Understanding Cardiovascular Autonomic Function in the Context of Concussion Injuries

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Presenter

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Dr. Michael F. La Fountaine

- Dr. La Fountaine received his Doctoral Degree in Applied Physiology from Columbia University in 2008. His current research agenda is focused on developing a more robust appreciation for how genetic variability of autonomic receptors, and their subtypes, contribute to cardiovascular autonomic control in otherwise healthy humans, and that which may occur in response to acute or chronic neurological injury.

Disclosures

- Dr. Michael F. La Fountaine has no relevant financial or non-financial relationships to disclose relating to the content of this activity.
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- The views expressed in this presentation are those of the author and do not necessarily reflect the official policy or position of the Department of Defense, or the U.S. Government.
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- Commercial support was not received for this activity.

Polling Question

- What is your profession?
  - ❑ Physician
  - ❑ Nurse
  - ❑ Psychologist
  - ❑ Physical Therapist
  - ❑ Social Worker
  - ❑ Certified Athletic Trainer
  - ❑ Clinical Researcher (human subjects)
  - ❑ Administrator
  - ❑ Other

Polling Question

- What role do you play in the evaluation of suspected head injuries?
  - ❑ Primary care and assessment
  - ❑ Post-acute medical and symptom management
  - ❑ Rehabilitation
  - ❑ Mental health care
  - ❑ Evaluation of return-to-duty
  - ❑ I am not involved with clinical activities for suspected head injuries

Learning Objectives

At the conclusion of this activity, participants will be able to:

1. Summarize the structure and function of the autonomic nervous system.
2. Describe the methods and outcomes from non-invasive assessments of cardiovascular autonomic function.
3. Define concussion injury and how the injury is speculated to impact cardiovascular autonomic function.
4. Identify signs and symptoms of cardiovascular autonomic dysfunction.

Concussions

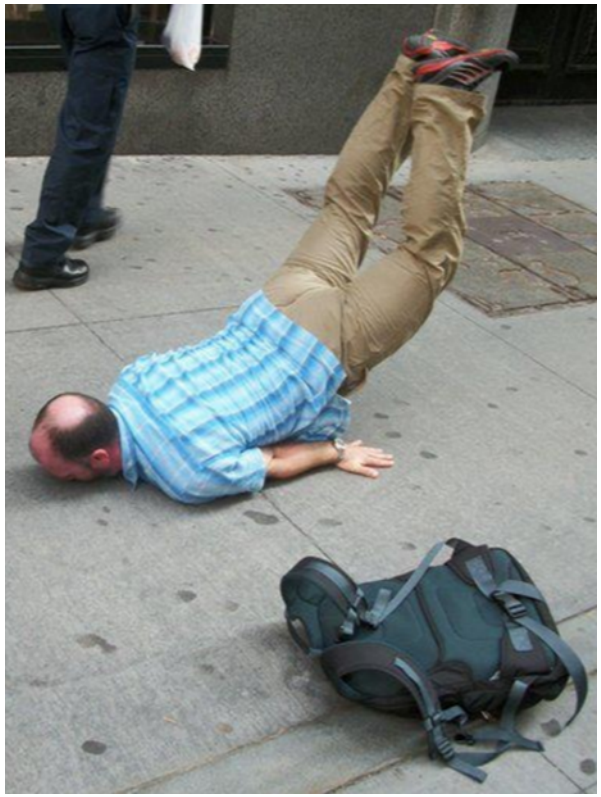
http://www.ccbarlow.com/wp-content/uploads/2012/07/Water-drop-in-pondFotolia_41270412_XS-300x200.jpg

Concussions

https://www.google.com/url?sa=i&source=images&cd=&ved=2ahUKEwjXhfeMx_PjAhViT9BKHcXxCiQQjRx6BAgBEAQ&url=https%3A%2F%2Frarehistoricalphotos.com%2Ftesting-football-helmets-1912%2F&psig=AOvVaw22Wtk4tpJBiv4zcISOWrxM&ust=1565633668820619

Concussions

https://www.google.com/url?sa=i&source=images&cd=&ved=2ahUKEwinwY-cxvPjAhULWNBKHXnAA-MQjRx6BAgBEAQ&url=https%3A%2F%2Fwww.youtube.com%2Fwatch%3Fv%3D72CfGmC9VuU&psig=AOvVaw3OWAuCjDVsKrfrja6TL2br&ust=1565363122249061



http://thejennyking.com/days-win-days-fail-hard-like-smack-face-pavement-hard/

Concussions

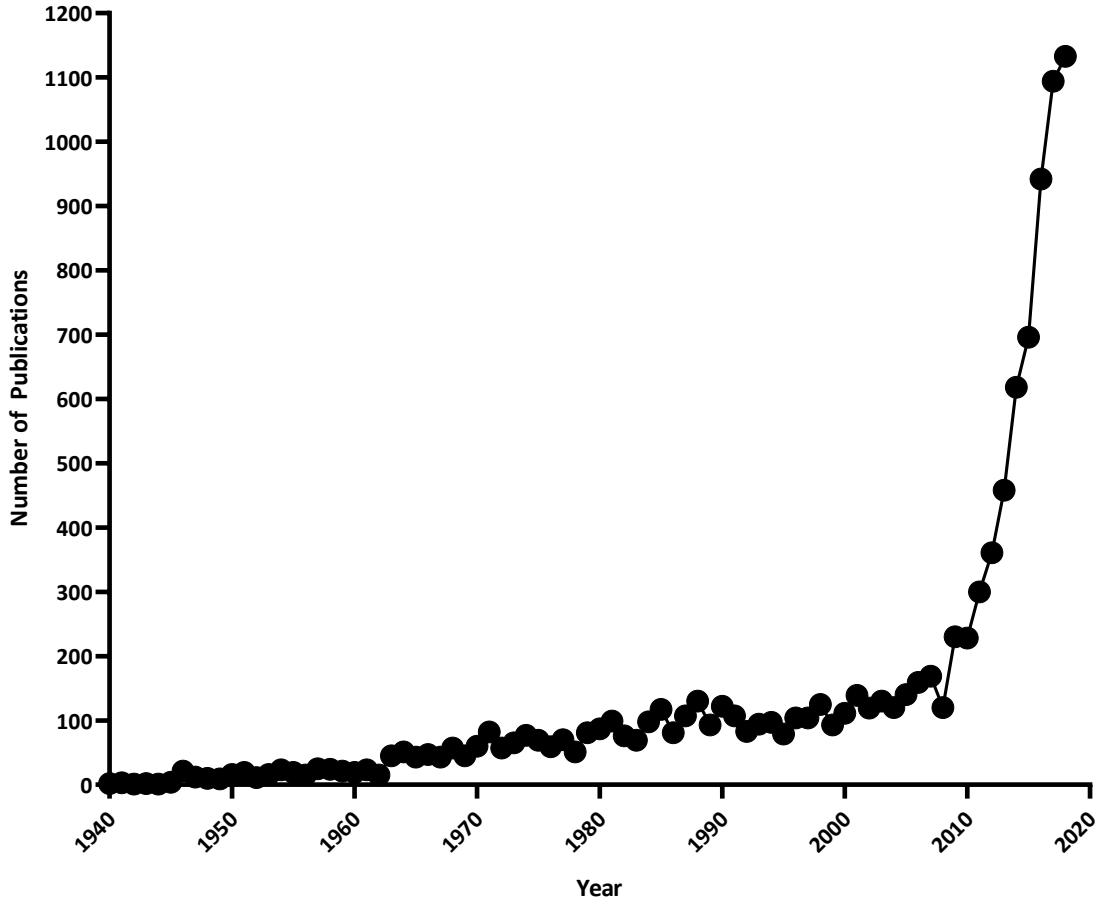
http://www.universityobserver.ie/wp-content/uploads/2012/11/Rugby-scrum.jpg

http://thesportsfanjournal.com/wp-content/uploads/2013/07/jadeveon-clowney-hit-600x398.jpg

Concussions

https://www.google.com/url?sa=i&source=images&cd=&ved=2ahUKEwjUuMOYyfPjAhUDn-AKHem5A6gQjRx6BAgBEAQ&url=https%3A%2F%2Fen.wikipedia.org%2Fwiki%2FExplosion&psig=AOvVaw0V5AC4NFzn8Vm8hq9fDHV5&ust=1565363914364782

Pubmed Results Mentioning Concussion



"Medically Ready Force…Ready Medical Force"

History of Concussion

- The first "modern" physician to discuss concussion as a separate entity was Lanfrancus, 13th Century
  - Taught that symptoms after a concussive injury could rapidly disappear and were the result of a transient paralysis of cerebral function caused by the brain being shaken
  - commotio cerebrum v. contusio cerebrum

History of Concussion

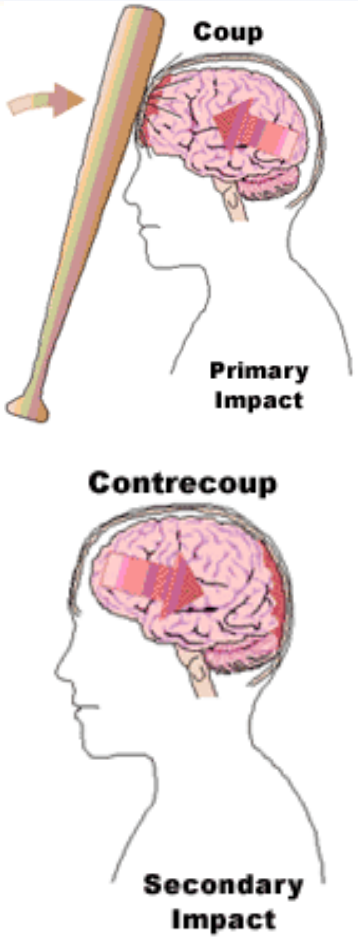
- da Carpi (1518)- “cerebrum commotum” caused by the thrust of the soft structure of the brain against the solid part of the skull
- Coiter (1573)- acute symptoms as “a faltering in the speech, impairing of memory, dullness of understanding, & weak judgement”
- Acquapendente (1578)- lethargy and vertigo
- Queyrat (1657)- injury due to “ebb and flow” of nervous tissue within the brain
- Marchetti (1665)- short duration of “…alienation of the mind, with a privation of sense and motion.”

