Dr. R. Garth Smith

KidsInclusive Centre for Child & Youth Developement

Dr. R. Garth Smith is a full-time Associate Professor of Pediatrics at Queen's University and a Developmental Pediatrician.

He is a graduate of the University of the West Indies and completed his residency training at the Hospital for Sick Children in Toronto in 1986. He did some additional Developmental Pediatric training at Chapel Hill, North Carolina. After working in Private Practice in Southwest Ontario for 9-years, he moved to Kingston to the Child Development Centre (now KidsInclusive Centre for Child & Youth Development) as a Developmental Pediatrician, and subsequently Medical Director. Since 1996, his work has included the assessment and treatment of autism spectrum disorders, acquired brain injuries, neuromuscular disorders and children with hearing impairment, among other neurobehavioral disorders.

He is an active educator of medical students and residents on developmental topics and a past member of the Examinations Committee of the Royal College of Physicians and Surgeons of Canada. He has presented at numerous conferences across Ontario in his areas of interest.

His research interests include the diagnosis and treatment of anxiety disorders in autism spectrum disorders, quality of life in children and youth with multiple concussions, and other.

Dr. Smith enjoys collecting and breeding Malawi Lake African Cichlids and watching English Premier League Soccer and other sports.

Adolescent Brain Injury - They Look Fine but is Seeing Believing?

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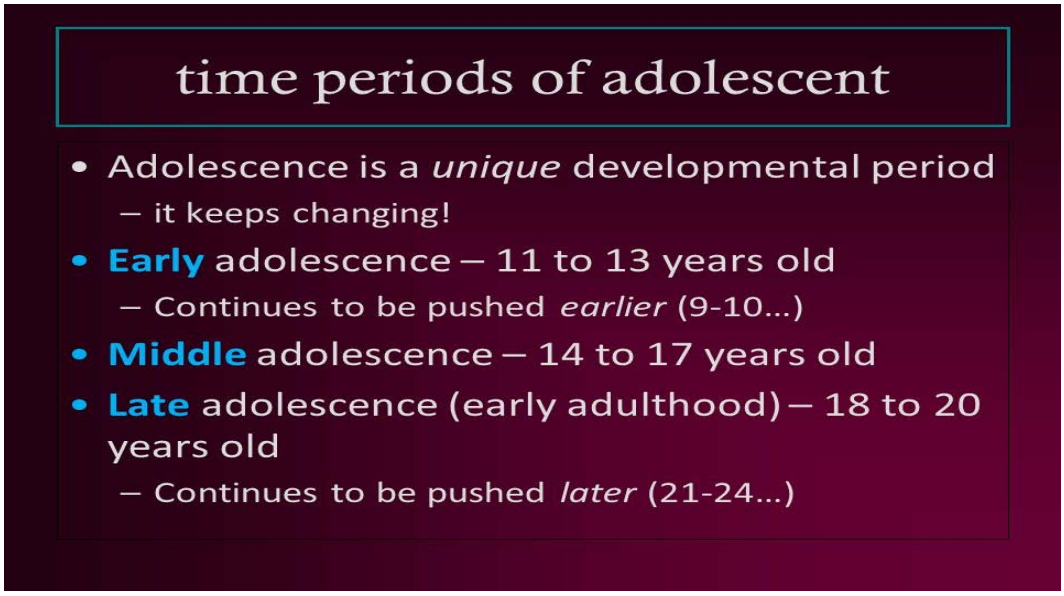
Objectives:

By the end of this presentation, attendees should:

- Appreciate the wide ranging impact of traumatic brain injury (TBI) on children and adolescents
- Recognize that, mild TBI's (concussions) can be extremely impactful
- Be aware that ADOLESCENTS are particularly at risk, and why
- Know the "protective" factors, and vulnerabilities
- Recognize that a multidisciplinary approach to management is essential to optimal recovery

