DEFENSE CENTERS OF EXCELLENCE

For Psychological Health & Traumatic Brain Injury

Hearing and Vision Impairment from Combat Trauma

July 24, 2014, 1-2:30 p.m. (EDT)

Presenters

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DVBIC Defense and Veterans Brain Injury Center

DHCC Deployment Health Clinical Center

National Center for Telehealth & Technology

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Moderator

Christian Shenouda, M.D.
TBI Physician
Contract support to the
Defense and Veterans Brain Injury Center
Silver Spring, Md.

DVBIC Defense and Veterans Brain Injury Center

DHCC

Webinar Details

- Live closed captioning is available through Federal Relay Conference Captioning (see the “Closed Captioning” box)
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  - Dial: CONUS **888-877-0398**; International **210-234-5878**
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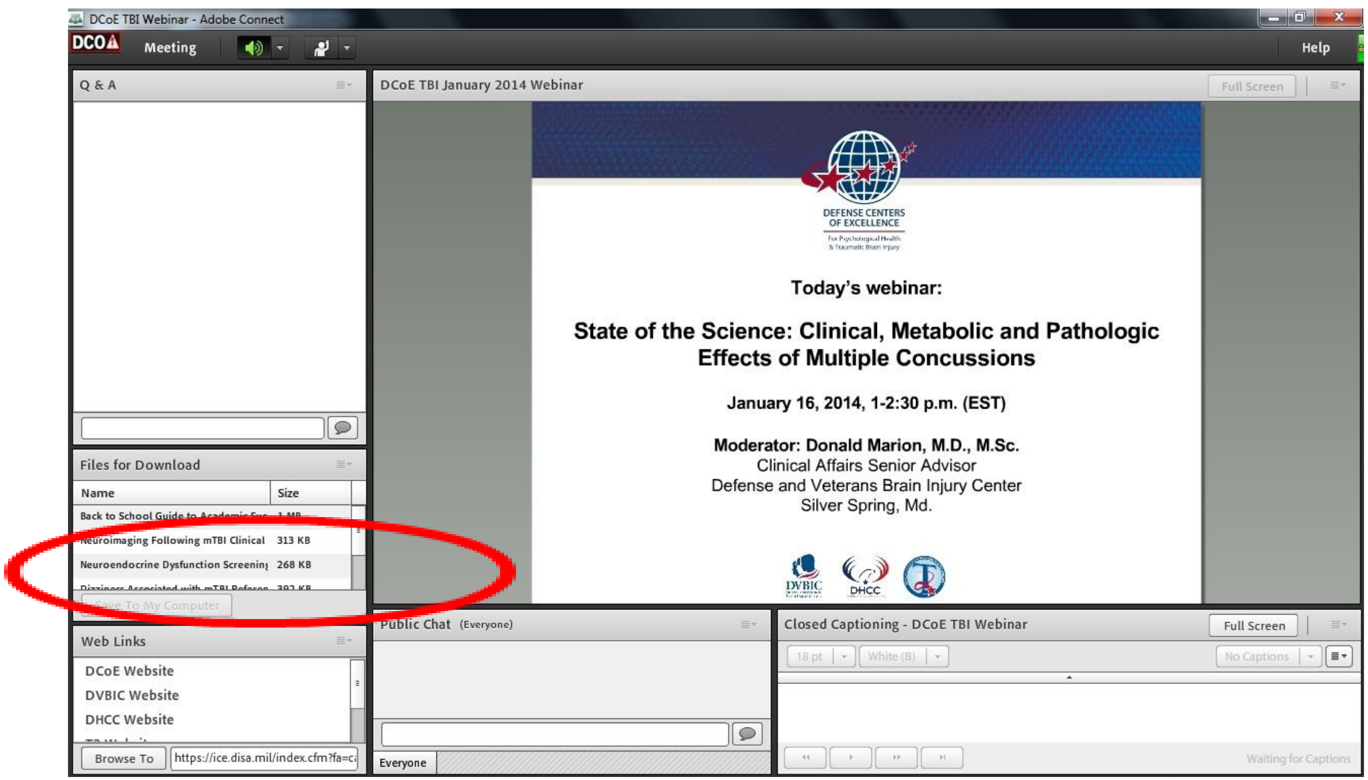

