THE SCIENCE OF CONCUSSION

A special edition of **UPMC Restore**

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Michael Collins, PhD, is a stockholder with ImPACT Applications, Inc., and has participated in speaking engagements for the company. All other contributors have reported no relationships with entities producing health care goods or services.

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Advancements in Assessment, Management, and Rehabilitation

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Concussion by the Numbers

- Between 1.7 and 3 million sports- and recreation-related concussions happen every year. On average, 300,000 are football-related.
- Five of 10 concussions go unreported or undetected.
- 90 percent of concussions occur without loss of consciousness.
- One in 10 high-school athletes who play contact sports — including soccer and lacrosse — will suffer a concussion this year.
- Girls' soccer sees the second-most concussions of all high school sports. Girls' basketball sees the third-most.

The UPMC Sports Medicine Concussion Program sees 18,000 concussion patients each year:

- 30 percent are from outside the state of Pennsylvania.
- About 70 percent are high school-aged.

Over the past 10 years, the field of concussion management has evolved rapidly. From professionals to youth athletes, significant emphasis has been placed on accurate diagnosis, management and rehabilitation, and return-to-play decisions. Scientific evidence regarding variation in symptom presentation and recovery process, as well as data regarding pathophysiology of concussion, injury subtypes, risk factors, outcome, effects of repetitive injury, and treatment protocols, are moving the practice of concussion management toward individualized assessment, comprehensive symptom evaluation, and a more conservative management approach — especially in young athletes. This publication will discuss the diagnosis and management of concussion, multidisciplinary rehabilitation strategies, as well as the body of concussion research.

Definition of Concussion

In 2001, 2004, and 2008, experts from around the world met to discuss specific issues related to concussion at the International Symposia on Concussion in Sport. One result of these meetings was the adoption of the following definition of concussion.

Concussion is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Several common features that incorporate clinical, pathological, and biomechanical injury constructs that may be utilized in defining the nature of a concussive head injury include:

1. Concussion may be caused either by a direct blow to the head, face, neck, or elsewhere on the body with an "impulsive" force transmitted to the head.
2. Concussion typically results in the rapid onset of short-lived impairment of neurologic function that resolves spontaneously.
3. Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury.
4. Concussion results in a graded set of clinical symptoms that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course; however, it is important to note that in a small percentage of cases postconcussive symptoms may be prolonged.
5. No abnormality on standard structural neuroimaging studies is seen in concussion.

Due to the signs, symptoms, and impairments associated with concussion, the Centers for Disease Control and Prevention has developed a comprehensive definition of concussion, classifying the diagnosis as a traumatic brain injury (TBI):

> A mild traumatic brain injury (mTBI), or concussion, is defined as a complex pathophysiologic process affecting the brain, induced by traumatic biomechanical forces secondary to direct or indirect forces to the head. MTBI is caused by a jolt to the head or body that disrupts the function of the brain. This disturbance of brain function is typically associated with normal structural neuroimaging findings (i.e., CT scan, MRI). MTBI results in a constellation of physical, cognitive, emotional and/or sleep-related symptoms that may or may not involve a loss of consciousness (LOC). Duration of symptoms is highly variable and may last from several minutes to days, weeks, months, or longer in some cases.

Pathophysiology

Concussion in athletes is typically produced by acceleration/deceleration forces (e.g., helmet-to-helmet contact in football or helmet-to-ice contact in hockey), with the injuries occurring along a continuum of severity dependent on the mechanical forces that caused them.[1,2]

The complex cellular and vascular changes that occur following concussion have been described as a multilayered neurometabolic cascade.[3] The primary mechanisms include ionic shifts, abnormal energy metabolism, diminished cerebral blood flow (CBF), and impaired neurotransmission. A brief summary of this process, derived from several sources,[3,4] is provided in the following two paragraphs:

The stretching of an axon results in an indiscriminate release of neurotransmitters and uncontrolled ionic fluxes. When ionic gradients are disrupted, cells respond by activating ion pumps in an attempt to restore the normal membrane potential. Pump activation increases glucose use. This contributes to dramatic increases in the local cerebral metabolic rate for glucose. This hypermetabolism occurs in tandem with mildly decreased CBF, which contributes to the disparity between glucose supply and demand.