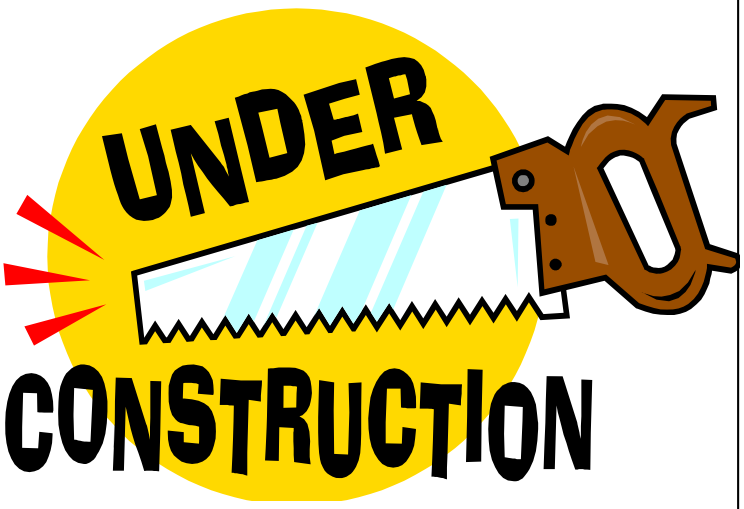
A Brief Look at Adolescent Development



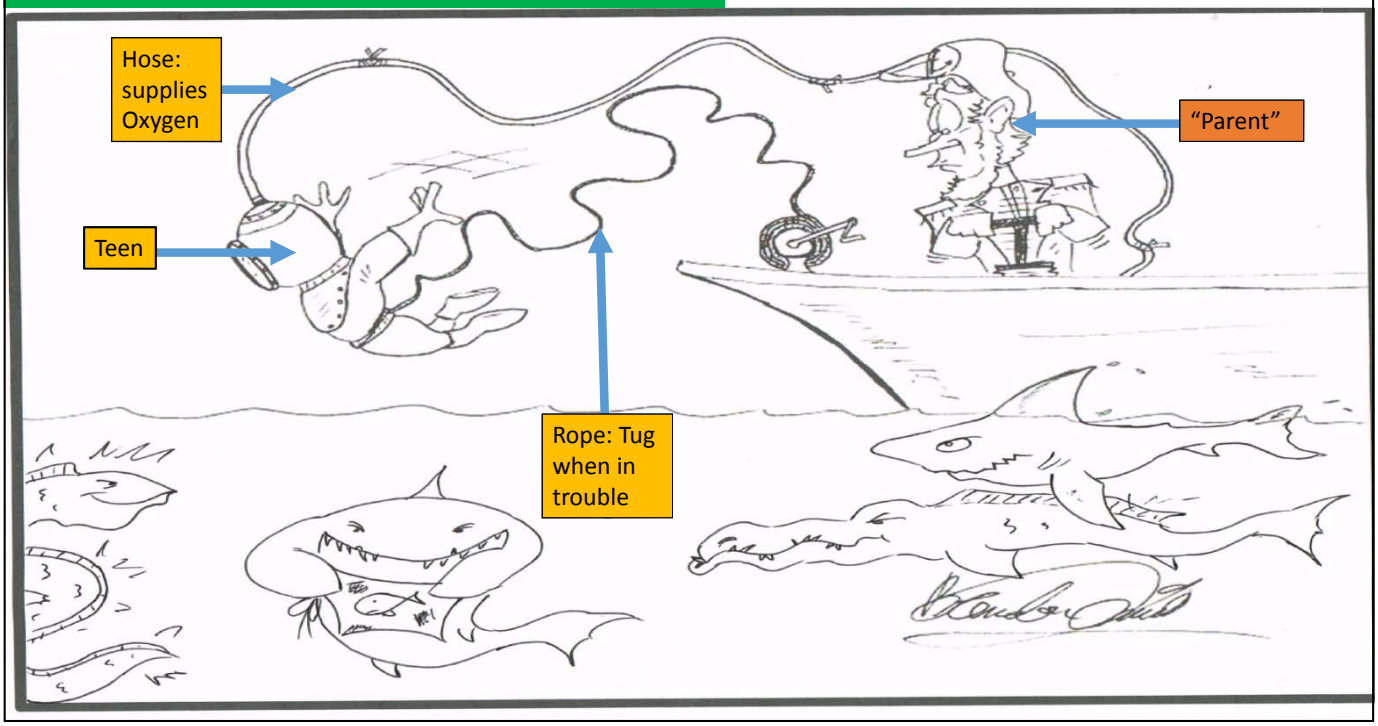
A Brief Look at Adolescent Development

Bottom Line? Adolescent Brain Development

- Develops from BACK to FRONT
  - Physical coordination (cerebellum)
  - Emotion (Amygdala-midbrain)
    - Passionate, committed, but over-reactive
  - Motivation (Nucleus Acumbens-midbrain)
    - Not always directed at best priorities
  - Judgement/Reasoning (Prefrontal Cortex)
    - Decisions, impulse control, forethought, planning



Struggles of Teenager-hood: Like a Deepsea Diver



When I was a boy of fourteen, my father was so ignorant I could hardly stand to have the old man around. But when I got to be twenty-one, I was astonished by how much he'd learned in seven years.

—MARK TWAIN

Definition of TBI

- "An acquired injury to the brain caused by an external physical force, resulting in temporary or permanent impairment of cognitive, physical or psychosocial functional or psychosocial impairment, or all three, often, but not always associated with a diminished or altered state of consciousness
- May be caused by bump, blow or jolt directly or indirectly to the head, OR a penetrating brain injury"

Adapted from CDC, IDEA definitions

Definition of Concussion

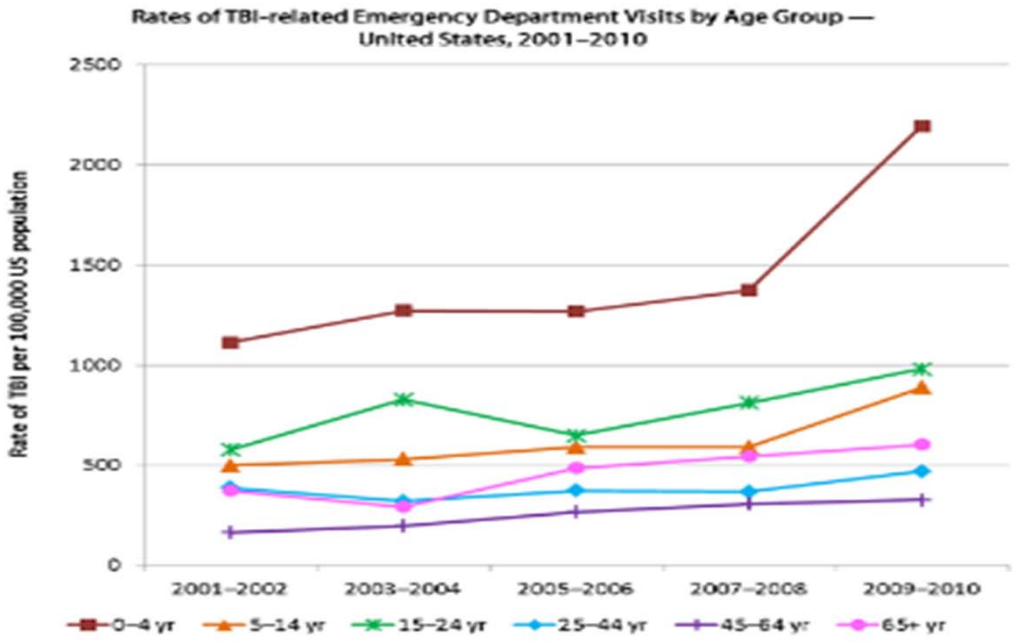
- A direct or INDIRECT blow to the brain resulting in changes in normal brain function, or deterioration of pre-existing brain dysfunction.

