognitive functions such as perception, attention, discrimination, organization, reasoning, memory, and self-regulation interfere with communication of basic needs and with communication in wider social contexts.  
          
        Social communication skills, also known as pragmatic language skills, encompass the meaning and use of language in social situations. They include the interpretation of contextual clues, non-verbal communications, and other interpersonal skills. Social communication skills training is also appropriate for these cases. There is some evidence that group instruction, 90 minutes weekly over 12 weeks, by a skilled leader, results in improved skills ([Dahlberg, 2007](#Dahlberg2007)).  
          
        Certified speech-language pathologists and occupational therapists are qualified to identify, evaluate, and determine the appropriateness of treatment for individuals with speech, language, and cognitive-communicative disorders. When treatment is indicated, speech-language pathologists develop, supervise, and/or implement a plan of treatment. Treatment of cognitive-communicative disorders has come to be included under labels such as cognitive retraining, cognitive rehabilitation, cognitive therapy, cognitive remediation, and neurotraining. Speech/language pathologists should be integral members of interdisciplinary teams engaged in the identification, diagnosis, and treatment of individuals with cognitive-communicative disorders. According to the American Speech-Language and Hearing Association (ASHA), certified Speech-Language Pathologists are qualified to identify, diagnose, and determine the appropriateness of treatment for individuals with speech, language, and cognitive-communicative disorders.  
          
        Interaction and consultation between the speech-language pathologist, medical specialists, and other members of the interdisciplinary treatment team is an essential part of the treatment of TBI-related communication disorders. There is extensive overlap in professional domains, making it important that team members from different clinical fields collaborate in their approach to assessment and intervention.  
          
        Speech-language evaluation is recommended when there is evidence to support the presence of communicative symptoms. The evaluation includes:

● A thorough review of relevant medical and social history.

● A comprehensive assessment of communication skills including standard and non-standard measures.

● Evidence of consultation with family members and/or support system.

● Diagnosis of communication disorder.

● Indication of the severity of the disorder, the individual’s candidacy for intervention, and the prognosis for improvement.

● An intervention plan that is coordinated and integrated with other services being received.

● Realistic functional goals and recommendations that reflect consideration of the pre-morbid level of function and input from the individual and family and/or support system to assure social and ecological validity.

● Estimate need of therapy frequency and duration with attention to the anticipated ultimate outcome.

● A plan for providing education and training to the individual’s family members and/or support system.

Constellations of communication-related deficits in TBI are extremely varied, depending on the characteristics of the individual who is injured, the nature, location, and severity of injury, and the post-trauma support systems. Coinciding with the great diversity within this group, there is a similar level of diversity in treatment approaches. These have been divided into various categories, such as “conventional” and “functional,” or those who seek to improve communicative functioning through a restorative, compensatory, or behavioral approach. Experienced therapists commonly use a combination of such approaches, depending on the needs of each individual.  
  
For certain individuals, prosthetic or alternative augmentative communication (AAC) devices may be necessary to optimize communicative success. These include, but are not limited to: (1) palatial lift prostheses for velopharyngeal dysfunction resulting in severe impairment in speech intelligibility; and (2) augmentative or alternative communication devices which may be indicated when speech is inadequate for functional communication. AAC may involve the use of simple gesture systems, alphabet boards, pictures, word books, or sophisticated use of computer technology (speech generation devices). AAC strategies may enhance communicative participation by replacing, supplementing or scaffolding residual natural speech and providing a means of repairing disrupted communication.   
  
Melodic Intonation Therapy is a structured therapy that trains verbal reproduction with melodically intoned phrases while tapping the patient’s hand. A number of case series have supported its use in cases with non-fluent aphasia and/or auditory communication deficits when there is minimal or no damage to the right hemisphere. The therapy can take place as late as six months or longer after injury ([van der Muelen, 2012](#VanderMeulen2012)). It is often done 3–5 hours per week for six weeks. Non-speech oral motor exercises on speech are another widely accepted therapy ([McCauley, 2009](#McCauley2009)).  
The process of deciding on these techniques or devices and the training in their use is integrated into the individual’s ongoing evaluation and therapy plan.  
  
For moderate/severe TBI, the following are recommended guidance:

* Frequency: (1) Acute setting – once to twice daily sessions; (2) Sub-acute or outpatient and home/community setting – 1 to 5 sessions per week.

❖ Optimum Duration: 12 weeks with re-evaluations at 4-week intervals. A minimum of 24 sessions for moderate/severe TBI.

❖ Maximum Duration: Intervention beyond 8 weeks requires documentation of continued functional progress towards established goals. Post-acute therapy could extend for 6 to 12 months, or more, if the individual with TBI has significant speech impairment and is making gradual documented improvement.

For MTBI, treatment may be focused on attention, memory, and speed of processing with use of compensatory aids. Treatment usually parallels the guidelines set forth under cognitive therapy (refer to Section [G.3. Cognition](#TBIG3Cognition)).  
  
Ongoing reassessment and modification of therapy approaches is a part of skilled therapy and is especially necessary with the dynamic nature of communication impairment that occurs with TBI. Goal setting is an evolving and dynamic process that is pivotal to each therapy session. Because of wide variability in type, nature, and severity of communication impairments common to TBI, and the lack of unanimity in the literature with respect to the nature and temporal course of post-TBI communicative dysfunction, there should be flexibility in frequency, intensity and duration of treatment. Many cases require follow-up visits at various points to assist individuals with changes in their life, such as increasing job demands.

1. NONOPERATIVE THERAPEUTIC PROCEDURES – RETURN TO WORK, DRIVING, & OTHER
   1. DRIVING: Independent driving is considered a complex activity of daily living. An individual’s potential for safe driving is influenced by an intricate interaction of physical, cognitive, visual, and behavioral impairments.   
        
      Self report of feeling confident with driving ability may not be reliable. Some studies of demential patients have demonstrated this ([Iverson, 2010](#Iverson2010)). An individual’s ability to drive is typically evaluated and treated under physician orders by a certified driver rehabilitation specialist. Physicians, neuropsychologists, or rehabilitation therapists can perform an initial screening to determine driving ability by assessing visual acuity, visual fields, memory, visual perception, visual processing, visual spatial skills, selective and divided attention, executive skills, motor and sensory function coordination, pain, and fatigue ([Wang, 2010](#Wang2010)); [Defense Centers for Excellence for Psychological Health and Traumatic Brain Injury, 2009](#DefCtrforExPsych2009)). The AMA suggests confrontational field testing, Snellen E acuity testing, Trail Making Test part B, clock drawing test, and rapid pace test (walk 10 feet back and forth in nine seconds) as an initial screening, along with ROM and motor strength testing ([Wang, 2010](#Wang2010)).  
        
      A thorough history should be taken which includes: (1) a review of all medication that might affect cognition or coordination; (2) screening for sleep apnea (BMI >35, neck size > 15.5 in, for female or 17 in. for males, daytime sleepiness, Eppworth Sleepiness Scale score of 10 or greater, two or more hypertension medications); (3) history of accidents and/or tickets; and (4) consultation with family and/or support system members or others regarding driving ability. Reluctance of others to ride with the patient may be an indication of problems. Patients may also fill out surveys that have some predictive abilities ([American Automobile Association [AAA], 2011](#AAA2011); [Eby, 2010](#Eby2010)). Unfortunately, at the time of this guideline, there is no evidence for the use of one system of assessment over another to predict driving skills ([Marino, 2012](#Marino2012); [[Cochrane] Martin, 2009](#Martin2009)).  
        
      In addition, the treatment and evaluation process may require the services of a:

● Commercial driver trainer for driving practice.

● Ophthalmologist or optometrist for visual evaluation.

● Commercial vendor and rehab engineer for adaptive equipment.

● Neuropsychologist for cognitive evaluation.

● Speech-language pathologist for communication evaluation and compensatory strategies.

● Occupational or physical therapist with expertise in acquired brain injury.

Public and personal safety and compliance with state department of motor vehicles procedures ultimately determine individual driving privileges. Evaluation and treatment typically occur during the post-acute phase of rehabilitation. Usually, successful driving results are obtained within the first two years post-injury, but this is not always the case.

❖ Frequency and Time to Produce Effect: Evaluation time of a minimum of 1 to 2 sessions to evaluate physical, perceptual, cognitive, and behavioral skills and for collaboration with other interdisciplinary team members.

❖ Optimum Duration: Between 2 to 6 sessions of behind-the-wheel driving evaluation and training on the road.

If the individual fails the evaluation, he or she may be required to participate in additional driving practice and repeat the behind-the-wheel test, or to wait three months or longer to repeat the evaluation. The evaluation may be repeated at 3- to 12-month intervals as determined by the evaluator and physician. Several repeat assessments may be necessary to determine safe driving readiness.

Recommendations and physician prescriptions for necessary adaptive equipment and vehicle modification for safe driving or for dependent passenger transport in vehicles may be necessary. Van lifts and other adaptive equipment and vehicle modifications may be required for dependent individuals in order to provide access to community services and activities. Therapeutic assistance is necessary to help the individual and physician comply with state department of motor vehicles standards for practices and procedures for driver’s licensure.  
  
Significant and multiple cognitive impairments, as well as motor and visual impairment, may decrease, delay, or prevent an individual from achieving functional driving independence. Important cognitive factors include ability to make complex judgments, organize information, anticipate and/or react quickly, maintain self-control, and other factors. Individuals with moderate/severe TBI may or may not be able to successfully compensate for these impairments.

* 1. RETURN TO WORK: In addition to the treatment strategies described below, practitioners should be familiar with how various state and federal statutes and regulations may impact return-to-work planning. These may include, but are not limited to, Family and Medical Leave Act (FMLA), Americans with Disabilities Act (ADA), Occupational Health and Safety Administration (OSHA), Federal Motor Carrier Safety Administration (FMCSA), and the Department of Transportation (DOT). One study found a relationship between perceived self-efficacy in cognitive areas and life satisfaction. The same study found a relationship with employed or voluntary work and satisfaction ([Cicerone, 2007](#Cicerone2007)). In places where the employer is unable to accommodate, other options include sheltered work shops.
     1. Return to Work – MTBI: During the first five days post-injury, symptoms can be severe and significantly disrupt normal daily function. Initial considerations should include lightening task load and allowing extra time to complete normal tasks. Thus, shortening the work day or adding breaks, along with decreased responsibility for the first several weeks are generally suggested. Driving, heavy lifting, working with dangerous machinery, use of ladders, and heights may be restricted because of possible safety risk ([Centers for Disease Control and Prevention. U.S. Department of Health and Human Services, n.d](#CDCnd).). For individuals with MTBI who have persistent deficits, or who have difficulty once back at work, a return to work program requires a carefully designed and managed plan involving the person with TBI, his/her employer, and the treatment team. Physicians should consider evaluation and treatment for co-morbid conditions such as chronic pain, stress level, pre-existing personality disorders, depression, anxiety, and/or substance abuse. Communication among all involved parties and the avoidance of fragmentation among treatment professionals is critical to successful outcome. Case management may be indicated to facilitate communication. Following return to work, maintenance support services are appropriate to best insure the durability of the outcome.  
          
        Following MTBI, many individuals are able to resume normal work duties with secondary prevention precautions and education requiring little or no additional therapeutic intervention. A smaller percentage of individuals with MTBI at the upper end of the definition, such as age greater than 40, prior TBI, loss of consciousness close to 30 minutes, or mental status changes lasting up to 24 hours, may require more assistance in return to work and accommodations. Individuals with MTBI should be instructed to temporarily reduce the amount, type, and/or intensity of their work duties or temporarily remain out of work entirely for the first three days and gradually increase complex cognitive and physical duties based on symptomology.  
          
        If workers with MTBI have any loss of consciousness or prolonged disorientation, providers should consider restricting higher risk job duties, such as working at heights, working with power tools and operating heavy machinery, until they have been free from the symptoms, including dizziness, and imbalance for two weeks. Second impact syndrome (refer to Section [C.10.b Secondary Prevention](#TBIC10bSecondaryPrevention)) has been seen in younger age groups who suffer severe life threatening effects after a second brain injury within a short time after the first TBI. Physicians should take this into account when writing work restrictions.  
          
        Return to full duty depends on the rate of decrease of symptoms. Generally, if symptoms recur during increasing job duties or exertion, duties should be decreased slightly ([Defense and Veterans Brain Injury Center, 2008](#DefandVetsTBIctr2008)). For cases with symptoms lasting longer than 15 minutes at the time of the injury, unconsciousness lasting minutes or prolonged amnesia, very gradual return to activity over weeks may be necessary ([Frey, 2009](#Frey2009)).  
          
        Post-concussion symptoms in workers with MTBI may include cognitive deficits in memory, attention, and executive function. Physicians should be aware of this, even if the worker has no complaints/symptoms. Memory, attention, and executive function should be tested by asking specific questions regarding recent events and having the individual perform specified tasks. Physicians should educate the individual with TBI and their supervisor to be aware of possible memory and attention deficits and to accommodate accordingly. Time to return to baseline function will differ according to the individual’s pre-accident condition, age, and medication, as well as other pre-injury, injury, and post-injury factors. The individual should be competent in most basic ADLs before return to work is considered.  
          
        Physicians should attempt to be clear and specific in documenting vocational restrictions and have a plan for re-entry to work and communication with the employer (e.g., supervisor, safety officer, employee health nurse). Having a significant physical disability, psychosocial impairment, cognitive impairment, or a history of alcohol and other substance abuse are factors that impede return to work. Other factors impeding return to work include difficulties regarding transportation, coordination, and vision. An interdisciplinary team approach may be recommended, which may include a neuropsychological assessment, vocational evaluation, job site analysis, early contact with employer, assessment of vocational feasibility, supervisor education, transferable skills analysis, skillful increased titration of job duties and demands, job coaching, physical therapy, occupational therapy, speech-language therapy, and psychological services.  
          
        For individuals with MTBI who have persistent deficits, or who have difficulty once back at work, a return-to-work program should occur, which requires a carefully designed and managed plan involving the person with TBI, his/her employer, and the treatment team. Physicians should consider evaluation and treatment for co-morbidities, such as chronic pain, stress level, pre-existing personality disorders, depression, anxiety, or substance abuse. Communication among all involved parties and the avoidance of fragmentation among treatment professionals is critical to successful outcome. Case management may be indicated to facilitate communication. Following return to work, maintenance support services are appropriate to best insure the durability of the outcome.
     2. Return to Work – Moderate/Severe TBI: Following moderate/severe TBI, some individuals are unable to return to work. Successful return to work among individuals with moderate/severe injury may require an interdisciplinary approach including neuropsychological assessment, speech-language assessment, functional capacity evaluation, job site analysis, early contact with employer, assessment of vocational feasibility, transferable skills analysis, supervisor education, job coaching, skillful increased titration of job duties and demands, mental health, family counseling, and follow-up services.
     3. The Following Should be Considered when Attempting to Return an Injured Worker with Moderate/Severe TBI to Work:
        1. Job History Interview: The authorized treating physician should perform a job history interview at the time of the initial evaluation and before any plan of treatment is established. Documentation should include the workers’ job demands, stressors, duties of current job, and duties of job at the time of the initial injury. In addition, cognitive and social issues should be identified, and treatment of these issues should be incorporated into the plan of care.
        2. Coordination of Care: Management of the case is a significant part of return to work and may be the responsibility of the authorized treating physician, occupational health nurse, risk manager, or others. Case management is a method of communication between the primary provider, referral providers, insurer, employer, and employee. Because case management may be coordinated by a variety of professionals, the case manager should be identified in the medical record.
        3. Communication: Essential between the patient, authorized treating physician, employer, and insurer. Employers should be contacted to verify employment status, job duties and demands, and policies regarding injured workers. In addition, availability and duration of temporary and permanent restrictions, as well as other placement options, should be discussed and documented. All communications in the absence of the patient are required to be documented and made available to the patient.
        4. Establishment of Return-To-Work Status: Return to work for persons with TBI should be thought of as therapeutic, assuming that work is not likely to aggravate the basic problem or increase discomfort. In most cases of TBI, the worker may not be currently working or even employed. The goal of return to work would be to implement a plan of care to return the worker to any level of employment with the current employer or to return them to any type of new employment.
        5. Establishment of Activity Level Restrictions: A formal job description for the injured/ill employee who is employed is necessary to identify physical and cognitive demands at work and assist in the creation of modified duty. A job site evaluation may be utilized to identify tasks such as pushing, pulling, lifting, reaching above shoulder level, grasping, pinching, sitting, standing, posture, balance, ambulatory distance and terrain, and if applicable, environment for temperature, air flow, noise, tolerance for scanning, scrolling and other computer use, cognitive activities, and the number of hours that may be worked per day. Due to the lack of predictability regarding exacerbation of symptoms affecting function, an extended and occupationally focused functional capacity evaluation may be necessary to determine the patient’s tolerance for job type tasks over a continuing period of time. Work capacity should usually be evaluated with an FCE or through assessment by occupational or physical therapists with experience in acquired brain injury treatment. Work restrictions assigned by the authorized treating physician may be temporary or permanent. The case manager should continue to seek out modified work until restrictions become less cumbersome or as the worker’s condition improves or deteriorates.
        6. Rehabilitation and Return to Work: As part of rehabilitation, every attempt should be made to simulate work activities so that the authorized treating physician may promote adequate job performance. The use of ergonomic or adaptive equipment, therapeutic breaks, assistive devices, and interventional modalities at work may be necessary to maintain employment.
        7. Vocational Assistance: Formal vocational rehabilitation is a generally accepted intervention and can assist disabled persons to return to viable employment. Assisting patients to identify vocational goals will facilitate medical recovery and aid in the maintenance of MMI by (1) increasing motivation towards treatment and (2) alleviating the patient’s emotional distress. TBI patients will benefit most if vocational assistance is provided during the interdisciplinary rehabilitation phase of treatment. To assess the patient’s vocational capacity, a vocational assessment utilizing the information from occupational and physical therapy assessments may be utilized to identify rehabilitation program goals, as well as optimize both patient motivation and utilization of rehabilitation resources. This may be extremely helpful in decreasing the patient’s fear regarding an inability to earn a living which can add to their anxiety and depression.
  2. VOCATIONAL REHABILITATION: A generally accepted intervention, but the Colorado Workers’ Compensation statute limits its use. In one study, an acquired brain injury vocational rehabilitation program was successful at returning 41% of clients to competitive employment. The majority of the cases were two years or more from date of injury and had injuries classified as severe (post-traumatic amnesia duration of one or more days). These cases were also without significant behavior or problems and able to function independently for ADLs. The program included cognitive training for those who had not previously received it and job trials with job coach support ([Murphy, 2006](#Murphy2006)). Initiation of vocational rehabilitation requires adequate evaluation of individuals with TBI for quantification of highest functional level, motivation and achievement of MMI. Vocational rehabilitation should involve a comprehensive job analysis and a carefully planned return to work strategy. In some instances, retraining may need to occur to access new job markets (refer to Section K.2 Return to Work).
  3. COMPLEMENTARY AND ALTERNATIVE MEDICINE (CAM): (as defined by the National Center for Complementary and Alternative Medicine [NCCAM]) A group of diverse medical and health care systems, practices, and products that are not presently considered to be part of conventional medicine. CAM includes a wide range of interventions, some of which have not been supported by empirical data. These alternative treatments include, but are not limited to: art therapy, craniosacral trauma release, EEG neuro feedback, dance therapy, hippotherapy, hypnosis, and horticulture therapy. CAM uses methods of treatment based on a broad range of knowledge with roots in both eastern and western medicine. Many providers may integrate more than one procedure. Some of these interventions, including the exercise-based procedures, are currently integrated into ongoing rehabilitation programs. In general, most approaches place major focus on the important relationship between physical and emotional well-being. Alternative therapies should not be employed as the primary treatment modality, but they may be considered for individual cases when other treatments have failed to produce functional gains, when there is a valid clinical rationale for their use, and when treatment goals are directed to documentable, functional improvement. CAM treatment requires prior authorization from the payer and agreement on fees in accordance with criteria in the Division of Workers’ Compensation Rules 16 and 18.

❖ Time to Produce Effect: 3 to 6 treatments.

❖ Optimum Duration: 4 to 6 weeks.

❖ Maximum Duration: Not well-established for CAM and should be based on specific CAM treatment, physician’s clinical judgment, and demonstration of positive symptomatic and functional gains.

Acupuncture, biofeedback, and cervical spinal manipulations are widely accepted and may be used for headaches or other painful conditions (refer to Sections F.1. Acupuncture, F.2. Biofeedback and F.14.c Manipulation in the Chronic Pain Guidelines).

* 1. OTHER TREATMENTS:
     1. Hyperbaric Oxygen: Studies in this area demonstrated a possible decrease in morbidity for severely injured patients but no clear overall improvement in outcome. It is also associated with possible long term pulmonary damage. It is considered investigational at this time and not recommended [([Cochrane] Bennett, 2004](#Bennett2004)).
     2. Deep Thalamic Stimulation: This technique has been used in some cases of stroke with motor and cognition problems. There are no studies reported on patients with TBI. It is considered investigational at this time and generally not recommended. It may be used for patients with severe spasticity or motor problems who have failed other treatments.
     3. Transcranial Magnetic Stimulation: This is a noninvasive treatment and exploratory diagnostic tool that is FDA approved for use in major depression resistant to other therapy. Some patients have experienced seizures as a side effect. There is no evidence for its use in TBI, and it is not recommended for TBI or for comatose or vegetative patients. It is considered experimental for these conditions.

1. OPERATIVE THERAPEUTIC PROCEDURES

It is not the intent of medical treatment guidelines to provide an exhaustive list of surgical procedures associated with TBI. Instead, an overview of the general categories is presented to illustrate the wide range of procedures that are widely accepted for treatment of individuals with TBI. Combinations and variations on procedures should be tailored to specific cases; hence, a variety of procedures based on the clinical judgment of the treating physician is to be expected. Common procedures include, but are not limited to:

* 1. EXTRACRANIAL SOFT TISSUE:
     1. Debridement and closure.
     2. Plastic or reconstructive.
  2. MAXILLOFACIAL:
     1. Repair and stabilization of fracture.
     2. Facial nerve decompression.
     3. Repair and/or reconstruction.
  3. SKULL:
     1. Debridement, elevation, and/or repair of fracture or defect including cranioplasty.
  4. BRAIN:
     1. Debride penetrating injury, gunshot wound, or foreign body.
     2. Decompression and evacuation.
        1. Hematoma: epidural, subdural, intraparenchymal.
        2. Contusion.
        3. Infections: abscess or empyema.
     3. Decompressive Craniectomy:
        1. Description: Removal of a large portion of the skull and dural opening to managecerebral edema causing increased intracranial pressure (ICP). The bone flap is then stored. Cranioplasty is later required to correct the skull defect deficit. When the autologous graft cannot be replaced, an alternative method allograft may be used ([Cabraja, 2009](#Cabraja2009)).  
             
