A Novel Acute Detection Method for Mild Traumatic Brain Injury

UNIVERSITY of WASHINGTON TACOMA

Austin Millen, John Tarasidis, Dr. Joyce Dinglasan-Panlilio, Dr. Patrick Reynolds

INTRODUCTION

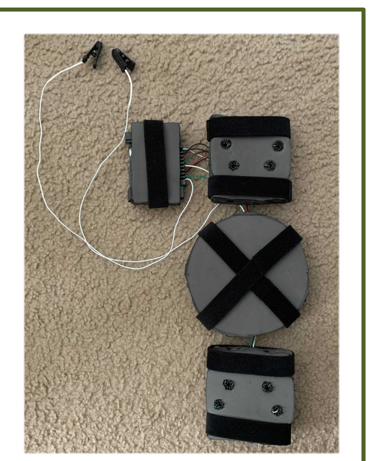
Traumatic Brain Injuries (TBIs) have been a source of focus for researchers since the earliest recorded incidences as "shell shock" seen in World War I soldiers with head trauma[8]. Today, TBIs are still a wide-spread occurrence with a reported incidence of 200/100,000 population, most often the result of head trauma from sports injuries, military conflicts, accidents, or abuse[1]. Approximately 50% to 90% of patients with mild TBIs (mTBIs) go unidentified or undiagnosed in healthcare settings, forming a vulnerable population susceptible to second trauma and life-long neurological deficits[2,7]. TBIs have become a source of focus for both the Health and Defense sectors, the main goals being development of high accuracy diagnostic techniques and an improved understanding of the pathophysiological cascade from the time of injury to complete brain recovery.

OBJECTIVES

- Perform miniature literature review on current understanding of TBI pathophysiology and care pathways
- Develop and test first iteration of a portable helmet electroencephalogram (EEG) device using dry electrodes

METHODS AND MATERIALS

- PubMed search and review of approximately 100 abstracts involving key words "Pathophysiology", "TBI", and "Standard of Care"
- Development of dry electrode form and coating
- Drop tests of inertial and pressure threshold sensors and electrode channel detection ability



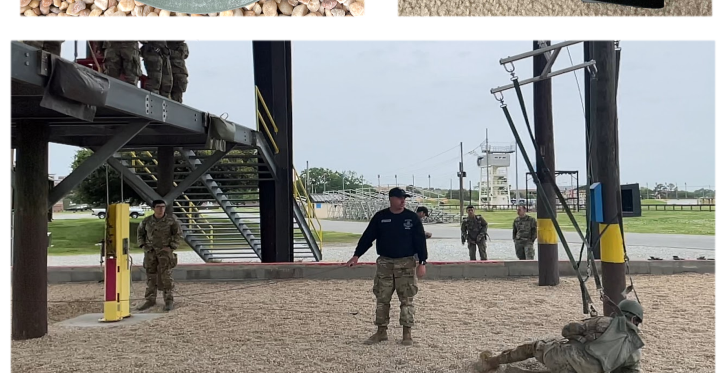


Figure 1 (top left): EEG device in helmet; Figure 2 (top right): EEG device stand-alone;
Figure 3: (bottom): Ft. Benning Drop Tests.
Pictures courtesy of John Tarasidis