The Extent of the Problem:

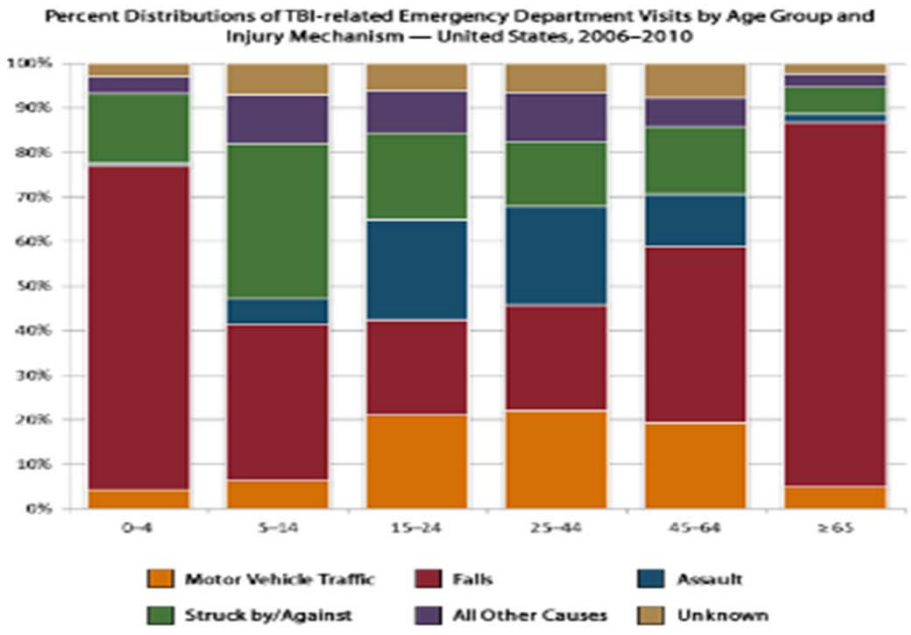
The Extent of the Problem:

- The majority (i.e., over 75%; Sosin, Sniezek, & Thurman,1996) of TBIs occurring in children are mild, and caused by…
  - falls,
  - transportation/motor vehicle collisions, and
  - sports-related injuries (Yeates, 2010)

Yeates, K. O. (2010). Traumatic brain injury. In K. O. Yeates, M. D. Ris, H. G. Taylor, & B. F. Pennington (Eds.), Pediatric neuropsychology: Research, theory, and practice (2nd ed.) New York, NY: Guilford Press.



From CDC website



From CDC website

Pathophysiology of Concussion

- A concussion may be caused by either a direct blow to the head (contact forces) or by a blow to elsewhere on the body with the forces being subsequently transmitted to the brain (inertial forces) (McLean, 1996; Teasdale and Matthew, 1996).
- Rotational forces around a defined axis are thought to be responsible for damage to deep white matter tracts, resulting in a diffuse axonal injury as well as causing damage to deep gray matter nuclei (McLean, 1996; Thibault and Gennarelli, 1990)

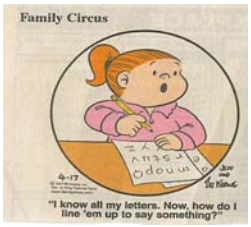
Cognitive Problems

- Problem solving
- Comprehension of abstract language
- Word retrieval
- Expressive language organization
- Pragmatics

(Yeates et al., 2002)

- Executive functions
- Memory
- Attention
- Concentration
- Information processing
- Sequencing

Work more effortful; more cognitive fatigue



Cognitive Problems

- Tend to improve slowly (6-18 months) with greatest rate of improvement in first 12 months
- Kids/teens often deny their cognitive problems due to lack of insight
- May not be noticeable to others which may cause anxiety or frustration

(Yeates et al., 2002)

Psycho-behavioral Changes

- **May occur as direct or indirect result of injury**
- Inability to express him/herself appropriately
- Decreased language production
- Concrete thinking and poor insight may interfere with compliance or ability to respond to interventions
- Frustration about being in the hospital and missing family
- Loss of former self
- Poor pragmatic skills
- Hard to differentiate between symptoms of TBI vs. somatic manifestations of depression

Li, L., & Liu, J. (2013)

Sex Differences Post mTBI/Concussion

Female Athletes

- Had a higher post-concussion symptom score 3 months post-injury (Bazarian et al, 2010) & 8 days PI *(Covassin T et al, 2013)
- More often complained of drowsiness and noise sensitivity (Frommer et al, 2011)
- Had a significantly greater neurocognitive decline and increased symptoms (Sandel NK et al, 2017)
- Reported more neurobehavioral and somatic symptoms (Frommer et al, 2011)

Male Athletes

- Complain of cognitive deficits and amnesia (Frommer et al., 2011).

*Reaction time & visual memory esp.