Concussion Mechanism

Coup injury: site where brain contacts the skull

Contre-coup: injury site opposite to where brain contacts the skull

- Occurs by lateral or anterior/posterior translation of brain within Cerebrospinal fluid (CSF)
- Rotation about the brain stem may occur
- Shearing: resulting from rapid acceleration of the head



Concussion Defined

Joint VA/DoD Clinical Practice Guidelines (2016)

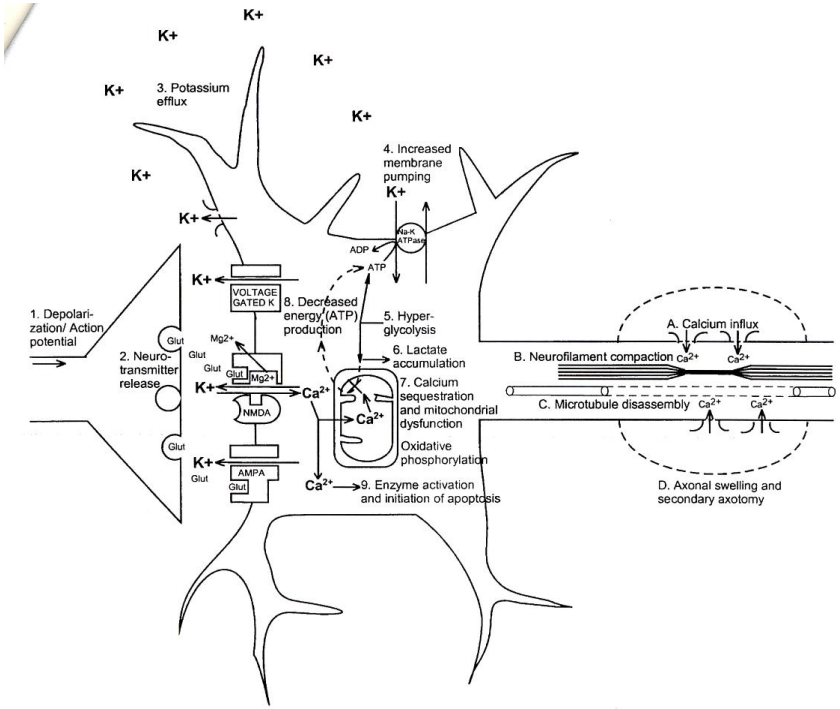
- ….a traumatically induced structural injury and/or physiological disruption of brain function as a result of an external force…indicated by new onset or worsening of at least one of the following clinical signs immediately following the event:
  - Any period of loss of or a decreased level of consciousness
  - Any loss of memory for events immediately before or after the injury (posttraumatic amnesia)
  - Any alteration in mental state at the time of the injury (e.g., confusion, disorientation, slowed thinking, alteration of consciousness/mental state)
  - Neurological deficits (e.g., weakness, loss of balance, change in vision, praxis, paresis/plegia, sensory loss, aphasia) that may or may not be transient
  - Intracranial lesion

Concussion Defined

5th International Conference on Concussion in Sport (2016)

- *Sport related concussion is a traumatic brain injury induced by biomechanical forces. Several common features that may be utilized in clinically defining the nature of a concussive head injury include:*
  - *…either by a direct blow to the head, face, neck or elsewhere on the body with an impulsive force transmitted to the head.*
  - *…results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously. However, in some cases, signs and symptoms evolve over a number of minutes to hours.*
  - *…may result in neuropathological changes, but the acute clinical signs and symptoms largely reflect a functional disturbance rather than a structural injury and, as such, no abnormality is seen on standard structural neuroimaging studies.*
  - *…results in a range of clinical signs and symptoms that may or may not involve loss of consciousness. Resolution of the clinical and cognitive features typically follows a sequential course. However, in some cases symptoms may be prolonged.*
  - *…the clinical signs and symptoms cannot be explained by drug, alcohol, or medication use, other injuries (such as cervical injuries, peripheral vestibular dysfunction, etc) or other comorbidities (eg, psychological factors or coexisting medical conditions).*

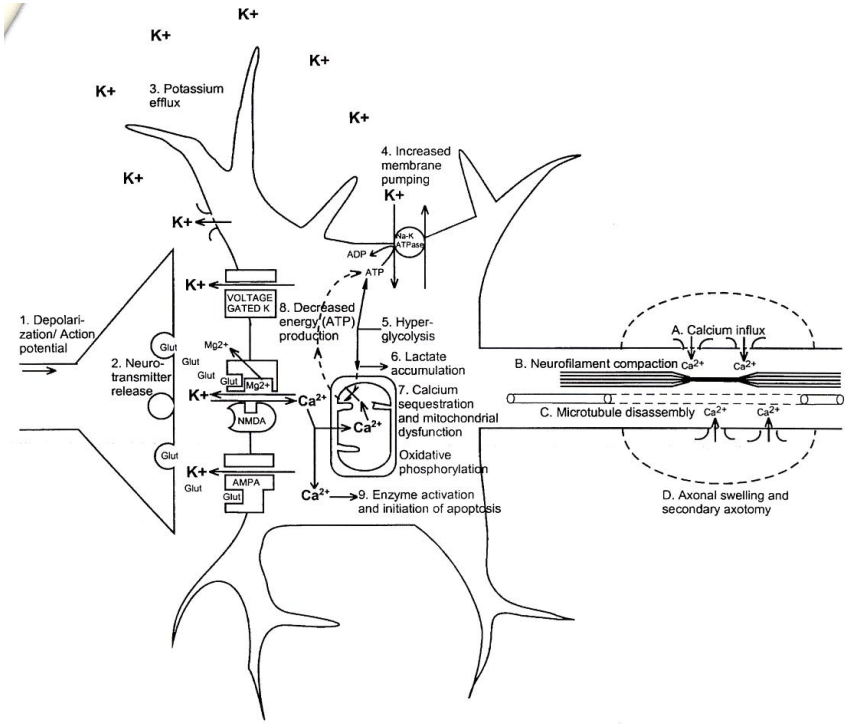
Neurometabolic Cascade of Concussion



1. Nonspecific depolarization and initiation of Action Potential (AP)
2. Release of excitatory Amino Acids (AA's)
3. Massive Efflux of K^+
4. Increased activity of membrane ionic pumps to restore homeostasis
5. Hyperglycolysis to generate more ATP
6. Lactate accumulation
7. Ca^{2+} and sequestration in mitochondria leading to impaired oxidative metabolism
8. Decreased ATP production
9. Calpain activation and initiation of apoptosis

*Giza CC and Hovda D. J Athl Train, 2001; 36 (3): 228-235

Physiological Basis of Concussion



A. Axolemmal disruption and calcium influx

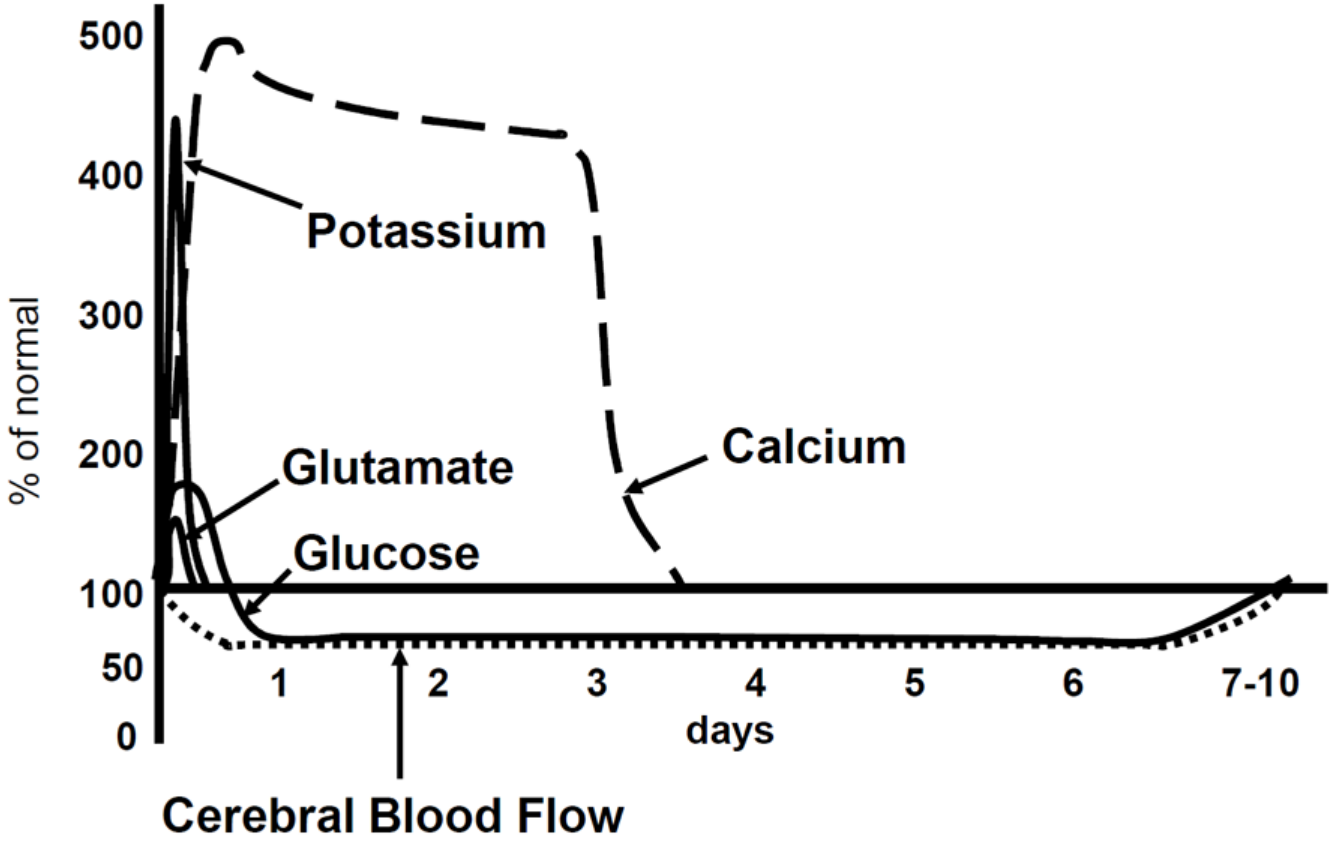
B. Neurofilament compaction via phosphorylation

