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LMA

Traumatic Brain Injury in Adults

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Traumatic Brain Injury in Adults

The signs and symptoms of TBI vary extensively in severity and combinations of domains impacted, depending on the site and extent of injury to the neural substrate. Examples of physical, sensory, neurobehavioral, cognitive-communication, and swallowing effects of TBI are listed below.

Physical Effects

Physical effects resulting from TBI include

- changes in level of consciousness (ranging from brief loss of consciousness to coma);
- seizures;
- headaches;
- dizziness;
- nausea;
- vomiting;
- fatigue;
- reduced muscle strength (paresis/paralysis);
- impairments in movement, balance, and/or coordination, including dyspraxia/apraxia;
- motor programing deficits (dyspraxia/apraxia).

Visual Effects

Visual effects resulting from TBI include

- changes in visual acuity,

- double vision (diplopia),
- problems with visual convergence and accommodation,
- sensitivity to light,
- visual field deficits/visual neglect.

Auditory and Vestibular Effects

Auditory and/or vestibular effects of TBI include

- auditory dysfunction stemming from mechanical injuries to the outer ear (debris, tears, etc.); middle ear (ruptured tympanic membrane or ossicular disarticulation); and/or inner ear (cochlear injury, trauma to the cochlear nerve, disruption of the membranous labyrinth, and vascular compromise); and temporal lobe lesions;
- central auditory dysfunction;
- difficulty hearing speech in noise;
- hearing loss that may be transient or permanent;
- hypersensitivity to sounds (hyperacusis);
- tinnitus;
- dizziness, vertigo, and/or imbalance.

In blast injuries, the severity of auditory and/or vestibular effects may depend on the size of the blast, distance from the blast, orientation of the ear canal to the blast, and the environment (e.g., reflective surfaces or enclosed spaces).

Neurobehavioral Effects

Neurobehavioral effects resulting from TBI include

- affective changes, including over-emotional or over-reactive affect or flat (i.e., emotionless) affect;
- agitation and/or combativeness;
- anxiety disorder;
- depression;
- difficulty identifying emotions in others (alexithymia);
- emotional lability and mood changes or mood swings;
- excessive drowsiness and changes in sleep patterns, including difficulty falling or staying asleep (insomnia), excessive sleepiness (hypersomnia);
- feeling of disorientation or fogginess;
- increased state of sensory sensitivity accompanied by exaggerated response to perceived threats (hypervigilance);
- impulsivity;
- irritability and reduced frustration tolerance;
- stress disorders.

Cognitive-Communication Effects

Cognition and language are intrinsically and reciprocally related in both development and function. An impairment of language may disrupt one or more cognitive processes, and, similarly, an impairment of one or more cognitive processes may disrupt language. Cognitive-communication effects post-brain injury include difficulty with

- cognitive processes and systems (e.g., attention, perception, memory, executive function);
- verbal as well as nonverbal communication (e.g., listening, speaking, gesturing, reading, and writing) in all domains of language (phonology, morphology, syntax, semantics, and pragmatics).

Cognitive Deficits

Cognitive deficits resulting from TBI include

- attention deficits, including
  - reduced attention span (easily distractible),
  - difficulty with selective attention,
  - impaired sustained attention for task completion or conversational engagement,
  - deficits in shifting attention between tasks;
- executive function deficits, including difficulty with
  - goal setting,
  - strategy selection,
  - initiating and self-directing,
  - planning and organization,
  - reasoning and problem solving;
- information processing impairments, including
  - reduced processing speed and processing length (e.g., difficulty with longer messages and rapid rate of speech),
  - increased processing time for auditory and visual input (e.g., increased response latency when responding to questions in a conversation);
- memory and learning deficits, including
  - post-traumatic amnesia marked by impaired memory of events that happened either before (anterograde) or after (retrograde) the injury,
  - deficits in both retrieval of previously acquired knowledge and creation of new memory traces —long-term memory is often less impaired than short-term memory;
- impaired metacognition, including
  - deficits in subjective knowledge and insight into one's own cognitive processes,
  - reduced awareness of deficits (anosagnosia),
  - impaired self-monitoring,
  - poor self-regulation;

- deficits in orientation to self, situation, location, and/or time;
- impaired spatial cognition, including functional deficits in activities, such as navigation, driving, ambulation, dressing, and self-care (independent of any comorbid motor deficits).