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- The authority for training of contractors is at the discretion of the chief contracting official.
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- Credit Designation – The Duke University School of Medicine designates this live webinar for:
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  - 0.15 IACET continuing education credit
  - 1.5 NBCC contact hours credit commensurate to the length of the program
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  - 1.5 NASW contact hours commensurate to the length of the program for those who attend 100% of the program

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- Psychology: This activity complies with all of the Continuing Education Criteria identified through the **North Carolina Psychology Board's Continuing Education Requirements** (21 NCAC 54.2104). Learners may take the certificate to their respective State Boards to determine credit eligibility for contact hours.
- NASW: **National Association of Social Workers (NASW),** North Carolina Chapter: Southern Regional AHEC will award contact hours commensurate to the length of the program to participants who attend 100% of the program.

NASW North Carolina Chapter
National Association of Social Workers
Approval Year
2014

Questions and Chat

Throughout the webinar, you are welcome to submit technical or content-related questions via the Q&A pod located on the screen. **Please do not submit technical or content-related questions via the chat pod**.

The Q&A pod is monitored during the webinar, and questions will be forwarded to our presenter for response during the question-and-answer session of the webinar.

Participants may also chat amongst each other during the webinar using the chat pod.

We will keep the chat function open 10 minutes after the conclusion of the webinar.

Webinar Overview

Consequences of blast exposure can lead to a variety of ocular, otologic and vestibular injuries. Serious combat eye trauma accounts for approximately 15 percent of all battlefield injuries and up to 75 percent of those affected experience short or long-term visual dysfunction. Hearing loss and tinnitus prevalence for service members and veterans is increasing 13-18 percent annually and account for the top two most common service-connected disabilities among veterans.

The majority of neurosensory disorders resulting from combat trauma are caused by mild traumatic brain injury. Understanding the mechanics of the injury and the associated research guides patient care and treatment recommendations. While there are assistive devices, technologies, training programs, and support groups, to assist those with vision or hearing loss, it is also important to provide psychological health support to the injured and their families.

This webinar will examine current research and evidence-based practices to reduce injury risk and enable prevention and treatment in both clinical and non-clinical settings.

During this webinar, participants will learn to:

- Describe hearing and balance disorders associated with combat trauma
- Articulate the basic science concepts of neurosensory disorders
- Incorporate evidence-based best practices into approaches supporting care and treatment methodologies
- Summarize methods and tools known to reduce risk of injury to hearing and vision in a combat setting

CAPT Michael E. Hoffer, M.D.

- CAPT Michael Hoffer directs the Spatial Orientation Center at Naval Medical Center San Diego that provides clinical care, performs basic research, translates research findings, and educates medical providers on balance and hearing issues.
- CAPT Hoffer graduated from Stanford University and attended the University of California San Diego, School of Medicine.
- After his internship in General Surgery and residency in Otolaryngology: Head and Neck Surgery, both at the University of Pennsylvania, CAPT Hoffer completed a fellowship in Otology/Neurotology at the Ear Research Institute in Sarasota, Florida.
- CAPT Hoffer is double boarded in Otolaryngology and Neurotology.
- His research work has focused on the basic science, diagnosis, and treatment of vestibular disorders.
- CAPT Hoffer and his colleagues at the Spatial Orientation Center have authored over 50 peer reviewed articles focusing on neurosensory issues after trauma.

Carey D. Balaban, Ph.D.

- Dr. Balaban is the Vice Provost for Faculty Affairs and Professor, Departments of Otolaryngology, Neurobiology, Communication Science & Disorders, and Bioengineering at the University of Pittsburgh.
- He holds a Ph.D. in anatomy from the University of Chicago and a Ph.D. in physiology from the University of Tokyo, Japan.
- He is a Potomac Institute for Policy Studies Academic Fellow at the Center for Neurotechnology Studies; Health Policy and Preparedness.
- Dr. Balaban is active in the National Institutes of Health research activities as the principal investigator, co-principal investigator and subproject director.
- He has authored well over 100 journal articles, monographs and book chapters.
- Dr. Balaban is the recipient of the NIH National Research Service Award and the NIH Research Career Development Award.

Hearing and Balance Disorders in Operational Environments

CAPT Michael E. Hoffer, M.D.
Carey Balaban, Ph.D.