There also appears to be impaired oxidative metabolism and diminished mitochondrial function, resulting in the overuse of anaerobic energy pathways and elevated lactate as a by-product. Moreover, intracellular magnesium levels appear to decrease significantly and remain depressed for several days following injury. This is important because magnesium is essential for the generation of adenosine triphosphate (ATP; energy production). Magnesium is also essential for the initiation of protein synthesis and the maintenance of the cellular membrane potential. A sustained initial influx of Ca2 can result in mitochondrial accumulations of this ion

contributing to metabolic dysfunction and energy failure. High intracellular Ca2 levels, combined with stretch injury, can initiate an irreversible process of destruction of microtubules within axons.

Diagnosis

A concussion can be difficult to diagnose due to the wide variation in how the injury is sustained, as well as how and when symptoms present. Following the injury, there may or may not be obvious signs that a concussion has occurred, even though some athletes with mild concussion may experience one of approximately 20 classic symptoms, including headaches, confusion, memory loss, or balance problems. To further complicate the diagnosis, some athletes may mask or minimize an injury, or symptoms, to avoid being removed from play. Undiagnosed concussion can significantly impact an athlete's ability to function at a high level and increases their risk for suffering a second concussion before the first has healed. Repeated concussions, without proper recovery can lead to chronic effects of injury.

Given that concussion is largely a functional injury, careful symptom evaluation is the best way to establish a diagnosis. Techniques such as CT, MRI and EEG, are almost always normal following a single concussion; however, they can be employed to rule out other pathologies, including intracranial bleed, swelling, or skull fracture.

Symptoms

A thorough assessment of symptoms in four areas is critical:

- Cognitive: fogginess, difficulty concentrating, memory deficits, cognitive fatigue
- Somatic: headaches, dizziness, nausea, light/sound sensitivity
- Mood disruption: irritability, sadness, anxiety
- Sleep dysregulation: difficulty falling asleep, fragmented sleep, too much/too little sleep.

How these symptoms might be observed or reported is summarized in UPMC's sideline concussion card. (See Table 1, above)

TABLE 1

Signs/Behaviors Observed by Staff	Symptoms Reported by Athlete
Loss of consciousness	Feeling "foggy" or groggy
Forgets events prior to play(retrograde)	Change in sleep pattern (appears later)
Forgets events after hit (post-traumatic)	Feeling fatigued
Appears to be dazed or stunned	Headache
Is confused about assignment	Nausea
Forgets plays	Balance problems or dizziness
Is unsure of game, score, or opponent	Double or fuzzy/blurry vision
Moves clumsily	Sensitivity to light or noise
Answers questions slowly	Feeling sluggish or slowed down
Shows behavior or personality change	Concentration or memory problems

Fewer than 10% of the more than two million concussions annually include a loss of consciousness. The most commonly reported symptoms are headaches and dizziness. Although a headache might not be present when a concussion has occurred, assessing this symptom can be made more difficult by existing headache conditions. However, a "concussion headache" is often uniquely described as pressure in the skull that may be located to one region, or more generalized.

Risk Factors

The diagnosis of concussion does not have some of the widely known established risk factors found in other conditions, like family history. Future research may focus on discerning additional risk factors, as well as those factors that could indicate length of recovery period. Current research indicates the following increase concussion risk, or impact symptom presentation and recovery time:

- Participation in high risk sports, such as soccer, hockey, or football
- Lack of proper, or properly fitted and worn, safety equipment
- Gender: Females seem to present with more symptoms that persist

- Previous concussion(s)
- Age: Athletes under 18 have been shown to exhibit longer recovery times when compared to college and professional athletes
- History of learning disabilities
- History of migraine headaches
- Returning to play too quickly
- Repeat concussion (occurring before a patient has recovered from the first one); this also increases the chances of long-term effects of injury.

Neuropsychological Assessment

The use of neurocognitive testing has advanced quickly in recent years and can be a valuable tool in assessing recovery from concussions. Together with comprehensive symptom evaluation and management, these scores also can be factored into return-to-play decisions.

The assessment tool most accepted and with the largest database is the ImPACT® Test. The ImPACT Test is used by the NFL, NHL, and other professional sports organizations, as well as more than 10,000 high schools and 1,000 colleges. ImPACT was developed by clinicians from UPMC and is currently headquartered in Pittsburgh, Pa.

There are two parts to the test, the Symptom Score component and the six part neurocognitive test component. Both component scores should return to baseline or normal before an athlete is allowed to resume playing a contact sport.