           The most recent randomized controlled trial comparing medical management for diffuse TBI with a bifrontotemporoparietal craniectomy showed similar rates of death and worse outcomes ([Cooper, 2011b](#Cooper2011b)). However the surgery was done early, and a number of the patients randomized to craniectomy had bilateral fixed dilated pupils. The study was also criticized for use of a 20mmHg ICP trigger and early surgery. ([Chi, 2011](#Chi2011); [Hutchinson, 2011](#Hutchinson2011); [Marion, 2011](#Marion2011)). A Cochrane evidence-based review in 2006 reached no conclusions about the efficacy of the procedure for diffuse TBI [([Cochrane] Sahuquillo, 2006).](#Sahuquillo2006) In a systematic review of the literature, the average 6 month mortality was 28%, and the mean quality of life outcome was independently functioning but debilitated ([Kakar, 2009](#Kakar2009)).
        2. Indications:   
             
           May be performed in conjunction with evacuation of mass lesions or may be performed for intracranial hypertension.
        3. Complications:
           1. Craniectomy – Subdural hygroma or hemorrhage, contra-lateral contusions, outcome of a vegetative state, cerebral herniation ([Stiver, 2009](#Stiver2009)).
           2. Cranioplasty – Infection, wound break down, bone resorption, increased rate of complication for patients with bilateral craniectomies ([Gooch, 2009](#Gooch2009)).
  5. CEREBRAL SPINAL FLUID (CSF):
     1. CSF Leak or Fistula: Lumbar spinal drain or serial lumbar puncture may be used as option to promote spontaneous resolution of CSF leak, or as adjunct to surgical repair. Repair of the leak or fistula may require surgical exploration of the anterior cranial fossa, the temporal bone, and/or sinuses to identify the CSF leak and seal it.
     2. Ventricular Shunting: The treatment of hydrocephalus may require ventricular shunting. Even though ventricular shunting is frequently regarded as a routine procedure, clinicians should recognize the possibilities of mechanical, biological or technical complications. The complications of ventricular shunting for hydrocephalus may include, but are not limited to, shunt failure, hemorrhage, delayed wound closure, infection, and seizures. Favorable outcome from CSF ventricular shunting in appropriately selected individuals will depend on the timing of intervention, the type of shunt valve used, seizure prophylaxis, and methods of long-term follow up management. A recent advancement in this type of intervention includes the use of programmable shunt valves. This may require periodic reprogramming of the shunt valve and is a generally accepted procedure.
     3. Ventriculostomy:
        1. Control of ICP.
        2. Acute hydrocephalus.
           1. Obstructive.
           2. Communicating (usually with sub-arachnoid hemorrhage).
  6. OPHTHALMOLOGIC:
     1. Direct trauma to globe and/or orbital contents.
     2. Repair orbital fractures, decompression of orbital contents.
     3. Optic nerve decompression: immediate surgery may be indicated if the trauma results in entrapment or compression of the nerve, or if a hematoma is present in the optic nerve sheath.
     4. Strabismus: surgery may be required to eliminate or decrease diplopia. Individuals may require several revision operations to achieve maximal results.
     5. Vitrectomy may be indicated in cases of vitreous hemorrhage.
     6. Surgery may be indicated in cases of eye-lid abnormalities, lacrimal disorders, and other traumas to the external ocular structures.
  7. OTOLOGIC:
     1. Direct Trauma Or Barotrauma:
        1. Ossicular Discontinuity: The mechanism of head trauma causing TBI may result in dislocation of the hearing bones, creating a conductive hearing loss. This would require an exploratory tympanostomy with ossicular replacement to correct.
        2. Tympanic Membrane Perforation: This would cause a conductive hearing loss. Tympanoplasty is indicated for correction.
     2. Tympanostomy: Tube placement alters pressure relationships in the middle and inner ear and can reduce dizziness in some patients with progressive vestibulopathy. It can be used to allow access to the middle ear for dizziness treatment devices and gentamicin perfusion. Individuals must be able to tolerate tympanostomy tubes and practice water precautions and aural hygiene to maintain tube patency.
     3. Middle Ear Exploration:
        1. Perilymphatic Fistula Repair: This presents as a sensorineural hearing loss and dizziness that usually worsens with exertion, straining or altitude changes. Exploratory tympanotomy with patching or round and oval window niches is indicated in these individuals. The operation itself is as much a diagnostic tool as a therapeutic one. The success rate for treating dizziness due to fistula is 80% ([Flint, 2010](#Flint2010)).
        2. Endolymphatic Sac Surgery: This is a non-destructive procedure performed in the operating room under general anesthesia. The surgeon removes the mastoid bone and uncovers the endolymphatic sac. A drain may or may not be placed in the sac at the time of surgery. This operation has a 65% success rate at controlling dizzy spells in patients with Ménière’s disease/endolymphatic hydrops ([Flint, 2010](#Flint2010)).
        3. Labyrinthectomy: This is a destructive procedure performed in the operating room under general anesthesia. The surgeon removes the semicircular canals using the operating drill. This procedure not only obliterates balance function on the operated side, but it also renders the individual deaf in that ear. Because of its destructive nature, it is not indicated in bilateral disease. This procedure has been largely supplanted by gentamicin perfusion for first-line ablation. It can be utilized when other ablative procedures fail to control symptoms. Use should be reserved for cases with documented progressive hearing loss and/or progressive vestibular damage.
     4. Vestibular Nerve Section: This is a destructive procedure performed in the operating room under general anesthesia. It is usually performed by a team including a neurootologist and a neurosurgeon. There are several approaches, but the final step is that of sectioning the vestibular nerve as it exits the brainstem. Being destructive in nature, it is not indicated in bilateral disease. This procedure has been largely supplanted by gentamicin perfusion for first-line ablation. It can be utilized when other ablative procedures fail to control symptoms. Use should be reserved for cases with documented progressive vestibular damage.
  8. DECOMPRESSION OF FACIAL NERVE: If there is immediate onset of total facial paralysis, or if the electroneuronography (EnoG) shows greater than 90% degeneration of the facial nerve, then exploration of the path of the facial nerve is indicated. This usually involves a middle fossa craniotomy and mastoidectomy in order to completely decompress the facial nerve.
  9. OTHER CRANIAL NERVE REPAIR OR DECOMPRESSION: May be required for functionally disabling conditions such as diplopia.
  10. VASCULAR INJURY:
      1. Endovascular procedures (i.e., stent, embolism).
      2. Direct repair.
      3. Occlusion, trapping, aneurysm repair.
  11. PERIPHERAL NERVE INJURY:
      1. May include decompression and repair and/or fracture management.
  12. ORTHOPEDIC:
      1. Fracture management.
      2. Adjunctive tenotomies and myotomies.
         1. Common upper extremity procedures may require pre-surgical evaluation inclusive of occupational therapy, ROM, function, diagnostic nerve blocks, and dynamic EMG. Definitive procedures include, but are not limited to:
            1. Shoulder muscle release.
            2. Functional elbow release: brachial radialis myotomy, biceps and brachialis lengthening.
            3. Fractional lengthening of wrist and/or finger flexors.
            4. Flexor digitorum superficialis (FDS) to flexor digitorum profundus (FDP) transfer.
            5. Intrinsic muscle contracture release.
            6. Surgical release of thenar muscles for thumb in palm deformity.
            7. Individualized and customized procedures for spastic upper extremity deformities with adjunctive selective musculotendinous transfers, neurotomy and neurectomies.
         2. Common lower extremity procedures include, but are not limited to:
            1. Fractional muscle lengthening of knee flexors/hamstrings.
            2. Hip flexor releases/myotomies.
            3. Percutaneous vs. open release of the hip adductors.
            4. Percutaneous tendon Achilles lengthening.
            5. Ankle/foot motor balancing surgery adjunctive to tendon-Achilles lengthening (TAL procedure) includes: (1) toe flexor release, (2) split anterior tibial tendon transfer (SPLATT procedure), (3) inter-phalangeal joint fusions, and (4) ankle fusions.
            6. Individualized and customized procedures for spastic lower extremity deformities with adjunctive selective musculotendinous transfers, neurotomy and neurectomies.
         3. Resection heterotopic ossification.
  13. SPASTICITY:
      1. Spinal cord procedures, including percutaneous and open selective dorsal rhizotomy (SDR).
      2. Intrathecal Baclofen (ITB) pump: The pump is surgically implanted in the abdomen (refer to Section [I.6.g. Intrathecal Baclofen Drug](2012%20TBI%20Guideline%20Draft-Post%20Advisory%20Panel.doc)).
      3. Other “tone management” procedures.

1. MAINTENANCE MANAGEMENT
   1. GENERAL PRINCIPLES: Most individuals following MTBI make a good neurological and functional recovery with minimal or no intervention, although the possibility of subtle residual impairments or functional limitations exists. Some individuals with MTBI experience impairments, functional limitations, and disabilities. Individuals with MTBI who have co-morbid conditions and/or have suffered a longer period of confusion or loss of consciousness are more likely to have a poorer outcome and require longer or maintenance care.  
        
      Individuals with moderate/severe TBI may experience lifetime impairment, functional limitations, and disabilities and are at risk the remainder of their lives for long term medical, psychiatric, physical, and cognitive complications. Subsequent brain injuries, the onset of seizures, endocrine or other medical conditions, maladaptive social skills, aggressive behaviors, substance abuse, and psychiatric disorders are common examples of some negative long-term consequences of TBI. Injured workers are entitled to lifetime medical benefits which are reasonable, necessary, and related to maintaining them at MMI. Therefore, individuals with moderate/severe TBI generally require long-term support to prevent secondary disability and to maintain an optimal level of medical and psychological health and functional independence achieved through rehabilitation. Health professionals with experience in life care plans are frequently involved in making assessments for long-term care. Providers and carriers should adopt a long-term case management model for these individuals. Common lifetime supports that are reasonable and necessary include, but are not limited to, physician oversight, nursing services, various periodic rehabilitation therapies, life skills training, supported living programs, attendant care, supported employment, productive activity recreation, transportation, medication, psychological services, and individual/family/support system education. Supported employment may assist in return to work outside a sheltered work setting. The specific type and amount of support necessary will vary in each individual case and may change over time. Practitioners are encouraged to analyze risk factors and to establish viable long-term maintenance plans. Long-term maintenance programs should be managed by an experienced certified case manager who may intervene quickly when necessary. Case management should not be discontinued when a person completes acute rehabilitation, but it should continue at a frequency necessary for successful long-term management.  
        
      Medical and rehabilitation providers are encouraged to educate individuals and their family and/or support systems regarding anticipated ongoing medical and rehabilitation needs. Because the long-term medical needs of individuals with moderate/severe TBI are uncertain, each individual, his/her family and/or support system and providers should plan for unforeseen medical, psychiatric, social, physical, and cognitive complications as individuals with TBI age. Failure to address long-term management as part of the overall treatment program may lead to higher costs and greater dependence on the health care system. Management of moderate/severe TBI continues after the individual has met the definition of MMI. MMI is reached when an individual’s condition has plateaued and the authorized treating physician believes no further medical intervention is likely to result in improved function. For moderate to severe patients, this is not likely to occur for at least two years. When the individual has reached MMI, a physician must describe in detail the plan for maintenance treatment, including the level and type of care and support services. (refer to Section [C.6. Course of Recovery](#TBIC6CourseOfRecovery)).  
        
      Maintenance care of individuals with moderate/severe TBI requires a close working relationship among the insurance carrier, the clinical providers, the family and/or support system, and the individual with TBI. Clinical providers have an obligation to design a cost-effective, medically appropriate program that is predictable and allows the carrier to set aside appropriate reserves. Insurers and adjusters have an obligation to assure that medically appropriate, cost effective programs are authorized in a timely manner. A designated primary physician for maintenance team management is recommended.  
        
      When developing a maintenance plan of care, the individual, his/her physician, and the insurer should attempt to meet the following goals:

● Maximum independence will be achieved through the use of home and community-based programs and services.

● Individuals with TBI shall maximally participate in decision-making, self-management and self-applied treatment.

Treatment involving more than one provider shall be coordinated through an authorized treating physician and case manager.

The authorized treating physician should reassess treatment at least every six months.  
  
Treatment by all practitioners should focus on establishing the highest possible level of self-sufficiency. Most passive modalities are oriented toward pain management. They should be limited and emphasize self-management and self-applied treatment with a demonstrated goal of increasing activity and function.  
  
Patients and families and/or support systems should understand that failure to comply with the elements of the self-management program or therapeutic plan of care may affect consideration of other interventions.  
  
Periodic reassessment of the individual’s condition will occur as appropriate. Overall maintenance plan should be reassessed at least annually by the authorized treating physician.   
  
Post-MMI treatment is alphabetically ordered. Programs should be individualized to specific needs.

* 1. COGNITIVE/BEHAVIORAL/PSYCHOLOGICAL MANAGEMENT: The maintenance program for individuals with moderate/severe TBIs should be oriented toward maintaining the highest level of independent function that he/she has been able to achieve. Developmental issues, changes in the individual’s support system, and development of or exacerbation of a mood or other psychiatric disorder may require psychological treatment to return the individual to the highest level of functioning possible. Individuals with or without TBI frontal involvement may need periodic reassessments, and psychiatric and/or psychological interventions. Some individuals with persistent behavioral problems (i.e. with impulsivity or other behavioral dyscontrol) may require regular psychological maintenance therapy to help the individual to function maximally in the community.   
       
     Where possible, the person with moderate/severe injury should be involved in social skills training, support groups, and/or other community-based activities to promote socialization. Some individuals with severe injuries will require periodic consultation to correct problems that have developed to allow them to continue to function in the community. Health care providers who provide services to maintain the functioning of individuals with TBI in the community are obligated to identify the specific diagnosis and symptoms on which treatment is focused and to document the ongoing results of such treatment. The number of sessions will depend on the individual and the situation. Aging or significant life change is likely to have an effect on cognitive, psychological and behavioral function and may require further treatment. Periodic assessment by the treating physician and/or an occupational, physical, or speech-language therapist may be necessary to maintain and/or upgrade the patient’s program and provide additional strategies if needed.  
       
     In the area of psychological function, researchers are learning more about long-term mood disorders, such as depression and anxiety, as well as executive dyscontrol, emotional dis-regulation, and all other disorders for which medication may be beneficial. Regaining insight or self-awareness into the changes caused by TBI is often accompanied by an increase in symptoms of depression. Depression is common following TBI. Increased suicidal ideation has also been reported to occur for many years following TBI. Psychosis is an uncommon but serious sequela of TBI that also requires psychotropic medication and close monitoring. Substance abuse, particularly alcohol abuse, can occur or recur after TBI, can worsen psychiatric and psychological co-morbidities, and should be screened for and treated if present.
  2. EXERCISE PROGRAMS REQUIRING SPECIAL FACILITIES: Some individuals with TBI may have higher compliance with an independent exercise program at a health club or a community activity-based wellness program versus participation in a home program, although individuals with TBI may require supervision or guidance. All exercise programs completed through a health club facility should be approved by the treating therapist and/or physician and focus on the same parameters of an age-adjusted and diagnosis-specific program for aerobic conditioning, flexibility, balance, stabilization, and strength. Prior to purchasing a membership, a therapist and/or exercise specialist who has treated the individual should visit the facility with the individual to assure proper use of the equipment. Periodic program evaluation and upgrading may be necessary by the therapist. The use of a personal trainer may be necessary.

❖ Frequency: Approximately 2 times per week. Regular attendance is necessary for continuation, with an exception for a medical or sufficient intervening cause.

❖ Maximum Maintenance Duration: Continuation beyond 3 months after MMI should be based on functional benefit and compliance. At MMI, health club membership should not extend beyond 3 months if attendance drops below 2 times per week on a regular basis without a medical cause.

* 1. HOME EXERCISE PROGRAMS AND EXERCISE EQUIPMENT: Most patients have the ability to participate in a home exercise program after completion of a supervised exercise rehabilitation program. Programs should incorporate an exercise prescription including the continuation of an age-adjusted and diagnosis-specific program for aerobic conditioning, flexibility, stabilization, balance, and strength. Some moderate/severe patients may benefit from the purchase or rental of equipment to maintain a home exercise program. Determination for the need of home equipment should be based on medical necessity to maintain MMI, compliance with an independent exercise program, and reasonable cost. Before the purchase or long-term rental of equipment, the patient should be able to demonstrate the proper use and effectiveness of the equipment. Effectiveness of equipment should be evaluated on its ability to improve or maintain functional areas related to ADLs or work activity. Home exercise programs are most effective when done three to five times a week. Prior to purchasing the equipment, a therapist and/or exercise specialist who has treated the patient should visit a facility with the patient to assure proper use of the equipment. Follow up evaluations in the home should occur to assure compliance and to upgrade the home program. Occasionally, compliance evaluations may be made through a four-week membership at a facility offering similar equipment. For chronic pain, refer to the Chronic Pain Guidelines.
  2. LONG-TERM RESIDENTIAL CARE: Some individuals with moderate/severe TBI may require long-term residential care due to the aging process, loss of a caregiver, becoming unsafe in their environment, or other similar changes. Such facilities or programs may provide the individual with TBI the necessary supervisory support so that he/she may safely maintain his/her maximum level of function in as least restrictive an environment as possible. In most cases, these individuals may be referred to Nursing Care Facilities (refer to Section [I.1.d. Nursing Care Facilities](#TBIG11dNursingCareFacilities), or Section [i. Supported Living Programs (SLP) or Long-Term Care Residential Services](#TBIG11iSLPorLongTermCare)).
  3. MAINTENANCE HOME CARE: Individuals with moderate/severe TBI may require ongoing home care to assist with a variety of services necessary to maintain their MMI. The type and frequency of the services required will be dependent on the nature and severity of residual deficits. Services may include skilled nursing, certified nursing assistants, life skills trainer, homemaker, and/or companion care or a combination of these services. Transportation services may also be required. Care may be necessary for limited periods of time, or in some cases may be required for the course of the individual’s lifetime.  
       
     It is essential for providers to be very specific as to the level and type of care necessary for each individual to maintain optimum health and safety. Long-term home health care is one of the most costly services of a maintenance program, and availability of professional resources may be limited. Physicians should prescribe only that care which is reasonably necessary to maintain the individual’s functional status or to cure and relieve the effects of the injury.  
       
     Over time, the individual’s status or family and/or support system’s status may change, resulting in the need to either increase or decrease the frequency, type, or level of care. Therefore, with each evaluation, or at least annually, providers shall assess any possible need for a change in home care.
  4. MEDICATION MANAGEMENT: Medications may be necessary for life long management of individuals with TBI. Medications may be used for medical, physical, perceptual, cognitive, neuroendocrine, and psychological reasons, and they should be prescribed by physicians experienced in TBI medication management. Reasons for possible medications and the types and names of medication are numerous, are individualized for each person, and are beyond the scope of these guidelines.  
       
     As with all prescriptive regimens, physicians periodically reassess the efficacy and side effects of each medication. This is particularly true for individuals who are on long-term medication use. Physicians must follow patients who are on any chronic medication or prescription regimen for compliance, efficacy, and side effects. Individuals with TBI are particularly susceptible to certain medication side effects, including compromised cognitive function, decreased seizure threshold, and other neurological effects. Follow-up visits should document the individual’s ability to perform routine functions. Laboratory or other testing is usually required on a regular basis to monitor medication effects on organ function. For some, medications and drug levels should be closely monitored. In situations where there are multiple providers for multiple clinical issues, coordination of the total medication regimen is essential. It is strongly recommended that changes in medication be discussed with the physician who is primarily managing the case. Individuals with TBI may forget to take medications and/or have difficulty with complicated medication regimens. They may need assistance with medication management, such as reminders, medication boxes, assistance with filling medication boxes, or medication administration supervision. Some medications may need to be prescribed in small amounts or locked due to safety in patients who are impulsive, forgetful, inconsistent, or otherwise unsafe in independent medication management.

❖ Maintenance Duration: Medication and medical management reviews may need to be monthly or more frequently if necessary for changes in medication. Frequency depends on the medications prescribed, with laboratory and other monitoring done as appropriate. As new medications become available and side effects of other medications are established, there may need to be changes in medical management

* 1. NEUROMEDICAL MANAGEMENT: Moderate/severe TBI patients and some MTBI patients will have ongoing medical issues requiring treatment on a regular basis. The frequency of follow up will vary according to the severity of the medical problem. Examples of related medical diagnoses include, but are not limited to: neuro-endocrine dysfunction, urinary incontinence, heterotrophic ossification, seizures, and other conditions described in the treatment sections of this guideline.

❖ Maintenance Duration: Medical management visit frequency will depend on the severity of the medical condition but may occur monthly or more frequently. Visits should occur at least at six-month intervals for extremely stable conditions.

* 1. PATIENT EDUCATION MANAGEMENT: Educational classes, sessions, or programs may be necessary to reinforce self-management techniques and social skills training and help the individual adjust to life changes. This may be performed as formal or informal programs, either group or individual.

❖ Maintenance Duration: 2 to 6 educational sessions during one 12-month period. Changes in life circumstances or the individual’s condition may require greater frequency of educational sessions.

* 1. PHYSICAL, OCCUPATIONAL, and Speech-Language THERAPY: Aggravation of the physical components of the injury may require short-term intensive treatment to return the individual to the post-MMI baseline. Therapy with the individual actively involved and/or passive therapy may be indicated on a continued basis if the therapy maintains objective physical function, decreases pain, or decreases medication use. There is good evidence that physical, occupational, or multi-disciplinary outpatient therapy reduces deterioration of ADLs and independence for stroke patients living in the community ([ [Cochrane] Outpatient Service Trialists, 2003](#OutpatientServTri2003)). Additionally, issues of aging that result in decreased function in mobility, balance, and overall physical function may require active or passive intervention. In those situations, frequency and duration parameters as defined (in Sections [G. Nonoperative Therapeutic Procedures – Initial Treatment Considerations,](#TBIGTherapeuticProceduresNonOp) [H. Non-operative Therapeutic Procedures – Neuromedical Conditions in Moderate/Severe Brain Injury](#TBIHNonOp), [I. Non-operative Therapeutic Procedures – Rehabilitation](#TBIINonOp), [J. Nonoperative Therapeutic Procedures – Vision, Speech, Swallowing, Balance & Hearing](#TBIJNonOp), and [K. Nonoperative Therapeutic Procedures – Return to Work, Driving, & Other](#TBIKNonOp)) apply. Over time, speech, language and/or cognitive functioning may deteriorate due to changes in the individual’s living situation, role and responsibilities at home or work, support systems, and/or life’s stressors. Short-term speech-language therapy emphasizing patient education, compensatory strategies, and functional goals measured objectively may be indicated. Aging issues of the individual or the caregiver may also result in a decline of speech, language and/or cognitive functioning requiring speech-language treatment.
  2. PURCHASE, RENTAL, AND MAINTENANCE OF DURABLE MEDICAL EQUIPMENT: It is recognized that some patients with TBI may require ongoing use of equipment for the purpose of maintaining MMI in the areas of strength, ROM, balance, tone control, functional mobility, ADLs, and/or analgesic effect. This may include, but is not limited to: exercise equipment; bathroom ADL equipment; assistive devices, such as shower/bath seats, assistive mobility devices, splints and/or braces, and assistive technology for memory and medication support; functional electrical muscle stimulators; TENS units and Continuous Positive Airway Pressure (C-PAP). Purchase or rental of this equipment should be done only if the assessment by the physician and/or therapist has determined the effectiveness, compliance, and improved or maintained function by its application. Periodic maintenance and replacement of the equipment may also be indicated and should be considered in the maintenance plan. It is generally felt that large expense purchases such as spas, whirlpools, and special mattresses, are not necessary to maintain function for MTBI patients.

❖ Maintenance Duration: Not to exceed 6 months for rental equipment. Purchase and maintenance should occur if effective.

**TRAUMATIC BRAIN INJURY**

**BIBLIOGRAPHY**

The bibliography for the Traumatic Brain Injury Medical Treatment Guidelines reflects the articles, abstracts, and literature reviewed during the Traumatic Brain Injury update process. Over 500 articles and literature were examined for consideration during the course of this update.   Literature that was used to support evidence statements is listed in the bibliography.   A limited number of articles qualified for evidence statements.  The designated strength of the evidence (eg. some, good, strong) may not coincide with acceptability of treatment.  Each level of evidence was assigned in accordance with the related Assessment Criteria.   
  
Where applicable, literature was given a designation of one of the following: high quality, adequate, inadequate, or not applicable.  It should be noted that some articles might have more than one assigned level, such as, ‘adequate’ on one concept and ‘high-quality’ on another concept.  The details regarding level of evidence assignment are discussed in the related critiques. Articles that were used to assign *some*, *good*, or *strong* evidence are identified in the bibliography with an asterisk.