C. Microtuble disassembly and accumulation of axonally transported organelles

D. Axonal swelling and eventual axotomy

*Giza CC and Hovda D. J Athl Train, 2001; 36 (3): 228-235

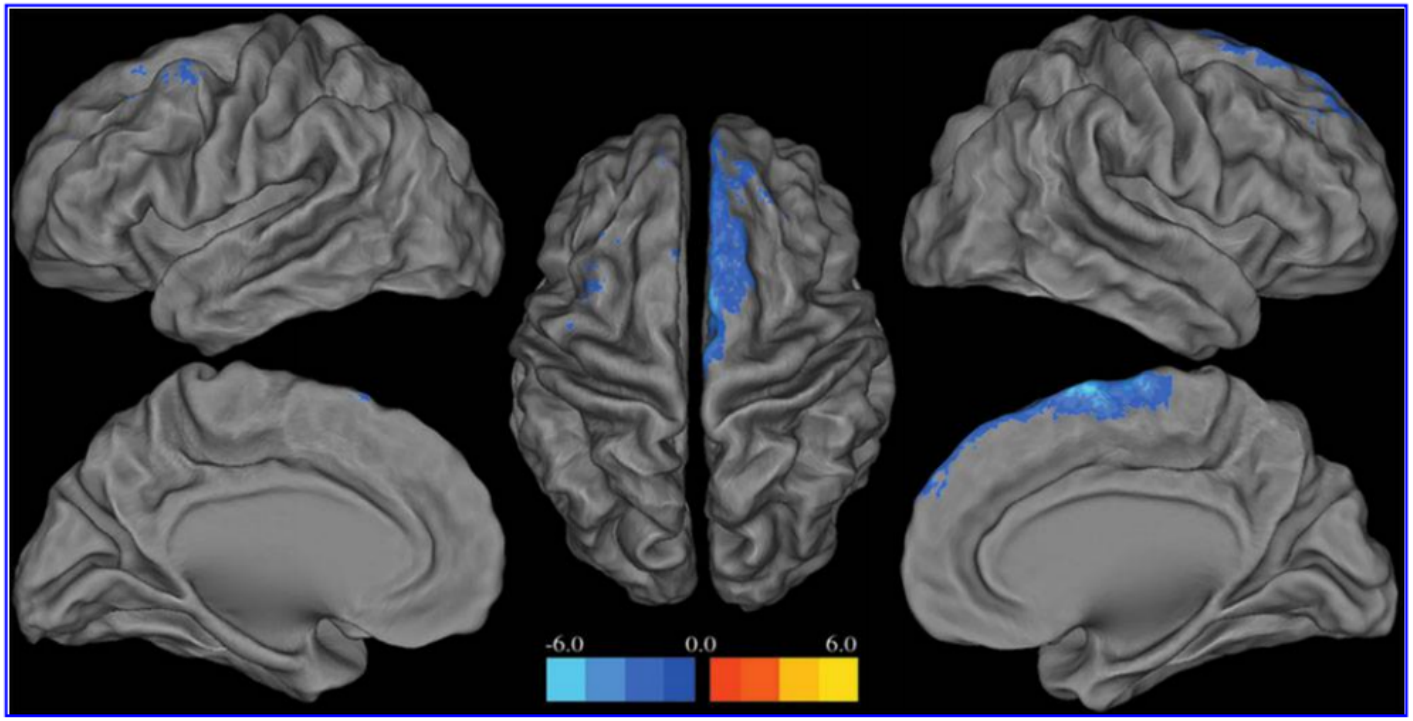
Physiological Basis of Concussion



*Giza CC and Hovda D. Neurosurgery, 2014; 75(4): S24-S32

Altered Cerebral Blood Flow (CBF) after Concussion

Concussion vs. Control @ 1 day



right and pre-supplementary motor area regions

FIG. 1. Regions (in blue color) show significantly less cerebral blood flow (CBF) in concussion group at 24 h after injury, compared with the control group. No region shows significantly more CBF in the concussion group compared to the control group. Images reflect family-wise error correction at $p < 0.05$. Color bar indicates the t score. Color image is available online at www.liebertpub.com/neu

*Used arterial spin labeling MRI

Wang Y, J Neurotrauma; 2016

Altered CBF after Concussion

Concussion vs. Control @ 8 days

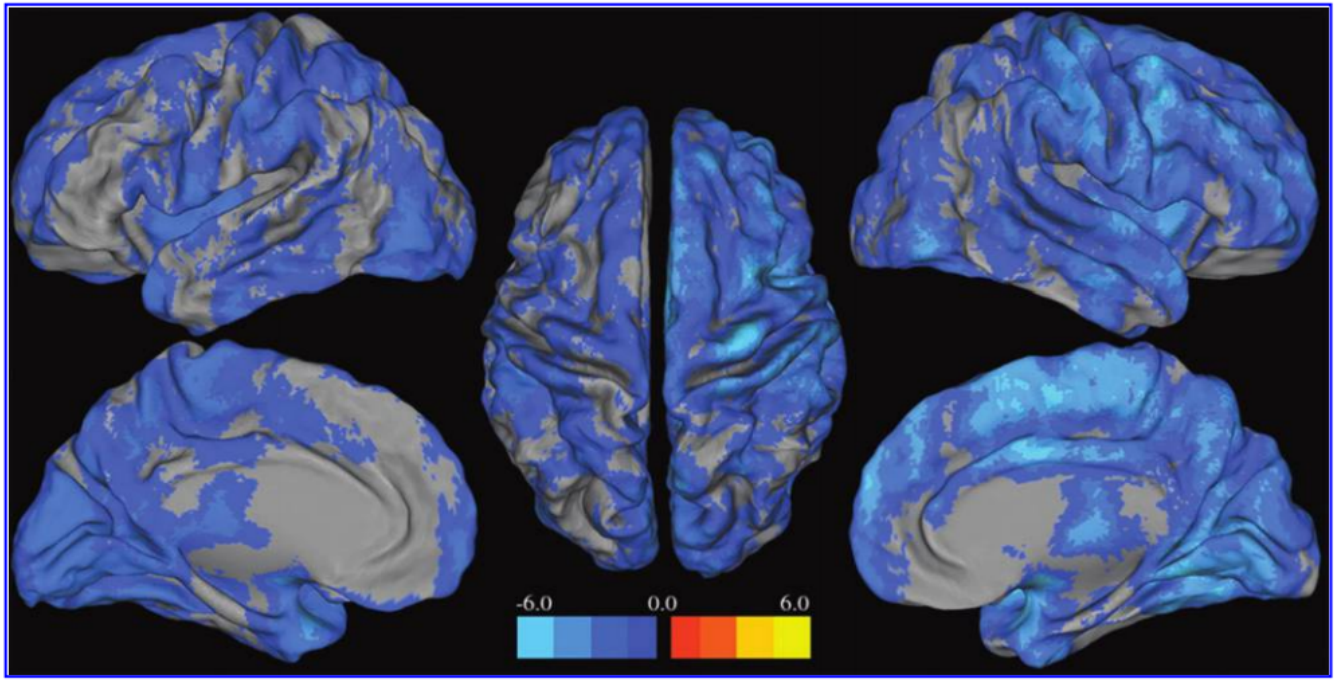


FIG. 2. Diffuse cortical and subcortical regions (in blue color) show significantly less cerebral blood flow (CBF) in concussion group at 8 days after injury, compared with the control group. No region shows significant more CBF in the concussion group compared to the control group. Images reflect family-wise error correction at $p < 0.05$. Color bar indicates the t scores. Color image is available online at www.liebertpub.com/neu

Diffuse cortical gray matter;

- bilateral prefrontal regions (cognition, personality, social behavior),
- temporal lobes (auditory),
- some parietal regions (sensory integration, visual processing, math), and
- thalamus (sleep, consciousness, sensory integration)