Language Deficits

Along with the typical language comprehension and production deficits associated with co-existing aphasia, individuals with TBI may also present with linguistic deficits in the domains listed below (See aphasia for information related to neurogenic language deficits in adults):

- comprehension deficits, including
  - deficits in processing abstract language/concepts (e.g., figurative speech);
  - difficulty in interpreting the subtleties of conversation (e.g., humor, sarcasm);
  - impaired interpretation of nonverbal communication, such as tone of voice, facial expression, and body language;
  - increased auditory processing time;
- verbal expression deficits, including
  - anomia or word retrieval deficits;
  - difficulty with discourse, including
    - coherence, confabulatory speech,
    - content,
    - story grammar;
  - increased response latencies;
  - perseveration of verbal responses;
  - reduced word fluency;
- difficulty with pragmatics/social communication, including
  - conversational topic selection and maintenance marked by verbosity,
  - initiating conversation,
  - producing/interpreting nonverbal communication, such as facial expressions and body language,
  - turn taking,
  - using an appropriate tone of voice;
- reading deficits, including difficulty in reading comprehension, especially with complex syntax and figurative language (e.g., idioms, metaphors, similes);
- writing deficits that may mirror deficits in verbal communication—writing difficulty may also be a result of motor deficits in the dominant hand and/or visuospatial deficits.

Considerations for Bi/Multilingual Speakers

Cognitive control deficits uniquely impact linguistic abilities in bi/multilingual speakers (Ansaldo & Marcotte, 2007), especially in individuals with frontal lobe and subcortical lesions (Price, Green, & von Studnitz, 1999). Difficulty in maintaining output in the target language is strongly influenced by the speaker's premorbid proficiency in the two languages. In addition to the language production errors noted in monolingual speakers, bi/multilingual individuals with acquired brain injury may also demonstrate

- code-mixing errors,
- language-switching errors,
- semantic/phonological paraphasias produced in the nontarget language,
- translation errors.

Speech-language pathologists consider variations in narrative structures secondary to cultural and linguistic factors to ensure that a communication difference is not inaccurately diagnosed as a disorder.

Motor Speech Deficits

Motor speech deficits of TBI include

- apraxia of speech marked by inconsistent error patterns in phoneme production due to deficits in motor planning;
- aprosodia/dysprosodia, including deficits in intonation, pitch, stress, and rate, marked by monotonous verbal output;
- dysarthria characterized by reduced respiratory support, articulatory imprecision, and/or vowel distortions that impact speech intelligibility and resonance disorders secondary to paresis or paralysis of musculature of the speech motor system.

Voice Deficits

Voice deficits resulting from TBI include

- aphonia/dysphonia consequent to intubation, tracheostomy, or dependence on mechanical ventilation;
- laryngeal hyper/hypofunction marked by
  - abnormalities in pitch;
  - poor control of vocal intensity (excessive loudness or whisper);
  - changes in vocal quality, such as harshness, hoarseness, strained-strangled voice quality, and glottal fry;
- psychogenic (related to post-traumatic stress disorder) or neurogenic (related to injury to sensory or motor innervations of the vocal folds) phonatory abnormalities.

Dysphagia

Typically, swallowing disorders in TBI are neurogenic in nature, secondary to cortical or subcortical damage, resulting in oral/pharyngeal sensory disorders and/or motor deficits (e.g., weakness or paralysis of oropharyngeal musculature, oral apraxia). Cognitive impairments, such as poor memory, reduced insight, limited attention, impulsivity, and agitation, in TBI survivors may affect swallowing and increase aspiration risk (Logemann, 2006; Morgan, Ward, & Murdoch, 2004).