When the evidence is conflicting or inconclusive, acceptability of treatment is determined by a combination of available medical literature and group consensus.  Some of the elements that are considered in making consensus determinations are:  level of functional benefit, acceptable risk/morbidity/mortality, and acceptable cost.  
  
A review of the Traumatic Brain Injury Medical Treatment Guidelines bibliography needs to coincide with a review of the General Guidelines Principles.  In particular, please review Guidelines Principle #12: Guidelines Recommendations and the Strength of Medical Evidence and Consensus Recommendations. All recommendations in the guidelines are considered to represent reasonable care in appropriately selected cases, regardless of the level of evidence or consensus attached to it.  Those procedures considered inappropriate, unreasonable, or unnecessary are designated in the guideline as 'not recommended.'

1. Aarabi, B., Chesler, D., Maulucci, C., Blacklock, T., & Alexander, M. (2009). Dynamics of subdural hygroma following decompressive craniectomy: a comparative study. *Journal of Neurosurgery, 26 (6)*, 1-12. DOI:10.3171/2009.3FOCUS0947.
2. Adler, R. H., & Herring, S. A. (2011). Changing the culture of concussion. Education meets legislation. *American Academy of Physical Medicine and Rehabilitation, 3,* S469-S470. DOI: 10.1016/j.pmrj.2011.08.006.
3. Aetna. Clinical policy bulletins: Vision therapy. No. 0489. Retreived from: http://www.aetna.com/cpb/medical/data/400\_499/0489.html
4. Alderson, P., & Roberts I. (2005). Corticosteroids for acute traumatic brain injury (Review). *Cochrane Database of Systematic Reviews, 1,*1-27. <http://www.thecochranelibrary.com>
5. Allum, J. H. J., & Shepard, N. T. (1999). An overview of the clinical use of dynamic posturography in the differential diagnosis of balance disorders. *Journal of Vestibular Research, 9,* 223-252. ISBN 0957-4271, IOS Press.
6. American Academy of Neurology. (1997). Practice parameter: The management of concussion in sports (summary statement). *Neurology, 48,* 581-585. ISSN:0028-3878.
7. American Automobile Association. (2011). Interactive Driving Evaluation. Retrieved October 2012, from http://seniordriving.aaa.com/evaluate-your-driving-ability/interactive-driving-evaluation.
8. American College of Emergency Physicians (ACEP), Centers for Disease Control and Prevention (CDC), Jagoda, A. S., Bazarian, J. J., Burns, J. J. Jr., Cantrill, S. V., Gean, A. D., Howard, P. K.,…& Whitson, R. R. (2008). Clinical policy: Neuroimaging and decision making in adult mild traumatic brain injury in the acute setting. *Annals of Emergency Medicine, 52,* 714-748. DOI:10.1016/j. annemergmed.2008.08.021.
9. American Congress of Rehabilitation Medicine; Mild Traumatic Brain Injury Committee. (1993). Definition of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation, 8 (3),* 86–87.
10. American Medical Association. (2010, March 15). AMA releases new older driver safety guide motor vehicle injuries a leading cause of injury-related deaths in seniors. 1995-2012. Retrieved December 2011, from <http://www.ama-assn.org/ama/pub/news/news/older-driver-safety.page>?
11. Anand, R., Chapman, S. B., Rackley, A. R., Keebler, M., Zientz, J., & Hart Jr., J. (2010). Gist reasoning training in cognitively normal seniors. *International Journal of Geriatric Psychiatry, 26,* 961-968. DOI: 10.1002/gps.2633.
12. Anderson, K. E., Hurley, R. A., & Taber, K. H. (2011). Functional imaging. *American Psychiatric Association, 6,* 1-23 DOI: 10.1176/appl. books.9781585624201.673855. <http://www.psychiatryonline.com>
13. Andrews, P., Sinclair, H. L., Battison, C. G., Polderman, K. H., Citerio, G., Mascia, L., … the Eurotherm3235Trial collaborators. (2011). European society of intensive care medicine study of therapeutic hypothermia (32-35°C) for intracranial pressure reduction after traumatic brain injury (the Eurotherm3235Trial). *European Society of Intensive Care Medicine, 12(8),* 1-12. <http://www.trialsjournal.com/content/12/1/8>
14. Andrews, S. M, & A Cheng. (2008). Modest cooling therapies (35˚C to 37.5˚C) for traumatic brain injury (Review). *Cochrane Database of Systematic Reviews, 3,*1-14<http://www.thecochranelibrary.com>
15. Angevaren, M., Aufdemkampe, G., Verhaar, H. J. J., Aleman, A., & Vanhees, L. (2008). Physical activity and enhanced fitness to improve cognitive function in older people without known cognitive impairment (review). *Cochrane Database of Systematic Reviews, 2,* 1-7. DOI: 10.1002/14651858.CD005381.pub2.
16. Arango, M. F., & Bainbridge, D. (2008). Magnesium for acute traumatic brain injury (Review). *Cochrane Database of Systematic Reviews, 4,* 1-18. DOI: 10.1002/14651858.CD005400.pub3. <http://www.thecochranelibrary.com>
17. Archer, T. (2011). Influence of physical exercise on traumatic brain injury deficits: Scaffolding effect. *Neurotox Res*. DOI: 10.1007/s12640-011-9297-0.
18. Archer, T., Svensson, K., & Alricsson, M. (2012). Physical exercise ameliorates deficits induced by traumatic brain injury. *Acta Neurologica Scandinavica,* 1-10. DOI: 10.1111/j.1600-0404.2011.01638. x.
19. Arciniegas, D. B. (2011). Clinical electrophysiologic assessments and mild traumatic brain injury: State-of-the-science and implications for clinical practice. *International Journal of Psychophysiology, 82,* 41-52. DOI: 10.1016/j. ijpsycho.2011.03.004.
20. Arciniegas, D. B., Anderson, C. A., Topkoff, J., & McAllister, T. W. (2005). Mild traumatic brain injury: a neuropsychiatric approach to diagnosis, evaluation, and treatment. *Neuropsychiatric Disease and Treatment, 1(4),* 311–327.
21. Arciniegas, D. B., Frey, K. L., Newman, J., & Wortzel, H. S. (2010). Evaluation and management of posttraumatic cognitive impairments. *Psychiatr Ann., 40(11),* 540-552. DOI: 10.3928/00485713-20101022-05.
22. Arciniegas, D. B., & Silver, J. M. (2007). Pharmacotherapy of cognitive impairment. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain Injury Medicine Principles and Practice* (995-1022). New York, NY: Demos.
23. Arlinghaus, K. A., Pastorek, N. J., & Graham, D. P. (2011). Neuropsychiatric assessment. *American Psychiatric Association, 4,* 1-17. DOI: 10.1176/appi. books.9781585624201.672629.
24. Arrich, J., Holzer M., Herkner H., & Müllner, M. (2010). Hypothermia for neuroprotection in adults after cardiopulmonary resuscitation (Review) *Cochrane Database of Systematic Reviews, 8,* 2-33.  [http://thecochranelibrary.com](%20http://thecochranelibrary.com)
25. Arundine, A., Bradbury, C. L., Dupuis, K., Dawson, D. R., Ruttan, L. A., & Green, R. E*.* (2012). Cognitive behavior therapy after acquired brain injury: maintenance of therapeutic benefits at 6 months posttreatment. *Journal of Head Trauma & Rehabilitation, 27(2),* 104-12. DOI: 10.1097/HTR.0b013e3182125591.
26. Ashman, T. A., Cantor, J. B., Gordon, W. A., Spielman, L., Flanagan, S., Ginsberg, A., … Greenwald, B. (2009). A randomized controlled trial of sertraline for the treatment of depression in persons with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 90,* 733-740. DOI:10.1016/j.apmr.2008.11.005.
27. Association of Rehabilitation Nurses. (2012). Definitions and Scope of Practice. Retrieved from http://www.rehabnurse.org/index.php/certification/content/definition.html.
28. Aurora, S. K., Dodick, D. W., Turkel, C. C., DeGryse, R. E.,. . . Brin, M. F. (2010). OnobotulinumtoxinA for treatment of chronic migraine: Results from the double-blind, randomized, placebo-controlled phase of the PREEMPT I trial. *Cephalalgia, 30 (7),* 793-814. DOI: 10.1177/0333102410364676.
29. Aurora, S. K., Winner, P. Freeman, M. C., Spierings, E. L., Heiring, J. O., DeGryse, R. E.,… Turkel, C. C. (2011). OnabotulinumtoxinA for treatment of chronic migraine: Pooled analyses of the 56-Week PREEMPT clinical program. *Headache, 51,*1358-1373. DOI: 10.1111/j.1526-4610.2011.01990.x.
30. Aziz, N. A., Leonardi-Bee, J., Phillips, M. F., Gladman, J., Legg, L. A., & Walker, M. (2008). Therapy-based rehabilitation services for patients living at home more than one year after stroke. *Cochrane Database of Systematic Reviews, 2,* 1-26. DOI: 10.1002/14651858.CD005952.pub2.
31. Badjatia, N. (2009). Hyperthermia and fever control in brain injury. *Society of Critical Care Medicine, 37(7),*S250-S257.
32. Barclay-Goddard, R. E., Stevenson, T. J., Poluha,W., Moffatt, M., Taback, S. P. (2004). Force platformfeedback for standing balance training after stroke. *Cochrane Database of Systematic Reviews, 4,* 1-24. DOI: 10.1002/14651858.CD004129.pub2.
33. Barclay-Goddard, R. E., Stevenson, T. J., Poluha, W., & Thalman, L. (2011). Mental practice for treating upper extremity deficits in individuals with hemiparesis after stroke. *Cochrane Database of Systematic Reviews, 5,* 1-45. DOI: 10.1002/14651858.CD005950.pub4.
34. Barkhoudarian, G., Hovda, D., & Giza, C. (2011). The molecular pathophysiology of concussive brain injury. *Clinical Sports Medicine, 30,* 33**-**48. DOI: 10.1016/j.csm.2010.09.001
35. Barnes, D. E., Yaffe, K., Satariano, W. A., & Tager, I. B. (2003). A longitudinal study of cardiorespiratory fitness and cognitive function in healthy older adults. *JAGS, 51(4),* 459-465.
36. Basso, A., Cattaneo, S., Girelli, L, Luzzatti, C., Miozzo, A., Modena, L., & Monti, A. (2011). Treatment efficacy of language and calculation disorders and speech apraxia: A review of literature. *European Journal of Physical and Rehabilitation Medicine, 47(1),* 101-121.
37. Belafsky, P., Gianoli, G., Soileau, J., Moore, D., & Davidowitz, S. (2000). Vestibular autorotation testing in patients with benign paroxysmal positional vertigo. *Otolaryngology -- Head and Neck Surgery, 122,* 163-167. DOI: 10.1016/S0194-5998(00)70233-6
38. Belanger, H. G., Curtiss, G., Demery, J. A., Lebowitz, B. K., & Vanderploeg, R. D. (2005). Factors moderating neuropsychological outcomes following mild traumatic brain injury: A meta- analysis. *Journal of the International Neuropsychological Society, 11,* 215-227. DOI: 10.1017/S1355617705050277.
39. Belanger, H. G., Vanderploeg, R. D., Curtiss, G., & Warden, D. L. (2007). Recent neuroimaging techniques in mild traumatic brain injury. *The Journal of Neuropsychiatry and Clinical Neurosciences 19:*5-20. <http://neuro.psychiatryonline.org>
40. Bell, K. R., Hoffman, J. M., Temkin, N. R., Powell, J. M., Fraser, R. T., Esselman, P. C.,… Dikmen, S. (2008). The effect of telephone counselling on reducing posttraumatic symptoms after mild traumatic brain injury: A randomised trial. *J Neurol Neurosurg Psychiatry, 79,* 1275–1281. DOI:10.1136/jnnp.2007.141762.
41. Bendtsen, L., Evers, S., Linde, M., Mitsikostas, D. D.,Sandrini, G., & Schoenen, J. (2010). EFNS guidelines on the treatment of tension-type headaches – Report of an EFNS task force. *European Journal of Neurology, 17,* 1318-1325. DOI:10.1111/j.1468-1331.2010.03070. x
42. Bendtsen, L., Evers, S., Linde, M., Mitsikostas, D. D.,Sandrini, G., & Schoenen, J. (2012). Treatment of tension-type headache. *European Handbook of Neurological Management, 2, Second Edition,* 225-238.
43. Bendtsen, L, Jensen, R., & Olesen, J. (1996). A non-selective (amitriptyline), but not a selective (citalopram), serotonin reuptake inhibitor is effective in the prophylactic treatment of chronic tension-type headache. *Journal of Neurology, Neurosurgery, and Psychiatry, 61,* 285-290. http://www. jnnp.bmj.com. *\*\*\*Used in Evidence Statement.*
44. Bennett, M. H., Trytko, B., & Jonker, B. (2004). Hyperbaric oxygen therapy for the adjunctive treatment of traumatic brain injury (Review). *Cochrane Database of Systematic Reviews, 4,* 1-31.<http://www.thecochranelibrary.com>
45. Bennett, M. H., Wasiak, J., Schnabel, A., Kranke, P., & French, C. (2005). Hyperbaric oxygen therapy for acute ischaemic stroke. *Cochrane Database of Systematic Reviews, 3,* 1-37. DOI: 10.1002/14651858.CD004954.pub2.
46. Bernard, S. (2009). Hypothermia after cardiac arrest: Expanding the therapeutic scope*. Critical Care Medicine, 37(7),* S227-S233.
47. Bernhardt, J., Thuy, M. N. T., Collier, J. M., & Legg, L. A. (2009). Very early versus delayed mobilisation after stroke. *Cochrane Database of Systematic Reviews, 1,* 1-19. DOI: 10.1002/14651858.CD006187.pub2.
48. Berrigan, L., Marshall, S., McCullagh, S., Velikonja, D., & Bayley, M. (2011). Quality of clinical practice guidelines for persons who have sustained mild traumatic brain injury. *Brain Injury, 25(7-8),* 742-751. DOI: 10.3109/02699052.2011.580317.
49. Bethge, M., Herbold, D., Trowitzsch, L., & Jacobi, C. (2011). Work status and health-related quality of life following multimodal work hardening: A cluster randomized trial. *Journal of Back and Musculoskeletal Rehabilitation, 24,* 161-172. DOI:10.3233/BMR-2011-0290, IOS Press.
50. Bhattacharyya, N., Baugh, R. F., Orvidas, L., Barrs, D., Bronston, L. J., Cass, S., …Haidari, J. (2008). Clinical practice guideline: Benign paroxysmal positional vertigo. *Otolaryngology–Head and Neck Surgery, 139,* S47-S81. DOI:10.1016/j. otohns.2008.08.022.
51. Bigler, E. D. (2011). Structural imaging. *American Psychiatric Association, 5,* 1-18. DOI: 10.1176/appl. books.971585624201.673559. htttp://www.psychiatryonline.com
52. Birks, J. (2006). Cholinesterase inhibitors for Alzheimer’s disease. *Cochrane Database of Systematic Reviews, 1,* 1-89. DOI: 10.1002/14651858.CD005593.
53. Black, F. O., Angel C. R., Pesznecker, S. C., & Gianna, C. (2000). Outcome analysis of individualized vestibular rehabilitation protocols. *The American Journal of Otology, 21,* 543-551.
54. Bland, D., Zampieri, C., & Damiano, D. (2011). Effectiveness of physical therapy for improving gait and balance in individuals with traumatic brain injury: A systematic review. *Brain Injury, 25(7–8)*, 664–679. DOI: 10.3109/02699052.2011.576306.
55. Blatt, P. J., Georgakakis, G. A., Herdman, S., J., Clendaniel, R., A., & Tusa, R. J. (2000). The effect of the canalith repositioning maneuver on resolving postural instability in patients with benign paroxysmal positional vertigo. *The American Journal of Otology, 21,* 356-363.
56. Bleiberg, J., & O’Shanick, G. (2009). The road to rehabilitation part 3: Guideposts to recognition: Cognition, memory & brain injury. *Brain Injury Association of America. The Voice of Brain Injury.* <http://www.biausa.org>
57. Block, A. R., Ohnmeiss, D. D., Guyer, R. D., Rashbaum, R. F., & Hochschuler, S. H. (2001). The use of presurgical psychological screening to predict the outcome of spine surgery. *The Spine Journal, 1,* 274-282. PII: S1529-9430(01)00054-7. *\*\*\*Used in Evidence Statement.*
58. Bowen, A., Knapp, P., Gillespie, D., Nicolson, D. J., & Vail, A. (2011), Non-pharmacological interventions for perceptual disorders following stroke and other adult-acquired, non-progressive brain injury. *Cochrane Database of Systematic Reviews, 4,*  1-50. DOI: 10.1002/14651858.CD007039.pub2.
59. Bowen, A., & Lincoln, N. (2007). Cognitive rehabilitation for spatial neglect following stroke. *Cochrane Database of Systematic Reviews, 2,* 1-51. DOI: 10.1002/14651858.CD003586.pub2.
60. Bradbury, C. L., Christensen, B. K., Lau, M. A., Ruttan, L. A., Arundine, A. L., & Green, R. E. (2008). The efficacy of cognitive behavior therapy in the treatment of emotional distress after acquired brain injury. *Arch Phys Med Rehabil. 89(12 Suppl),* S61-S68. DOI:10.1016/j.apmr.2008.08.210.
61. Bradt, J., Magee, W. L., Dileo, C., Wheeler, B. L., & McGilloway, E. (2010). Music therapy for acquired brain injury (Review). *Cochrane Database of Systematic Reviews, 7,* 1-42. DOI: 10.1002/14651858.CD006787.pub2. <http://www.thecochranelibrary.com> *\*\*\*Used in Evidence Statement*
62. Brain Injury Association of America. (2009). *Conceptualizing Brain Injury as a Chronic Disease.*
63. Brasure, M., Lamberty, G. J., Sayer, N. A., Nelson, N. W., MacDonald, R., Ouellette, J.,… Wilt, T. J. (2012). Multidisciplinary postacute rehabilitation for moderate to severe traumatic brain injury in adults. (Prepared by the Minnesota Evidence-based Practice Center under Contract No. 290-2007-10064-I.) *AHRQ,* *12-EHC101-EF*, E1-E32. www.effectivehealthcare.ahrq.gov/reports/final.cfm.
64. Bratton, S. L., Chestnut, R. M., Ghajar, J., Hammond, F. F. M., Harris, O. A., Hartl, R.,… Wright, D. W. (2007a). I. Blood pressure and oxygenation. *Journal of Neurotrauma, 24(Supplement 1),*S7-S13. DOI:10.1089/neu.2007.9995.
65. Bratton, S. L., Chestnut, R. M., Ghajar, J., Hammond, F. F. M., Harris, O. A., Hartl, R.,… Wright, D. W. (2007b). II. Hyperosmolar therapy*. Journal of Neurotrauma, 24(Supplement 1),*S14-S20. DOI:10.1089/neu.2007.9994.
66. Bratton, S. L., Chestnut, R. M., Ghajar, J., Hammond, F. F. M., Harris, O. A., Hartl, R.,… Wright, D. W. (2007c). III Prophylactic hypothermia. *Journal of Neurotrauma, 24(Supplement 1),* S21-S25. DOI:10.1089/neu.2007.9993.
67. Bratton, S. L., Chestnut, R. M., Ghajar, J., Hammond, F. F. M., Harris, O. A., Hartl, R.,… Wright, D. W. (2007d). VI. Indications for intracranial pressure monitoring. *Journal of Neurotrauma, 24(Supplement 1),* S37-S44. DOI:10.1089/neu.2007-9990.
68. Bratton, S. L., Chestnut, R. M., Ghajar, J., Hammond, F. F. M., Harris, O. A., Hartl, R.,… Wright, D. W. (2007e). XIV. Hyperventilation. *Journal of Neurotrauma, 24(Supplement 1),* S87-S90. DOI:10.1089/neu.2007.9982.
69. Bratton, S. L., Chestnut, R. M., Ghajar, J., Hammond, F. F. M., Harris, O. A., Hartl, R.,… Wright, D. W. (2007f). XV. Steroids. *Journal of Neurotrauma, 24(Supplement 1),* S91-95. DOI: 10.1089/neu.2007.9981.
70. Broglio, S. P., Sosnoff, J. J., Rosengren, K. S., & McShane, K. (2009). A comparison of balance performance: Computerized dynamic posturography and a random motion platform. *American College of Rehabilitation Medicine, 90,* 145-150. DOI:10.1016/J. apmr.2008.06.025.
71. Broglio, S. P., Tomporowski, P. D., & Ferrara, M. S. (2005). Balance performance with a cognitive task: A dual-task testing paradigm. *American College of Sports Medicine*. DOI: 10.1249.01. MSS.0000159019.14919.09.
72. Bronfort, G., Nilsson, N., Haas M., Evans, R. L., Goldsmith, C. H., Assendelft, W. J. J., & Bouter, L. M. (2004). Non-invasive physical treatments for chronic/recurrent headache. *Cochrane Database of Systemic Reviews, 3,*1-70. DOI:10.1002/14651858. CD001878. pub2. *\*\*\*Used in Evidence Statement*
73. Brown, T. H., Mount, J., Rouland, B. L., Kautz, K. A., Barnes, R. M., & Kim, J. (2005). Body weight-supported treadmill training versus conventional gait training for people with chronic traumatic brain injury. *Journal of Head Trauma Rehabilitation, 20(5),* 402-415.
74. Bruns, D., & Disorbio, J. M. (2009). Assessment of biopsychosocial risk factors for medical treatment: A collaborative approach. *Journal of Clinical Psychological Medicine Settings, 16,* 127–147. DOI 10.1007/s10880-009-9148-9.
75. Buki, A., & Povlishock, J. T. (2006). All roads lead to disconnection? – Traumatic axonal injury revisited. *Acta Neurochir, 148,* 181–194. DOI 10.1007/s00701-005-0674-4.
76. Bullock, M. R., & Povlishock, J. T. (2007). Editor’s commentary. *Journal of Neurotrauma, 24(1).* DOI: 10.1089/neu.2007.9998.
77. Cabraja, M., Klein, M., & Lehman, T. (2009). Long-term results following titanium cranioplasty of large skull defects. *Journal of Neurosurgery, 26(6),* 1-7. DOI: 10.3171/2009.3FOCUS091.
78. Cantu, R. C., & Register-Milhalik, J. K., (2011). Considerations for return-to-play and retirement decisions after concussion. *American Academy of Physical Medicine and Rehabilitation, 3,* S440-S444. DOI: 10.1016/j. pmrj.2011.07.013.
79. Carnaby-Mann, G. D., & Crary, M. A.(2007). Examining the evidence on neuromuscular electrical stimulation for swallowing: A meta-analysis. *Archives of Otolaryngol – Head and Neck Surgery, 133,* 564-571.
80. Carney, N. A. (2007a). Methods. *Journal of Neurotrauma, 24(Supplement 1),* S3-S6. DOI: 10.1089/neu.2007.9996.
81. Carney, N. A., & Ghajar, J. (2007b). Introduction. *Journal of Neurotrauma, 24(Supplement 1),* S1-S2. DOI:10.1089/neu.2007.9997.
82. Carroll, L. J., Cassidy, J. D., Peloso, P. M., Borg, J., von Holst, H., Holm, L.,… Pepin, M. (2004). Prognosis for mild traumatic brain injury: Results of the WHO collaborating centre task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine, Suppl. 43,* 84–105. DOI 10.1080/16501960410023859.
83. Case Management Society of America. (2012). What is a case manager?. Retrieved October 2011,  from http://www.cmsa.org/Home/CMSA/WhatisaCaseManger/tabid/224/Default.aspx