*Used arterial spin labeling MRI

Wang Y, J Neurotrauma; 2016

Altered CBF after Concussion

Concussion 1 day vs. Concussion 8 days

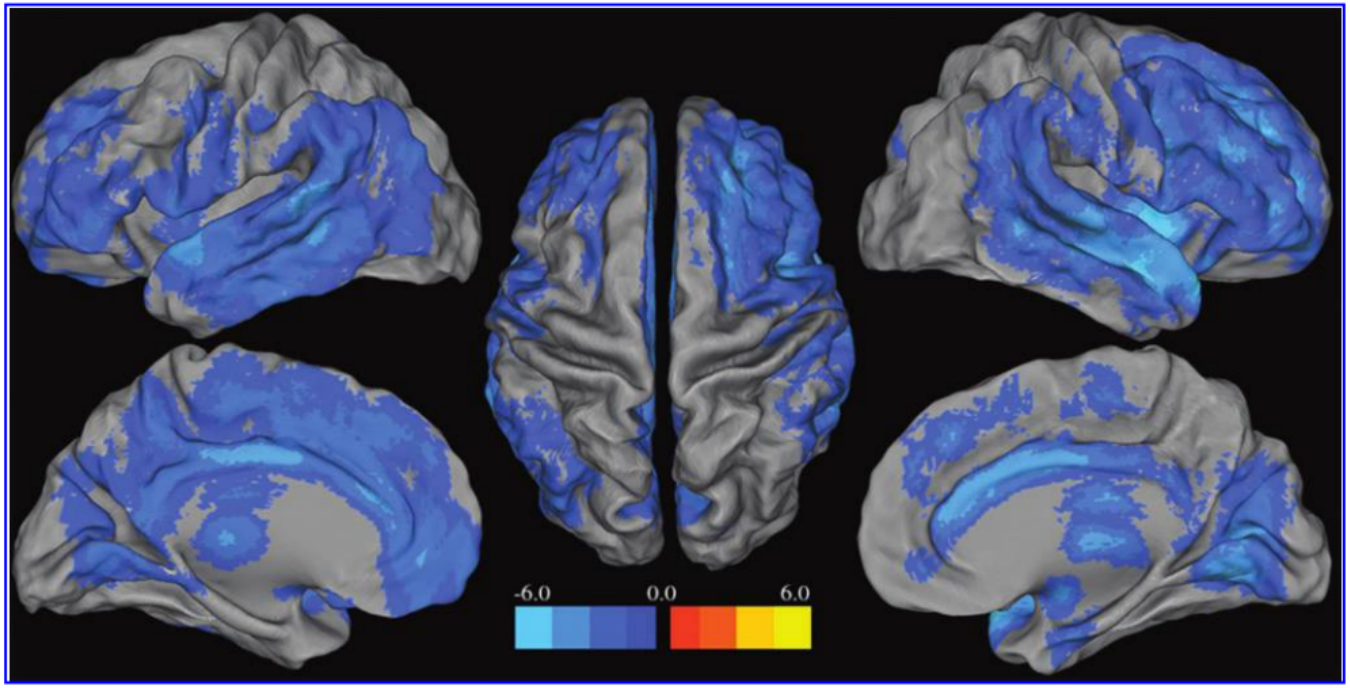


FIG. 3. Spread cortical and subcortical regions (in blue color) show significantly decreased cerebral blood flow (CBF) in concussion group at 8 days compared to 24 h after injury. No region shows significantly increased CBF in the concussion group at 8 days. Images reflect family-wise error correction at $p < 0.05$. Color bar indicates the t scores. Color image is available online at www.liebertpub.com/neu

Frontal and temporal lobes

*Used arterial spin labeling MRI

Wang Y, J Neurotrauma; 2016

Postconcussive Pathophysiology

Defense Health Agency

- Points of consideration from the literature:

- Hypothalamic [catecholamine] may represent activation of noradrenergic and dopaminergic fiber tracts that innervate various hypothalamic nuclei and cerebral blood vessels
- Circulating [catecholamine] reported to be higher in patients with more severe brain injury
  - Correlates with neurological outcome scores
- Does this contribute to pituitary dysfunction in the short and long term?
  - Most hypothalamic releasing factors are regulated by catecholaminergic innervation of specific hypothalamic nuclei, the changes in levels of hypothalamic catecholamines observed in the present study may play a role in the neuroendocrine response to brain injury

Physiological Uncoupling

Uncoupling of the autonomic and cardiovascular systems in acute brain injury.

- Severity of neurological injury and outcome are inversely associated with HR and BP variability
- Complete uncoupling during brain death

Goldstein B. Am J Physiol, 1998; 275 (4 pt 2): R1287-1292

The Central Autonomic Network

- *"…part of the internal regulatory system of the brain and is involved in visceromotor, neuroendocrine, complex motor, and pain modulating control mechanisms essential for adaption and survival."*

Genarroch EE, Mayo Clin Proc, 1993; 68 (10): 988-1000.

- Insular Cortex
  - left insular cortex: stimulation leads to bradycardia and depressor responses
  - right insular cortex: stimulation resulted in tachycardia and pressor responses (sympathetic pre-dominant)

Oppenheimer SM, Neurology, 1992; 42(9): 1727-32.

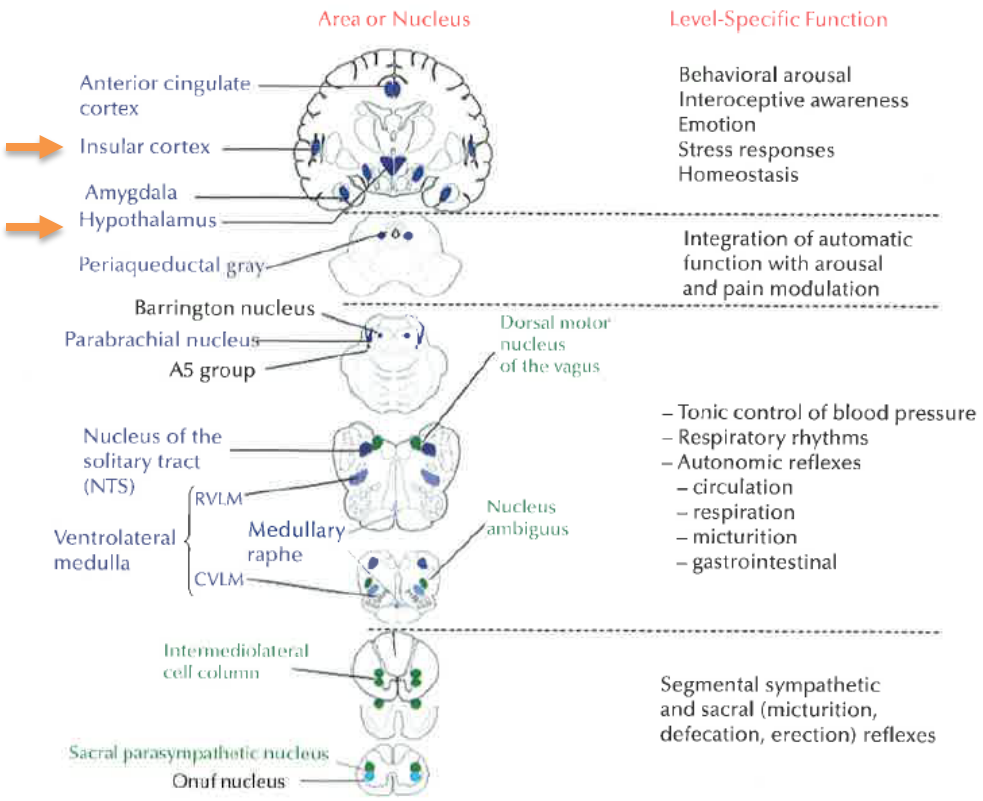


Figure 1.1. Central autonomic control network. Distribution of autonomic control areas in the central nervous system and level-specific organization of autonomic control. Abbreviations: CVLM, caudal ventrolateral medulla; RVLM, rostral ventrolateral medulla.

The Central Autonomic Network

- Controls preganglionic sympathetic and parasympathetic motor neurons
  - Reciprocal interconnections, parallel organization, and state-dependent activity

Genarroch EE, 1993.

- CN IX and X synapse with the NTS, which is also the 1º termination for ascending afferents from the arterial and cardiac baroreceptors, arterial chemoreceptors and lung stretch receptors.

Seller H, 1969.

- Excitatory neurons project from the NTS to the insular cortex and CVLM, which tonically inhibits the RVLM.

Torrealba F, 1996.; Guyenet PG, 1990.

- RVLM is the primary regulatory center for sympathetics; origination site for descending efferent excitation signals through the spinal cord giving rise to the preganglionic nerve fibers, the sympathetic chain ganglia and post-ganglionic projections to the end organs.

Guyenet PG, 1990. Roberston D, 2012.