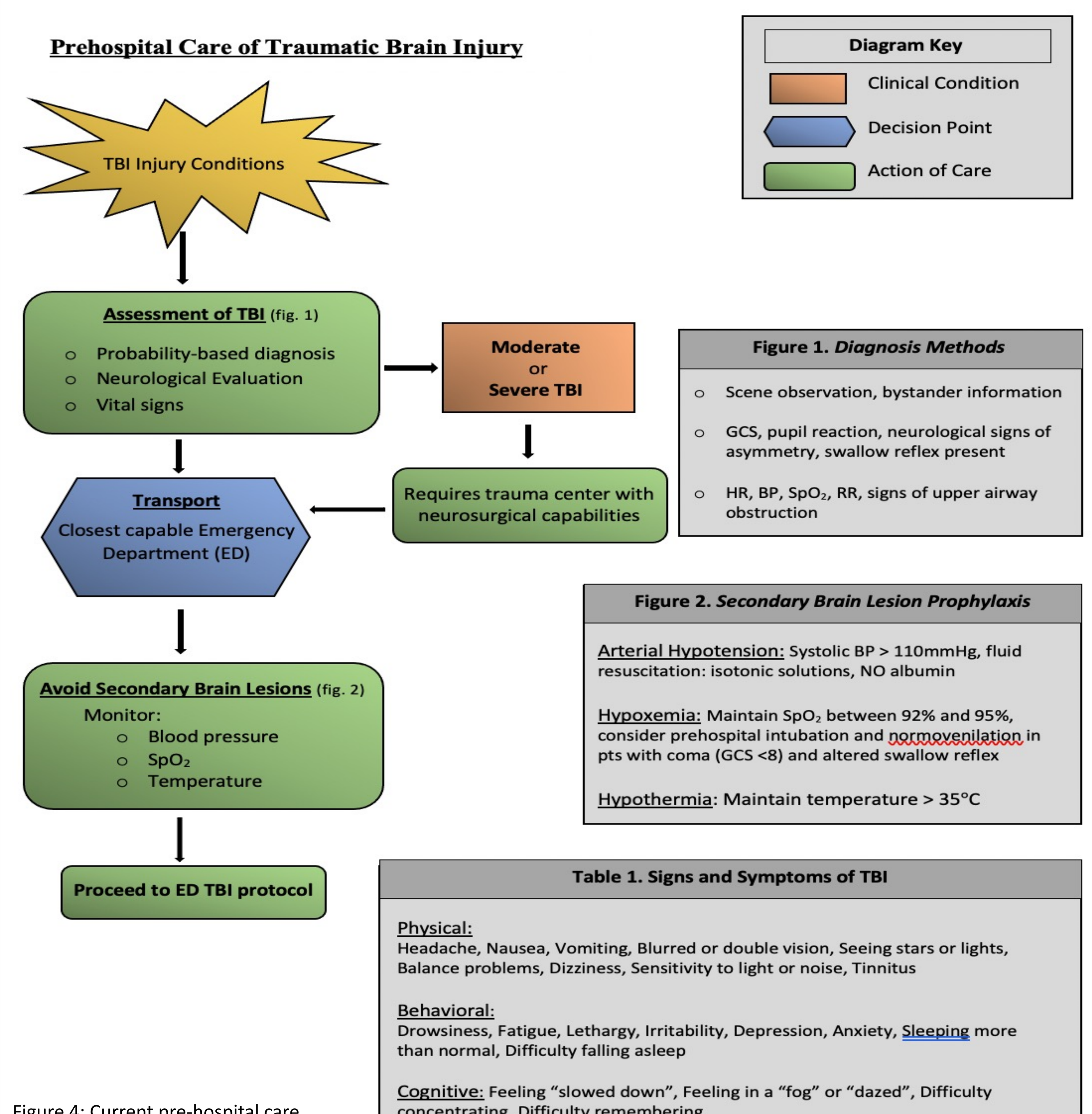


Figure 4: Current pre-hospital care algorithm. Adapted from Pélieu et al.[6]