Table 6. All Concussion Symptoms Reported: Year 2[a]

Symptom	Males (n = 327), No. (%)	Females (n = 94), No. (%)	Exact *P* Value
Amnesia	**84 (26)**	**10 (11)**	**.0020**
Concentration difficulty	166 (51)	44 (47)	.50
Confusion/disorientation	**175 (54)**	**33 (35)**	**.0017**
Dizziness/unsteadiness	252 (77)	72 (77)	.92
Drowsiness	**64 (20)**	**29 (31)**	**.02**
Headache	311 (95)	91 (97)	.59
Hyperexcitability	8 (2)	2 (2)	1.00
Irritability	22 (7)	3 (3)	.20
Loss of consciousness	12 (4)	5 (5)	.55
Nausea	108 (33)	34 (36)	.57
Tinnitus	43 (13)	10 (11)	.52
Sensitive to light/visual disturbance	103 (32)	26 (28)	.48
Sensitive to noise	**15 (5)**	**13 (14)**	**.0015**

[a] Bold font indicates significant findings.

Can We Predict Persistence of Post-Concussion Symptoms?

- Possibly: (Zemek et al: JAMA. 2016;315(10): 1014-1025).
- Next slide…

Clinical Score for Persistent Postconcussion Symptoms (PPS) Among Children with Acute Concussion in the ED

Table 5. Selected Predictor Variables for Multivariable Model of Persistent Postconcussive Symptoms (PPCS) at 28 Days in the Derivation Cohort[a]

	No. of Risk Points for PPCS	Odds Ratio (95%CI)	*P* Value
Age group, y			
5-7	0	1 [Reference]	<.001
8-12	1	1.54 (1.09-2.19)	
13-<18	2	2.31 (1.62-3.32)	
Sex			
Male	0	1 [Reference]	<.001
Female	2	2.24 (1.78-2.82)	
Prior concussion and symptom duration			
No prior concussion; symptom duration <1 wk	0	1 [Reference]	.01
Prior concussion; symptom duration ≥1 wk	1	1.53 (1.10-2.13)	
Physician-diagnosed migraine history			
No	0	1 [Reference]	.001
Yes	1	1.73 (1.24-2.43)	
Answering questions slowly			
No	0	1 [Reference]	.008
Yes	1	1.37 (1.08-1.74)	
Balance Error Scoring System tandem stance No. of errors			
0-3	0	1 [Reference]	.02
≥4 or Physically unable to undergo testing	1	1.31 (1.04-1.66)	
Headache			
No	0	1 [Reference]	.01
Yes	1	1.66 (1.11-2.48)	
Sensitivity to noise			
No	0	1 [Reference]	.002
Yes	1	1.47 (1.15-1.87)	
Fatigue			
No	0	1 [Reference]	<.001
Yes	2	1.84 (1.37-2.46)	

[a] There were 1701 patients in the derivation cohort included in the primary analysis.

From: Zemek et al: JAMA. 2016;315(10): 1014-1025.

Table 6. Risk Categories for Persistent Postconcussive Symptoms (PPCS) in the Derivation Cohort[a]

PPCS Risk Category	Total No. of Risk Points	Estimated Risk of PPCS, % (95% CI)	No. With PPCS/ Total No. of Patients (%)
Low risk	0	4.1 (2.4-6.7)	0/6 (0)
	1	5.8 (3.9-9.5)	6/37 (16.2)
	2	8.3 (6.0-13.2)	11/98 (11.2)
	3	11.8 (8.5-17.8)	15/165 (9.1)
Medium risk	4	16.4 (11.9-22.4)	41/239 (17.2)
	5	22.3 (16.7-29.7)	71/289 (24.6)
	6	29.7 (22.7-37.9)	90/299 (30.1)
	7	38.2 (30.1-46.9)	96/243 (39.5)
	8	47.6 (38.9-57.1)	80/172 (46.5)
High risk	9	57.1 (48.2-65.6)	58/103 (56.3)
	10	66.1 (57.2-74.4)	30/43 (69.8)
	11	74.1 (65.8-81.5)	9/13 (69.2)
	12	80.8 (74.6-88.3)	3/3 (100)

From: Zemek et al: JAMA. 2016;315(10): 1014-1025

[a] There were 1701 patients in the derivation cohort included in the primary analysis.

Other Important Factors:

- (Too) Early Participation in Physical activity (Grool et al. JAMA 2016; 316,(23):2504-2514)
- Being symptomatic at 1 month PREDICTED being symptomatic at 1 year (Waljas et al, J Neurotrauma: 2015 Apr 15;32(8):534-47)
- PPCS (defined as smx >3months post SRI) patients were more likely than control patients to have…
  - a concussion history (p = 0.010), (2x the risk – McCrea et al.2013. *J Internat Neuropsychological Soc.* 19 (1):22-33.)
  - premorbid mood disorders (p = 0.002),
  - other psychiatric illness (p = 0.039),
  - or significant life stressors (p = 0.036).
  - Delayed onset of immediate PCS
  - Other factors that increased the likelihood of PCS development were **a *family history* of mood disorders, other psychiatric illness, and migraine** (Morgan, et al, J Neurosurg Pediatr. 2015 Jun;15(6):589-98)-Vanderbilt Sports Concussion Center.)

What are the new findings?

- For the majority of predictors, the literature is mixed with positive and negative findings.
- Preinjury mental health problems and prior concussions appear to be risk factors for persistent symptoms.
- Greater acute and subacute symptoms are a consistent predictor of worse clinical outcome.
- The teenage years might be a particularly vulnerable time for having persistent symptoms—with greater risk for girls than boys.