Generally, the symptoms of a concussion disappear before the neurocognitive findings return to normal, although occasionally, this can be reversed.

Migraine symptoms following concussion, as opposed to the usual generalized headache of a concussion, are predictive of poorer scores on ImPACT testing, and are associated with a longer recovery time. The components measured by the test — verbal and visual memory, processing speed, and reaction time — assess different aspects of brain function to determine the number of deficits/symptoms affecting the patient.

Individuals who score well below expected levels of function initially should be monitored very carefully as they will usually take longer to heal and may be more prone to developing Post-Concussion Syndrome. Initial scores, and changes in scores, also will help indicate which patients need extended activity modification or therapies.

Therapies Used in Concussion Treatment

In the days and weeks following a concussion, the brain undergoes a dynamic restorative process. The ionic shifts, abnormal energy metabolism, diminished CBF, and impaired neurotransmission associated with sports concussions support the importance of immediate rest — physical and cognitive — following the injury. During this initial recovery period, vigorous exercise, intensive academic work, or blows to the head or body could exacerbate the pathophysiology of the injury.

Many patients recover from a concussion with rest, over the course of a few weeks; however, up to 20% of athletes will experience more chronic and lingering symptoms beyond a few weeks. In these cases, the recovery period may be protracted since additional therapies are needed to support the patient's rehabilitation. These interventions are called for if the patient shows no marked progress at three weeks post-concussion.

Concussion rehabilitation therapies often include vestibular, ocular, medical, and exertional therapies. Used in combination, and over a period of time, these therapies help the brain recover from the pathophysiologic abnormalities, and allow the patient to return to their regular activities.

Vestibular Rehabilitation Following Concussion

The primary functions of the vestibular system are to stabilize vision while the head moves, and to provide information to help maintain balance. The brain and the peripheral organ that is housed in the bony labyrinth of the inner ear comprise the vestibular system. If one or both of these components are compromised as a result of concussion, the patient's vestibular system may be affected, and vestibular rehabilitation may be necessary.

Vestibular therapy is a branch of neurological physical therapy that addresses post-concussion symptoms such as dizziness, difficulty focusing, and difficulty maintaining balance. Simple activities such as moving quickly in the environment can be compromised after concussion due to issues with the vestibular and ocular systems. Patients with protracted recovery often have vestibular dysfunction caused by several factors: posttraumatic benign paroxysmal positional vertigo, a co-existing labyrinthine concussion, posttraumatic migraine related dizziness, and central vestibular dysfunction from a brainstem concussion.

Patients typically present with one or all of the following symptoms: dizziness (55% of all patients), visual blurring and double vision (49% of all patients), and disruptions in balance (43% of all patients). Difficulty focusing, motion discomfort, height phobia, and difficulty functioning in busy environments also are symptoms that often point to vestibular dysfunction.

It is important to screen for vestibular symptoms at the first subacute assessment of the patient. Screening should consist of a thorough patient interview in which a variety of qualitative information is obtained, as well as ocular-motor testing, vestibular-ocular testing, and evaluation of balance.

Common Ocular-Motor Tests

- "H-Test" — Smooth Pursuits
- Vertical/Horizontal Saccades, paying special attention to any dizziness, blurriness, and over/under shoots that may occur.

Vestibular-Ocular Tests

- Vertical/Horizontal Gaze Stability, observing any nystagmus, provocative dizziness/blurriness, and slowed movements
- Optokinetic Sensitivity
- Ocular Convergence and Accommodation; near point of conversion should be less than 6cm.

Balance Examination

- Romberg
- Patient Standing on Foam Cushion with eyes open and closed, arms folded. Results are abnormal if patient cannot execute for 30 seconds, or if there is excessive sway.

Based on evaluation results, patients are given a variety of therapies to be completed at home daily and are seen by therapists at regular intervals in order to assess progress and make adjustments in the treatment plan. While time varies from patient to patient, patients are typically discharged after five to six visits to vestibular therapy. Vestibular measures for success include:

- Self-reporting — Patient is confident in their ability to balance and no longer reports dizziness.
- Clinical testing — Patient is in the normal range.
- No vestibular problems reported during exertional and cardiovascular screenings.