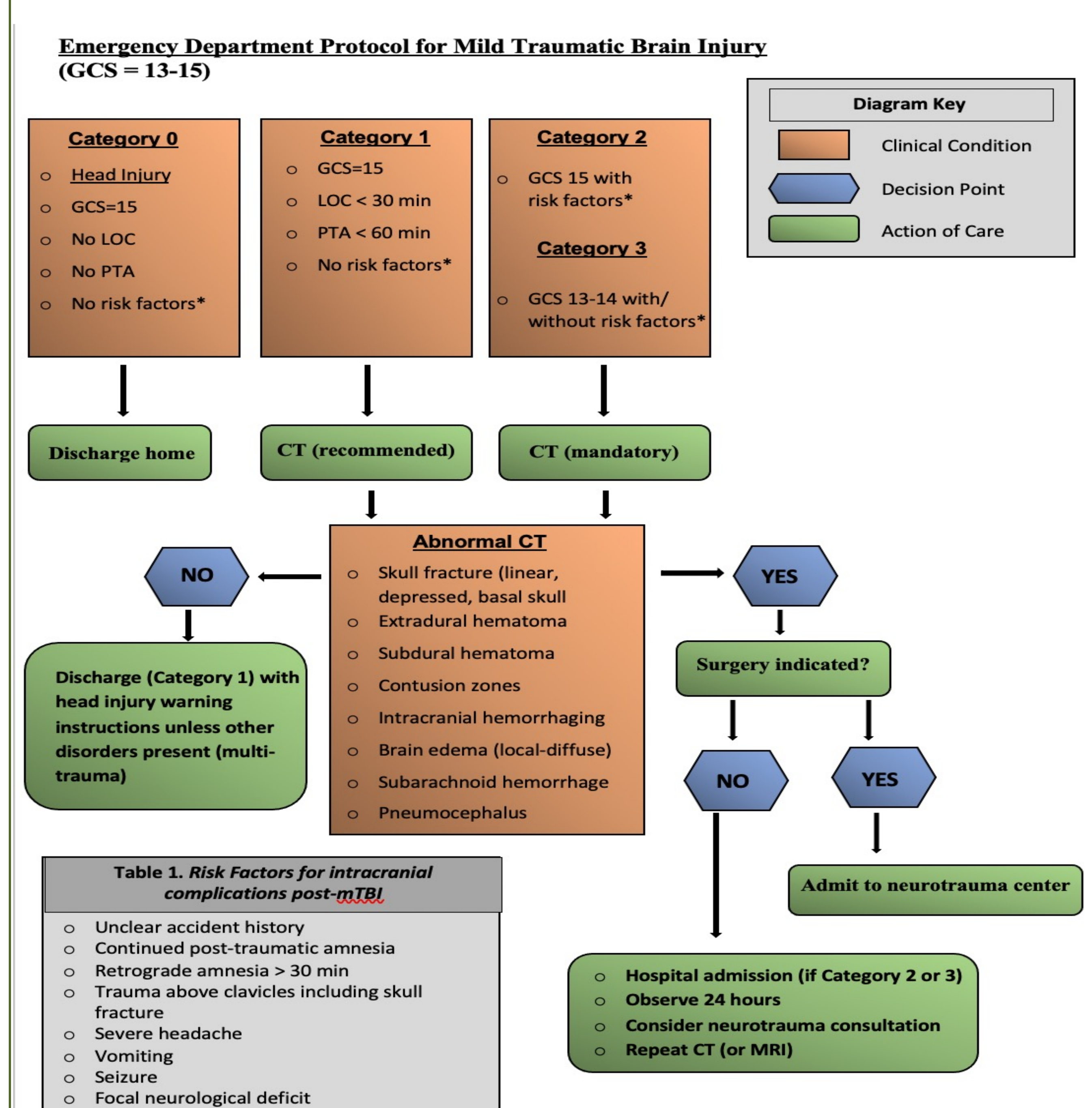


Figure 5: Current Emergency Department care algorithm. Adapted from Vos et al.[11]

RESULTS

- A suspected unregulated influx of ions into neurons occur from mechanical shearing/stretching brain tissue[4,5]
- Indiscriminate release of neurotransmitters result, kickstarting the metabolic cascade of the injury[3,5]
- Subsequent metabolic cascade is likely primary injury mechanism in TBIs post-initial insult[9]
- Biomarker assays to detect TBIs are being investigated, but brain scans using CT continue to be gold standard of care[10]