Iverson GL, *et al*. *Br J Sports Med* 2017;**51**:941–948. doi:10.1136/bjsports-2017-097729

Return to Learn/Return to Play Protocols

- For concussions, clear protocols have been formulated for a GRADED re-integration into school AND sports: (http://www.parachutecanada.org/downloads/programs/activeandsafe/Concussion_Guidelines_for_Physicians.pdf; http://onf.org/system/attachments/266/original/GUIDELINES_for_Diagnosing_and_Managing_Pediatric_Concussion_Recommendations_for_HCPs__v1.1.pdf)
- See handout for examples of these; the "cornerstone" of recovery from concussion

Return to Learn/Return to Play Protocols

- Are there problems with following these?
- Challenges:
  - How much rest is needed?
  - How much time off from school is recommended?
  - What to do when kids have difficulty with school routines?
  - When should physical activity be recommenced, and what type?

DeMatteo et al: Clin Pediatr 2015. 54(8):783-792
Grool et al: JAMA. 2016; 316(23):2504-2514

Return to School/Learn Protocol

- Too early return to school can result in
  - Exacerbation of symptoms, prolonged recovery, increased risk of REINJURY
- However, prolonged rest and school absence can be devastating
  - Loss of academic standing, social isolation resulting in (increased) anxiety/depression (DeMatteo et al, 2015)
  - Particularly impactful on "high-achieving" individuals (personal unpublished data)
- Recommendation:
  - Return to school with APPROPRIATE modifications and recognition of symptom exacerbation – delicate balanced approach (DeMatteo et al, 2015)
  - This return to school should be "conservative & individualized" (Purcell L; *Pediatr Child Health.*2012;17:31)

Return to School/Learn Stages (DeMatteo et al, 2015; *Tamara et al,2017)

- **Stage 1:** Brain Rest – No School (at LEAST one week; NO TV, video games, texting, reading!). When symptom-free, or at 2 weeks maximum, start **Stage 2!**
- **Stage 2:** Getting Ready to Go Back! Start 2 days prior to returning to school. Include walking, 15 mins screen time 2x/day, begin reading. (Reduce activity if symptoms worsen. Move to Stage 3 when symptoms abate, or stay max 2 wks if persist)
- **Stage 3:** Back to school/modified program: may last days to months! See references.

*Tamara et al, *J Athl Train.* 2017 Mar;52(3):262-287

Return to "Play" Protocols (Almost entirely based on expert consensus vs. research)

- Balanced, graded, conservative and INDIVIDUALIZED approach generally recommended.
- Risk of too early Return to Activity:
  - Exacerbation or return of symptoms
  - Risk of RE-INJURY: Poor judgement, balance issues, reaction time, multitasking ability, etc. (Not consistently found to be true)
- Risk of TOO prolonged restriction of activity (DiFazio et al. 2016)
  - Prolongation of PCS
  - Social isolation, increased anxiety, depression

DiFazio, et al: Clin Pediatr 2016, 55(5): 443-451

Return to "Play" Protocols

- Adult studies post-mTBI, showed NO benefit of 5-7 days of rest vs. early graded mobilization (de Kruijk et al. *J Neurol Neurosurg Psychiatry:* 2002; 73:167-172)
- Adolescent studies show MIXED evidence for benefits of time-limited prescribed rest (Gibson et al. *Brain Inj.* 2013; 27:839-842; Moser et al. *J Pediatr.* 2012; 161:922-6; Moser et al. *Brain Inj.* 2015;29:58-63.)
- One randomized controlled study (Thomas et al. *Pediatrics.* 2015; 135: 213-223.) showed
  - strict bed rest (5-6 days) did NOT provide BETTER symptom resolution than early (1-2 day) rest then graded activity
  - Instead, bed rest group reported MORE PCS!

DiFazio, et al: Clin Pediatr 2016, 55(5): 443-451

Problems with RTP Protocols

- Compliance to rest difficult to ascertain and definition variable in studies
- DOES one REALLY rest the brain even when lying with eyes closed? fMRI studies say "NO".
- Determining when rest should end:
  - PCS are non-specific and can be mimicked by anxiety, depression, family/school/social stress
- Prolonged rest can itself be harmful (next slide)

DiFazio, et al: Clin Pediatr 2016, 55(5): 443-451

Prolonged rest can itself be harmful?

- Anxiety, Expectations AND NOCEBO effect: Exaggerated reaction due to belief of harm ("Texting could harm your brain")
- Depression from "removal" from normal life activities (social isolation) (Activity Restriction Model of depression)
- In teens, inability to engage in social, recreational, & sporting activities devastating and counter-intuitive (viz Infectious Mono, cancer, TBI, etc)
- Physical deconditioning from short or extended periods of rest , can mimic PCS

DiFazio, et al: Clin Pediatr 2016, 55(5): 443-451;
Silverberg & Iversen: J Head trauma Rehabil 2013; 28: 250-9

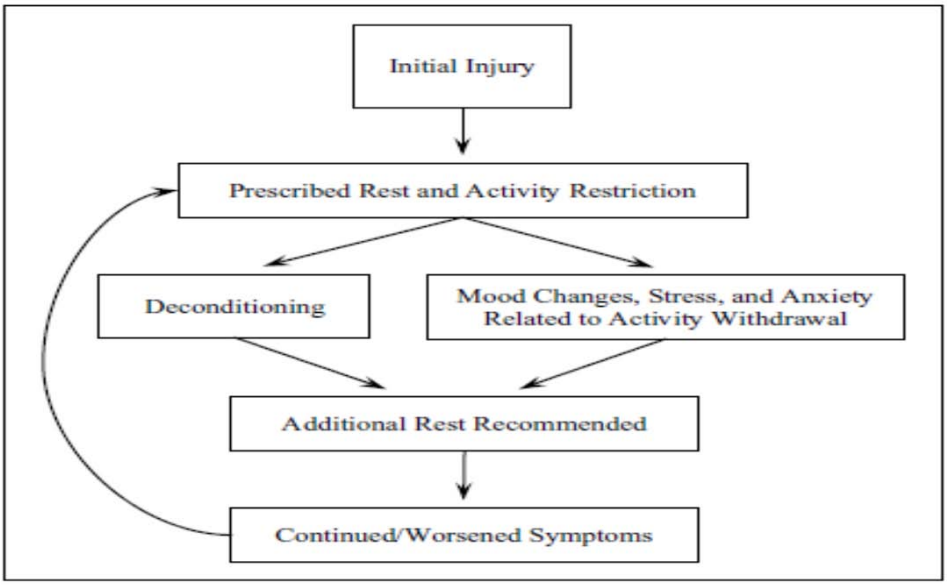


Figure 1. Theoretical model for prolonged rest and activity restriction contributing to persistent symptoms.