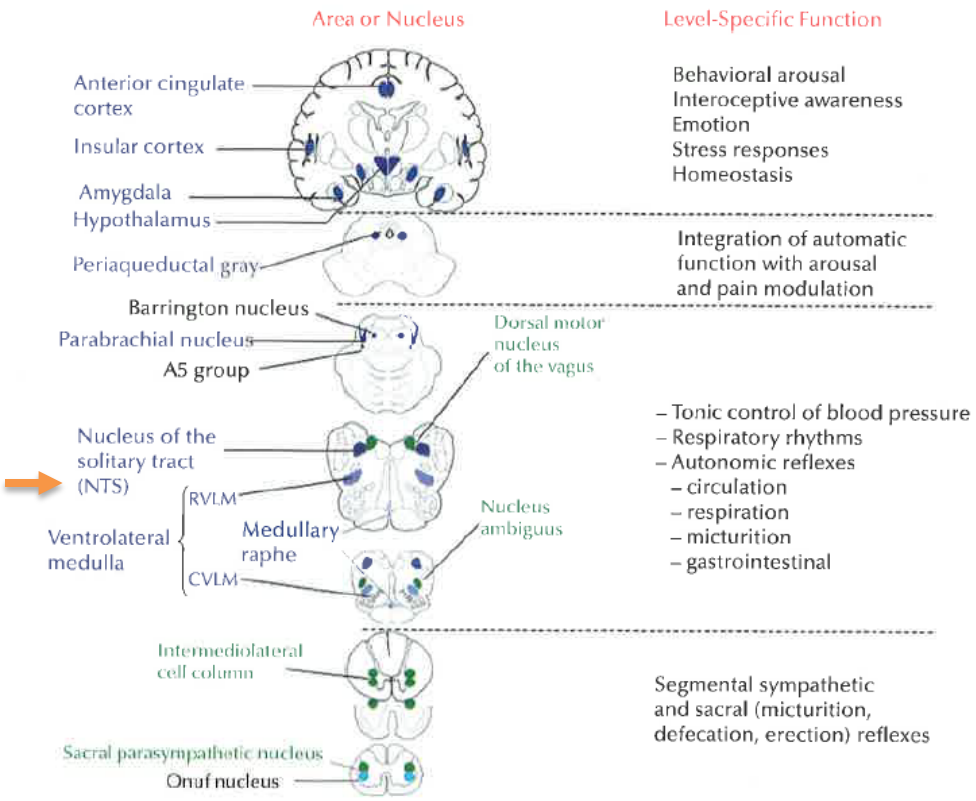
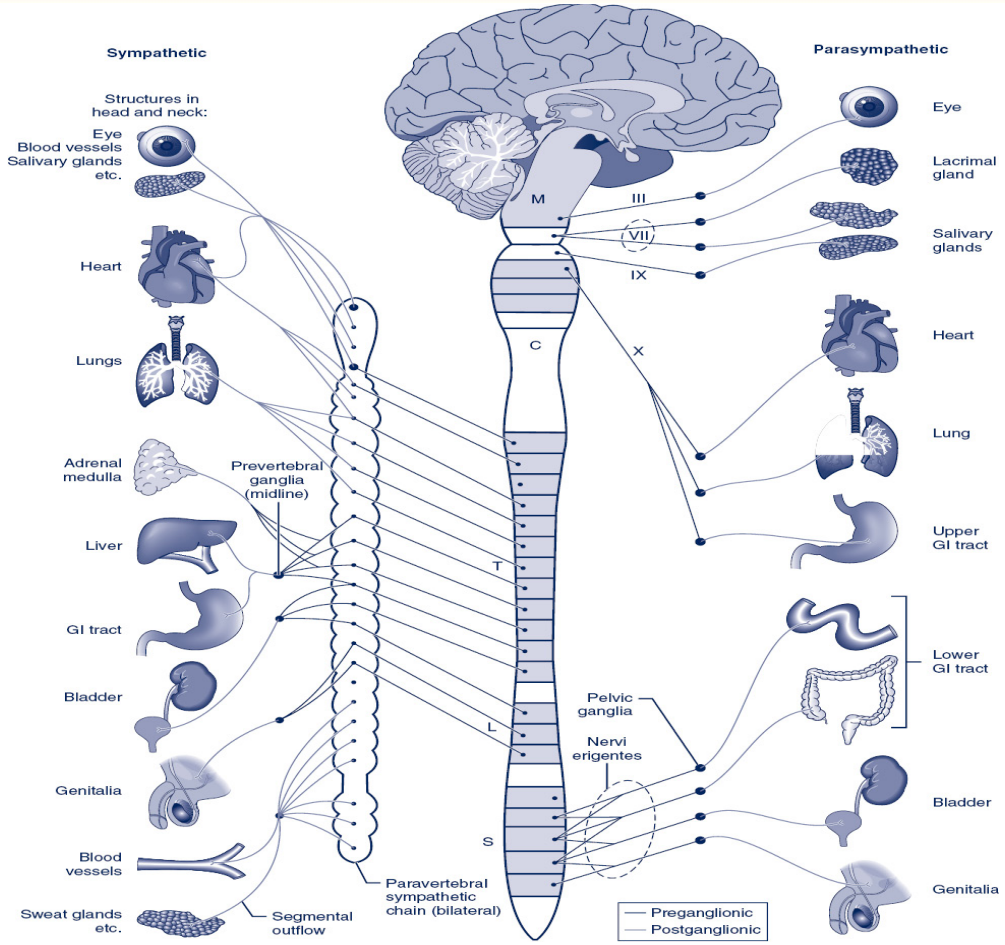
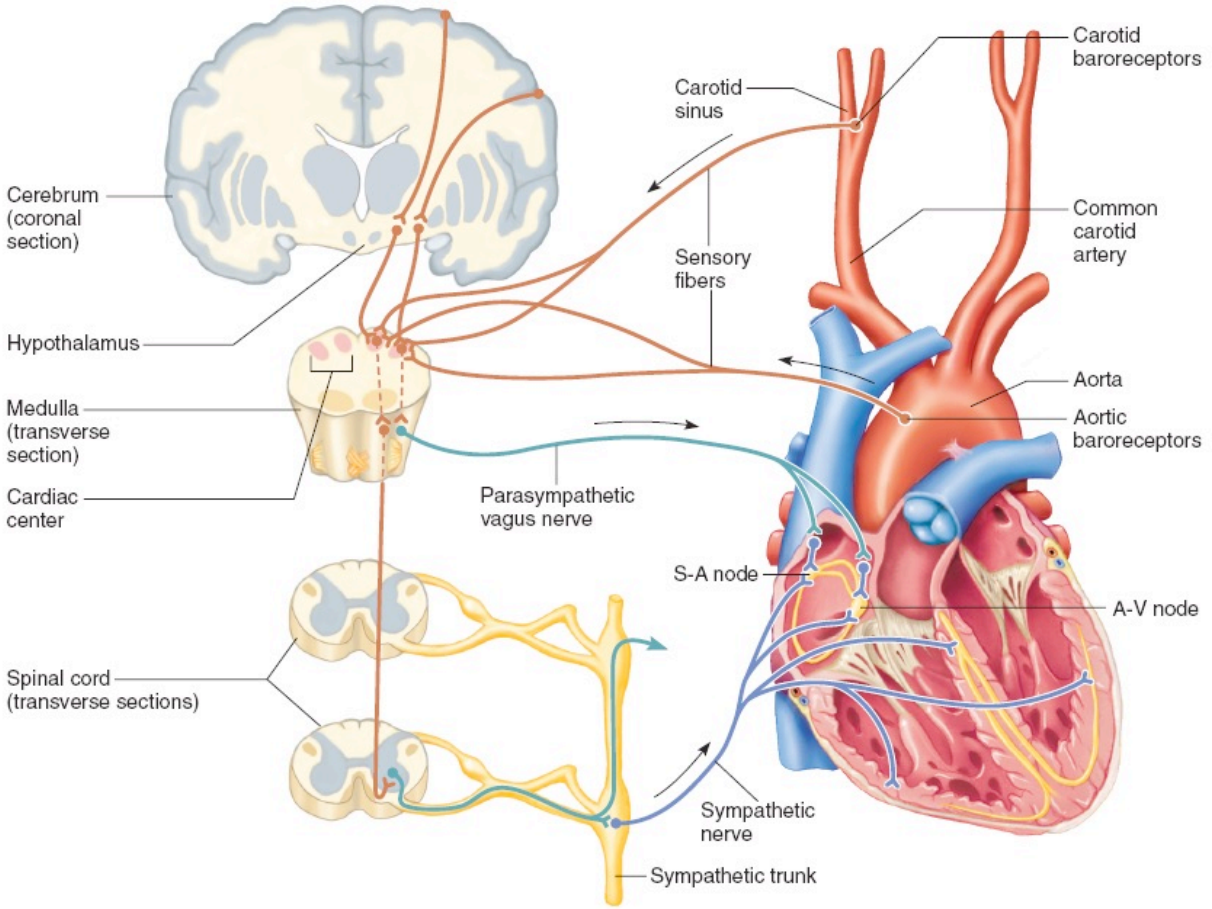


Figure 1.1. Central autonomic control network. Distribution of autonomic control areas in the central nervous system and level-specific organization of autonomic control. Abbreviations: CVLM, caudal ventrolateral medulla; RVLM, rostral ventrolateral medulla.

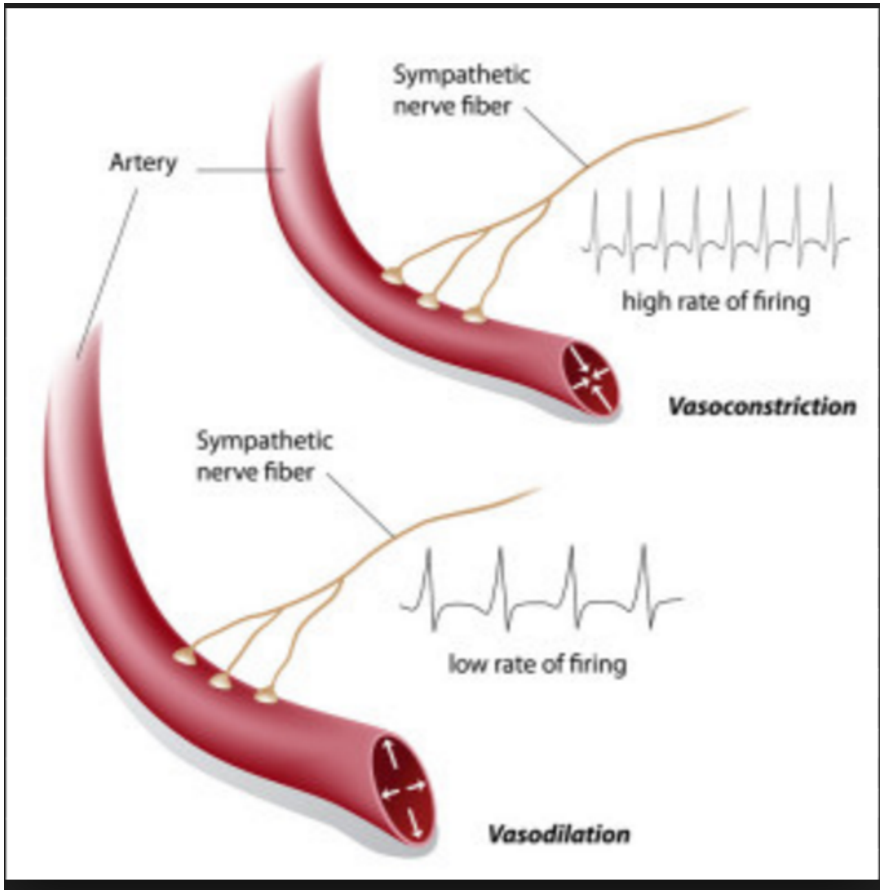
Organization of the Autonomic Nervous System



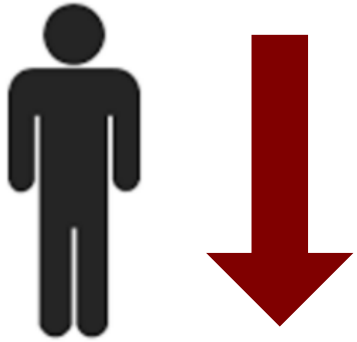
Cardiac Conduction System



Sympathetic Outflow to the Vasculature



Positional Considerations



- Baroreceptor activation resulting from a fall in blood pressure leads to a withdrawal of vagal activity to the sinoatrial node permitting an increase in heart rate, and a reduction of NTS inhibition of the CVLM.
- This will unencumber the RVLM to augment sympathetic outflow to increase peripheral vascular resistance (MAP), myocardial contractility and, ultimately, cardiac output.