84. Centers for Disease Control and Prevention. U.S. Department of Health and Human Services. (n.d.). Heads up: Facts for physicians about mild traumatic brain injury (MTBI). *Facts for Physicians,* 1-23.
85. Chang, B., & Lowenstein, D., (2003). Practice parameter: Antiepileptic drug prophylaxis in severe traumatic brain injury: Report of the quality standards subcommittee of the American Academy of Neurology. *Neurology, 60,* 10-16. DOI: 10.1212/01. WNL.0000031432.05543.14
86. Chang, V. H., Lombard, L., & Greher, M. R. (2011). Mild traumatic brain injury in the occupational setting. *American Academy of Physical Medicine and Rehabilitation, 3,* S387-S395. DOI: 10.1016/j. pmrj.2011.08.007.
87. Chen, Y. –H., Kang, J. –H., & Lin, H. –C. (2011). Patients with traumatic brain injury: Population-based study suggests increased risk of stroke. *Stroke*, *42,* 2733-2739. DOI: 10.1161/STROKEAHA.111.620112.
88. Chi, J. H. (2011). Craniectomy for traumatic brain injury: Results from the DECRA trial. *Neurosurgery, 68(6),* N19-N20.
89. Chiarovano, E., Zamith, F., Vidal, P. -P., & de Waele, C. (2011). Ocular and cervical VEMPs: A study of 74 patients suffering from peripheral vestibular disorders. *Clinical Neurophysiology, 122,* 1650–1659. DOI:10.1016/j. clinph.2011.01.006.
90. Chronicle, E. P., & Mulleners, W. M. (2004). Anticonvulsant drugs formigraine prophylaxis. *Cochrane Database of Systematic Reviews, 3,* 1-60. DOI: 10.1002/14651858.CD003226.pub2. *\*\*\*Used in Evidence Statement*
91. Cicerone, K. D., & Azulay, J. (2007). Perceived self-efficacy and life satisfaction after traumatic brain injury. *Journal of Head Trauma Rehabilitation, 22(5),* 257-266.
92. Cicerone, K. D., Dahlberg, C., Kalmar, K., Langenbahn, D. M., Malec, J. F., Bergquist, T. F.,… Morse, P. A. (2000). REVIEW ARTICLE Evidence-based cognitive rehabilitation: Recommendations for clinical practice. *Archives of Physical Medicine and Rehabilitation, 81,* 1596-1615. DOI:10.1053/apmr.2000.19240.
93. Cicerone, K. D., Dahlberg, C., Malec, J. F., Langenbahn, D. M., Felicetti, T., Kneipp, S.,… Catanese, J. (2005). REVIEW ARTICLE Evidence-based cognitiver rehabilitation: updated review of the literature from 1998 through 2002. *Archives of Physical Medicine and Rehabilitation, 86,* 1681-1692. DOI:10.1016/j.apmr.2005.03.024.
94. Cicerone, K. D., Langenbahn, D. M., Braden, C., Malec, J. F., Kalmar, K., Fraas, M.,…Ashman, T. (2011). REVIEW ARTICLE (META-ANALYSIS) Evidence-based cognitive rehabilitation: Updated review of the literature from 2003 through 2008. *Archives of Physical Medicine and Rehabilitation, 92,* 519-530. DOI:10.1016/j.apmr.2010.11.015.
95. Cicerone, K. D., Mott, T., Azulay, J., Sharlow-Galella, M. A., Ellmo, W. J., Paradise, S., & Friel, J. C. (2008). A randomized controlled trial of holistic neuropsychologic rehabilitation after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 89,* 2239-2249. DOI:10.1016/j.apmr.2008.06.017. *\*\*\*Used in Evidence Statement.*
96. Ciuffreda, K. J., Kapoor, N., Rutner, D., Suchoff, I. B.,…Craig, S. (2007). Occurrence of oculomotor dysfunctions in acquired brain injury: A retrospective analysis. *American Optometric Association, 78,* 155-161. DOI:10.1016/j.optm.2006.11.011.
97. Ciuffreda, K. J., Ludlam, D., COVT, & Thiagarajan, P. (2011a). Oculomotor diagnostic protocol for the mTBI population. *Elsevier Inc. for American Optometric Association,* 61-63. DOI:10.1016/j.optm.2010.11.011.
98. Ciuffreda, K. J., & Ludlam, D. (2011b). Objective diagnostic and interventional vision test protocol for the mild traumatic brain injury population. *American Optometric Association,* 337-339. DOI:10.1016/j.optm.2011.03.006.
99. Ciuffreda, K. J., Rutner, D., Kapoor, N., Suchoff, I. B., & Han, M. E. (2008). Vision therapy for oculomotor dysfunctions in acquired brain injury: A retrospective analysis. *Optometry, 79,* 18-22. DOI:10.1016/j.optm.2007.10.004.
100. Clark, H., Lazarus, C., Arvedson, J., Schooling, T., & Frymark, T. (2009). Evidence-based systematic review: Effects of neuromuscular electrical stimulation on swallowing and neural activation. *American Journal of Speech-Language Pathology, 18,* 361–375. DOI:10.1058-0360/09/1804-0361.
101. Clifton, G. L., Miller, E. R., Choi, S. C., Levin, H. S., McCauley, S., Smith, K. R., …Schwartz, M. (2001). Lack of effect of induction of hypothermia after acute brain injury. *The New England Journal of Medicine, 344(8),* 556-563. <http://www.nejm.org>
102. Clifton, G. L., Valadka, A., Zygun, D., Coffey, C. S., & Okonkwo, D. O. (2011). Very early hypothermia induction in patients with severe brain injury (the National Acute Brain Injury Study: Hypothermia II): a randomized trial. *The Lancet, 10,* 131-139. [www. thelancet.com/neurology](http://www.thelancet.com/neurology).
103. Coburn, K., Lauterbach, E. C., Boutros, N. N., Black, K. J., Arciniegas, D. B., & Coffey, C. E. (2006). The value of quantitative electroencephalography in clinical psychiatry: A report by the committee on research of the American Neuropsychiatric Association. *The Journal of Neuropsychiatry and Clinical Neurosciences, 18,* 460-500. <http://neuro.psychiatryonline.org>
104. Cohen, H. S. (2006). Disability and rehabilitation in the dizzy patient. *Current Opinion in Neurology, 19,* 49–54.
105. Cohen H. S., & Kimball, K. T. (2008). Usefulness of some current balance tests for identifying individuals with disequilibrium due to vestibular impairments*. Journal of Vestibular Research, 18,* 1-16. [hcohen@bem.tmc.edu](mailto:hcohen@bem.tmc.edu)
106. Cohen, H. S., & Sangi-Haghpeykar, H. (2010). Canalith repositioning variations for benign paroxysmal positional vertigo. *Otolaryngology Head Neck Surgery, 143(3),* 1-12. [hcohen@bem.tmc.edu](mailto:hcohen@bem.tmc.edu)
107. Colcombe, S., & Kramer, A. F. (2003). Fitness effects on the cognitive function of older adults: A meta-analytic study. *Psychological Science, 14(2),* 125-130.
108. Colcombe, S, Kramer, A. F., McAuley, E., Erickson, K. I., & Scalf, P. (2004). Neurocognitive aging and cardiovascular fitness recent findings and future directions. *Journal of Molecular Neuroscience, 24,* 9-14.
109. Commission for Case Manager Certification. (n.d) Professional case manager. Retrieved October 2012,  from <http://ccmcertification.org/case-managers/professional-case-manger>.
110. Congressional Record E462 (March 23, 2010). Extensions of Remarks. Speech of Honorable Bill Pascrell Jr.
111. Cooper, D. J., Rosenfeld, J. V., & Davies, A. R. (2011a) Correspondence craniectomy in diffuse traumatic brain injury. *The New England Journal of Medicine, 365(4),* 373-376. <http://www.nejm.org>
112. Cooper, D. J., Rosenfeld, J. V., Murray, L., Arabi, Y. M., Davies, A. R., D’Urso, P.,… Wolfe, R. (2011b). Decompressive craniectomy in diffuse traumatic brain injury. *The New England Journal of Medicine, 364(16),* 1493-1502. DOI: 10.1056/NEJMoa1102077.
113. Costa, J., Espírito-Santo, C. C., Borges, A. A., Ferreira, J., Coelho, M. M., Moore, P., & Sampaio, C. (2005). Botulinum toxin type A therapy for cervical dystonia (Review). *Cochrane Database of Systematic Reviews, 1,* 1-71. DOI: 10.1002/14651858.CD003633.pub2. <http://www.thecochranelibrary.com>. *\*\*\*Used in Evidence Statement*
114. Coupar, F., Pollock, A., van Wijck, F., Morris, J., & Langhorne, P. (2010). Simultaneous bilateral training for improving arm function after stroke. *Cochrane Database of Systematic Reviews, 4,* 1-62. DOI: 10.1002/14651858.CD006432.pub2.
115. CRASH trial collaborators. (2004). Effect of intravenous corticosteroids on death within 14 days in 10,008 adults with clinically significant head injury (MRC CRASH trial): randomized placebo-controlled trial. *The Lancet, 364,* 1321-1328. *\*\*\*Used in Evidence Statement.*
116. CRASH trial collaborators. (2005). Final results of MRC CRASH, a randomized placebo-controlled trial of intravenous corticosteroid in adults with head injury – outcomes at 6 months. *The Lancet, 365,* 1957-1959. DOI:10.1016/50140-6736(05)6652X.
117. Cullen, N., Bayley, M., Bayona, N., Hilditch, M., & Aubut, J. (2007). Management of heterotopic ossification and venous thromboembolism following acquired brain injury. *Brain Injury, 21(2),* 215-230. DOI: 10.1080/02699050701202027.
118. Currie, SR, & Wilson, KG. (2000). Cognitive-Behavioral Treatment of Insomnia Secondary to Chronic Pain.  *Journal of Consulting and Clinical Psychology. 68*; 3, 407-416. Assessment: Adequate. *\*\*\*Used in Evidence Statement*
119. Dahlberg, C., Cusick, C. P., Hawley, L. A., Newman, J. K., Morey, C. E. Harrison-Felix, C. L., Whiteneck, G. G., (2007). Treatment efficacy of social communication skills training after traumatic brain injury: A randomized treatment and deferred treatment controlled trial. *Archives of Physical Medicine and Rehabilitation, 88,* 1561-1573. DOI: 10.1016/j.apmr.2007.07.033. *\*\*\*Used in Evidence Statement.*
120. Danish, S. F., Barone, D., Lega, B. C., & Stein, S. C. (2009). Quality of life after hemicraniectomy for traumatic brain injury in adults. *Journal of Neurosurgery, 26(6),* E2. DOI: 10.3171/2009.3FOCUS945.
121. das Nair, R., & Lincoln, N. (2007). Cognitive rehabilitation for memory deficits following stroke. *Cochrane Database of Systematic Reviews, 3,* 1-20. DOI: 10.1002/14651858.CD002293.pub2.
122. Dash, P. K., Zhao, J., Hergenroeder, G., & Moore, A. N. (2010). Biomarkers for the diagnosis, prognosis, and evaluation of treatment efficacy for traumatic brain injury. *The American Society for Experimental Neurotherapeutics, Inc., 7,* 100-114.
123. Dean, P J. A., O’Neill, D., & Sterr, A. (2012). Post-concussion syndrome: Prevalence after mild traumatic brain injury in comparison with a sample without head injury. *Brain Injury, January, 26(1),* 14–26. DOI: 10.3109/02699052.2011.635354.
124. Dean, C. M., Richards, C. L., & Malhouin, F. (2001). Walking speed over 10 metres overestimates locomotor capacity after stroke. *Clinical Rehabilitation, 15,* 415-421. DOI: 10.1191/026921501678310216.
125. Defense and Veterans Brain Injury Center. (2008). Defense and Veterans Brain Injury Center Consensus Conference on the acute management of concussion/mild traumatic brain injury (mTBI) in the deployed setting. 1-10.
126. Defense Centers for Excellence for Psychological Health and Traumatic Brain Injury. (2010a). Case management of concussion/mild TBI: Guidance document. <http://www.dcoe.health.mil.ForHealthPros/TBIInformation.aspx>
127. Defense Centers for Excellence for Psychological Health and Traumatic Brain Injury. (2010b). Portable, field-based devices for the early diagnosis of mild traumatic brain injury. [*http://www.dcoe.health.mil.ForHealthPros/TBIInformation.aspx*](http://www.dcoe.health.mil.ForHealthPros/TBIInformation.aspx)
128. Defense Centers for Excellence for Psychological Health and Traumatic Brain Injury. (2009). Driving following traumatic brain injury: Clinical recommendations. [*http://www.dcoe.health.mil.ForHealthPros/TBIInformation.aspx*](http://www.dcoe.health.mil.ForHealthPros/TBIInformation.aspx)
129. Den Hertog, H. M., van der Worp, H. B., Tseng, M. C., & Dippel, D. W. J. (2009). Cooling therapy for acute stroke. *Cochrane Database of Systematic Reviews, 1,* 1-38. DOI: 10.1002/14651858.CD001247.pub2.
130. Denise, P., Vouriot, A., Normand, H., Golding, J. F., & Gresty, J. A. (2009). Effect of temporal relationship between respiration and body motion on motion sickness. *Autonomic Neuroscience: Basic and Clinical, 151,*142-146. DOI:10.1016/j. autney.209.06.007.
131. Derry, S., Moore, R. A., & McQuay, H. J. (2010). Paracetamol (acetaminophen) with or without an antiemetic for acute migraine headaches in adults. *Cochrane Database of Systematic Reviews, 11,* 1-55. DOI:10.1002/14651858. CD008040. pub2. *\*\*\*Used in Evidence Statement*
132. Diener, H. C., Dodick, D. W., Aurora, S. K., Turkel, C. C., DeGryse, R. E., Lipton, R. B., …Brin, M. F., (2010) OnabotulinumtoxinA for treatment of chronic migraine: Results from the double-blind, randomized, placebo-controlled phase of the PREEMPT 2 trial. *Cephalalgia, 30(7),* 804-814.DOI: 10.1177/0333102410364677.
133. Dietrich, W. D., & Bramlett, H. M. (2010). The evidence for hypothermia as a neuroprotectant in traumatic brain injury*. Neurotherapeutics, 7(1),* 1-13. DOI:10.1016/j.nurt.2009.10.015.
134. DiFabio, R. (1995). Sensitivity and specificity of platform posturography for identifying patients with vestibular dysfunction. *Physical Therapy, 75(4),* 290-305.
135. Ditunno, Jr. J. F. (1992). Functional assessment measures in CNS trauma. *Journal of Neurotrauma, 9(1),* S301-305.
136. Dobie, R. A. (1997). Does computerized dynamic posturography help us care for our patients? *The American Journal of Otology, 18,* 108-112.
137. Doble, J. E., Feinberg, D. L., Rosner, M. S., & Rosner, A. J. (2010). Identification of binocular vision dysfunction (vertical heterophoria) in traumatic brain injury patients and effects of individualized prismatic spectacle lenses in the treatment of postconcussive symptoms: a retrospective analysis. *American Academy of Physical Medicine and Rehabilitation, 2,* 244-253. DOI: 10:1016/j.pmrj-2010.01.011.
138. Donaldson, C. J., Hoffer, M. E., Balough, B. J., & Gottshall, K. R. (2010). Prognostic assessments of medical therapy and vestibular testing in post-traumatic migraine-associated dizziness patients. *Otolaryngology-Head and Neck Surgery, 143,* 820-825. DOI:10.1016/j. otohns.2010.09.024.
139. Doyle, S., Bennett, S., Fasoli, S. E., & McKenna, K. T. (2010). Interventions for sensory impairment in the upper limb after stroke. *Cochrane Database of Systematic Reviews, 6,* 1-57. DOI: 10.1002/14651858.CD006331.pub2.
140. Dutton, R. P., Prior, K., Cohen, R., Wade, C., Sewell, J., Fouche, Y.,…& Scalea, T. M. (2011). Diagnosing mild traumatic brain injury: Where are we now? *Journal of* *Trauma, 70,* 554-559. DOI:10.97/ta.0B013e3 1820d1062. *\*\*\*Used in Evidence Statement.*
141. Eby, D. W., Molnar, L. J., & Shope, J. T. (2010) Driving decisions workbook. *Social and Behavioral Analysis Division. University of Michigan. Transportation Research Institute.* Retrieved October 2012, from www.umtri.umich.edu/library/pdf/2000-14.pdf.
142. Eicher, V., Murphy, M. P., Murphy, T. F., & Malec, J. F. (2012). Progress assessed with the Mayo-Portland adaptability inventory in 604 participants in 4 types of post-inpatient rehabilitation brain injury programs. *Arch Phys Med Rehabil, 93,* 100-107. DOI: 10.1016/j.apmr.2011.06.038.
143. El-Kashlan, H. K., Shepard, N. T., Asher, A. M., Smith-Wheelock, M., & Telian, S. A. (1998). Evaluation of clinical measures of equilibrium. *The Laryngoscope, 108,* 311-319. <http://www.laryngoscope.com>
144. Elkind, A. H., O’Carroll, P., Blumenfeld, A., DeGryse, R., & Dimitrova, Rozalina. (2006). A series of three sequential, randomized, controlled studies of repeated treatments with botulinum toxin type A for migraine prophylaxis. *Journal of Pain, 7(10),* 688-696.
145. Ellemberg, D., Henry, L. C., Macciocchi, K. M., & Broglio, S. P. (2009). Advances in sport concussion assessment: From behavioral to brain imaging measures. *Journal of Neurotrauma, 26,* 2365-2382. DOI: 10.1089/neu.2009.0906.
146. Engberg, A. W., Liebach, A., & Nordenbo, A. (2006). Centralized rehabilitation after severe traumatic brain injury – a population-based study. *Acta Neurol Scand, 113,* 178–184. DOI: 10.1111/j.1600-0404.2005.00570.x.
147. English, C., & Hillier, S. L. (2010). Circuit class therapy for improving mobility after stroke. *Cochrane Database of Systematic Reviews, 7,* 1-29. DOI: 10.1002/14651858.CD007513.pub2.
148. Erickson, K. I., Voss, M. W., Shaurya Prakash, R., Basake, C., Szabo, A., Chaddock, L., …Kramer, A. F., (2011). Exercise training increases size of hippocampus and improves memory. *PNAS, 108(7),* 3017-3022. DOI: 10.1073/pnas.1015950108.
149. Evans, M. K., & Krebs, D. E. (1999). Posturography does not test vestibulospinal function. *Otolaryngology Head Neck Surgery, 120,*1640173.
150. Evers, S., Afra, J., Frese, A., Goadsby, P.,. . . Sandor, P. S. (2009). EFNS guideline on the drug treatment of migraine – revised report of an EFNS task force. *European Journal of Neurology, 16,* 968-981 DOI:10.1111/j.1468-1331.2009.02748. x
151. Evers, S., Goadsby, P., Jensen, R., May, A., Pascual, J., & Sixt, G. (2011). Treatment of miscellaneous idiopathic headache disorders (group 4 of the HIS classification) – Report of an EFNS task force. *European Journal of Neurology, 18,* 803-812. DOI:1111/j.1468-1331.2011.03389. x.
152. Evers, S., Goadsby, P., Jensen, R., May, A., Pascual, J., & Sixt, G. (2012). Treatment of miscellaneous idiopathic headache disorders. *European Handbook of Neurological Management, 2, second edition,* 321-335.
153. Fann, J. R., Hart, T., & Schomer, K. G. (2009). Treatment for depression after traumatic brain injury: A systematic review. *Journal of Neurotrauma, 26,* 2383-2402. DOI: 10.1089/neu.2009.1091
154. Ferris, L. T., Williams, J. S., & Shen, C. –L. (2007). The Effect of acute exercise on serum brain-derived neurotrophic factor levels and cognitive function. *Journal of the American College of Sports Medicine,* 728-734. DOI: 10.1249/mss.0b013e31802f04c7.
155. Fife, T. D., Iverson, D. J., Lempert, T., et. al (2008). Practice parameter: Thereapies for benign paroxysmal positional vertigo (an evidence-based review): Report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology, 70,* 2067. DOI 10l.0000313378.77444. ac.
156. Fife, T. D., Tusa, R. J., Furman, J. M., Zee, D. S., Frohman, E., Baloh, R. W., …Eviatar, L. (2000). Assessment: Vestibular testing techniques in adults and children: Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology, 55,* 1-12. <http://www.neurology.org/content/55/10/1431.full.html>
157. Finnoff, J. T., Jelsing, E. J., & Smith, J. (2011). Biomarkers, genetics, and risk factors for concussion. *American Academy of Physical Medicine and Rehabilitation, 3,* S452-S459. DOI: 10.1016/j. pmrj.2011.07.014.
158. Flint, P. W., Haughey, B. H., Lund, V. J., Niparko, J. K., Richardson, M. A., Robbins, K. T., & Thomas, J. R. (2010). Surgery for vestibular disorders. In S. A. Telian (Ed.), *Cummings Otolaryngology: Head & Neck Surgery, 5th edition,* (ch 176). Retrieved from <http://www.mdconsult.com>
159. Ford-Smith, C. D., Wyman, J. F., Elswick, Jr., R. K., Frenandez, T., & Newton, R. A. (1995). Test-retest reliability of the sensory qrganization test in noninstitutionalized older adults. *Archives of Physical Medicine and Rehabilitation, 76,* 76-81.
160. Forsyth, R. J., Jayamoni, B., Paine, T. C., Mascarenas, S. (2006). Monoaminergic agonists for acute traumatic brain injury (Review). *Cochrane Database of Systematic Reviews, 4,* 1-17. <http://www.thecochranelibrary.com>
161. Forsyth, R. J., Wolny, S., & Rodrigues, B. (2010), Routine intracranial pressure monitoring in acute coma. *Cochrane Database of Systematic Reviews, 2,* 1-13. DOI: 10.1002/14651858.CD002043.pub2.
162. Freed, S., & Hellerstein, L. F. (1997). Visual electrodiagnostic findings in mild traumatic brain injury. *Brain Injury, 11(1),* 25-36.
163. French, B., Thomas, L. H., Leathley, M. J., Sutton, C. J., McAdam, J., Forster, A.,… Watkins, C. L. (2007). Repetitive task training for improving functional ability after stroke. *Cochrane Database of Systematic Reviews, 4,*  1-77. DOI: 10.1002/14651858.CD006073.pub2.
164. Frey, W. F., Savage, R. C., & O’Shanick, G. (2009). The road to rehabilitation part 8: Journey toward understanding: Concussion & mild brain injury. *Brain Injury Association of America.The Voice of Brain Injury.* <http://www.biausa.org>
165. Furman, J. M. (1994). Posturography: Uses and limitations. *Bailli*ẻ*re’s Clinical Neurology, 3,* 501-513. ISBN 0-7020-1951-8.
166. Furman, J. M. (1995). Role of posturography in the management of vestibular patients. O*tolaryngology Head Neck Surgery, 112,* 8-15.
167. Gallagher, C. N., Hutchinson, P. J., & Pickard, J. D. (2007). Neuroimaging in trauma. *Curr Opin Neurol, 20,* 403-409. [galclare@gmail.com](mailto:galclare@gmail.com)
168. Garcia, A. N., Shah, M. A., Dixon, C. E., Wagner, A. K., & Kline, A. E. (2011). Biologic and plastic effects of experimental traumatic brain injury treatment paradigms and their relevance to clinical rehabilitation. *Physical Medicine and Rehabiliation, 3(6 Suppl),* S18–S27. DOI:10.1016/j.pmrj.2011.03.017.