The Role of Exertional Therapy in Concussion Rehabilitation

Exertional therapy takes patients through a variety of physical activities, the goal of which is a return to life prior to concussion. Using a variety of aerobic activities, strengthening exercises, and stretching/range of motion (ROM) drills, exertional therapy rehabilitates the vestibular system to allow therapists to gauge a patient's progress as they work to return to a preconcussive state.

At the initial visit, the therapist screens for vestibular issues. This exam consists of an aerobic conditioning circuit, a dynamic circuit (lunges, squats, and rotations), and a functional circuit (running with head turns, steps, side to sides, jumping up to catch a ball).

It is also important to evaluate the patient for anxiety at this time. If a patient is anxious about their condition or their ability/inability to function, they may perceive that their symptoms are not improving or getting worse. Medications

TABLE 2

Stage	Target Heart Rate	Activities	Recommendations
Stage One	30-40% of maximum exertion	Very light aerobic conditioning, sub-max strength training, ROM/stretching, very low level balance activities	10 to 15 minutes of cardio only, low stimulus environment, no impact activities, limit head movement/ position change, limit concentration
Stage Two	40-60% of maximum exertion	Moderate aerobic conditioning, light weight strength exercise, stretching (active stretching initiated), low level balance activities	20 to 30 minutes of cardio, exercise in gym areas, use various exercise equipment, allow some positional changes and head movements, low level concentration activities
Stage Three	60-80% of maximum exertion	Moderately aggressive aerobic exercise, all forms of strength exercise (80% max), active stretching exercise, impact activities such as running, plyometrics (no contact), challenging proprio-balance activities	Any environment permitted for exercise (indoor or outdoor), integrate strength, conditioning, and balance/proprioceptive exercise; incorporate concentration challenges
Stage Four	80-90% of maximum exertion	Non-contact physical training, aggressive strength exercise, impact activities/plometrics, sports-specific training	Avoid contact activity, resume aggressive training in all environments (indoor and outdoor)
Stage Five	Full exertion	Full training activities with contact, continue aggressive strength/conditioning exercise, sport-specific activities	Initiate contact activities as appropriate, full exertion for sport

to ease anxiety are available and may be worthy of discussion at this time. In fact, UPMC encourages patients to begin exertional therapy as early as possible to avoid feelings of anxiety and depression.

Exertional therapy consists of a cardio workout consisting of aerobic conditioning, strength exercises, ROM/stretching, and balance activities. Newly diagnosed patients (Stage 1) often perform for a shorter period of time. As patients recover, heart rates and duration are increased (Stage 5). The goal for return to work/play/sport is completion of 25 minutes of exertional therapy without interruption.

Stages are shown in Table 2, above.

Before returning to work/sport/play, it is important that the patient completes all five stages of exertional therapy to help ensure all vestibular issues are addressed and have returned to preconcussive levels.

The Role of Medical Therapy in Concussion Rehabilitation

Over time and with significant rest, the brain will often repair abnormalities on its own. In some cases, however, medical intervention is necessary. Typically, if in three weeks following the event, the patient exhibits little or no improvement, he/she should be referred for medical therapy.

Medical therapy for concussion focuses on four areas:

- Neuropsychiatric symptoms (anxiety, depression, nervousness, emotional instability, irritability)
- Migraine and somatic symptoms (visual challenges, dizziness, light/noise sensitivity)
- Cognitive slowing (memory dysfunction, fogginess)
- Sleep dysregulation/disturbance.

Neuropsychiatric Symptoms

Pharmacotherapy varies based on the patient's phase of concussion recovery. SSRIs, such as escitalopram (Lexapro®) and sertraline (Zoloft®) are often prescribed for patients several months into recovery. If anxiety presents and is coupled with vestibular issues, a low dose (0.25 mg/2Xday) of clonazepam (Klonopin®) may be prescribed. Behavior modification therapy also can be incorporated into concussion rehabilitation if neuropsychiatric symptoms persist. For newly diagnosed patients, a medical consultation may occur at the first evaluation but they may not be placed on any drug right away as patients can recover on their own.

Migraine and Somatic Symptoms

Patients presenting with persistent headache can be placed on a course of amitriptyline. While this is classified as a tricyclic antidepressant, UPMC uses it for its migraine prophylaxis capabilities. Additionally, it can induce drowsiness, which will help regulate sleep and allow the brain to rest.