Disclosure

- These views expressed in this presentation are those of the presenters and do not reflect the official policy of any military Service, the Department of Defense, or the U.S. Government.
- The presenters have no relevant financial relationships to disclose.
- The presenters do not intend to discuss the off-label, investigative, or other unapproved uses of commercial products or devices.

Threats to hearing and balance

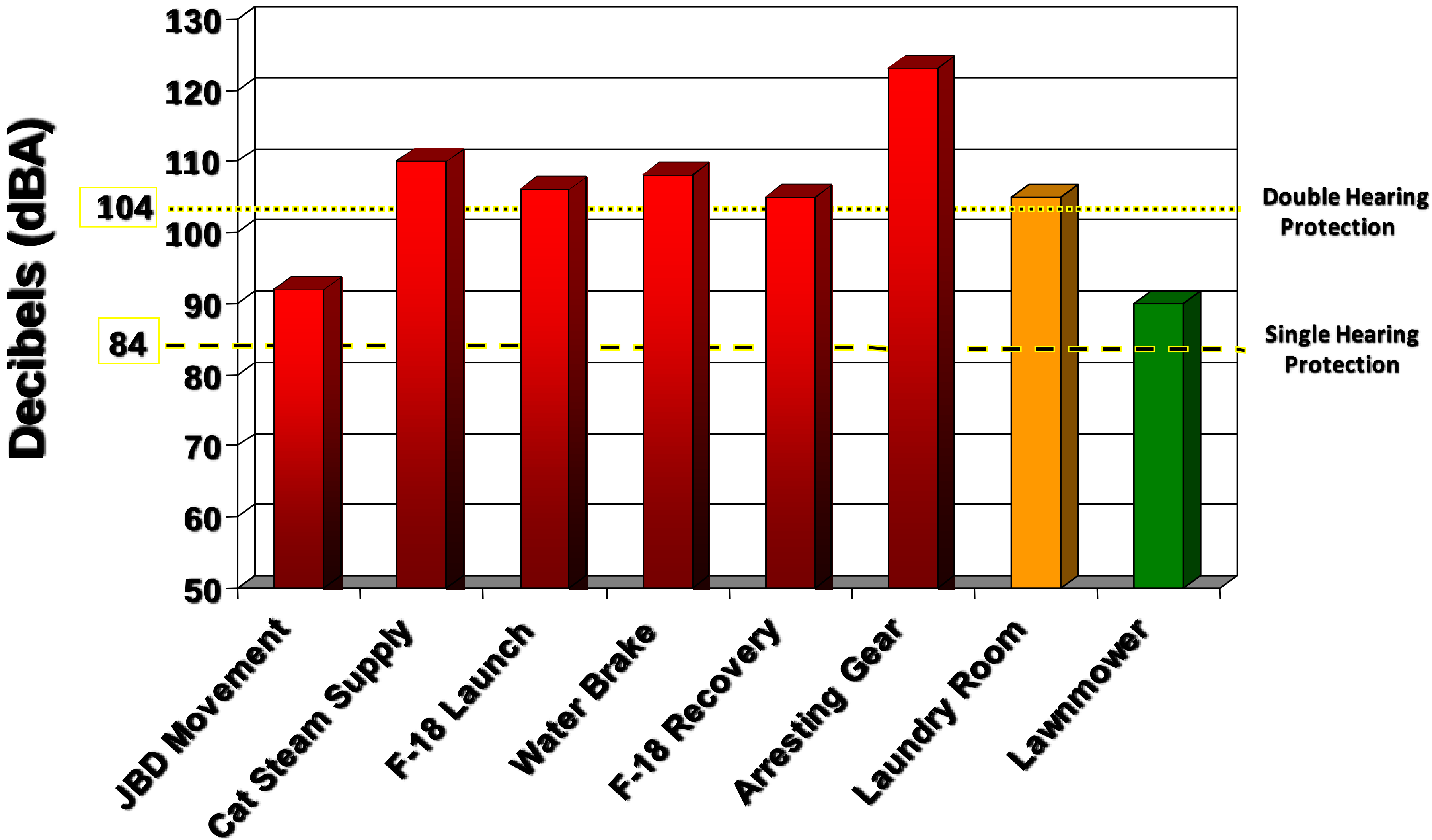
- Penetrating Head Injuries and Open Head Injuries
- Noise
  - Continuous
  - Impulse
- Traumatic Brain Injury
  - Blunt
  - Blast