169. Gates, G. A., Green, J. D., Tucci, D. L., & Telian, S. A. (2004). The effects of transtympanic micropressure treatment in people with unilateral meniere’s disease. *Arch Otolaryngol Head Neck Surg.,130,* 718-725. <http://www.archoto.com>. *\*\*\*Used in Evidence Statement*
170. Gates, G. A., Verrall, A., Green, Jr, J. D., Tucci, D. L., & Telian, S. A. (2006). Meniett clinical trial: Long-term follow-up. *Arch Otolaryngol Head Neck Surg., 132,* 1311-1316. <http://www.archoto.com>
171. Gavett, B. E., Cantua, R. C., Shentonb, M., Linb, A. P., Nowinskia, C. J., McKeea, A., C., & Sterna, R. A. (2011). Clinical appraisal of chronic traumatic encephalopathy: current perspectives and future directions. *Current Opinion in Neurology, 24,* 525–531. DOI:10.1097/WCO.0b013e32834cd477
172. Gennarelli, T. A., & Graham, D. I. (1998). Neuropathology of the head injuries. *Seminars in Clinical Neuropsychiatry, 3,* 160-175.
173. Ghaffar, O., McCullagh, S., Ouchterlony, D., & Feinstein, A. (2006). Randomized treatment trial in mild traumatic brain injury. *Journal of Psychosomatic Research, 61,* 153–160. DOI:10.1016/j.jpsychores.2005.07.018. *\*\*\*Used in Evidence Statement.*
174. Ghigo, E., Masel, B., Aimaretti, G., Leon-Carrion, J., Casaneuva, F. F., Dominguez-Morales, M. R.,… Urban, R. (2005). Consensus guidelines on screening for hypopituitarism following traumatic brain injury. *Brain Injury, 19(9),* 711-724. DOI: 10.1080/02699050400025315.
175. Giacino, J. T., Whyte, J., Bagiella, E., Kalmar, K., Childs, N., Khademi, A.,… Sherer, M. (2012a). Placebo-controlled trial of amantadine for severe traumatic brain injury. *The New England Journal of Medicine, 366(9),* 819-826. <http://www.nejm.org>. *\*\*\*Used in Evidence Statement.*
176. Giacino, J. T., Whyte, J., Bagiella, E., Kalmar, K., Childs, N., Khademi, A.,… Sherer, M. (2012b). Protocol for: Placebo-controlled trial of amantadine for severe traumatic brain injury. *The New England Journal of Medicine, 366(9),* 1-70. <http://www.nejm.org>. *\*\*\*Used in Evidence Statement.*
177. Giacino, J. T., Whyte, J., Bagiella, E., Kalmar, K., Childs, N., Khademi, A.,… Sherer, M. (2012c). Supplementary material for: Placebo controlled trial of amantadine for traumatic brain injury (MS# 11-ｭ‐02609.R1). *The New England Journal of Medicine, 366(9),* 1-13. <http://www.nejm.org>. *\*\*\*Used in Evidence Statement.*
178. Gianoli, G., Mcwilliams, S., Soileau, J., & Belafsky, (2000). Posturographic performance in patients with the potential for secondary gain. *Otolaryngology -- Head and Neck Surgery, 122,* 11-18. DOI:10.1016/SO194-5998(00)70137-9.
179. Gilman, S. (2002). Joint position sense and vibration sense: anatomical organization and assessment*. Journal of Neurol Neurosurg Psychiatry, 72,* 473-477. <http://www.jnnp.com>
180. Giza, C. C., Griesbach, G. S., Hovda, D. A. (2005). Experience-dependent behavioral plasticity is disturbed following traumatic injury to the immature brain. *Behavioural Brain Research, 157,* 11–22. DOI:10.1016/j.bbr.2004.06.003.
181. Goebel, J. A., Sataloff, R., Hanson, J. M., Nashner, L. M.,…Sokolow, C. (1997). Posturographic evidence of nonorganic sway patterns in normal subjects, patients, and suspected malingerers. *Otolaryngology-Head and Neck Surgery, 117(4)*, 293-302.
182. Gonzalez, P. G., & Walker, M. T. (2011). Imaging modalities in mild traumatic brain injury and sports concussion. *American Academy of Physical Medicine and Rehabilitation, 3,* S413-S424. DOI: 10.1016/j. pmrj.2011.08.536.
183. Gonzalez-Frenandez, M., Gil-Gomez, J., Alcaniz, M., Noe, E. & Colomer, C. (2010). eBaViR, easy balance virtual rehabilitation system: a Study with patients. *Annual Review of* *Cybertherapy and Telemedicine,*61-66*.* DOI:10.3233/978-1-60750-561-7-61.
184. Gooch, M. R., Gin, G. E., Kenning, T. J., & German, J. W. (2009). Complications of cranioplasty following decompressive craniectomy: analysis of 62 cases. *Journal of Neurosurgery, 26(6),* E9. DOI: 10.3171/2009.3. FOCUS0962.
185. Gordon, W.A., Zafonte, R., Cicerone, K., Cantor, J., Brown, M., Lombard, L.,… Chandna, T., (2006). Traumatic brain injury rehabilitation: State of the science. *American Journal of Physical Medice and Rehabilitation, 85,* 343-382. DOI: 10.1097/01.phm.0000202106.01654.61.
186. Gottshall, K. (2011). Vestibular rehabilitation after mild traumatic brain injury with vestibular pathology. *NeuroRehabilitation, 29,* 167-171. DOI: 10.3233/NRE-2011-0691.
187. Gowda, N. K., Agrawal, D., Bal, C., Chandrashekar, N., Tripati, M., Bandopadhyaya, G. P.,… Mahapatra, A. K. (2006). Technetium Tc-99m ethyl cysteinate dimer brain single-photon emission CT in mild traumatic brain injury: A prospective study. *American Journal of Neuroradiology 27,* 447– 451. <http://www.ajnr.org>
188. Granacher, R. P. Jr. (2008). Commentary: Applications of functional neuroimaging to civil litigation of mild traumatic brain injury. *Journal American Academy of Psychiatry Law, 36(3),* 323-328.
189. Gray, J. M., Robertson, I., Pentland, B., & Anderson, S. (1992). Microcomputer-based attentional retraining after brain damage: A randomized group controlled trial. *Neuropsychological Rehabilitation, 2(2),* 97-115.
190. Green, W., Ciuffreda, K. J., Thiagarajan, P., Optom, B. S.,…Kapoor, N. (2010a). Accommodation in mild traumatic brain injury. *Journal of Rehabilitation Research & Development, 47(3),* 183-200.
191. Green, W., Ciuffreda, K. J., Thiagarajan, P., Optom, B. S.,. . . Kapoor, N. (2010b). Static and dynamic aspects of accommodation in mild traumatic brain injury: A review*. Optometry, 81,* 129-136. DOI:10.1016/j. optm.2009.07.015.
192. Greener, J., Enderby, P., & Whurr, R. (2010). Pharmacological treatment for aphasia following stroke (Review). *Cochrane Database of Systematic Reviews, 5,* 1-59. DOI: 10.1002/14651858.CD000424. <http://www.thecochranelibrary.com>
193. Griesbach, G. S. (2011a). Exercise after traumatic brain injury: Is it a double-edged sword? *Physical Medicine and Rehabilitation, 3,* S64-S72. DOI: 10.1016/j.pmrj.2011.02.008
194. Griesbach, G. S., Gomez-Pinilla, F., & Hovda, D. A. (2004a). The upregulation of plasticity-related proteins following TBI is disrupted with acute voluntary exercise. *Brain Research, 1016,* 154–162. DOI:10.1016/j.brainres.2004.04.079
195. Griesbach, G. S., Gomez-Pinilla, F., & Hovda, D. A. (2007). Time window for voluntary exercise–induced increases in hippocampal neuroplasticity molecules after traumatic brain injury is severity dependent. *Journal of Neurotrauma, 24(7),* 1161-1171. DOI: 10.1089/neu.2006.0255.
196. Griesbach, G. S., Hovda, D. A., & Gomez-Pinilla, F. (2009). Exercise-induced improvement in cognitive performance after traumatic brain-injury in rats is dependent on BDNF Activation. *Brain Research*, *1288*, 105–115. DOI:10.1016/j.brainres.2009.06.045.
197. Griesbach, G. S., Hovda, D. A., Molteni, R., Wu, A., & Gomez-Pinilla, F. (2004b). Voluntary exercise following traumatic brain injury: Brain- derived neurotrophic factor upregulation and recovery of function. *Neuroscience,125,* 129–139. DOI: 10.1016/j.neuroscience.2004.01.030
198. Griesbach, G. S., Hovda, D. A., Tio, D. L., & Taylor, A. N. (2011b). Heightening of the stress response during the first weeks after a mild traumatic brain injury*. Neuroscience, 178,*147–158. DOI:10.1016/j.neuroscience.2011.01.028.
199. Griesbach, G. S., Tio, D. L., Vincelli, J., McArthur, D. L., & Taylor, A. N. (2012). Differential effects of voluntary and forced exercise after traumatic brain injury on stress responses. *Journal of Neurotrauma,* 1-33. DOI: 10.1089/neu.2011.2229.
200. Grossmann, W., & Schmidramsl, H. (2001). An extract of petasites hybridus is effect in the prophylaxis of migraine. *Alternative Medicine Review, 6(3),* 303-310. *\*\*\*Used in Evidence Statement*
201. Guresir, E., Schuss, P., Vatter, H., Raabe, A., Seifert, V., & Beck, J. (2009). Decompressive craniectomy in subarachnoid hemorrhage. *Journal of Neurosurgery, 26(6),* E4. DOI: 10.3171/2009.3. FOCUS0954.
202. Gurkov, R., Filipe Mingas, L. B. Rader, T., Louza, J., Olzowy, B., & Krause, E. (2012). Effect of transtympanic low-pressure therapy in patients with unilateral Menière’s disease unresponsive to betahistine: A randomised, placebo-controlled, double-blinded, clinical trial. *The Journal of Laryngology & Otology*, 1-7. DOI:10.1017/S0022215112000102. *\*\*\*Used in Evidence Statement*
203. Guskiewicz, K. M., Bruce, S. L., Cantu, R. C., Ferrara, M. S., Kelly, J. P., McCrea, M.,… Valovich McLeod, T. C. (2006). Research based recommendations on management of sport related concussion: summary of the National Athletic Trainers’ Association position statement. *British Journal of Sports Medicine,* 6-10. <http://www.bjsportmed.com>
204. Guskiewicz, K. M., Perrin, D. H., & Gansneder, B. M. (1996). Effect of Mild Head Injury on Postural Stability in Athletes. *Journal of Athletic Training, 31(4),* 300-306.
205. Guskiewicz, K. M., & Register-Mihalik, J. K. (2011). Postconcussive Impairment Differences Across a Multifaceted Concussion Assessment Protocol. *American Academy of Physical Medicine and Rehabilitation, 3,* S445-S451. DOI: 10.1016/j. pmrj.2011.08.009.
206. Hackett, M. L., Anderson, C. S., House, A., & Halteh, C. (2008). Interventions for preventing depression after stroke. *Cochrane Database of Systematic Reviews, 3,* 1-79. DOI: 10.1002/14651858.CD003689.pub3.
207. Hall, K. M., Hamilton, B. B., Gordon, W. A., & Zasler, N. D. (1993). Characteristics and comparisons of functional assessment indices: Disability Rating Scale, Functional Independence Measure, and Functional Assessment Measure. *Journal Head Trauma Rehabilitation, 8(2),* 60-72.
208. Harrington, A. L., Blount, P. J., & Bockenek, W. L. (2008). Heterotopic ossification. In W. R. Frontera, J. K. Silver, & T. D. Risso Jr. (Ed.), *Essentials of Physical Medicine and Rehibilitation Musculoskeletal Disorders, Pain, and Rehabilitation, 2nd edition,* (ch 123, 691-695).
209. Harwood, M., Weatherall, M., Talemaitoga, A., Barber, P. A.,. . . McNaughton, H. (2011). Taking charge after stroke: promoting self-directed rehabilitation to improve quality of life – a randomized controlled trial. *Clinical Rehabilitation, 11,* 1-9 DOI: 10.1177/0269215511426017. <http://cre.sagepu.com/content/early2011/11/11/0269215511426017>
210. Hassett, L., Moseley, A. M., Tate, R., Harmer, A. R. (2008). Fitness training for cardiorespiratory conditioning after traumatic brain injury (Review). *Cochrane Database of Systematic Reviews, 2,* 1-52. DOI:10.1002/14651858.CD006123.pub2.
211. Hassett, L. M., Moseley, A. M., Tate, R. L., Harmer, A. R., Fairbairn, T. J., & Leung, J. (2009). Efficacy of a fitness centre-based exercise programme compared with a home-based exercise programme in traumatic brain injury: A randomized controlled trial. *Journal of Rehabilitation Medicine, 41,* 247-255. DOI: 10.2340/16501977-0316.
212. Headache Classification Committee: Olesen, J., Bousser, M. –G., Diener, H. –C., Dodick, D., First, M., Goadsby, P. J.,… Steiner, T. J. (2006) New appendix criteria open for a broader concept of chronic migraine. *Cephalalgia, 26,* 742–746.
213. Headache Classification Subcommittee of the International Headache Society. (2004). International Classification of Headache Disorders: 2nd Edition. *Cephalalgia, 24(Supp.1),* 9-160.
214. Hendrick, R. E., Dodd III, G. D., Fullerton, G. D., Hendee, W. R., Borgstede, J. P., & Larke, F. (2011). The University of Colorado radiology adult dose-risk smartcard. *Journal of the American College of Radiology,* 290-292. DOI: 10.1016/j.jacr.2011.12.034.
215. Herring, S. A., Cantu, R. C., Guskiewicz, K. M., Putukian, M., & Kibler, W. B. (2011). Concussion (mild traumatic brain injury) and the Team Physician: A consensus statement—2011 Update. *Official Journal of the American College of Sports Medicine,* 2412*-2*422. DOI: 10.1249/MSS.0b013e3182342e64.
216. Hillier, S. L., & McDonnell, M., (2011). Vestibular rehabilitation for unilateral peripheral vestibular dysfunction (Review). *The Cochrane Database of Systematic Reviews, 2,* 1-72. DOI: 10.1002/14651858.CD005397.pub3. *\*\*\*Used in Evidence Statement*
217. Hobson, J., Chisholm, E., & El Refaie, A. (2010). Sound therapy (masking) in the management of tinnitus in adults. *Cochrane Database of Systematic Reviews, 12,* 1-24. DOI: 10.1002/14651858.CD006371.pub2.
218. Hoffman, J. M., Bell, K. R., Powell, J. M., Behr, J., Dunn, E. C., Dikmen, S., & Bombardier, C. (2010). A randomized controlled trial of exercise to improve mood after traumatic brain injury. *Physical Medicine and Rehabilitation, 2,* 911-919. DOI: 10.1016/j.pmrj.2010.06.008.
219. Hoffman, J. M., Dikmen, S., Temkin, N., & Bell, K. R. (2012). Development of posttraumatic stress disorder after mild traumatic brain injury. *Arch Phys Med Rehabil, 93,* 287-292. DOI: 10.1016/j.apmr.2011.08.041.
220. Hoffmann, T., Bennett, S., Koh, C. L., & McKenna, K. T. (2010). Occupational therapy for cognitive impairment in stroke patients. *Cochrane Database of Systematic Reviews, 9,* 1-18. DOI: 10.1002/14651858.CD006430.pub2.
221. Holland, S., Silberstein, S. D., Freitag, F., Dodick, D. W., Argoff, C., & Ashman, E. (2012). Evidence-based guideline update: NSAIDs and other complementary treatments for episodic migraine prevention in adults : Report of the Quality Standards Subcommittee of the American Academy of Neurology and the American Headache Society. *Neurology,* 78, 1346-1354. DOI 10.1212/WNL.0b013e3182535d0c.
222. Honaker, J. A., Converse C. M., & Shepard, N. T. (2009). Modified head shake computerized dynamic posturography. *American Journal of Audiology, 18,* 108-113. DOI:10.1044/1059-0889(2009/09-0012).
223. Hoofien, D., Gilboa, A., Vakil, E., & Donovick, P. J. (2001). Traumatic brain injury (TBI) 10-20 years later: a comprehensive outcome study of psychiatric symptomatology, cognitive abilities and psychosocial functioning. *Brain Injury,15(3),* 189-209.
224. Hornstein, A. (2011). Social Aspects. *American Psychiatric Association, 33,* 1-8. DOI: 10.1176/appl. books.97815856224201.687604. <http://www.psychiatryonline.com>
225. Horak, F. B., Jones-Ryce C., Black, O., & Shumway-Cook, A. (1992). Effects of vestibular rehabilitation on dizziness and imbalance. *Otolaryngol Head Neck Surgery, 106(2), 175-180*.
226. Hou, R., Moss-Morris, R., Peveler, R., Mogg, K, Bradley, P. B., & Belli, A. (2012). When a minor head injury results in enduring symptoms: a prospective investigation of risk factors for postconcussional syndrome after mild traumatic brain injury. *J Neurol Neurosurg Psychiatry, 83,* 217-223. DOI:10.1136/jnnp-2011-300767.
227. Hudak, A. M., Hynan, L. S., Harper, C. R., & Diaz-Arrastia, R. (2012). Association of depressive symptoms with functional outcome after traumatic brain injury. *Journal of Head Trauma and Rehabilitation, 27(2),* 87-98. DOI: 10.1097/HTR.0b013e3182114efd.
228. Hurkmans, J., de Bruijn, M., Boonstra, A. M., Jonkers, R., Bastiaanse, R., Arendzen, H., & Reinders-Messelink, H. A. (2012). Music in the treatment of neurological language and speech disorders: A systematic review, *Aphasiology, 26(1),* 1-19. DOI: 10.1080/02687038.2011.602514.
229. Hutchinson, J. S., Ward, R. E., Lacroix, J., Hebert, P. C., Barnes, M. A., Bohn, D. J.,… Skippen, P. W. (2008). Hypothermia Therapy after Traumatic Brain Injury in Children. *The New England Journal of Medicine,358,* 2447-2456. <http://www.nejm.org>
230. Hutchinson, P. J., Timofeev, I., Kolias, A. G., Corteen, E. A., Czosnyka, M., Menon, D. K.,… Kirkpatrick, P. J. (2011). Decompressive craniectomy for traumatic brain injury: The jury is still out. *British Journal of Neurosurgery* – Letter to the Editor, *25(3),* 441-442.
231. Intiso, D., Santilli, V, Grasso, M. G., Rossi, R., & Caruso, I. (1994). Rehabilitation of walking with electromyographic biofeedback in foot-drop after stroke. *Stroke, 25(6),* 1189-1192.
232. Iverson, D. J., Gronseth, G. S., Reger, M. A., Classen, S., Dubinsky, R. M., & Rizzo, M. (2010). Practice Parameter update: Evaluation and management of driving risk in dementia. *American Academy of Neurology*, Abstract, 1316. <http://www.neurology.org>
233. Iverson, G. L. (2006). Complicated vs uncomplicated mild traumatic brain injury: Acute neuropsychological outcome. *Brain Injury, 20(13–14),* 1335–1344. DOI: 10.1080/02699050601082156.
234. Jackson, J. L., Kuriyama, A., & Hayashino, Y. (2012a) Botulinum toxin A for prophylactic treatment of migraine and tension headaches in adults: a meta-analysis. *Journal of the American Medical Association*, *307(16),* 1736-1745.
235. Jackson, J. L., Kuriyama, A., & Hayashino, Y. (2012b) Supplementary online content for: Botulinum toxin A for prophylactic treatment of migraine and tension headaches in adults: a meta-analysis. *Journal of the American Medical Association*, *307(16),* 1736-1745.
236. Jacobs, A., Put, E., Ingels, M., Put, T., & Bossuyt, A. (1996). One-year follow-up of Technetium-99m-HMPAO SPECT in mild head injury. *J Nucl Medicine, 37,* 1605-1609. *\*\*\*Used in Evidence Statement.*
237. Jakal, A., & von Hauenschild, P. (2011). Therapeutic effects of cranial osteopathic manipulative medicine: A systematic review. *J Am Osteopath Assoc., 111(12),* 685-693.
238. Jha, A., Weintraub, A., Allshouse, A., Morey, C., Cusick, C., Kittelson, J., … Gerber, D. (2008). A randomized trial of Modafinil for the treatment of fatigue and excessive daytime sleepiness in individuals with chronic traumatic brain injury. *Journal of Head Trauma Rehabilitation, 23(1),* 52–63. <http://www.headtraumarehab.com>
239. Jiang, J.-Y. (2009). Clinical study of mild hypothermia treatment for severe traumatic brain injury. *Journal of Neurotrauma, 26,* 399–406. DOI: 10.1089/neu.2008.0525.
240. Jiang, J.-Y., Xu, W., Li, W.-P., Gao, G.-Y., Boa, Y.-H., Liang, Y.-M., & Luo, Q.-Z. (2006). Effect of long-term mild hypothermia or short-term mild hypothermia on outcome of patients with severe traumatic brain injury. *Journal of Cerebral Blood Flow & Metabolism, 26,* 771–776. <http://www.jcbfm.com>
241. Johnson, E. W., Lovell, M. R. (2011). Neuropsychological assessment. *American Psychiatric Association, 8,* 1-25. DOI: 10.1176/appi. books.9781585624201.674504. <http://www.psychiatryonline.com>
242. Junpeng M., Huang S., & Qin S. (2011). Progesterone for acute traumatic brain injury (Review). *Cochrane Database of Systematic Reviews, 1,* 1-25. DOI: 10.1002/14651858.CD008409.pub2. <http://www.thecochranelibrary.com>. *\*\*\*Used in Evidence Statement.*
243. Kaiser, P. R., Valko, P. O., Werth, E., Thomann, J., Meier, J., Stocker, R.,… Baumann, C. R. (2010). Modafinil ameliorates excessive daytime sleepiness after traumatic brain injury. *Neurology,75,* 1780-1786. DOI 10.1212/WNL.0b013e3181fd62a2.
244. Kakar, V., Nagaria, J., & Kirkpatrick, P. J. (2009). The current status of decompressive craniectomy. *British Journal of Neurosurgery, 23(2),* 147-157.
245. Kapoor, N., Ciuffreda, K. J., & Han, Y. (2004). Oculomotor rehabilitation in acquired brain injury: A case series. *Archives Physical Medicine and Rehabilitation, 85,* 1667-1678. DOI:10.1016/j. apmr.2003.12.044.
246. Karlin, A. M. (2011). Concussion in the pediatric and adolescent population: “Different population, different concerns”. *American Academy of Physical Medicine and Rehabilitation, 3,* S369-S379. DOI: 10.1016/j. pmrj.2011.07.015.
247. Karnath, H-O., Dieterich, M. (2006). Spatial neglect – a vestibular disorder? *Brain, 129,* 293-305. DOI:10.1093/brain/awh698.
248. Kaufman, K. R., Brey, R. H., Chou, L-S., Rabatin, A.,…Basford, J. R. (2006). Comparison of subjective and objective measurements of balance disorders following traumatic brain injury. *Medical Engineering and Physic, 28,* 234-239. DOI:10.1016/j. medengphy.2005.05.005.
249. Kelly, H., Brady, M. C., & Enderby, P. (2010) Speech and language therapy for aphasia following stroke. *Cochrane Database of Systematic Reviews, 5,* 1-172. DOI: 10.1002/14651858.CD000425.pub2. <http://www.thecochranelibrary.com>
250. Kenning, T. J., Gandhi, R. H., & German, J. W. (2009). A comparison of hinge craniotomy and decompressive craniectomy for the treatment of malignant intracranial hypertension: early clinical and radiographic analysis. *Journal of Neurosurgery, 26(6),* E6. DOI: 10.3171/2009.4. FOCUS0960.
251. Ker, K., & Blackhall, K. (2008). Bradykinin beta-2 receptor antagonists for acute traumatic brain injury (Review). *The Cochrane Database of Systematic Reviews, 1,* 1-22. DOI: 10.1002/14651858.CD006686.pub2.