In adults or children over 12, a typical course may be:

1. 10mg in the evening/3 days, then
2. 20mg in the evening/3 additional days, then
3. 30mg in the evening/3 additional days

A course of amitriptyline therapy may take at least four weeks to see its full effect, after the patient reaches full dosing of 30mg. There are a number of special circumstances that affect dosage, especially in children, and patients should be monitored for mood changes, especially those under age 19. This population appears more susceptible to black box warnings on antidepressant medications.

Other headache prophylactics include: propranolol, topamax, verapamil, escitalopram, valproic acid, gabapentin, and sertraline. Vestibular therapy also should a be employed to reduce headache symptoms, if warranted.

Cognitive Slowing

The UPMC Sports Medicine Concussion Program has completed several studies investigating the off-label use of amantadine as a neurostimulant for concussed patients who experience symptoms of fogginess or an inability to focus. Originally developed as an antiviral for the flu, this drug affects the dopamine receptors in the brain and works on pre and post synaptic receptors, increasing the patient's ability to focus, feel less tired, and reduce the feeling of cognitive slowing. Standard dosing is:

- 100mg at breakfast for five days, then
- 100mg at breakfast and lunch

The standard course of amantadine therapy is four to six weeks, however some patients may require a longer course. Patients should be seen regularly for evaluation and medication adjustment.

Under special circumstances, more traditional neuro-stimulants, such as methylphenidate, dextroamphetamine or amphetamine-salts can be utilized (although off-label) to minimize cognitive fatigue and improve concentration.

Sleep Dysregulation/Disturbance

Sleep dysregulation is often an unwanted co-traveler of concussion. The first line of therapy should be a discussion of the importance of good sleep hygiene. Physicians should recommend that patients go to bed at the same time and wake up at the same time every day. This often helps regulate their sleep cycle and eliminates the need for sleep aids.

Should sleep disturbance persist, 3mg of melatonin can be recommended. Like other sleep medicines, this should not be used on a daily basis. In some cases, trazadone (100mg), Ambien,® or Restoril® also can be prescribed. It is important, however, to know how these drugs work in concert with other medications the patient may be taking and to take into consideration the side effects and addictive complications sometimes related to these drugs.

The course of pharmacotherapy significantly varies from patient to patient. It is important that patients are monitored every four to six weeks. In some cases, four to six weeks of drug therapy may be adequate; if the patient also has neuropsychiatric symptoms, he or she may need to stay on the sleep medicine for a slightly longer period of time. Cognitive therapy also may be added if sleep dysregulation persists.

Decisions for return to work/play/sport are made in conjunction with other team members based on close evaluation of ImPACT testing and other clinical evaluations.

The Role of Ocular Therapy in Concussion Rehabilitation

For some patients, specialized ocular therapy is needed in order for recovery to progress. This therapy is coordinated by an optometrist who specializes in neuro-optometry. Through the use of a specialized computer and optical devices, ocular therapy seeks to correct visual-motor or perceptual-cognitive deficiencies. As the patient improves, the visual skills are reinforced through repetition, and integrated with cognitive and motor skills until they become automatic. Vision therapy sessions include procedures designed to enhance the brain's ability to control eye alignment, teaming, and movements, as well as focusing abilities and visual processing.

Return to Play

Return-to-play decisions are an important aspect of concussion management, and should take into consideration all aspects of the concussion diagnosis and recovery period. Athletes should not be returned to play until they are symptom free with both physical and cognitive exertion, and neurocognitive functioning has returned to baseline levels or estimates of premorbid functioning.

Patients should have no symptoms when resting — with full academic or professional activities — and during noncontact exertion. Once asymptomatic during these activities, patients should proceed through increasing amounts of noncontact physical exertion and noncontact sport-specific training. Finally recovery of full neurocognitive functioning should be exhibited — typically through performance on a neurpsychological testing battery.

As with establishing a diagnosis of concussion, comprehensive symptom assessment, and individualized treatment plans are crucial to determining whether a patient is fully recovered and can return to play. These evidence-based models are becoming the mode for best practices, and they will continue to evolve through ongoing research, larger clinical trials, and the development of more advanced techniques for detecting and diagnosing concussion, managing concussion rehabilitation, and determining length of recovery period and the appropriate time to return to play.