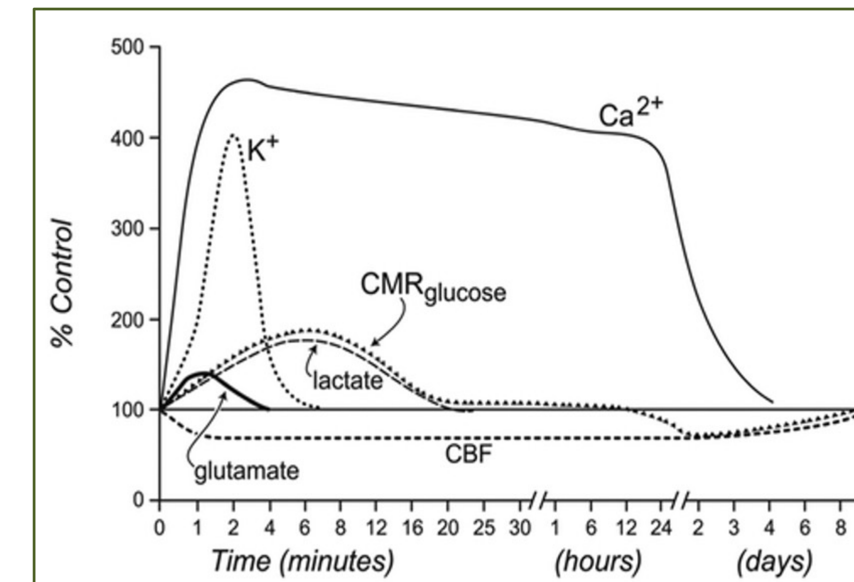


Figure 6: Neurometabolic cascade following concussion in mouse models
K+, potassium; CMRglucose, cerebral metabolic rate of glucose utilization; Ca2+, calcium; CBF, cerebral blood flow. Image credit: Hovda et al. (https://journals-sagepub-com.offcampus.lib.washington.edu/doi/full/10.1177/1941738111433673 [5]. accessed 22June2021.)

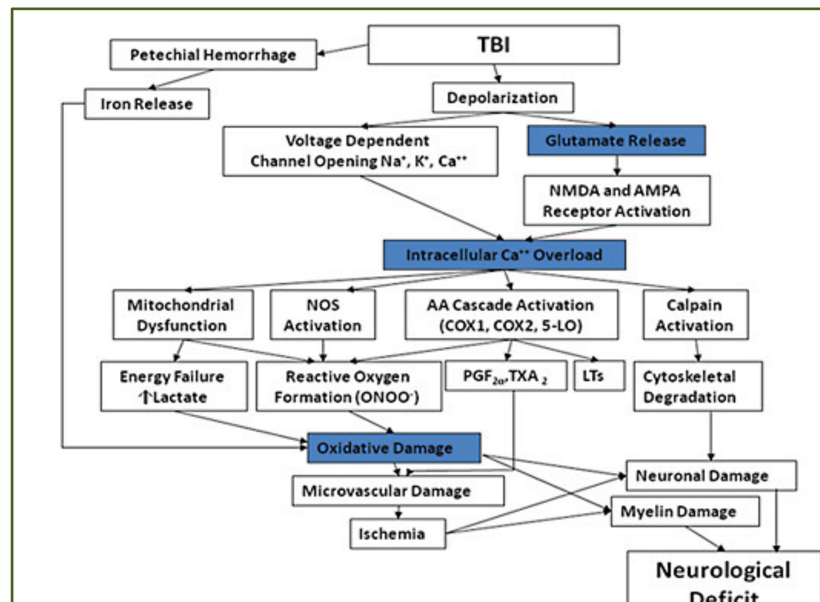


Figure 7: Schematic of posttraumatic secondary injury showing the cascade of pathophysiological and pathochemical players and their inter-relationships. Image credit: Hall, 2007. (https://www.nap.edu/read/13121/chapter/26#383 [3]. Accessed 15June2021)