252. Kerr, A., Cheng, S.-Y., & Jones, T.A., (2011). Experience-dependent neural plasticity in the adult damaged brain*. Journal of Communication Disorders, 44,* 538–548. DOI:10.1016/j.jcomdis.2011.04.011
253. Khan, S., Khan, A., & Feyz, M. (2002). Decreased length of stay, cost savings and descriptive findings of enhanced patient care resulting from an integrated traumatic brain injury programme. *Brain Injury, 16(6),* 537-554.
254. Kilani R. K., Paxton, B. E., Stinnett, S. S., Barnhart, H. X., Bindal, V., & Lungren, M. P. (2011). Self-referral in medical imaging: A meta-analysis of the literature. *American College of* *Radiology, 8,* 469-476. DOI:10.1016/J. Jacr.2011.01.016.
255. Kim, Y.-H., Ko, M.-H., Na, S.-Y., Park, S.-H., & Kim, K.-W. (2006). Effects of single-dose methylphenidate on cognitive performance in patients with traumatic brain injury: a double-blind placebo-controlled study*. Clinical Rehabilitation, 20,* 24-30. DOI: 10.1191/0269215506cr927oa.
256. Kirthi, V., Derry S., Moore R. A., & McQuay, H. J., (2010). Aspirin with or without an antiemeticfor acute migraine headaches in adults*. Cochrane Database of Systematic Reviews, 4,* 1-77. DOI:10.1002/14651858. CD008041. pub2. *\*\*\*Used in Evidence Statement.*
257. Kleffelgaard, I., Roe, C., Soberg, H. L., & Bergland, A. (2012). Associations among self-reported balance problems, post-concussion symptoms and performance-based tests: a longitudinal follow-up study. *Disability & Rehabilitation, 34(9),* 788-794. DOI: 10.3109/09638288.2011.619624.
258. Kleim, J. A. (2011). Neural plasticity and neurorehabilitation: Teaching the new brain old tricks. *Journal of Communication Disorders, 44,* 521–528. DOI:10.1016/j.jcomdis.2011.04.006.
259. Klonoff, P. S., Lamb, D. G., & Henderson, S. W. (2001). Outcomes from milieu-based neurorehabilitation at up to 11 years post-discharge. *Brain Injury, 15(5),* 413-428.
260. Knaepen, K., Goekint, M., Heyman, E. M., & Meeusen, R. (2010). Neuroplasticity - exercise-induced response of peripheral brain-derived neurotrophic factor A systematic review of experimental studies in human subjects. *Sports Medicine, 40(9),* 766-801.
261. Kochanek, P. M., & Safar, P. J. (2003). Therapeutic hypothermia for severe traumatic brain injury. *Journal of the American Medical Association, 289(22),* 3007-3009.
262. Koeler, R., Wilhelm, E., & Shoulson, I. (2011). Cognitive rehabilitation therapy for traumatic brain injury; Evaluating the evidence. *National Academy of Sciences, 280. ISBN 978-0-309-218184*. <http://www.nap.edu/catalog.php?record_id=13220>
263. Kornbluth, J. & Bhardwaaj, A. (2011). Evaluation of coma: A critical appraisal of popular scoring systems. *Neurocrit Care, 14,* 134-143. DOI:10.1007/S12028-010-9409-3.
264. Kou, Z.,Wu, Z., Tong, K. A., Holshouser, B., Benson, R. R., Hu, J., & Haacke, M. (2010). The role of advanced MR Imaging findings as biomarkers of traumatic brain injury. *Journal of Head Trauma Rehabilitation, 25(4),* 267-282.
265. Kramer, A. F., Hahn, S., Cohen, N. J., Banich, M. T., McAuley, E., Harrison, C. R.,… Colcombe, A. (1999). Ageing, fitness and neurocognitive function. *Nature, 400,* 418-419.
266. Kraus, M. F., Susmaras, T., Caughlin, B. P., Walker, C. J., Sweeney, J. A., & Little, D. M. (2007). White matter integrity and cognition in chronic traumatic brain injury: a diffusion tension imaging study. *Brain, 130,* 2508-2519. DOI:10.1093/brain/awm216.
267. Krawczyk, D. (n.d.). Brain training to enhance frontal lobe reasoning in soldiers with TBI. *Technical Abstract.* Proposal Number: PT100084 University of Texas.
268. Krishnan, A. & Silver, N. (2009). Headache (chronic tension-type). *BMJ Publishing Group Ltd.; Clinical Evidence, 7,* 1-22. <http://www.clinicalevidence.bmj.com>
269. Kulcu, D. G., Yanik, B., Boynukalin, S., & Kurtais, Y. (2008). Efficacy of a home-based exercise program on benign paroxysmal positional vertigo compared with betahistine. *Journal of Otolaryngology Head and Neck Surgery, 37(3),* 373-379. DOI: 10.2310/7070.2008.0063.
270. Kumar, S. K, & Macaden A. S. (2009). Cognitive rehabilitation for occupational outcomes after traumatic brain injury (Protocol). *Cochrane Database of Systematic Reviews, 3,* 1-7. <http://www.thecochranelibrary.com>
271. Laatsch, L. (2007). The use of functional MRI in traumatic brain injury diagnosis and treatment. *Physical Medicine and Rehabilitation Clinics of North America, 18,* 69-85. DOI:10.1016/j. pmr.2006.11.00.
272. Laker, S. R. (2011a). Concussion supplement: Introduction. *American Academy of Physical Medicine and Rehabilitation, 3,* S351-S353. DOI: 10.1016/j. pmrj.2011.08.004.
273. Laker, S. R. (2011b). Concussion supplement: Epidemiology of concussion and mild traumatic brain injury. *American Academy of Physical Medicine and Rehabilitation 3:*S354-S358. DOI: 10.1016/j. pmrj.2011.07.017.
274. Lane-Brown, A., & Tate, R. (2009). Interventions for apathy after traumatic brain injury (Review). *The Cochrane Database of Systematic Reviews, 2,* 1-27. DOI: 10.1002/14651858.CD006341.pub2.
275. Lange, R. T., Iverson, G. L., & Franzen, M. D. (2009). Neuropsychological functioning following complicated vs. uncomplicated mail traumatic brain injury. *Brain Injury, 23(2),* 83-91. DOI:10.1080/02699050802635281.
276. Langevin P., Peloso P. M. J., Lowcock J., Nolan M., Weber, J., Gross, A.,… Haines, T. (2011). T. Botulinum toxin for subacute/chronic neck pain. *Cochrane Database of Systematic Reviews, 7,* 1-71. DOI:10.1002/14651858. CD008626pub2. *\*\*\*Used in Evidence Statement*
277. Langham, J., Goldfrad, L. J., Teasdale, C., Shaw, G., & Rowan K. (2003). Calcium channel blockers for acute traumatic brain injury (Review). *Cochrane Database of Systematic Reviews, 4,* 1-23. <http://www.thecochranelibrary.com>
278. Larrosa, F., Dura, M. J., Menacho, J., Gonza´lez-Sabate´, L., Cordo´n, A., Hernandez, A., & Garcı´a-Iba´n˜ez, L. (2012). Aphysiologic performance on dynamic posturography in workrelated patients. *European Archives of Otorhinolaryngol,* DOI 10.1007/s00405-012-1930-x.
279. Larson, E. B., & Zollman, F. S. (2010). The effect of sleep medications on cognitive recovery from traumatic brain injury. *Journal of Head Trauma Rehabilitation,*  *25(1)*, 61–67.
280. Laver, K. E., George, S., Thomas, S., Deutsch, J. E., & Crotty, M. (2011). Virtual reality for stroke rehabilitation. *Cochrane Database of Systematic Reviews, 9,* 1-70. DOI: 10.1002/14651858.CD008349.pub2.
281. Lavrich, J. B. (2010). Convergence insufficiency and its current treatment. *Current Opinion in Ophthalmology, 21,* 356-360. DOI:10.1097/ICU. ObO13e32833cf03a.
282. Lee, H., Kim, S. –W., Kim, J. –M., Shin, I. –S., Yang, S. –J., & Yoon, J. –S. (2005). Comparing effects of methylphenidate, sertraline and placebo on neuropsychiatric sequelae in patients with traumatic brain injury. *Human Psychopharmacol Clin Exp, 20,* 97–104. DOI: 10.1002/hup.668.
283. Lee, L. –C., Lieu, F. –K., Chen, Y. –H., Hung, T. –H., & Chen, S. –F. (2012). Tension pneumocephalus as a complication of hyperbaric oxygen therapy in a patient with chronic traumatic brain injury. *American Journal of Physical Medicine & Rehabilitation, 91(4),* 1-5. DOI: 10.1097/PHM.0b013e31824ad556.
284. Legg, L., Drummond, A., & Langhorne, P. (2006). Occupational therapy for patients with problems in activities of daily living after stroke. *Cochrane Database of Systematic Reviews, 4,* 1-45. DOI: 10.1002/14651858.CD003585.pub2. *\*\*\*Used in Evidence Statement*
285. Lehmann, J. F., Boswell, S., Price, R., Burleigh, A.,. . . Hertling, D. (1990). Quantitative evaluation of sway as an indicator of functional balance in post-traumatic brain injury. *Archives of physical medicine and rehabilitation, 71(12),* 955-962.
286. Leker, R. R. & Shohami, E. (2002). Cerebral ischemia and trauma – different etiologies yet similar mechanisms: neuroprotective opportunities. *Brain Research Reviews, 39,* 55-73.
287. Levin, M., & Ward, T. (2011). Headaches. *American Psychiatric Association, 21,* 1-8. DOI: 10.1176/appi. books.9781585624201.680643. <http://www.psychaitryonline.com>
288. Li, Y., Zheng, H., Witt, C. M., Roll, S., Yu, S. –G., Yan, J.,… Liang, F. –R. (2012). Acupuncture for migraine prophylaxis: a randomized controlled trial. *CMAJ, 184(4),* 401-410. DOI:10.1503/cmaj.110551.
289. Lincoln, N., Majid, M., & Weyman, N. (2000). Cognitive rehabilitation for attention deficits following stroke. *Cochrane Database of Systematic Reviews, 4,* 1-11. DOI: 10.1002/14651858.CD002842.
290. Linde, K., Allais, G., Brinkhaus, B., Manheimer, E., Vickers, A., & White, A. R. (2009). Acupuncture for migraine prophylaxis. *Cochrane Database of Systematic Review, 1,* 1-107. DOI:10.1002/14651858. CD001218. pub.2. *\*\*\*Used in Evidence Statement*
291. Linde, K., & Rossnagel, K. (2004). Propranolol for migraine prophylaxis (Review). *Cochrane Database of Systematic Reviews, 2,* 1-117. DOI: 10.1002/14651858.CD003225.pub2. *\*\*\*Used in Evidence Statement.*
292. Linde, K., Streng, A., Jürgens, S., Hoppe, A., Brinkhaus, B., Witt, C.,… Melchart, D. (2005). Acupuncture for patients with migraine a randomized controlled trial. *Journal of the American Medical Association, 293*, 2118-2125. http:// [www.jama.ama-assn.org](http://www.jama.ama-assn.org)
293. Linde, M., Hagen, K., Salvesen, O., Gravdahl, G. B., Helde, G., & Stovner, L., J. (2011). Onobotulinum toxin A treatment of cervicogenic headache: A randomized, double blind, placebo-controlled study. *Cephalalgia, 31,* 797-807. *\*\*\*Used in Evidence Statement*
294. Linscott, R. J, Knight, R. G., & Godfrey, H. P. D. (1996). The profile of functional impairment in communication (PFIC): A measure of communication impairment for clinical use. *Brain Injury, 10(6),* 397-412. <http://www.informahealthcare.com>
295. Lipp, M. & Longridge, N. S. (1994). Computerized dynamic posturography: Its place in the evaluation of patients with dizziness and imbalance. *Journal of Otolaryngology, 23(3),* 177-183.
296. Lipton, R. B., Göbel, H., Einhäupl, K. M., Wilks, K., & Mauskop, A. (2004). Petasites hybridus root (butterbur) is an effective preventive treatment for migraine. *Neurology, 63,* 2240-2245. DOI 10.1212/01.WNL.0000147290.68260.11. *\*\*\*Used in Evidence Statement*
297. Lloyd, F. J., Reyna, V. F. (2009). Clinical gist and medical education: Connecting the dots. *Journal of the American Medical Association, 302(12),* 1332-1333.
298. Loader, B., Gruther, W., Mueller, C. A., Neuwirth, G.,…Mittermaier, C. (2007). Improved postural control after computerized optokinetic therapy based on stochastic visual stimulation in patients with vestibular dysfunction. *Journal of Vestibular Research, 17,*131-136. ISBN 0957-4271/07.
299. Lojovich, J. (2010). The relationship between aerobic exercise and cognition: Is movement medicinal?. *Head Trauma Rehabilitation, 25(3),* 184-192.
300. Lombardi, F. F. L., Taricco, M., De Tanti, A., Telaro, E., & Liberati, A. (2002). Sensory stimulation for brain injured individuals in coma or vegetative state. *Cochrane Database of Systematic Reviews, 2,* 1-13. DOI: 10.1002/14651858.CD001427.
301. Longridge, N. S., & Mallinson, A. I. (2005a). “Across the Board” posturography abnormalities in vestibular injury. *Otology & Neurotology, 26,* 695-698.
302. Longridge, N. S., & Mallinson, A. I. (2005b). Visual vestibular mismatch in work-related vestibular injury. *Otology & Neurotology, 26,* 691-694.
303. Lucas, S. (2011). Headache manaagement in concussion and mild traumatic brain injury. *American Academy of Physical Medicine and Rehabilitation, 3,* S406-412. DOI: 10.1016/j. pmrj.2011.07.016.
304. Maas, A., & Stocchetti, N. (2011). Hypothermia and the complexity of trials in patients with traumatic brain injury. *The Lancet Neurology, 10,* 111-113. <http://www.thelancet.com/neurology>
305. Maas, A. I. R., Marmarou, A., Murray, G. D., Teasdale, Sir G. M., & Steyerberg, E. W. (2007). Prognosis and clinical trial design in traumatic brain injury: The IMPACT study*. Journal of Neurotrauma, 24(2),* 232-238. DOI:10.1089/neu.2006.0024.
306. Maas, A. I. R., Roozenbeel. B., & Manley, G. T. (2010a). Clinical trials in traumatic brain injury: Past experience and current developments. *The American Society for Experimental NeuroTherapeutics, Inc., 7,* 115-126.
307. Maas, A. I. R., Steyerberg, E. W., Marmarou, McHugh A., G. S., Lingsma, H. F., Butcher, I., & Murray, G. D. (2010b). IMPACT recommendations for improving the design and analysis of clinical trials in moderate to severe traumatic brain injury. *The American Society for Experimental NeuroTherapeutics, Inc., 7,* 127-134.
308. Maconochie, I., & Ross, M. (2010) Head injury (moderate to severe). *Clinical Evidence 6(1210),* 1-12.
309. Majerske, C. W., Mihalik, J. P., Ren, D., Collins, M. W.,…Wagner, A. K. (2008). Concussion in sports: Postconcussive activity levels, symptoms, and neurocognitive performance. *Journal of Athletic Training, 43(3),* 265-274. <http://www.nata.org/jat>
310. Makdissi, M., Darby, D., Maruff, P., Ugoni, A., Brukner, P. & McCrory, P. R. (2010). Natural history of concussion in sport: Markers of severity and implications for management. *The American Journal of Sports Medicine, 38,* 464-471, appendix. DOI: 10.1177/0363546509349491.
311. Malec, J. F. (2001). Impact of comprehensive day treatment on societal participation for persons with acquired brain injury. *Arch Phys Med Rehabil, 82,* 885-895. DOI: 10.1053/apmr.2001.23895.
312. Mallison, A. I., & Longridge, N. S. (2005). A new set of criteria for evaluating malingering in work-related vestibular injury. *Otology & Neurotology, 26(4),* 686-690.
313. Malykh, A. G., & Sadaie, M. R. (2010). Piracetam and piracetam-like drugs from basic science to novel clinical applications to CNS disorders. *Drugs, 70(3)* 287-312.
314. Manley, G. T. (2009). Introduction: Decompressive craniectomy for trauma and cerebrovascular disease. *Journal of Neurosurgery, 26(6),* E1. PMID: 19485713. DOI: 10.3171/2009.4. FOCUS. JUNE09. INTRO.
315. Marino, M., de Belvis, A., Basso, D., Avolio, M., Pelone, F., Tanzariello, M., & Ricciardi, W. (2012) Interventions to evaluate fitness to drive among people with chronic conditions: Systematic review of literature. *Accid. Anal. Prev.,* 1-20. DOI: 10.1016/j.aap.2012.05.010.
316. Marion, D. W. (2011). Decompressive craniectomy in diffuse traumatic brain injury. *The Lancet, 10,* 497-498.
317. Marshall, S., Teasell, R., Bayona, N., Lippert, C., Chundamala, J., Villamere, J., Mackie, D., Cullen, N., & Bayley, M. (2007). Motor impairment rehabilitation post acquired brain injury. *Brain Injury, 21,* 133-160. DOI: 10.1080/02699050701201383.
318. Martin, A. J., Marottoli, R., & O’Neill, D. (2009). Driving assessment for maintaining mobility and safety in drivers with dementia. *Cochrane Database of Systematic Reviews, 1,* 1-22. DOI: 10.1002/14651858.CD006222.pub2.
319. Marzo, S. J., Leonetti, J. P., Raffin, M. J., & Letarte, P. (2004). Diagnosis and management of post-traumatic vertigo. *Laryngoscope, 114,* 1720-1723.
320. Masel, B. E. (2012) *Brain Injury As A Chronic Disease*. Transitional Learning Center Galveston, Texas.
321. Masel, B. E., & DeWitt, D. S. (2010). Traumatic brain injury: A disease process, not an event. *Journal of Neurotrauma, 27,* 1529-1540. DOI: 10.1089/neu.2010.1358.
322. Maskell, F., Chiarelli, P., & Isles, R. (2006). Dizziness after traumatic brain injury: Overview and measurement in the clinical setting. *Brain Injury, 20(3),* 293-305. DOI:10.1080/02699050500488041.
323. Maskell, F., Chiarelli, P., & Isles, R. (2007). Dizziness after traumatic brain injury: Results from an interview study. *Brain Injury, 21(7),* 741-752. DOI:10.1080/02699050701672109.
324. Mathew, N. T., Frishberg, B. M., Gawel, M.,. . . Turkel, C. (2005). Botulinum toxin type A (BOTOX®) for the prophylactic treatment of chronic daily headache: A randomized, double-blind, placebo-controlled trial. *Headache, 12,* 293-307.
325. Matsushita, M., Hosoda, K., Naitoh, Y., Yamashita, H., & Kohmura, E. (2011). Utility of diffusion tensor imaging in the acute stage of mild to moderate traumatic brain injury for detecting white matter lesions and predicting long-term cognitive function in adults. *Journal of Neurosurgery, 115,* 130-139.
326. McCauley, R. J., Strand, E., Lof, G. L., Schooling, T., & Frymark, T. (2009). Evidence-based systematic review: Effects of nonspeech oral motor exercises on speech. *American Journal of Speech-Language Pathology, 18,* 343-360.
327. McCrea, M., Guskiewicz, K. M., Marshall, S. W., Barr, W., Randolph, C., Cantu, R. C., …Kelly, J. P. (2003). Acute effects and recovery time following concussion in collegiate football players: The NCAA concussion study. *Journal of the American Medical Association, 290(19),* 2556-2563. <http://www.jama.com>
328. McCrea, M., Iverson, G. L., McAllister, T. W., Hammeke, T. A., Powell, M. R., Barr, W. B. & Kelly, J. P. (2009). An integrated review of recovery after mild traumatic brain injury (MTBI): Implications for clinical management. *The Clinical Neuropsychologist, 23,* 1368-1390. ISSN:1385-4046 print/1744-4144 online, DOI: 10.1080/13854040903074652.
329. McCrory, D. C., & Gray R. N., (2003). Oral sumatriptan for acute migraine. *Cochrane Database of Systematic Reviews, 3,* 1-91. DOI:10.1002/14651858. CD002915. *\*\*\*Used in Evidence Statement.*
330. McCrory, P., Meeuwisse, W., Johnston, K., Dvorak, J., Aubry, M., Molloy, M., & Cantu, R. (2009). Consensus statement on concussion in sport 3rd international conference on concussion in sport held in Zurich, November 2008. *Clinical Journal of Sport Medicine, 19,* 185–200.
331. McDowell, S., Whyte, J., & D’Esposito, M. (1998). Differential effect of a dopaminergic agonist on prefrontal function in traumatic brain injury patients. *Brain, 121,* 1155-1164.
332. McFadden, K. L., Healy, K. M., Dettmann, M. L., Kaye, J. T., Ito, T. A., & Hernandez, T. D. (2011). Acupressure as a non-pharmacological intervention for traumatic brain injury (TBI). *Journal of Neurotrauma, 28,* 21-34. DOI: 10.1089/neu.2010.1515.
333. McFadden, K. L., & Hernandez, T. D. (2010). Cardiovascular benefits of acupressure (Jin Shin) following stroke. *Complementary Therapies in Medicine, 18,* 42-48. DOI:10.1016/j.ctim.2010.01.001.
334. McFadzean, R. M. (2006). NovaVision: vision restoration therapy. *Current Opinion in Ophthalmology, 17,* 498–503.
335. McIntyre, L. A., Fergusson, D. A., Hebert, P. C., Moher, D., & Hutchison, J. S. (2003). Prolonged therapeutic hypothermia after traumatic brain injury in adults a systematic review. *Journal of the American Medical Association, 289(22),* 2992-2999. <http://www.JAMA.com>
336. Mehrazin, M., Nezameddini-Kachooer, S. A., Fallahi, B., Derakhshanl, M. K., Beiki, D., Ghodsi, S. M., Assadi, M., Pooyafard, F., & Eftekhari, M. (2011). Prospective evaluation of technetium-99m ECD SPET in mild traumatic brain injury for the prediction of sustained neuropsychological sequels. *Hellenic Journal of Nuclear Medicine, 74(3),* 243-250.
337. Mehrholz, J., Kugler, J., & Pohl, M. (2011). Water-based exercises for improving activities of daily living after stroke. *Cochrane Database of Systematic Reviews, 1,* 1-26. DOI: 10.1002/14651858.CD008186.pub2.
338. Meyer, G. J., Finn, S. E., Eyde. L, D., Kay, G. G., Moreland, K. L., Dies, R. R.,… Reed, G. M. (2001). Psychological testing and psychological assessment a review of evidence and issues. *American Psychologist, 56(2),* 128-165. DOI: 10.1037//OOO3-O66X.56.2.128.
339. Meythaler, J. M., Brunner, R. C., Johnson, A., & Novack, T. A. (2002). Amantadine to improve neurorecovery in traumatic brain injury – associated diffuse anoxal injury: A pilot double-blind randomized trial. *Journal of Head Trauma Rehabilitation, 17(4),* 300-313.
340. Mishra, A., Davis S., Speers, R., & Shepard, N. T. (2009). Head shake computerized dynamic posturography in peripheral vestibular lesions. *American Journal of Audiology, 18,* 53-59. DOI: 10.1044/1059-0889(2009/06/0024).
341. Mittenberg, W., Canyock, E. M., Condit, D., & Patton, C. (2001). Treatment of post-concussion syndrome following mild head injury*. Journal of Clinical and Experimental Neuropsychology, 23(6),* 829-836).
342. Moja, L., Cusi C., Sterzi R., & Canepari C. (2005). Selective serotonin re-uptake inhibitors (SSRIs) for preventing migraine and tension-type headaches. *Cochrane Database of Systematic Reviews, 3,* 1-69, DOI:10.1002/14651858. CD002919. pub 2.
343. Mooney, G. F., & Haas, L. J. (1993). Effect of methylphenidate on brain injury-related anger. *Archeives of Physical Medince and Rehabilitation, 74(2),* 153-160.