DiFazio, et al: Clin Pediatr 2016, 55(5): 443-451;

Mood & Anxiety in PCS

- Pre-injury mental health problems are a risk factor for PPCS (McNally et al. Neuropsychology. 2013;27: 1-12; Peterson et al. J Neuropsychiatry Clin Neurosci. 2015; 27: 280-6)
- Anxiety & depression can mimic PPCS and be secondary
- Risk factors for post-injury psychiatric outcome include:
  - Female sex
  - Higher initial PCSS subscore
  - Teens have higher incidence of BOTH internalizing and externalizing behaviors (Dykeman, 2003)
  - Presence of pre-injury
  - Family history of psychiatric illness
  - Multiple concussions
- ~12% in one study met criteria

Ellis et al. *J Neurosurg Pediatr.2015;16:709-718*)

What's Different in More Severe TBI's

- More severe and pervasive symptoms, including:
  - **Physical:** (motor, endocrine, hearing, vision, speech, seizures, etc.)
  - **Emotional:** (Anxiety, depression, suicidal ideation, poor impulse control, PTSD, decreased HRQoL, etc)(Rhine et al, *J Head Trauma Rehabil.* 2017 May 17; Max et al, Int J Devl Neuroscience 30(2012) 239-245; Battista et al, PLoS ONE 9(7)2014)
  - **Cognitive:** ("Gaps"in neurocognitive skills, severe language/speech issues, secondary "ADHD", slow problem-solving & motor output, impaired "new" learning, executive function, etc.) (Levin & Hanten, 2005. *Pediat Neurol* 33, 79-93.)
  - **Social:** A major area of concern!! Impaired empathy, nonverbal cognition (sarcasm, facial expression, gestures, etc) results in social isolation, & impaired QoL. (Turkstra et al. 2008, *NeuroRehabilitation* 23: 501-9)
  - Duration of coma, PTA, and alteration in consciousness may inform neurobehavioral outcome (Yeates, 2010)
  - Lesion burden correlates with morbidity (Babikian & Asarnow, 2009)

Teens: "They Look Fine but is Seeing Believing?"

- Over 50% of teens do not immediately report symptoms (Asken et al, J Athl Training. 2016;51(4):329-335)
- Most teens with mild-moderate don't "show" their symptoms
  - Likely due to pressures (intrinsic & extrinsic) to return to school or activities prematurely
- Those that acknowledge and report symptoms often accused of "faking" by peers, or teachers
- Possibly some "USE" symptoms to avoid school, etc.
- Increased "dependency" on, or restrictions by parents, caregivers or teachers INTERFERES with need for independence
  - Likely CREATES more stress for teens.

Summary

- Teenage years are potentially tumultuous at best
- Any issues that work against their achievement of independence and social engagement with peers, only adds to this
- Further research is imperative in trying to better identify risk factors & intervention that can mitigate these challenges

- THANKS FOR YOUR KIND ATTENTION!!

References

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TABLE 1 — E. Farace and W. M. Alves (2000)