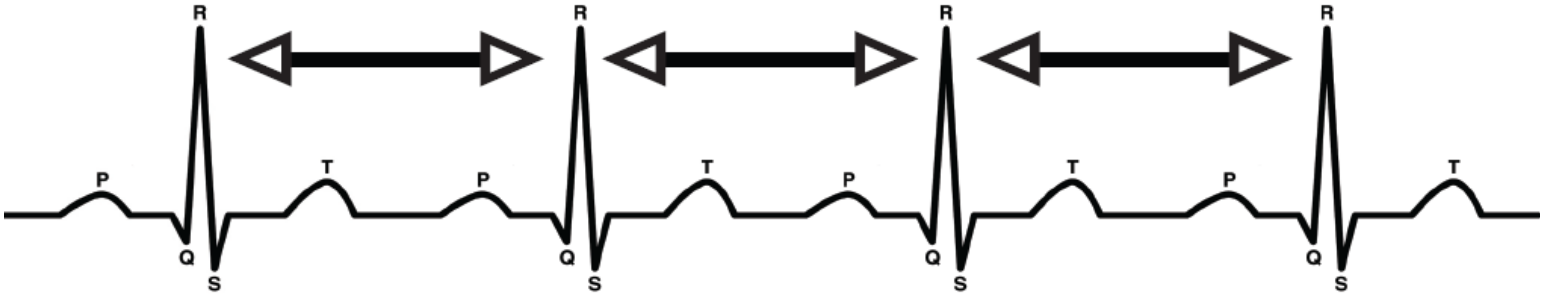
Additional Readings

Int J Psychophysiol. 2018 Oct;132(Pt A):155-166. doi: 10.1016/j.ijpsycho.2017.11.016. Epub 2017 Nov 29.

An anatomical and physiological basis for the cardiovascular autonomic nervous system consequences of sport-related brain injury.

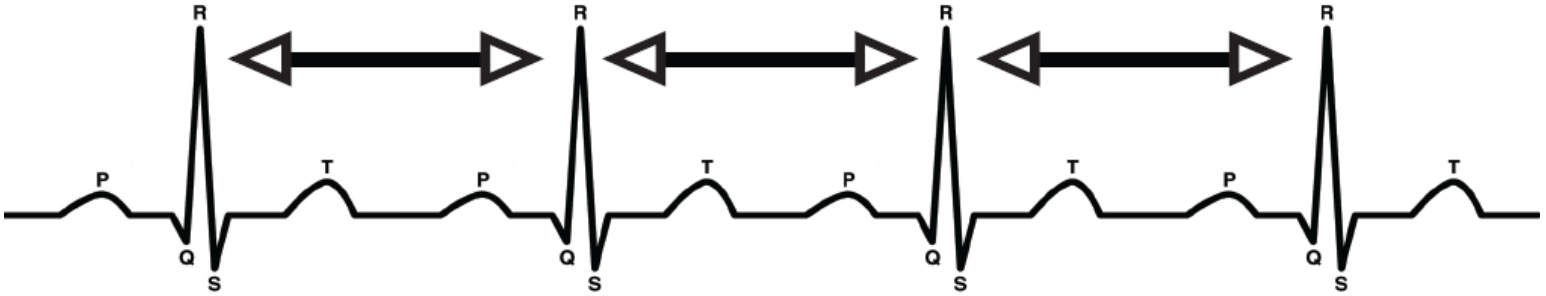
La Fountaine MF[1].

Cardiac Autonomic Modulation



- Acute
  - Altered physical or mental state
  - Medications, ingested substances
- Chronic
  - Subclinical/clinical morbidity
  - Predictive of arrhythmia and sudden cardiac death

Cardiac Autonomic Modulation



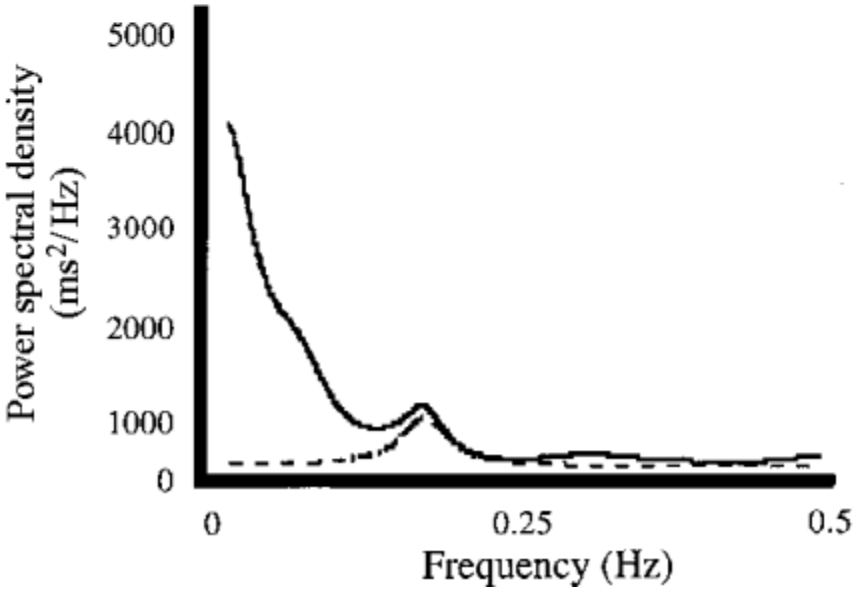
- Time domain parameters

Parameter	Unit	Description
SDNN	ms	Standard deviation of NN intervals
SDRR	ms	Standard deviation of RR intervals
SDANN	ms	Standard deviation of the average NN intervals for each 5 min segment of a 24 h HRV recording
SDNN index (SDNNI)	ms	Mean of the standard deviations of all the NN intervals for each 5 min segment of a 24 h HRV recording
pNN50	%	Percentage of successive RR intervals that differ by more than 50 ms
HR Max – HR Min	bpm	Average difference between the highest and lowest heart rates during each respiratory cycle
RMSSD	ms	Root mean square of successive RR interval differences
HRV triangular index		Integral of the density of the RR interval histogram divided by its height
TINN	ms	Baseline width of the RR interval histogram

Cardiac Autonomic Modulation

Computation methods

- Linear
  - fast Fourier transform
    - Low Frequency (LF) oscillations (0.04-0.15 Hz)
    - High Frequency (HF) oscillations (0.15-0.4 Hz)
    - Sympathovagal balance (LF:HF ratio)
- Non-linear
  - Entropy
  - Complexity
  - Fractal scaling dimension



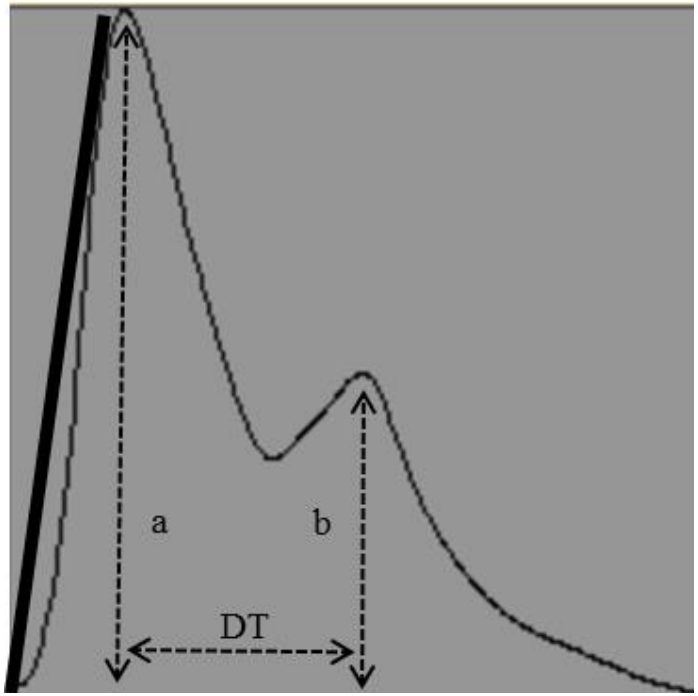
Cardiac Autonomic Modulation

Brain Inj. 2016;30(2):132-45. doi: 10.3109/02699052.2015.1093659. Epub 2015 Dec 15.

The impact of concussion on cardiac autonomic function: A systematic review.