344. Morgan, S. S., Beck, W. G., & Dobie, R. A. (2002). Can posturography identify informed malingerers? *Otology & Neurotology, 23,* 214-217.
345. Morillo, L. E. (2004). Migraine headache in adults. *BMJ Publishing Group, Ltd., Clinical Evidence, 5,* 2-21.
346. Moseley, A. M., Stark, A., Cameron, I. D., & Pollock, A. (2005). Treadmill training and body weight support for walking after stroke. *Cochrane Database of Systematic Reviews, 4,* 1-89. DOI: 10.1002/14651858.CD002840.pub2.
347. Müller, R., & Büttner, P. (1994). A critical discussion of intraclass correlation coefficients. *Statistics in Medicine, 13,* 2465-2476.
348. Murphy, L., Chamberlain, E., Weir, J., Alister, B., Nathaniel-James, D., & Agnew, R. (2006). Effectiveness of vocational rehabilitation following acquired brain injury: Preliminary evaluation of a UK specialist rehabilitation programme. *Brain Injury, 20(11),* 1119-1129. DOI: 10.1080/02699050600664335.
349. Musicco, M., Emberti, L., Nappi, G., & Caltagirone, C. (2003). Early and long-term outcome of rehabilitation in stroke patients: The role of patient characteristics, time of initiation, and duration of interventions. *Arch Phys Med Rehabil, 84,* 551-558. DOI:10.1053/apmr.2003.50084.
350. Nagamatsu, L. S., Handy, T. C., Laing Hsu, C., Voss, M., & Liu-Ambrose, T. (2012) Resistance training promotes cognitive and functional brain plasticity in seniors with probable mild cognitive impairment. *Arch Intern Med, 172(8),* 666-668.
351. Narayan, R. K. (2001). Hypothermia for traumatic brain injury a good idea proved ineffective. *The New England Journal of Medicine, 344(8),* 602-603. <http://www.nejm.org>
352. Nashner, L. M. (1993). Computerized dynamic posturography: Clinical applications. *Handbook of Balance Function Testing, 13,14,* 308-318. ISBN: 0-8016-6814-X.
353. Nasreddine, Z., Phillips, N., & Chertkow, H. (n.d.). Montreal Cognitive Assessments (MOCA) Ver.7.1, 7.2, & 7.3. <http://www.mocatest.org>
354. National Institute for Health and Clinical Excellence [NICE], (2007a). Head injury: Triage, assessment, investigation and early management of head injury in infants, children and adults. *National Health Services, 56,* 1-56. <http://www.nice.org.uk>
355. National Institute for Health and Clinical Excellence [NICE]. (2007b). Head injury: Triage, assessment, investigation and early management of head injury in infants, children and adults, Methods, Evidence & Guidance. *National Collaborating Center for Acute Care,* 1-230, ISBN 0-9549760-5-3.
356. National Institute for Health and Clinical Excellence [NICE]. (2007c). Head injury: triage, assessment, investigation and early management of head injury in infants, children and adults, APPENDICES. *National Collaborating Center for Acute Care,* 1-226, ISBN 0-9549760-5-3.
357. Naumann, M., So, Y., Argoff, C. E., Childers, M. K., Dykstra, D. D., Gronseth, G. S.,… Simpson, D. M. (2008). Assessment: Botulinum neurotoxin in the treatment of autonomic disorders and pain (an evidence-based review): Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology, 70,* 1707. DOI 10.1212/01. wnl.0000311390.87642. d8.
358. Neeper, S. A., Gomez-Pinilla, F., Choi, J., & Cotman, C. (1995). Exercise and brain neurotrophins. *Nature, 373,* 109.
359. Nestoriuc, Y., Martin, A., Rief, W., & Andrasik, F. (2008a). Biofeedback treatment for headache disorders: A comprehensive efficacy review. *Appl Psychophysiol Biofeedback, 33,* 125–140. DOI 10.1007/s10484-008-9060-3.
360. Nestoriuc, Y., Rief, W., & Martin, A. (2008b). Meta-analysis of biofeedback for tension-type headache: Efficacy, specificity, and treatment moderators. *Journal of Consulting and Clinical Psychology, 76(3),* 379–396. DOI: 10.1037/0022-006X.76.3.379.
361. Novack, T. A., Banos, J. H., Brunner, R., Renfroe, S., & Meythaler, J. M. (2009). Impact of early administration of sertraline on depressive symptoms in the first year after traumatic brain injury. *Journal of Neurotrauma, 26,* 1921-1928. DOI: 10.1089=neu.2009.0895.
362. Nuwer, M. R., Hovda, D. A., Schrader, L. M., & Vespa, P. M. (2005). Invited review routine and quantitive EEG in mild traumatic brain injury. *Clinical Neurophysiology, 116,* 2001-2025. DOI:10.1016/j.clinph.2005.05.008.
363. O’Neill, D. E, Gill-Body, K. M., & Krebs, D. E. (1998). Posturography changes do not predict functional performance changes. *The American Journal of Otology, 19,* 797-803.
364. Orff, H. J., Ayalon, L., & Drummond, S. P. A. (2009). Traumatic brain injury and sleep disturbance: A review of current research. *Journal of Head Trauma Rehabilitation, 24(3),* 155-165.
365. Orman, J. A. L., Kraus, J. F., Zaloshnja, E. & Miller, T. (2011). Epidemiology. *American Psychiatric Association, 1,* 1-9. DOI: 10.1176/appl. books.9781585624201.670001. <http://www.psychiatryonline.com>
366. Outpatient Service Trialists. (2003). Therapy-based rehabilitation services for stroke patients at home. *Cochrane Database of Systematic Reviews, 1,* 1-68. DOI: 10.1002/14651858.CD002925. *\*\*\*Used in Evidence Statement*
367. Ozdemir, F., Birtane, M., Tabatabaei, R., Kokino, S., & Ekuklu, G. (2001). Comparing stroke rehabilitation outcomes between acute inpatient and nonintense home settings. *Arch Phys Med Rehabil, 82,* 1375-1379. DOI:10.1053/apmr.2001.25973.
368. Padula, W. V., & Argyris, S. (1996). Post trauma vision syndrome and visual midline shift syndrome. *NeuroRehabilitation, 6,* 165-171. http://www.padulainstitute.com/post\_trauma\_vision\_syndrome.htm
369. Pang, M. Y. C., Lam, F. M., Wong, G. H., Au I. H, & Chow, D. L. (2011). Balance performance in head-shake computerized dynamic posturography: Aging effects and test-retest reliability. *Physical Therapy, 91(2),* 246-253. DOI:10.2522/ptj.20100221.
370. Patel, R., Ciuffreda, K. J., Tannen, B., & Kapoor, N. (2011). Elevated coherent motion thresholds in mild traumatic brain injury. *American Optometric Association, 82,* 284-289. DOI:10.1016/J. OPTM.2010.10.012.
371. Pavlou, M., Lingeswaran, A., Davies, R. A., Gresty, M. A., & Bronstein, A. M. (2004). Simulator based rehabilitation in refractory dizziness. *Journal or Neurology, 251,* 983-995. DOI: 10.1007/s00415-004-0476-2.
372. Pelak, V. S., Dubin, M, & Whitney, E. (2007). Homonymous hemianopia: A critical analysis of optical devices, compensatory training, and NovaVision. *Current Treatment Options in Neurology, 9(1),* 41-47. DOI: 10.1007/s11940-007-0029-y.
373. Perel, P., Roberts, P. P., Shakur, I., Thinkhamrop H., Phuenpathom, B., & Yutthakasemsunt S. (2010). Haemostatic drugs for traumatic brain injury (Review). *Cochrane Database of Systematic Reviews, 1,* 1-17 <http://www.thecochranelibrary.com>
374. Perel, P., Yanagawa, T., Bunn, F., Roberts, I. G., & Wentz, R. (2006). Nutritional support for head-injured patients (Review). *Cochrane Database of Systematic Reviews, 4,* 1-22. <http://www.thecochranelibrary.com>
375. Pẻrez, N., Martin, E., & Garcia-Tapia, R. (2003). Dizziness relating the severity of vertigo to the degree of handicap by measuring vestibular impairment. *Otolaryngology – Head and Neck Surgery, 128,* 372-381. DOI:10.1067/mhn.2003.102.
376. Perez-Lloret, S., & Rascol, O. (2010). Dopamine receptor agonists for the treatment of early or advanced Parkinson's Disease. *CNS Drugs, 24(11),* 941-968.
377. Permsirivanich, W., Tipchatyotin, S., Wongchai, M., Leelamanit, V., Setthawatcharawanich, S., Sathirapanya, P.,… Boonmeeprakob, A. (2009). Comparing the effects of rehabilitation swallowing therapy vs. neuromuscular electrical stimulation therapy among stroke patients with persistent pharyngeal dysphagia: A randomized controlled study. *J Medicine Assoc Thai, 92(2),* 259-265.
378. Polderman, K. H. (2008a). Induced hypothermia and fever control for prevention and treatment of neurological injuries. *The Lancet, 371,* 1955-1969. <http://www.thelancet.com>
379. Polderman, K. H. (2009). Mechanisms of action, physiological effects, and complications of hypothermia. *Society of Critical Care Medicine, 37(7),* S186-S202. DOI: 10.1097/CCM.0b013e3181aa5241
380. Polderman, K. H., Mayer, S. A., & Menon, D. (2008b). Hypothermia therapy after traumatic brain injury in children. *The New England Journal of Medicine, 359(11),* 1178-1180. <http://www.nejm.org>
381. Pollock, A., Baer, G., Pomeroy, V. M., & Langhorne, P. (2007). Physiotherapy treatment approaches for the recovery of postural control and lower limb function following stroke. *Cochrane Database of Systematic Reviews, 1,* 1-90. DOI: 10.1002/14651858.CD001920.pub2.
382. Pollock, A., Hazelton, C., Henderson, C. A., Angilley, J., Dhillon, B., Langhorne, P.,… Shahani, U. (2011a). Interventions for disorders of eye movement in patients with stroke. *Cochrane Database of Systematic Reviews, 10,* 1-33. DOI: 10.1002/14651858.CD008389.pub2.
383. Pollock, A., Hazelton, C., Henderson, C. A., Angilley, J., Dhillon, B., Langhorne, P.,… Shahani, U. (2011b). Interventions for visual field defects in patients with stroke. *Cochrane Database of Systematic Reviews*, *10,* 1-84. DOI: 10.1002/14651858.CD008388.pub2.
384. Ponsford, J. L., Ziino, C., Parcell, D. L., Shekleton, J. A., Roper, M., Redman, J. R.,… Rajaratnam, S. M. W. (2012) Fatigue and sleep disturbance following traumatic brain injury—their nature, causes, and potential treatments. *Journal of Head Trauma and Rehabilitation, 27(3),* 224-233. DOI: 10.1097/HTR.0b013e31824ee1a8.
385. Possl, J, Jurgensmeyer, S., Karlbauer, F., Wenz, C., & Goldenberg, G. (2001). Stability of employment after brain injury: a 7- year follow-up study. *Brain Injury, 15(1),* 15-27.
386. Povlishock, J. T., & Wei, E. P. (2009). Posthypothermic rewarming considerations following traumatic brain injury. *Journal of Neurotrauma, 26,* 333-340. DOI: 10.1089=neu.2008.0604.
387. Powell, J., Heslin, J., & Greenwood, R. (2002). Community based rehabilitation after severe traumatic brain injury: a randomised controlled trial. *J Neurol Neurosurg Psychiatry, 72,* 193–202.
388. Prvu Bettger, J. A., Stineman, M. G. (2007). Effectiveness of multidisciplinary rehabilitation services in postacute care: State-of-the-Science. A review. *Arch Phys Med Rehabil, 88,* 1526-1534. DOI:10.1016/j.apmr.2007.06.768.
389. Putukian, M. (2011). Neuropsychological testing as it relates to recovery from sports-related concussion. *American Academy of Physical Medicine and Rehabilitation, 3,* S425-S432. DOI: 10.1016/j. pmrj.2011.08.003.
390. Qiu, W., Zhang, Y., Sheng, H., Zhang, J., Wang, W., Liu, W., … Xu, Z. (2007). Effects of therapeutic mild hypothermia on patients with severe traumatic brain injury after craniotomy. *Journal of Critical Care, 22,* 229-236. DOI:10.1016/j.jcrc.2006.06.011.
391. Rabbie R., Derry S., Moore R. A., & McQuay, H. J. (2010). Ibuprofen with our without an antiemetic for acute migraine headaches in adults. *Vovhtsnr Fsysnsdr og Dydyrmsyiv* *Trbired,* *10,* 1-66. DOI:10.1002/14651858. CD008039. pub2. *\*\*\*Used in Evidence Statement.*
392. Randolph, C., Lovell, M., Laker, S. R. (2011). Neuropsychological testing point/counterpoint. *American Academy of Physical Medicine and Rehabilitation, 3,* S433-S439. DOI: 10.1016/j. pmrj.2011.08.002.
393. Rappaport, M., Hall, K. M., Hopkins, I., Belleza, T., & Cope, D. N. (1982). Disability rating scale for severe head trauma: Coma to community. *Archives Physical Medicine Rehabilitation, 63(3),* 118-123.
394. Reddy, C. C. (2011). Postconcussion syndrome: A physiatrist’s approach. *American Academy of Physical Medicine and Rehabilitation, 3,* 396-S405. DOI: 10.1016/j. prnrj.2011.07.012.
395. Reger, M. L., Poulos, A. M., Buen, F., Giza, C. C., Hovda, D. A., & Fanselow, M. S. (2012). Concussive brain injury enhances fear learning and excitatory processes in the amygdala. *Society of Biological Psychiatry, 71,* 335-343. DOI:10.1016/j.biopsych.2011.11.007.
396. Reich, B. A. (1989). Non-Invasive treatment of vascular and muscle contraction headache: A comparative longitudinal clinical study. *Headache, 29(1),* 34-41.
397. Reinhard, J., Schreiber, A., Schiefer, U., Kasten, E., Sabel, B. A., Kenkel, S.,… Trauzettel-Klosinski, S. (2005). Does visual restitution training change absolute homonymous visual field defects? A fundus controlled study. *British Journal of Ophthalmology, 89,* 30–35. DOI: 10.1136/bjo.2003.040543.
398. Relja, M., Poole, AC, Schoenen, J.,. . . Thompson, C. (2007). A multicentre, double-blind randomized, placebo-controlled, parallel group study of multiple treatments of botulinum toxin, type A (BoNTA) for the prophylaxis of episodic migraine headaches. *Cephalalgia, 27,* 492-503. DOI: 10.1111/j.1468-2982.2007.01315. x.
399. RESCUE icp study. (2009). Randomized evaluation of surgery with craniectomy for uncontrollable elevation of intra-cranial pressure.
400. Reyna, V. F., (2008) A theory of medical decision making and health: Fuzzy trace theory. *Medical Decision Making, 28(6),* 850-865. DOI: 10.1177/0272989X08327066.
401. Richards, P. M., & Ruff, R. M. (2010). Introduction to special issue. *Psychological Injury and Law, 3,* 1–2. DOI 10.1007/s12207-010-9070-3.
402. Rigg, J. L. & Mooney, S. R. (2011). Concussions and the military: Issues specific to servive members. *American Academy of Physical Medicine and Rehabilitation, 3,* S380-386. DOI: 10.1016/j. prmrj.2011.08.055.
403. Ripley, D. L. (2011). Endocrinopathy after traumatic brain injury. *Physical Medicine and Rehabilitation, 3(3),* 268-273. DOI:10.1016/j.pmrj.2011.01.008.
404. Roberts, I. (1999a). Aminosteroids for acute traumatic brain injury (Review). *Cochrane Database of Systematic Reviews, 3,* 1-17. <http://www.thecochranelibrary.com>
405. Roberts, I., & Schierhout, G. (2009). Hyperventilation therapy for acute traumatic brain injury. *Cochrane Database of Systematic Reviews, 4,* 1-14. DOI: 10.1002/14651858.CD000566.
406. Roberts, I., & Sydenham, E. (1999b). Barbiturates for acute traumatic brain injury. *Cochrane Database of Systematic Reviews, 3,* 1-23. DOI: 10.1002/14651858.CD000033.
407. Robertson, D. D., & Ireland, D. J. (1995). Dizziness handicap inventory correlates of computerized dynamic posturography. *The Journal of Otolaryngology, 24(2),* 118-124.
408. Rockswold, S. B., Rockswold, G. L., Zaun, D. A., Zhang, X., Cerra, C. E., Bergm an, T. A., & Liu, J. (2010). A prospective, randomized clinical trial to compare the effect of hyperbaric to normobaric hyperoxia on cerebral metabolism, intracranial pressure, and oxygen toxicity in severe traumatic brain injury. *Journal of Neurosurgery, 112,* 1080–1094.
409. Rohling, M. L., Faust, M. E., Beverly, B., & Demakis, G. (2009). Effectiveness of cognitive rehabilitation following acquired brain injury: A meta-analytic re-examination of Cicerone et al.’s (2000, 2005) Systematic Reviews. *Neuropsychology, 23(1),* 20–39 DOI:10.1037/a0013659.
410. Rosenberg, J. B., Shiloh, A. L., Savel, R. H., & Eisen, L. A. (2011) Non-invasive methods of estimating intracranial pressue. *Neurocritical Care.* DOI:10.1007/s12028-011-9545-4.
411. Rosenfeld, J. V., Maas, A. I., Bragge, P., Morganti-Kossman, M. C., Manley, G. T., & Gruen, R. L. (2012). Early management of severe traumatic brain injury. *Lancet, 380,* 1088-1098.
412. Rosengren, S. M., Welgampola, M. S., & Colebatch, J. G. (2010). Invited review vestibular evoked myogenic potentials: Past, present and future. *Clinical Neurophysiology, 121,* 636–651. DOI:10.1016/j.clinph.2009.10.016.
413. Rubin, A. M., Woolley, S. M., Dailey, V. M., & Goebel J. A. (1995). Postural stability following mild head or whiplash injuries. *The American Journal of Otology, 16(2),* 216-221.
414. Ruff, R. M., (2011). Mild traumatic brain injury and neural recovery: Rethinking the debate. *NeuroRehabilitation, 28, 167*-180. DOI: 10.3233/NRE-2011-0646.
415. Ruff, R. M., Iverson, G. L., Barth, F. T., Bush, S. S., Broshek, D. K., & the NAN Policy and Planning Committee. (2009). Recommendations for diagnosing a mild traumatic brain injury: A National Academy of Neuropsychology Education paper. *Archives of Clinical Neuropsychology, 24,* 3-10. DOI:10.1093/arclin/acp006.
416. Ruff, R. M., & Richards, P. M. (2003). Neuropsychological assessment and management of patients with persistent postconcussional disorders. *Clinical Neuropsychology and Cost Outcome Research,* (ch 4).
417. Sahuquillo, J. (2006). Decompressive craniectomy for the treatment of refractory high intracranial pressure in traumatic brain injury (Review). *Cochrane Database of Systematic Reviews, 1, 1-41.* <http://www.thecochranelibrary.com>
418. Salazar, A. M., Warden, D. L., Schwab, K., Spector, J., Braverman, S., Walter, J., …Ellenbogen, R. G. (2000). Cognitive rehabilitation for traumatic brain injury a randomized trial. *Journal of the American Medical Association, 283(23),* 3075-3081. <http://www.JAMA.com>. *\*\*\*Used in Evidence Statement.*
419. Sander, A. M., Roebuck, T. M., Struchen, M. A., Sherer, M., & High, W. M. Jr. (2001). Long-term maintenance of gains obtained in postacute rehabilitation by persons with traumatic brain injury. *Journal of Head Trauma Rehabilitation, 16(4),* 356–373.
420. Sanders, L. (2009). Trawling the brain. *Science News, 176(13),* 16-20.
421. Sarajuuri, J. M., Kaipio, M. –L., Koskinen, S. K., Niemelä, M. R., Servo, A. R., & Vilkki, J. S. (2005). Outcome of a comprehensive neurorehabilitation program for patients with traumatic brain injury. *Arch Phys Med Rehabil, 86,* 2296-2302. DOI:10.1016/j.apmr.2005.06.018.
422. Saxena, M., Andrews P., & Cheng A. (2008). Modest cooling therapies (34˚C to 37.5˚C) for traumatic brain injury. *Cochrane Database of Systematic Reviews, 3,* 1-15. <http://www.thecochranelibrary.com>
423. Scheiman, M., Mitchell, G. L., M., Cotter, S., Kulp, M. T.,…Vensveen, J. (2005). A randomized clinical trial of vision therapy/orthoptics versus pencil pushups for the treatment of convergence insufficiency in young adults. *Optometry and Vision Science, 82(7),* E583-E595. DOI:10.1040-5488/8207-0583/0.
424. Schneider, H. J., Kreitschmann-Andermahr, I., Ghigo, E., Stalla, G. K., & Agha, A. (2007). Hypothalamopituitary dysfunction following traumatic brain injury and aneurysmal subarachnoid hemorrhage a systematic review. *Journal of the American Medical Association, 298(12),* 1429-1438.
425. Schreiber, A., Vonthein, R., Reinhard, J., Trauzettel-Klosinski, S., Connert, C., & Schiefer, U. (2006). Effect of visual restitution training on absolute homonymous scotomas. *Neurology, 67,* 143–145. DOI 10.1212/01.wnl.0000223338.26040.fb.
426. Schultheis, M. T., Himelstein, J., & Rizzo, A. A. (2002). Virtual reality and neuropsychology: Upgrading the current tools. *Journal of Head Trauma, 17(5),* 378-394. DOI:10.1097/00001199-200210000-00002.
427. Scottish Intercollegiate Guidelines Network. (2009a). Early management of adult patients with a head injury; quick reference guide. *(NHS) National Health Services, Quality Improvement Scotland, 110,* 1-14. <http://www.SIGN.AC.UK>
428. Scottish Intercollegiate Guidelines Network. (2009b). Early management of adult patients with a head injury; Recommendations online: Clinical knowledge evidence translation. *(NHS) National Health Services, Quality Improvement Scotland, 110,* 1-14. <http://www.SIGN.AC.UK>
429. Scottish Intercollegiate Guidelines Network. (2009c). Early management of patients with a head injury; a national clinical guideline. *(NHS) National Health Services, Quality Improvement Scotland, 110,* 1-78. ISBN: 978 1 905813 46 9.
430. Seder, D. B., & Van der Kloot, T. E. (2009). Methods of cooling: Practical aspects of therapeutic temperature management. *Critical Care Medicine, 37(7),* S211-S222. DOI: 10.1097/CCM.0b013e3181aa5bad.
431. Sellars, C., Hughes, T., & Langhorne, P. (2005). Speech and language therapy for dysarthria due to non-progressive brain damage. *Cochrane Database of Systematic Reviews, 3,* 1-13. DOI:10.1002/14651858.CD002088.pub2.
432. Shuhendler, A. J., Lee, S., Siu, M., Ondovcik, S., Lam, K., Alabdullatif, A., … Einarson, T. R. (2009). Efficacy of botulinum toxin type A for the prophylaxis of episodic migraine headaches: A meta-analysis of randomized, double-blind, placebo-controlled trials. *Pharmacotherapy,29(7),* 784–791. *\*\*\*Used in Evidence Statement*
433. Signoretti, S., Lazzarino, G., Tavazzik B, & Vagnozzi, R. (2011). Concussion supplement: The pathophysiology of concussion*. American Academy of Physical Medicine and Rehabilitation, 3,* S359-368. DOI: 1016/jlpmrj-2011-07-018.