*Gender differences in TBI outcome**

Authors & Year	Outcome Variable	TBI Severity/Type	Time Since Injury	No. in Study M	No. in Study F	No. in Study Total	No. Reporting Symptom M	No. Reporting Symptom F	Statistical Analysis	Effect Size†	Worse Outcome
initial injury severity											
Wilberger, et al., 1990	death	GCS 3–7, ASDH	18 mos	77	24	101	50	17	$\chi^2 = 0.29$	w = –0.10	women
Kaplan & Corrigan, 1992	days of PTA	CHI w/ some PTA	chart review	62	26	88			F = 4.02‡	f = –0.22	women
	length of hospitalization	CHI w/ some PTA	chart review						F = 3.51	f = –0.20	women
somatic/PCSs											
Rutherford, 1977	≥1 PCS	minor CHI w/ concussion; no op	6 wks	92	53	145	37	37	$\chi^2 = 11.79$‡	w = –0.43	women
Edna & Cappelen, 1987	impaired memory	GCS 3–15, CHI w/ LOC, skull fx, or ICH	3–5 yrs	351	134	485	68	31	$\chi^2 = 0.84$	f = –0.07	women
	dizziness	GCS 3–15, CHI w/ LOC, skull fx, or ICH	3–5 yrs	351	134	485	48	42	$\chi^2 = 20.03$‡	w = –0.35	women
	fatigue	GCS 3–15, CHI w/ LOC, skull fx, or ICH	3–5 yrs	351	134	485	61	28	$\chi^2 = 0.80$	w = –0.07	women
	irritability (noise, light)	GCS 3–15, CHI w/ LOC, skull fx, or ICH	3–5 yrs	351	134	485	56	32	$\chi^2 = 4.10$‡	w = –0.16	women
	impaired concentration	GCS 3–15, CHI w/ LOC, skull fx, or ICH	3–5 yrs	351	134	485	46	22	$\chi^2 = 0.88$	w = –0.07	women
	insomnia	GCS 3–15, CHI w/ LOC, skull fx, or ICH	3–5 yrs	351	134	485	38	27	$\chi^2 = 7.26$‡	w = –0.21	women
	tinnitus	GCS 3–15, CHI w/ LOC, skull fx, or ICH	3–5 yrs	351	134	485	45	16	$\chi^2 = 0.07$	w = 0.02	men
	hearing defect	GCS 3–15, CHI w/ LOC, skull fx, or ICH	3–5 yrs	351	134	485	42	9	$\chi^2 = 2.84$	w = 0.13	men
	double vision	GCS 3–15, CHI w/ LOC, skull fx, or ICH	3–5 yrs	351	134	485	8	4	$\chi^2 = 0.20$	w = –0.04	women
	headache	GCS 3–15, CHI w/ LOC, skull fx, or ICH	3–5 yrs	351	134	485	62	51	$\chi^2 = 22.57$‡	w = –0.37	women
Jensen & Nielsen, 1990	headache	LOC 24 hrs, concussion	9–12 mos	96	72	168	52	56	$\chi^2 = 9.99$‡	w = –0.35	women
return to work											
McMordie, et al., 1990	no return to work	any TBI	mean 6.7 yrs	138	39	177	832	14	$\chi^2 = 7.22$‡	w = 0.39	men
Cifu, et al., 1997	no return to work	any TBI	1 yr	106	26	132	64	19	$\chi^2 = 1.44$	w = –0.22	women
new psychiatric symptoms											
Edna & Cappelen, 1987	anxiety	GCS 3–15, CHI w/ LOC, skull fx, or ICH	3–5 yrs	351	134	485	21	16	$\chi^2 = 4.88$‡	w = –0.17	women
	depression	GCS 3–15, CHI w/ LOC, skull fx, or ICH	3–5 yrs	351	134	485	27	18	$\chi^2 = 3.80$	w = –0.15	women
Levin, et al., 1987	depression	any TBI	≥6 mos	78	23	101			F = 13.77	f = –0.37	women

* ASDH = acute subdural hematoma; CHI = closed head injury; f = effect size index for F statistic; fx = fracture; GCS = Glasgow Coma Scale; ICH = intracranial hemorrhage; LOC = loss of consciousness; PCS = postconcussive symptom; PTA = posttraumatic amnesia; w = chi-square effect size index.

† See *Magnitude of Effect Sizes* for definitions.

‡ $p < 0.05$

Table 2
Accommodations for postconcussion effects affecting school

Postconcussion Effect	Functional School Problem	Accommodation/ Management Strategy
Neuropsychological deficits		
Attention/concentration	Short focus on lecture, classwork, homework	Shorter assignments, break down tasks, lighter work load
Working memory	Holding instructions in mind, reading comprehension, mathematics calculation, writing	Repetition, written instructions, use of calculator, shorter reading passages
Memory consolidation/ retrieval	Retaining new information, accessing learned information when needed	Smaller chunks to learn, recognition cues
Processing speed	Keep pace with work demand, process verbal information effectively	Extended time, slow down verbal information, comprehension checking
Fatigue	Decreased arousal/activation to engage basic attention, working memory	Rest breaks during classes, homework, and examinations
Symptoms		
Headaches	Interferes with concentration	Rest breaks
Light/noise sensitivity	Symptoms worsen in bright or loud environments	Wear sunglasses, seating away from bright sunlight or other light. Avoid noisy/crowded environments such as lunchroom, assemblies, and hallways
Dizziness/balance problems	Unsteadiness when walking	Elevator pass, class transition before bell
Sleep disturbance	Decreased arousal, shifted sleep schedule	Later start time, shortened day
Anxiety	Can interfere with concentration, student may push through symptoms to prevent falling behind	Reassurance from teachers and team about accommodations, workload reduction, alternate forms of testing
Depression/withdrawal	Withdrawal from school or friends because of stigma or activity restrictions	Time built in for socialization
Cognitive symptoms	Concentrating, learning	See specific cognitive accommodations (above)
Symptom sensitivity	Symptoms worsen with overactivity, resulting in any of the earlier-mentioned problems	Reduce cognitive or physical demands below symptom threshold, provide rest breaks, complete work in small increments until symptom threshold increases

From: Sady et al*: Phys Med Rehabil Clin N Am*: Nov: 22(4):701-19.

Table 5. Selected Predictor Variables for Multivariable Model of Persistent Postconcussive Symptoms (PPCS) at 28 Days in the Derivation Cohort[a]

	No. of Risk Points for PPCS	Odds Ratio (95%CI)	P Value
Age group, y			
5-7	0	1 [Reference]	<.001
8-12	1	1.54 (1.09-2.19)	
13-<18	2	2.31 (1.62-3.32)	
Sex			
Male	0	1 [Reference]	<.001
Female	2	2.24 (1.78-2.82)	
Prior concussion and symptom duration			
No prior concussion; symptom duration <1 wk	0	1 [Reference]	.01
Prior concussion; symptom duration ≥1 wk	1	1.53 (1.10-2.13)	
Physician-diagnosed migraine history			
No	0	1 [Reference]	.001
Yes	1	1.73 (1.24-2.43)	
Answering questions slowly			
No	0	1 [Reference]	.008
Yes	1	1.37 (1.08-1.74)	
Balance Error Scoring System tandem stance No. of errors			
0-3	0	1 [Reference]	.02
≥4 or Physically unable to undergo testing	1	1.31 (1.04-1.66)	
Headache			
No	0	1 [Reference]	.01
Yes	1	1.66 (1.11-2.48)	
Sensitivity to noise			
No	0	1 [Reference]	.002
Yes	1	1.47 (1.15-1.87)	
Fatigue			
No	0	1 [Reference]	<.001
Yes	2	1.84 (1.37-2.46)	

[a] There were 1701 patients in the derivation cohort included in the primary analysis.