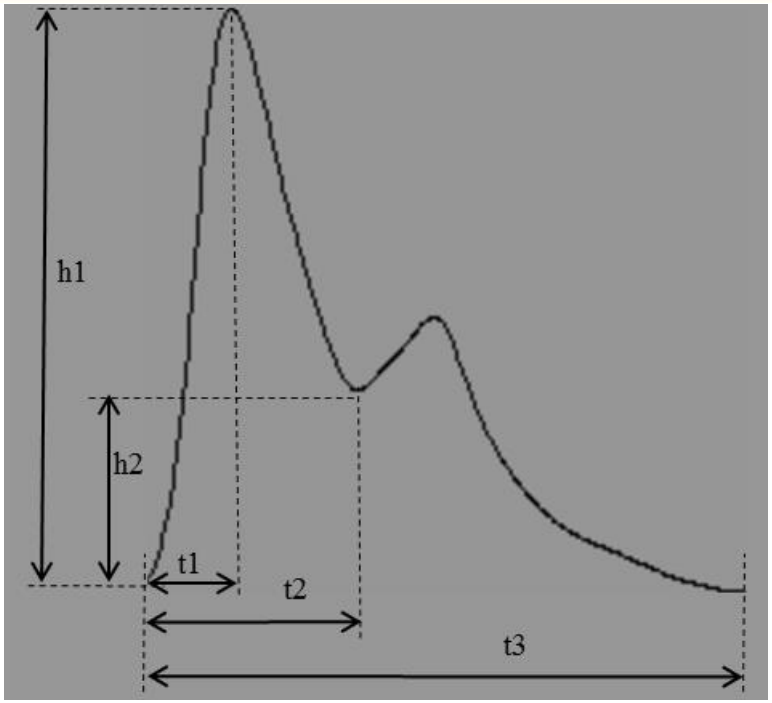
Blake TA[1], McKay CD[2], Meeuwisse WH[1,3], Emery CA[1,4,5].

Vascular Sympathetic Outflow



Finger Arterial Pulse Contour Characteristics

Systolic slope (ΔmmHg/Δtime)
Reflection Index (b/a)
Stiffness Index (height/DT).

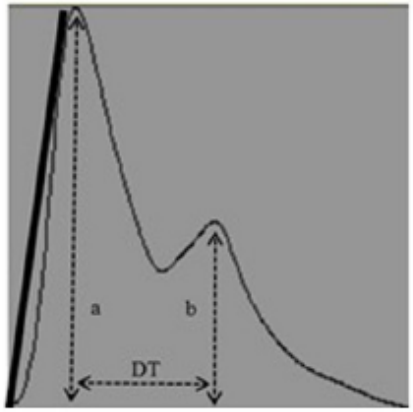


Dichrotic Notch Characteristics

Peak Latency (t1/t3),
Notch Relative Amplitude (h2/h1),
Notch Latency (t2/t3),
Peak-to-Notch Latency [(t2-t1)/t3)],
Notch Index [t2/(t3-t2)].

La Fountaine MF, 2016.

Vascular Sympathetic Outflow



	SysSlope (mmHg/s)		RI (%)		SI (m/s)		DT (ms)	
	Rest	First 60	Rest	First 60	Rest	First 60	Rest	First 60
	48 Hrs							
Control	458 ± 236*	486 ± 222*	74 ± 13	74 ± 11	5.8 ± 0.5	6.0 ± 0.7	312 ± 28	299 ± 15
Concussion	408 ± 132	370 ± 101	79 ± 4	78 ± 4	6.0 ± 0.5	6.2 ± 0.5	288 ± 16	302 ± 31
	Week 1							
Control	435 ± 132†	434 ± 133†	77 ± 7	76 ± 6	5.9 ± 0.4	5.9 ± 0.5	307 ± 20	299 ± 15
Concussion	419 ± 167	401 ± 119	77 ± 6	79 ± 4	6.0 ± 0.6	5.9 ± 0.6	301 ± 19	309 ± 21

FIGURE 2 | Finger arterial pulse contour characteristics of groups by condition and visit. The Systolic slope (SysSlope; black line, left panel) is calculated from the rate of rise (change in pressure/change in time) of the systolic upstroke. The reflection index (RI) is calculated as b/a. The stiffness index (SI) is calculated as body height/DT. Data are presented as group mean ± SD in table (right panel). *Control vs. concussion: $p < 0.0001$; †control vs. concussion: $p < 0.01$.

La Fountaine MF, 2016.

Baroreceptor Sensitivity

- The ability and tolerance of the baroreflex arc to buffer changes in arterial blood pressure. Chapleau, 2001
- Robust indicator of cardiovascular autonomic function in a range of health and disease states. Chapleau, 2001
- Quantified from beat-to-beat variations of simultaneous digital recordings of heart rate and arterial blood pressure at rest (spontaneous), or during hemodynamic provocations (e.g., physical, pharmaceutical). Bristow, 1969; Chen, 1982; Hughson, 1993

Baroreceptor Sensitivity

TABLE 1. Demographic characteristics of participants.

	Control	Concussion
n	10	10
Age (yr)	19.8 ± 1.0	19.6 ± 1.0
Height (m)	1.78 ± 0.10	1.79 ± 0.14
Weight (kg)	79.1 ± 13.0	83.1 ± 9.5
BMI ($kg \cdot m^{-2}$)	24.8 ± 1.7	25.3 ± 3.5
Ethnicity (AA/C/H) (*n*)	1/7/2	2/7/1
Sports		
Basketball (*n*)	2	3
Baseball (*n*)	1	0
Soccer (*n*)	7	6
Swimming (*n*)	0	1

Data are presented as group mean ± SD.
AA, African American; BMI, body mass index; C, Caucasian; H, Hispanic.

TABLE 2. Hemodynamic and cardiovascular autonomic characteristics by visit.

	Visit 1		Visit 2	
	Control	Concussion	Control	Concussion
HR (bpm)	64 ± 6	61 ± 11	60 ± 6	66 ± 8
SBP (mm Hg)	116 ± 17	121 ± 20	121 ± 10	122 ± 16
DBP (mm Hg)	59 ± 5	65 ± 10	58 ± 9	66 ± 10
MAP (mm Hg)	80 ± 7	84 ± 12	82 ± 11	84 ± 11
Respiration rate (per min)	13.9 ± 0.1	14.7 ± 0.1	14.1 ± 0.1	13.7 ± 0.1
HF-HRV ($ms^2 \cdot Hz^{-1}$)	3.36 ± 0.50	3.26 ± 0.58	3.50 ± 0.42	3.10 ± 0.59
LF-HRV ($ms^2 \cdot Hz^{-1}$)	3.63 ± 0.48	3.59 ± 0.40	3.61 ± 0.28	3.43 ± 0.35
LF-SBP ($mm\ Hg^2 \cdot Hz^{-1}$)	1.28 ± 0.70	1.52 ± 0.99	1.20 ± 0.44	1.34 ± 0.49

Data are presented as group mean ± SD.
MAP, mean arterial pressure.

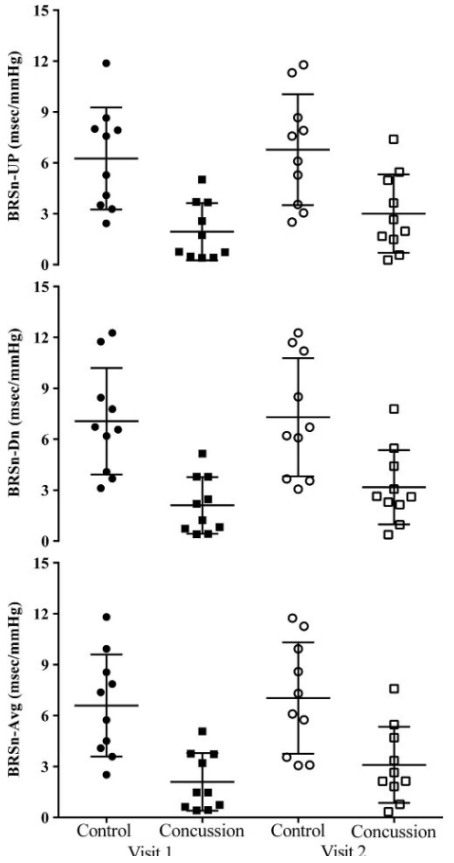
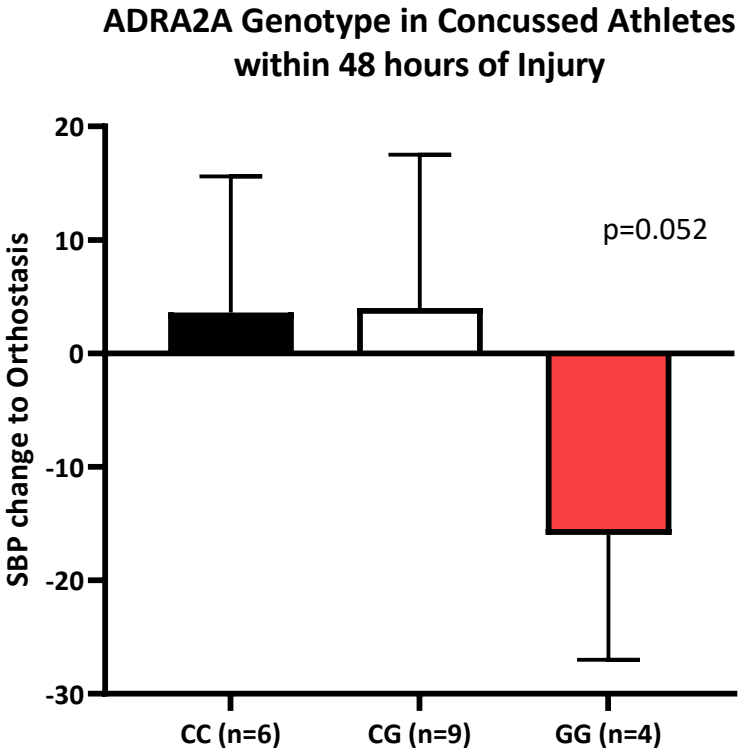


FIGURE 1—Normalized BRS dot plots by group and visit. Data are presented as group mean ± SD. Group means (collapsed across visit) were significantly lower in concussion vs controls, and all standardized mean differences showed large effect sizes (all $d > 1.20$).

La Fountaine MF, 2019.