434. Silberstein, S. D., Holland, S., Freitag, F., Dodick, D. W., Argoff, C., & Ashman, E. (2012). Evidence-based guideline update: Pharmacologic treatment for episodic migraine prevention in adults : Report of the Quality Standards Subcommittee of the American Academy of Neurology and the American Headache Society. *Neurology, 78,* 1337-1346. DOI 10.1212/WNL.0b013e3182535d20.
435. Silberstein, S. D., Stark, S. R., Lucas, S. M.,. . . Turkel, C. C. (2005). Botulinum toxin type A for the prophylactic treatment of chronic daily headache: A randomized, double-blind, placebo-controlled trial. *Mayo Clin Proc., 80(9),* 1126-1137. www. mayoclinicproceedings.com
436. Silver, J. M., Koumaras, B., Chen, M., Mirski, D., Potkin, S. G., Reyes, P., …Gunay, I. (2006). Effects of rivastigmine on cognitive function in patients with traumatic brain injury. *Neurology, 67,* 748–755. DOI 10.1212/01.wnl.0000234062.98062.e9. <http://www.neurology.org/content/67/5/748.full.html>
437. Silver, J. M., Koumaras, B., Meng, X., Potkin, S. G., Reyes, P. F., Harvey, P. D.,… Arciniegas, D. B. (2009). Long-term effects of rivastigmine capsules in patients with traumatic brain injury. *Brain Injury, 23(2),* 123-32. DOI: 10.1080/02699050802649696.
438. Sinikallio, S., Aalto, T., Koivumaa-Honkanen, H., Airaksinen, O., Herno, A., Kroger, H., & Viianamaki, H. (2009). Life dissatisfaction is associated with a poorer surgery outcome and depression among lumbar spinal stenosis patients: a 2-year prospective study. *European Spine Journal, 18,* 1187–1193. DOI 10.1007/s00586-009-0955-3. *\*\*\*Used in Evidence Statement.*
439. Sinikallio, S., Aalto, T., Lehto, S., Airaksinen, O., Herno, A., Kroger, H., & Viianamaki, H. (2010). Depressive symptoms predict postoperative disability among patients with lumbar spinal stenosis: A two-year prospective study comparing two age groups. *Disability and Rehabilitation, 32(6),* 462–468. DOI: 10.3109/09638280903171477. *\*\*\*Used in Evidence Statement.*
440. Sirtori, V., Corbetta, D., Moja, L., & Gatti, R. (2009). Constraint-induced movement therapy for upper extremities in stroke patients. *Cochrane Database of Systematic Reviews, 4,* 1-60. DOI: 10.1002/14651858.CD004433.pub2. *\*\*\*Used in Evidence Statement*
441. Sivan, M., Neumann, V., Kent, R., Stroud, A., & Bhakta, B. (2010). Pharmacotherapy for treatment of attention deficits after non-progressive acquired brain injury. A systematic review. *Clinical Rehabilitation, 24,* 110-121. DOI: 10.1177/0269215509343234.
442. Slobounov, S.M., Zhang, K., Pennell, D., Ray, W., Johnson, B., & Sebastianelli, W. (2009). Functional abnormalities in normally appearing athletes following mild traumatic brain injury: a functional MRI study. *Exp Brain Res., 202(2),* 341-354. DOI: 10.1007/s00221-009-2414-6.
443. Smith, C. (2011). Neuropathology. *American Psychiatric Association, 2,* 1-21 DOI: 10.1176/appi. books.9781585624201.671834. <http://www.psychiatryonline.com>
444. Sohlberg, M. M., & Mateer, C. A., (1987). Effectiveness of an attention-training program. *Journal of Clinical and Experimental Neuropsychology, 9(2),* 117-130.
445. Soo, C., Tate, R. (2007). Psychological treatment for anxiety in people with traumatic brain injury (Review). *Cochrane Database of Systematic Reviews, 3,* 1-23. DOI: 10.1002/14651858.CD005239.pub2. <http://www.thecochranelibrary.com>
446. Spikeman, J. M., Boelen, D. H. E., Lamberts, K., Brouwer, W. H., & Basotti, L., (2010). Effects of a multifaceted treatment program for executive dysfunction after acquired brain injury on indications of executive functioning in daily life. *Journal of the International Neuropsychological Society, 16,* 118-129. DOI:10.1017/S13555617709991020. *\*\*\*Used in Evidence Statement.*
447. Spoor, T. C. (2008). Traumatic Optic Neuropathies.In Yanoff, M., & Duker, J. S. (Ed.) *Ophthalmology, 3rd edition,* (ch 9.10).Retreived from http://www.mdconsult.com.
448. Staab J. P. (2006). Chronic dizziness: the interface between psychiatry and neuro-otology. *Current Opinion in Neurology, 19,* 41-48.
449. Staab J. P., & Ruckenstein, M. J. (2007). Expanding the differential diagnosis of chronic dizziness. *Archives Otolaryngol Head Neck Surg., 133,* 170-176. <http://www.archoto.com>
450. Stern, R. A., Riley, D. O., Daneshavar, D. H., Nowinski, C. J.,… McKee, A. C. (2011) Long-term consequences of repetitive brain trauma: Chronic traumatic encephalopathy. *American Academy of Physical Medicine and Rehabilitation, 3,* S460-S467 DOI: 10.1016/j. -pmrj.2011.08.008.
451. Stetler, C. B., Damschroder, L, J., Helfrich, C. D., & Hagedorn, H. J. (2011). A Guide for applying a revised version of the PARIHS framework for implementation. *Implementation Science, 6(99),* 1-29. doi:10.1186/1748-5908-6-99. <http://www.implementationscience.com/content/6/1/99>
452. Stewart, M. G., Chen, A. Y., Wyatt, R., Favrot, S., Jenkins, H. A. (1999). Cost-effectiveness of the diagnostic evaluation of vertigo*. Laryngoscope, 109,* 600-605.
453. Stiver, S. I. (2009).complications of decompressive craniectomy for traumatic brain injury. *Journal of Neurosurgery, 26(6),* E7. DOI: 10.3171/2009.4. FOCUS0965.
454. Stroke Unit Trialists’ Collaboration. (2007). Organised inpatient (stroke unit) care for stroke. *Cochrane Database of Systematic Reviews, 4,* 1-71. DOI: 10.1002/14651858.CD000197.pub2. *\*\*\*Used in Evidence Statement*
455. Sydenham, E., Roberts, I., & Alderson, P. (2009). Hypothermia for traumatic head injury (Review). *Cochrane Database of Systematic Reviews, 2,* 1-48. DOI: 10.1002/14651858.CD001048.pub4. <http://www.thecochranelibrary.com>
456. Tanriverdi, F., Agha, A., Aimaretti, G., Casanueva, F. F., Kelestimur, F., Klose, M.,… Schneider, H. J. (2011). Manifesto for the current understanding and management of traumatic brain injury-induced hypopituitarism. *J. Endocrinol. Invest., 34,* 541-543. DOI: 10.3275/7805.
457. Tendal, B., Nüesch, E., Higgins, J. P. T., Jüni, P., & Gotzsche, P. C. (2011). Multiplicity of data in trial reports and the reliability of meta-analyses: empirical study. *British Medical Journal, 343,* 1-13. DOI10.1136/bmj. d4829.
458. Tennant, A., Penta, M., Tesio, L., Grimby, G., Thonnard, J.-L., Slade, A., … Phillips, S. (2004). Assessing and adjusting for cross-cultural validity of impairment and activity limitation scales through differential item functioning within the framework of the Rasch Model The PRO-ESOR project. *Medical Care, 42(1 suppl),* I-37–I-48. DOI: 10.1097/01.mlr.0000103529.63132.77.
459. Tenovuo, O. (2005). Central acetylcholinesterase inhibitors in the treatment of chronic traumatic brain injury—clinical experience in 111 patients. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 29,* 61-67. DOI:10.1016/j.pnpbp.2004.10.006.
460. Tenovuo, O., Alin, J., & Helenius, H. (2009). A randomized controlled trial of rivastigmine for chronic sequels of traumatic brain injury—What it showed and taught? *Brain Injury, 23(6),* 548-558. DOI: 10.1080/02699050902926275.
461. Thiagarajan, P., Ciuffreda, K. J., & Ludlam D. P. (2011). Vergence dysfunction in mild traumatic brain injury (MTBI): a review. *Ophthalmic & Physiological Optics, 31,* 456-468. DOI:10.1111/j.1475-1313.2011.00831. x.
462. Thieme, H., Mehrholz, J., Pohl, M., Behrens, J., & Dohle, C. (2012). Mirror therapy for improving motor function after stroke. *Cochrane Database of Systematic Reviews, 3,* 1-65. DOI: 10.1002/14651858.CD008449.pub2. *\*\*\*Used in Evidence Statement*
463. Thomsen, J., Sass, K., Odkvist, L., & Arlinger, S. (2005). Local overpressure treatment reduces vestibular symptoms in patients with Ménière’s disease: A clinical, randomized, multicenter, double-blind, placebo-controlled study. *Otology & Neurotology, 26,*68–73.
464. Trelle, S., Reichenbach, S., Wandel, S., Hildebrand, P., Tschannen, B., Villiger, P.,… Juni, P. (2011). Cardiovascular safety of non-steroidal anti-inflammatory drugs: Network meta-analysis. *BMJ,* 1-11. DOI: 10.1136/bmj.c7086. *\*\*\*Used in Evidence Statement.*
465. Trzepacz, P. T., Kean, J., & Kennedy, R. E. (2011). Delirium and posttraumatic confusion: More than posttraumatic amnesia. Posttraumatic amnesia: *History and Hazards. Textbook of Traumatic Brain Injury, 9,* 1-16. DOI: 10:1176/appl. books.8781585624201.674873. [http://www. psychiatryonline.com](http://www.psychiatryonline.com).
466. Turner-Stokes, L. (2007). Cost-efficiency of longer-stay rehabilitation programmes: Can they provide value for money? *Brain Injury, 21(10),* 1015–1021. DOI: 10.1080/02699050701591445.
467. Turner-Stokes, L. (2008). Evidence for the effectiveness of multi-disciplinary rehabilitation following acquired brain injury: A synthesis of two systematic approaches. *J Rehabil Med, 40,* 691–701. DOI: 10.2340/16501977-0265.
468. Turner-Stokes, L., Nair A, Sedki, I., Disler, P. B., & Wade, D. T. (2005). Multi-disciplinary rehabilitation for acquired brain injury in adults of working age (Review). *Cochrane Database of Systematic Reviews, 3,* 1-46. DOI: 10.1002/14651858.CD004170.pub2. <http://www.thecochranelibrary.com>. *\*\*\*Used in Evidence Statement.*
469. Turner-Stokes, L., Paul, S., & Williams, H. (2006). Efficiency of specialist rehabilitation in reducing dependency and costs of continuing care for adults with complex acquired brain injuries. *J Neurol Neurosurg Psychiatry, 77,* 634–639. DOI: 10.1136/jnnp.2005.073411.
470. van der Kuy, P. –H. M., & Lohman J. J. H. M. (2002). A quantification of the placebo response in migraine prophylaxis. *Cephalalgia, 22,* 265–270. London. ISSN 0333-1024
471. van der Meulen, I,. van de Sandt-Koenderman, M. E., & Ribbers, G. M. (2012). SPECIAL COMMUNICATION Melodic intonation therapy: Present controversies and future opportunities. *Archives Physical Medicine Rehabilitation, 93, Suppl 1,* S46-S52. DOI:10.1016/j.apmr.2011.05.029.
472. van der Worp, H. B., & Kappelle, L, J. (2011). Early decompressive hemicraniectomy in older patients with nondominant hemispheric infarction does not improve outcome. *Stroke, 42,* 845-846. DOI: 10.1161/STROKEAHA.110.603605.
473. Vanderploeg, R. D., Schwab, K., Walker, W. C., Fraser, J. A., Sigford, B. J., Date, E. S., …Warden, D. L. (2008). Rehabilitation of traumatic brain injury in active duty military personnel and veterans: Defense and veterans brain injury center randomized controlled trial of two rehabilitation approaches. *Archives Physical Medicine Rehabilitation, 89,* 2227-2238. DOI:10.1016/j.apmr.2008.06.015.
474. Vas, A. K., Chapman, S. B., Cook, L. G., Elliot, A. C., & Keebler, M., (2011). Higher-order reasoning training years after traumatic brain injury in adults. *J Head Trauma Rehabilitation, 26,* 3*.2*24-239. DOI: 10.1097/HTR.0b013e318218dd3d. *\*\*\*Used in Evidence Statement.*
475. Veterans Affairs, Department of Defense (2009). VA/DoD Clinical practice guideline for management of concussion/mild traumatic brain injury. DOI:10:1682/*JRRD* 6:76.
476. Veterans Affairs, Technology Assessment Program, & Adams, E. (2009). Visual problems in traumatic brain injury: A systematic review of sequelae and interventions for the veteran population. http://www.va.gov/vatap
477. Vouriot, A., Gauchard G. C., Chau, N., Benamghar, L., & Perrin, P. P. (2003) Sensorial organization favouring higher visual contribution is a risk facto of falls in an occupational setting. *Neuroscience Research, 48,* 239-247. DOI:10.1016/j. neures.2003.11.001.
478. Wakai, A., Roberts, I. G., & Schierhout, G. (2007), Mannitol for acute traumatic brain injury. *Cochrane Database of Systematic Reviews, 1,* 1-16. DOI: 10.1002/14651858.CD001049.pub4.
479. Wang, C. C., Kosinski, C. J., Schwartzberg, J. G., and Shanklin, A., (2010). Assessing and counseling older drivers, 2nd ed. *American Medical Association IX*: 1-196.
480. Wang, D, Te-Chun, L, Chen, J, & Allan, S. (2011). Prescription benchmarks for Washington. *Workers Compensation Institut,* 1-117. ISBN 978-1-935325-96-3.
481. Warden, D. L., Gordon, B., McAllister, T. W., Silver, J. M., Barth, J. T., Bruns, J., …Zitnay, G. (2006). Guidelines for the pharmacologic treatment of neurobehavioral sequelae of traumatic brain injury. *Journal of Neurotrauma, 23(10).* 1468-1501.
482. Watkins, C. L., Auton, M. F., Deans, C. F., Dickinson, H. A., Jack, C. I. A., Lightbody, C. E.,… Leathley, M. J. (2007). Motivational interviewing early after acute stroke: A randomized, controlled trial. *Stroke, 38*, 1004-1009. DOI: 10.1161/01.STR.0000258114.28006.d7.
483. Webb, C. (1985). COWS Caloric test. *Annals of Emergency Medicine, 14(9),* 938.
484. Weightman, M. M., Bolgla, R., McCulloch, K. L., & Peterson, M. D. (2010). Physical therapy recommendations for service members with mild traumatic brain injury. *Journal of Head Trauma Rehabilitation, 25(3),* 206-218. <http://www.headtraumarehab.com>
485. Weinberg, J., Diller, L., Gordon, W. A., Gerstman, L. J., Lieberman, A., Lakin, P.,… Ezrachi, O. (1977). Visual scanning training effect on reading-related tasks in acquired right brain damage. *Archives of Physical Medicine and Rehabilitation, 58,* 479- 486. *\*\*\*Used in Evidence Statement*
486. West, C., Bowen, A., Hesketh, A., & Vail, A. (2008) Interventions for motor apraxia following stroke. *Cochrane Database of Systematic Reviews, 1,* 1-20. DOI: 10.1002/14651858.CD004132.pub2.
487. West, C., Hesketh, A., Vail, A., & Bowen, A. (2005) Interventions for apraxia of speech following stroke. *Cochrane Database of Systematic Reviews, 4,* 1-12. DOI:10.1002/14651858.CD004298.pub2. <http://www.thecochranelibrary.com>
488. Weuve, J, Kang, J. H., Manson, J. E., Breteler, M. M. B., Ware, J. H., & Grodstein, F. (2004). Physical activity, including walking, and cognitive function in older women. *Journal of the American Medical Association, 292(12),* 1454-1461.
489. Wheaton, P., Mathias, J. L., & Vink, R. (2011). Impact of pharmacological treatments on cognitive and behavioral outcome in the postacute stages of adult traumatic brain injury. *Journal of Clinical Psychopharmacol, 31(6),* 745-757. DOI: 10.1097/JCP.0b013e318235f4ac.
490. Whitney, S. L., Marchetti, G. R., & Schade, A. I. (2006). The Relationship between falls history and computerized dynamic posturography in persons with balance and vestibular disorders. *Archives of Physical Medicine and Rehabilitation, 87,* 402-407. DOI: 10.1016/j. apmr.2005.11.002
491. Whyte, J., Hart, T., Vaccaro, M., Grieb-Neff, P., Risser, A., Polansky, M., & Coslett, H. B. (2004). Effects of methylphenidate on attention deficits after traumatic brain injury: A multidimensional, randomized, controlled trial. *American Journal Physical Medicine and Rehabilitation, 83,* 401–420. DOI: 10.1097/01.PHM.0000128789.75375.D3. *\*\*\*Used in Evidence Statement.*
492. Whyte, J., Vaccaro, M., Grieb-Neff, P., Hart, T., Polansky, M., & Coslett, H. B. (2008). The effects of Bromocriptine on attention deficits after traumatic brain injury: A placebo-controlled pilot study. *American Journal of Physical Medicine and Rehabilitation, 87,* 85–99. DOI: 10.1097/PHM.0b013e3181619609.
493. Wilkinson, C. W., Pagulayan, K. F., Petrie, E. C., Mayer, C. L., Colasurdo, E. A., Shofer, J. B.,… Peskind, E. R. (2012). High prevalence of chronic pituitary and target-organ hormone abnormalities after blast-related mild traumatic brain injury. *Frontiers in Neurology, 3(11),* 1-12. DOI: 10.3389/fneur.2012.00011.
494. Williams, G., Clark, R., Schache, A., Fini, N. A., Moore, L, Morris, M. E., & McCrory, P. R. (2011). Training conditions influence walking kinematics and self-selected walking speed in patients with neurological impairments. *Journal of Neurotrauma, 28,* 281–287. DOI: 10.1089/neu.2010.1649.
495. Willis, C., Lybrand, S., & Bellamy, N. (2003). Excitatory amino acid inhibitors for traumatic brain injury (Review). *Cochrane Database of Systematic Reviews, 1*, 1-17. DOI: 10.1002/14651858.CD003986.pub2. <http://www.thecochranelibrary.com>
496. Willmott, C., & Ponsford, J. (2009). Efficacy of methylphenidate in the rehabilitation of attention following traumatic brain injury: a randomised, crossover, double blind, placebo controlled inpatient trial. *J Neurol Neurosurg Psychiatry, 80,* 552–557. DOI:10.1136/jnnp.2008.159632. *\*\*\*Used in Evidence Statement.*
497. Winter, J., Hunter, S., Sim, J., & Crome, P. (2011). Hands-on therapy interventions for upper limb motor dysfunction following stroke. *Cochrane Database of Systematic Reviews, 6,* 1-33. DOI: 10.1002/14651858.CD006609.pub2.
498. Wolf, S. L., Winstein, C. J., Miller, J. P., Taub, E., Uswatte, G., Morris, D.,… Nichols-Larsen, D. (2006). Effect of constraint-induced movement therapy on upper extremity function 3 to 9 months after stroke the EXCITE randomized clinical trial. *Journal of the American Medical Association, 296,* 2095-2104.
499. Wolf, S. L, Winstein, C. J., Miller, J. P., Thompson, P. A., Taub, E., Uswatte, G.,… Nichols-Larsen, D. (2008). The EXCITE trial: Retention of improved upper extremity function among stroke survivors receiving CI Movement Therapy. *Lancet Neurol., 7(1),* 33–40. *\*\*\*Used in Evidence Statement*
500. Wong, A. M. K., Lee, M. –Y., Kuo, J. –K., & Tang, F. –T. (1997). The development and clinical evaluation of a standing biofeedback trainer. *Journal of Rehabilitation Research and Development, 34(3),* 322-327.
501. Wong, V., Cheuk, D. K. L., Lee, S., & Chu, V. (2011). Acupuncture for acute management and rehabilitation of traumatic brain injury (Review) *Cochrane Database of Systematic Reviews, 5,* 1-35<http://www.thecochranelibrary.com>
502. Woodford, H. J., & Price, C. I. M. (2007). EMG biofeedback for the recovery ofmotor function after stroke. *Cochrane Database of Systematic Reviews, 2,* 1-26. DOI: 10.1002/14651858.CD004585.pub2.
503. Wortzel H. S, Filley, C. M, Anderson C. A, Oster, T, & Arciniegas, D. B (2008). Forensic applications of cerebral single photon emission computed tomography in mild traumatic brain injury. *The Journal of the American Academy of Psychiatry and the Law, 36,* 310-322.
504. Wortzel, H. S., Kraus, M. F., Filley, C. M., Anderson, C. A., Arciniegas, D. B. (2011). Diffusion tensor imaging in mild traumatic brain injury litigation. *The Journal of the American Academy of Psychiatry and the Law, 39,* 511-523.
505. Wu, H. M., Tang, J. L., Lin, X. P., Lau, J. T. F., Leung, P. C., Woo, J., & Li, Y. (2006). Acupuncture for stroke rehabilitation. *Cochrane Database of Systematic Reviews*, *3,* 1-25. DOI: 10.1002/14651858.CD004131.pub2.
506. Yardley, L, Beech, S., Zander, L., Evans, T., & Weinman, J. (1998). A randomized controlled trial of exercise therapy for dizziness and vertigo in primary care. *British Journal of General Practice, 48,* 1136-1140.
507. Yardley, L., Donovan-Hall, M., Smith, H. E., Walsh, B. M., Mullee, M., & Bronstein, A. M., (2004). Effectiveness of primary care-based vestibular rehabilitation for chronic dizziness*. Annals of Internal Medicine, 141(8),* 598-119.
508. Yardley, L. & Kirby, S. (2006). Evaluation of booklet-based self-management of symptoms in Meniere disease: A randomized controlled trial. *Psychosomatic Medicine, 68,* 762-769. DOI: 10.1098/0.1psy.0000232269.92.
509. Yeo, S. S., Kim, S. H., Kim, O. L., Kim, M. –S., & Jang, S. H. (2012). Optic radiation injury in a patient with traumatic brain injury. *Brain Injury, 26(6),* 891-895. DOI: 10.3109/02699052.2012.661119.
510. Yuh, E. L., Cooper, S. H., Ferguson, A. R., & Manley, G. T., (2011). Quantitative CT improves outcome prediction in acute traumatic brain injury. *Journal of Neurotrauma, 28(1),* 1-12. DOI: 10.1089/neu.2011.2008.
511. Zafonte, R. D. (2006). Update on biotechnology for TBI rehabilitation a look at the future. *Journal of Head Trauma Rehibilitation, 21(5),* 403-407.
512. Zafonte, R. D., Bagiella, E., Ansel, B. M., Novack, T. A., Friedewald, W. T., Hesdorffer, D. C.… Dikmen, S. S. (2012). Effect of Citicoline on functional and cognitive status among patients with traumatic brain injury Citicoline Brain Injury Treatment Trial (COBRIT). *Journal of the American Medical Association*, *308(19),* 1993-2000.
513. Zhang, L., Plotkin, R. C., Wang, G., Sandel, M. E., & Lee, S. (2004). Cholinergic augmentation with Donepezil enhances recovery in short-term memory and sustained attention after traumatic brain injury. *Archives Physical Medicine Rehabilitation, 85,* 1050-1055. DOI:10.1016/j.apmr.2003.10.014. *\*\*\*Used in Evidence Statement.*
514. Zygun, D. (n.d.). Commentary hypothermia in severe traumatic brain injury: Questions remain. *Journal of Critical Care.* 235-236.