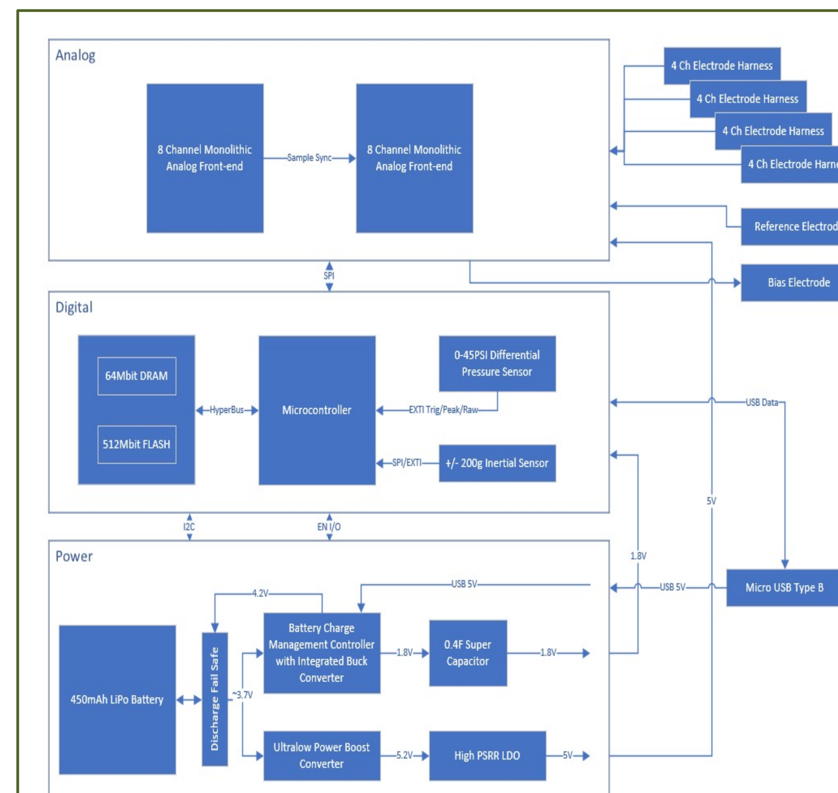


Figure 8: Iteration 1 of Automatic Concussion Evaluation (ACE) device.
Photo courtesy of John Tarasidis

- Electrodes redesigned using flexible silicone structure with Ag/AgCl coating for improved dry contact
- Channels now feature modular design and raised to 16 channels, approaching 32 channel standard clinical setup

REFERENCES

1. Carrone, S., Alwis, D., & Rajan, R. (2016). Traumatic Brain Injury and Neuronal Functionality Changes in Sensory Cortex. *Front Syst Neurosci, 10*(47)10.3389/fnsys.2016.00047
2. Giza, C., & Choe, M. "Diagnosis and Management of Acute Concussion," *Seminars in Neurology*, vol. 35, no. 01, pp. 029–041, 2015.
3. Hall, E. (1993). Lipid antioxidants in acute central nervous system injury. *Annals of Emergency Medicine, 22*(6), 1022-1027. S0196-0644(05)82745-3
4. Katayama, Y., Becker, D., Tamura, T., & Hovda, D. (1990). Massive increases in extracellular potassium and the indiscriminate release of glutamate following concussive brain injury. *Journal of Neurosurgery, 73*(6)10.3171/jns.1990.73.6.0889
5. Newer, M., Hovda, D., Schrader, L., & Vespa, P. (2005). Routine and quantitative EEG in mild traumatic brain injury. *Clinical Neurophysiology, 116*(9), 2001-2025. j.clinph.2005.05.008
6. Pélieu, I., Kull, C., & Walder, B. (2019). Prehospital and Emergency Care in Adult Patients with Acute Traumatic Brain Injury. *Med Sci(Basel), 7*(1), 12. 10.3390/medsci7010012
7. Prince, C., & Bruhns, M. "Evaluation and Treatment of Mild Traumatic Brain Injury: The Role of Neuropsychology," *Brain Sciences*, vol. 7, no. 12, p. 105, 2017.
8. Shively, S., & Perl, D. (2012). Traumatic brain injury, shell shock, and post-traumatic stress disorder in the military-- past, present, and future. *Journal of Head Trauma Rehabilitation, 27*(3)10.1097/htr.0b013e318250e9dd
9. Signoretti, S., Lazzarino, G., Tavazzi, B., & Vagnozzi, R. (2011). The Pathophysiology of Concussion. *Physical Medicine and Rehabilitation, 3*(10), S359-S368
10. Vagnozzi, R., Signoretti, S., Cristofori, L., Alessandrini, F., Floris, R., Isgrò, E., Lazzarino, G. (2010). Assessment of metabolic brain damage and recovery following mild traumatic brain injury: a multicentre, proton magnetic resonance spectroscopic study in concussed patients. *Brain, 133*(11), 3232-3242. 10.1093/brain/awq200
11. Vos, P., Battistin, L., Birbamer, G., Gerstenbrand, F., Potapov, A., Prevec, T., Von Wild, K. (2002). EFNS guideline on mild traumatic brain injury: report of an EFNS task force. *European Journal of Neurology, 9*, 207-219

ACKNOWLEDGEMENTS

Special thanks to mentors Joyce Dinglasan-Panlilio, Ph.D. of UWT and Patrick Reynolds, Ph.D. of NSIN. In addition, thanks to NSIN for their X-Force Fellowship that made this project possible.

A portion of this project was funded by NSIN, and the continuing project is a partnership with Georgia Institute of Technology

Georgia Institute of Technology
NSIN