Research

The UPMC Sports Medicine Concussion Program understands the importance of evidence-based research. Our legacy includes developing the most widely used neurocognitive testing tool to assess concussion (ImPACT), helping to refine return-to-play guidelines, and creating personalized treatment plans that cross a variety of clinical specialties. Our commitment to quantitative research has made the UPMC Sports Medicine Concussion Program an international leader in the area of concussion management and our standard of care is now adopted at facilities throughout the United States. Our findings have led to better assessment and education, and are helping to establish new treatments and approaches to post-concussion rehabilitation that may help patients return to their a preconcussive state.

A sample of recent research can be found by visiting **UPMCPhysicianResources.com/UPMCConcussionResearch**.

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Case Studies

15-year-old softball player

Overview: A 15-year-old fast-pitch softball player sustained a concussion in July 2012. She was batting and received a pitched ball to the posterior region of her helmeted head. No acute signs or symptoms were reported, and she continued to play. Onset of headaches and fatigue occurred approximately one hour later. She was first seen in clinic in August 2012. At the time, she was restricted from activity by her pediatrician and was reporting continued headaches daily, that were exacerbated by reading and visual activity. She also reported continued fatigue and memory problems. Her past medical history was unremarkable for concussion or migraine headache. She was identified as an average student with no history of attention deficit or learning problems. Neurocognitive testing was initially below expectation in the low average range. No vestibular concerns were noted during initial screening.

Treatment: When first seen in clinic, she was approximately one month post-injury and still experiencing fairly significant headaches and fatigue that increased with cognitive exertion. Given the duration since injury and nature of symptoms, she was referred for medication and placed on amantadine. The medication made school work more tolerable and reduced the frequency and duration of headaches. In October 2012, she was demonstrating neurocognitive improvements with scores generally in the average range. She began to increase her level of non-contact physical exertion beginning with light static activity and progressing to heavy cardiovascular exertion. She was supervised by physical therapists throughout the exertion process, and was eventually advised her to begin some non-contact softball specific training with her team. She remained on amantadine until she was able to achieve moderate to heavy non-contact exertion with no symptoms, eventually weaning off the medication in November 2012.

Outcome: She continued with structured physical exertion as well as off-season softball conditioning with her team. She was able to engage in these activities with no return of symptoms throughout the month of December, and was eventually seen in clinic in early January 2013 for clearance. At the time, her neurocognitive testing was in the average to high average range. She reported no difficulty with physical exertion and was performing well academically. Since she was asymptomatic with heavy physical exertion and demonstrated neurocognitive functioning within her expected baseline range, she met international criteria to return-to-play, and was cleared for full softball participation. *(see ImPACT® Clinical Report A below)*

ImPACT® Clinical Report A: 15-year-old softball player

Exam Type	Post-Injury 1	Post-Injury 2	Post-Injury 3	Post-Injury 4	Post-Injury 1
Date Tested	8/22/12	9/12/12	10/30/12	11/21/12	1/9/13
Last Concussion	7/30/12				
Composite Scores	Percentile scores if available are listed in **red type**				
Memory composite (verbal)	76 17%	76 17%	75 15%	73 11%	93 71%
Memory composite (visual)	57 10%	70 43%	71 47%	76 58%	86 88%
Visual motor speed composite	29.23 10%	33.2 22%	29.98 11%	34.5 28%	41.88 62%
Reaction time composite	0.67 16%	0.65 22%	0.73 7%	0.61 38%	0.56 51%
Impulse control composite	4	2	3	4	4
Total Symptom Score	13	20	8	0	0

28-year-old nurse

Overview: A 28-year-old nurse sustained a concussion in December 2011 when she fainted during a break at work. She struck the right frontal area of her face and head on the ground with 30 to 60 seconds loss of consciousness. She was taken to the hospital's emergency room where a CT scan indicated no intracranial abnormalities. Initial symptoms included persistent headaches, fatigue, hypersomnia, dizziness, and anxiety in busy environments. She was first seen in clinic in February 2012. At the time, she was missing a significant amount of work due to continued headaches, dizziness, and fatigue. She demonstrated neurocognitive functioning in the low average to impaired range. Vestibular screening was notably provocative for dizziness and discomfort when examining gaze stability, saccades, and VOR cancellation. Visual accommodation was also notably outside of normal limits. Her medical history was significant for migraine headaches and previous fainting spells. Previous medical tests ruled out seizure disorder and cardiovascular problems as causes for her fainting spells.