Response to Orthostasis after Concussion (Preliminary Work)

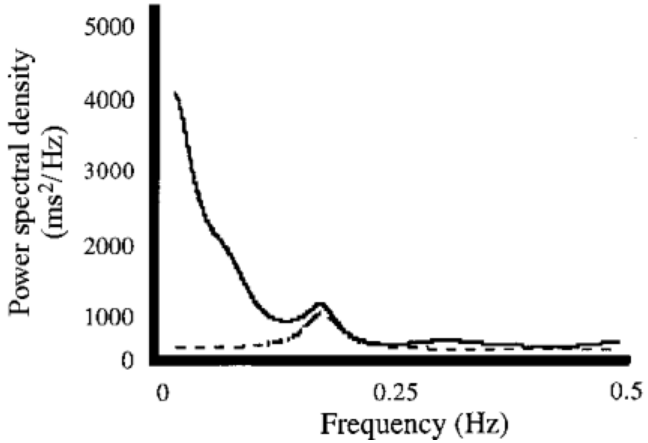


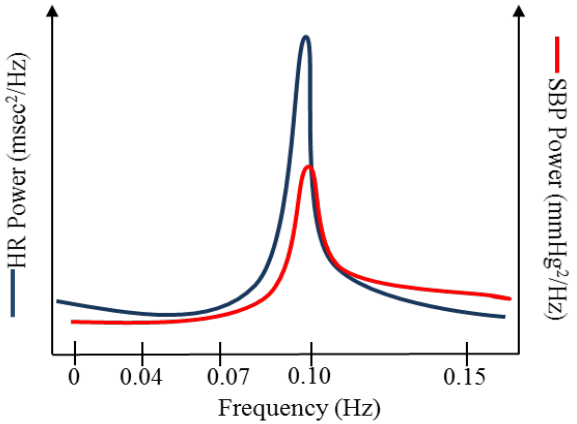
Presented at 2019 American College of Sports Medicine Annual Meeting. Orlando, FL.

La Fountaine MF, Hohn AN, Leahy CL, Testa AJ, Weir JP. Systolic Blood Pressure Response to Orthostasis after Concussion is Related to ADRA2A Gene Receptor Subtypes.

Mayer Waves

- Mayer waves (MW) likely arise through baroreflex adjustments to blood pressure oscillations and appear in the LF band of the HR and BP power spectra at a ~0.1 hertz (Hz) in humans. (Mayer, 1876; Julien, 2006)



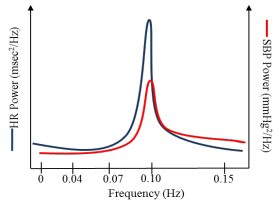


Mayer Waves (Preliminary Work)

Table 2. Cardiovascular Autonomic and Hemodynamic Profile of Participants by Visit

	Visit 1			Visit 2		
	Control	Concussion	p value	Control	Concussion	p value
Heart Rate (bpm)	63 ± 7	61 ± 10	NS	64 ± 9	64 ± 8	NS
Systolic Blood Pressure (mmHg)	112 ± 20	118 ± 18	NS	114 ± 12	117 ± 17	NS
Diastolic Blood Pressure (mmHg)	62 ± 5	66 ± 9	NS	66 ± 10	65 ± 11	NS
Heart Period Power Spectrum (msec^2/Hz)						
Low Frequency (0.04 - 0.15 Hz)	3.55 ± 0.52	3.65 ± 0.46	NS	3.49 ± 0.43	3.44 ± 0.44	NS
Mayer Wave Amplitude (~0.1 Hz)	4.42 ± 0.61	4.40 ± 0.61	NS	4.09 ± 0.65	4.00 ± 0.80	NS
Systolic Blood Pressure Power Spectrum (mmHg^2/Hz)						
Low Frequency (0.04 - 0.15 Hz)	0.99 ± 0.45	0.94 ± 0.57	NS	0.96 ± 0.37	0.93 ± 0.51	NS
Mayer Wave Amplitude (~0.1 Hz)	1.92 (1.64, 2.21)	1.39 (1.11, 1.68)	<0.05	1.75 (1.50, 2.00)	1.77 (1.52, 2.02)	NS

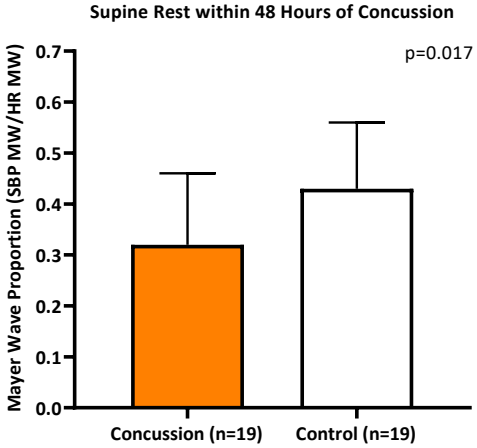
Data are presented as group mean ±SD or group estimated marginal mean (95% confidence interval). Abbreviations: bpm= beats per minute; Hz= hertz; mmHg= millimeters of mercury; NS= not significant. P values represent significant main effects for each visit.

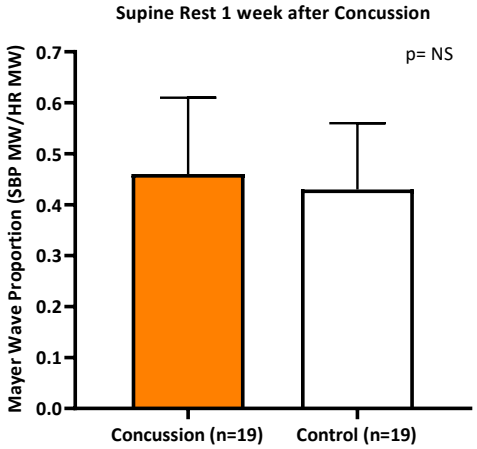


Presented at 2018 American College of Sports Medicine Annual Meeting. Denver, CO.

La Fountaine MF, Testa AJ, Weir JP. Discordance of Autonomic Discharge to the Cardiovascular System Following Concussion.

Manuscript submitted for peer review

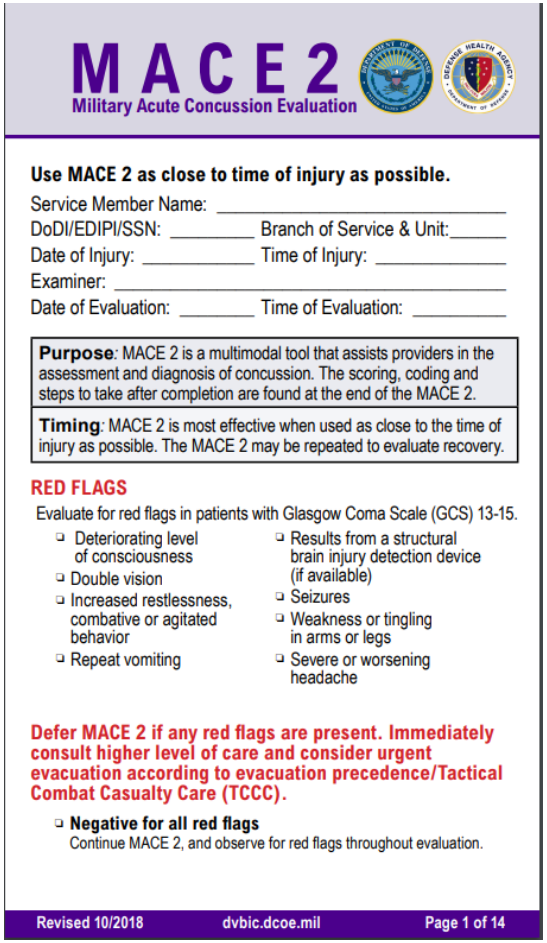




Relevance

- Military Acute Concussion Evaluation 2

https://dvbic.dcoe.mil/system/files/resources/MACE2.pdf



MACE 2
Military Acute Concussion Evaluation

Use MACE 2 as close to time of injury as possible.

Service Member Name: _____
DoDI/EDIPI/SSN: _____ Branch of Service & Unit: _____
Date of Injury: _____ Time of Injury: _____
Examiner: _____
Date of Evaluation: _____ Time of Evaluation: _____

Purpose: MACE 2 is a multimodal tool that assists providers in the assessment and diagnosis of concussion. The scoring, coding and steps to take after completion are found at the end of the MACE 2.

Timing: MACE 2 is most effective when used as close to the time of injury as possible. The MACE 2 may be repeated to evaluate recovery.

RED FLAGS

Evaluate for red flags in patients with Glasgow Coma Scale (GCS) 13-15.

- ❑ Deteriorating level of consciousness
- ❑ Double vision
- ❑ Increased restlessness, combative or agitated behavior
- ❑ Repeat vomiting
- ❑ Results from a structural brain injury detection device (if available)
- ❑ Seizures
- ❑ Weakness or tingling in arms or legs
- ❑ Severe or worsening headache

Defer MACE 2 if any red flags are present. Immediately consult higher level of care and consider urgent evacuation according to evacuation precedence/Tactical Combat Casualty Care (TCCC).

- ❑ **Negative for all red flags**
  Continue MACE 2, and observe for red flags throughout evaluation.

Revised 10/2018 dvbic.dcoe.mil Page 1 of 14

Relevance

- Return-to-duty

MILITARY MEDICINE, 184, 3/4:160, 2019

Evaluation of the Military Functional Assessment Program for Return-to-Duty Decision Making: A Longitudinal Study

Amanda M. Kelley; Mark Showers†*

Key Takeaways

- Cardiovascular autonomic dysfunction can present in a myriad ways following concussive head trauma.
- Abnormal heart rates both at rest and/or in response to psychological and physiological load.
- Paradoxical or augmented arterial blood pressure responses to orthostasis or exertional tasks.
- Resolution of autonomic dysfunction following concussion often mirrors that of affective and somatic symptoms, but may persist for months after injury in absence of other symptoms.
- Not discussed….could influence sleep/wake cycles

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