Table 6. Risk Categories for Persistent Postconcussive Symptoms (PPCS) in the Derivation Cohort[a]

PPCS Risk Category	Total No. of Risk Points	Estimated Risk of PPCS, % (95% CI)	No. With PPCS/ Total No. of Patients (%)
Low risk	0	4.1 (2.4-6.7)	0/6 (0)
	1	5.8 (3.9-9.5)	6/37 (16.2)
	2	8.3 (6.0-13.2)	11/98 (11.2)
	3	11.8 (8.5-17.8)	15/165 (9.1)
Medium risk	4	16.4 (11.9-22.4)	41/239 (17.2)
	5	22.3 (16.7-29.7)	71/289 (24.6)
	6	29.7 (22.7-37.9)	90/299 (30.1)
	7	38.2 (30.1-46.9)	96/243 (39.5)
	8	47.6 (38.9-57.1)	80/172 (46.5)
High risk	9	57.1 (48.2-65.6)	58/103 (56.3)
	10	66.1 (57.2-74.4)	30/43 (69.8)
	11	74.1 (65.8-81.5)	9/13 (69.2)
	12	80.8 (74.6-88.3)	3/3 (100)

[a] There were 1701 patients in the derivation cohort included in the primary analysis.

From: Zemek et al: JAMA. 2016;315(10): 1014-1025

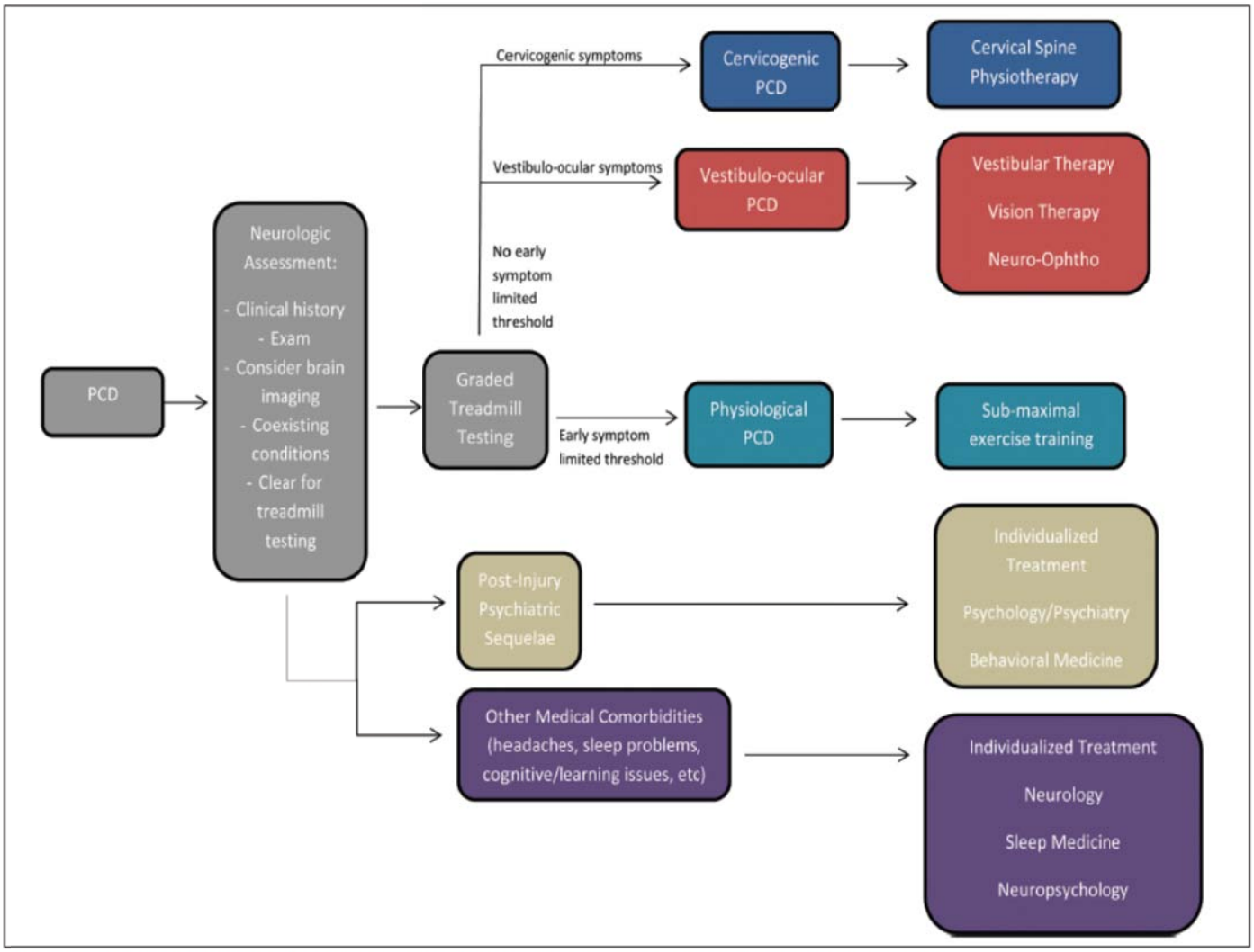

Figure 1. Flowchart of proposed multidisciplinary approach to postconcussion disorder (PCD). Adapted from Ellis et al.[22]

From: Jeff Strelzik, MD; and Raquel Langdon, MD; Pediatric Annals 2017; 46(4):e139-e144