Treatment: Initial treatment included removal from work in order to facilitate recovery, as well as prescription of amitriptyline, which was modestly effective in controlling her headaches. She also began vestibular therapy with exercises focusing on gaze stability and visual accommodation. She also received some adjustments for BPPV. Her medication was changed from amitriptyline to amantadine in May 2012, around which time she began to experience a significant reduction in headaches and overall improvement in functioning. Her neurocognitive testing also improved and was generally in the average to high average range. Given her improvements, a gradual return-to-work plan was developed, which included progressively increasing the number of hours worked, as well as the task demands of her position. She was quite apprehensive about returning to work. She remained on amantadine and was eventually transferred from vestibular to vision therapy in order to focus specifically on her lingering accommodative insufficiency.

Outcome: She gradually transitioned back to a full-time work schedule during June to October 2012. Given her level of anxiety and apprehension about returning to work, a gradual and structured plan was beneficial to her successful transition. She discontinued all medication and completed vision therapy in approximately September 2012. Her neurocognitive testing was stable and generally in the high average range throughout the last two months of treatment. At the time of discharge, she continued to report occasional migraine headaches up to a couple times per month. However, she had a migraine history and felt that her headaches had returned to baseline levels. *(see ImPACT® Clinical Report B below)*

16-year-old football player

Overview: A 16-year-old football player sustained a concussion in October 2012. While playing in a game as backup quarterback, he was struck early in the first quarter and experienced some unilateral blurriness in his left eye as well as mild headache. He continued to play, and later in the fourth quarter he was struck in the posterior region of his helmet on a blindside tackle. Immediate symptoms included bilateral blurred vision, headache, and photosensitivity. Upon evaluation at the hospital, he exhibited significant disorientation and confusion, which necessitated a CT scan that was negative. At the time of his first evaluation in clinic he had not been trialed on any medications and had not had any therapies. The primary intervention had been academic accommodations in the form of half days of school. His primary complaints at the time were fatigue, headaches, dizziness, fogginess, and concentration problems. His past medical history was unremarkable for concussion or migraine headache but notable in that he is a cancer survivor. He was identified as an above-average student. Neurocognitive testing indicated impaired verbal memory, visual motor speed, and reaction time. Visual memory was borderline impaired. Vestibular screening was provocative for dizziness and there was a notable convergence insufficiency at 16cm.

Treatment: He was approximately one month post-injury when first seen in clinic. Given the nature of his symptoms, he was referred for vestibular therapy, vision therapy, and medication management. Homebound instruction was also initiated as the primary academic intervention. He was placed on amantadine which helped reduce the severity of his headaches and made school work more tolerable. By his second evaluation, approximately one month later, he reported feeling somewhat better. He also demonstrated neurocognitive improvement in all areas. He began to expose himself to the academic setting and was attending school for two

ImPACT® Clinical Report B: 28-year-old nurse*

Exam Type	Post-Injury 1		Post-Injury 2		Post-Injury 3		Post-Injury 1		Post-Injury 3	
Date Tested	2/23/12		4/16/12		5/21/12		7/26/12		10/22/12	
Last Concussion	12/5/11									
Composite Scores	Percentile scores if available are listed in **red type**									
Memory composite (verbal)	55	<1%	75	15%	83	43%	90	73%	93	82%
Memory composite (visual)	60	22%	49	6%	73	47%	81	80%	75	55%
Visual motor speed composite	33.28	17%	37.63	39%	44.08	74%	44.63	75%	51.65	99%
Reaction time composite	0.83	<1%	0.65	23%	0.56	65%	0.52	85%	0.49	94%
Impulse control composite	3		4		6		4		8	
Total Symptom Score	68		47		33		21		1	

*Represents a sampling of this patient's clinical reports.

classes in conjunction with homebound instruction. At this time he was also referred for exertion therapy to help him become more physically active.

Outcome: At the time of his most recent evaluation, he was continuing to progress in his recovery. He had increased his attendance in school to three periods and was attending basketball practice and doing light conditioning. His neurocognitive data was continuing to improve with average visual memory, low average verbal memory and visual motor speed, and borderline reaction time. His vestibular examination was within normal limits, as was his visual accommodation and convergence. He reported experiencing headaches, fogginess, and dizziness and acknowledged not being fully compliant with his vestibular exercises. It was recommended that he continue in his therapies and increase his physical exertion in the form of non-contact conditioning with his football team. The importance of attending school more regularly was also discussed and it was recommended that he add another class to attend, with the goal of being in school full-time by the start of the next semester. Finally, he expressed anxiety about his ability to attend school and navigate its complex environment. It was recommended that he discuss this with his prescribing clinician to determine whether he might benefit from medication. *(see ImPACT® Clinical Report C above)*

ImPACT® Clinical Report C: 16-year-old football player

Exam Type	Post-Injury 4		Post-Injury 3		Post-Injury 2	
Date Tested	11/13/12		12/4/12		1/8/13	
Last Concussion	10/2/12					
Composite Scores	Percentile scores if available are listed in **red type**					
Memory composite (verbal)	54	<1%	71	10%	73	13%
Memory composite (visual)	54	6%	68	26%	71	35%
Visual motor speed composite	15.93	<1%	22.78	<1%	30.73	9%
Reaction time composite	1.49	<1%	1.04	<1%	0.72	8%
Impulse control composite	11		9		8	
Total Symptom Score	73		28		41	

*Patient rehabilitation continues; he has not be released at the time this publication was printed.

13-year-old football player

Overview: A 13-year-old football player sustained a concussion in October 2012. While running back a kickoff, he was tackled and struck his head on the ground. He denied onset of symptoms during the game, but noticed headache, disorientation, and difficulty maintaining attention during the bus ride home. He was managed by his pediatrician at first, but was eventually referred to clinic in early December 2012. At that time, he was taking amantadine (100mg at breakfast; 100mg at lunch), which he felt was beneficial to his headache presentation and improved cognitive functioning initially, but was not noticing significant benefit after approximately one month on the medication. His primary complaints at the time of his first evaluation were headaches and visual strain, as well as

some mild dizziness. His neurocognitive testing indicated intact verbal and visual memory, with scores in the average range. However, his visual motor speed and reaction time were low average to borderline. Convergence and accommodation of the eyes were measured at approximately 10 to 12cm, indicating mild insufficiency. He was referred for vestibular therapy.

Treatment: He progressed quickly with vestibular therapy. His exercises focused on the mild convergence and accommodative insufficiency that was observed during initial evaluation. His symptoms diminished, with mild headaches continuing through the end of December. Given his improvements, he began weaning off the amantadine, reducing to 100mg at breakfast, and fully weaned off the medication by early January 2013. His vestibular therapists were also able to incorporate some light to moderate physical exertion into his therapy program. He hoped to return to wrestling in January 2013. He was evaluated via computerized gaze stability testing in mid-January, demonstrated performance within normal limits, and was cleared from vestibular therapy. Convergence and accommodation of the eyes also returned to normal range at approximately 6cm. Neurocognitive testing improved and was predominantly average to high average during both evaluations in January 2013.

Outcome: With his successful completion of vestibular therapy and improvements in neurocognitive functioning, he was allowed to continue with wrestling conditioning. He was able to increase his exertion to heavy conditioning that included running, jumping rope, push-ups, and sit-ups during wrestling practice. He was able to tolerate heavy exertion with no return of symptoms, and, coupled with baseline neurocognitive functioning, met international criteria to return-to-play. Given the high likelihood of contact in wrestling, it was recommended that he first engage in some controlled contact in practice under the supervision of his athletic trainer. He was able to tolerate controlled levels of contact in practice and has successfully returned to competition. *(see ImPACT® Clinical Report D below)*

ImPACT® Clinical Report D: 13-year-old football player

Exam Type	Baseline		Post-Injury 4		Post-Injury 3		Post-Injury 4		Post-Injury 1	
Date Tested	7/21/10		11/8/12		12/11/12		1/8/13		1/24/13	
Last Concussion					10/3/12					
Composite Scores	Percentile scores if available are listed in **red type**									
Memory composite (verbal)	83	50%	79	36%	91	80%	100	100%	91	80%
Memory composite (visual)	80	72%	54	7%	82	72%	88	86%	83	75%
Visual motor speed composite	21.55	<1%	21.75	<1%	31	21%	31.92	25%	30.92	19%
Reaction time composite	0.62	77%	0.76	6%	0.72	10%	0.65	28%	0.59	56%
Impulse control composite	8		9		10		10		9	
Total Symptom Score	0		17		19		2		0	

Scores in **green type** exceed the Reliable Change Index (RCI) when compared to the baseline score. However, scores that do not exceed the RCI index may still be clinically significant.

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