Environmental and Operational Noise

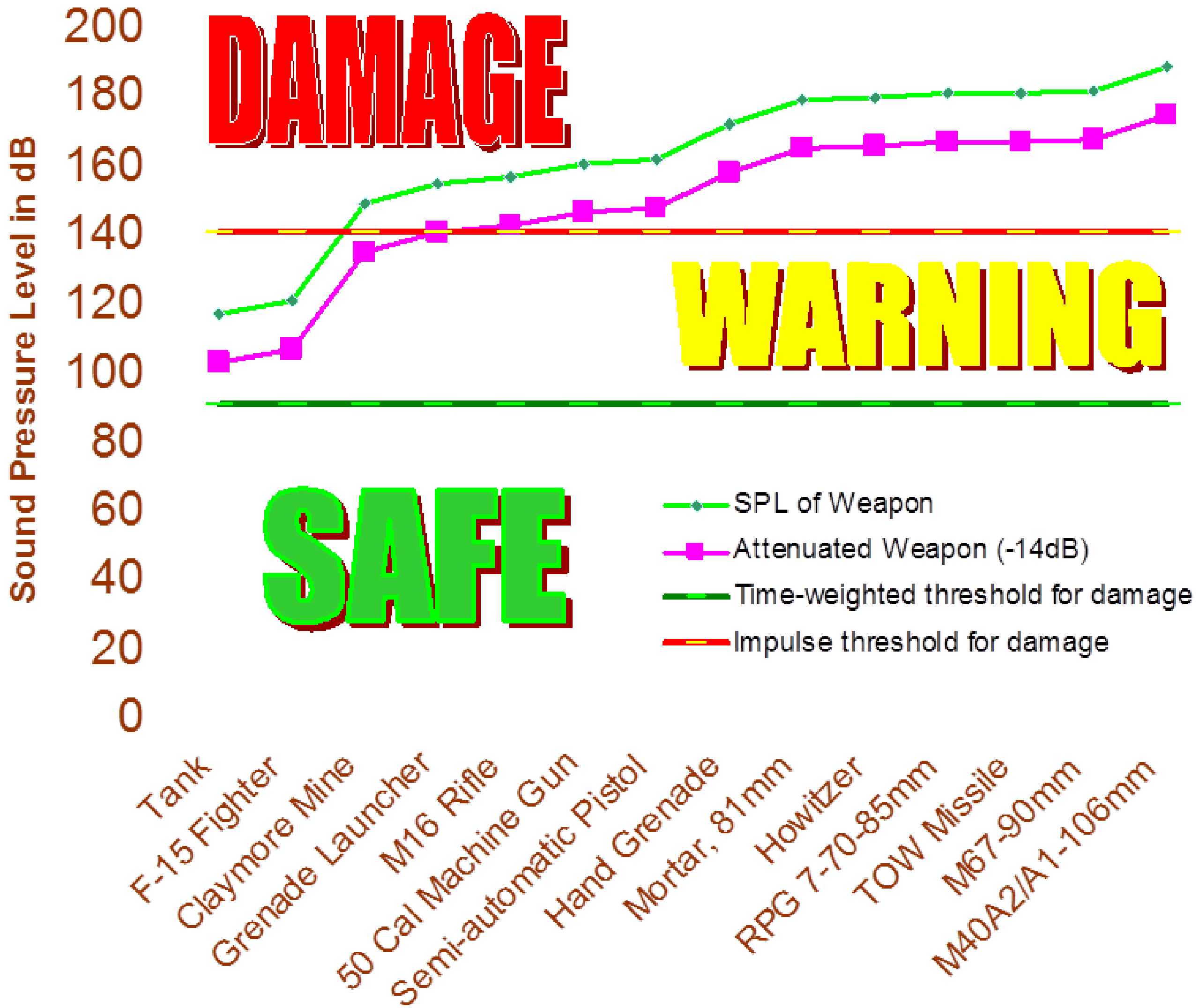
- Ubiquitous Hazard that mainly produces hearing loss and tinnitus
  - Two most common VA disabilities
  - Disability claims well over $1 Billion per year
- Effects of hearing loss
  - Impaired mission accomplishment
  - Loss of Key personnel
  - Affected individual, family, and friends suffer

(Hearing Loss 101: Stats and Figures)

CVN Airborne Noise (Gallery Deck)

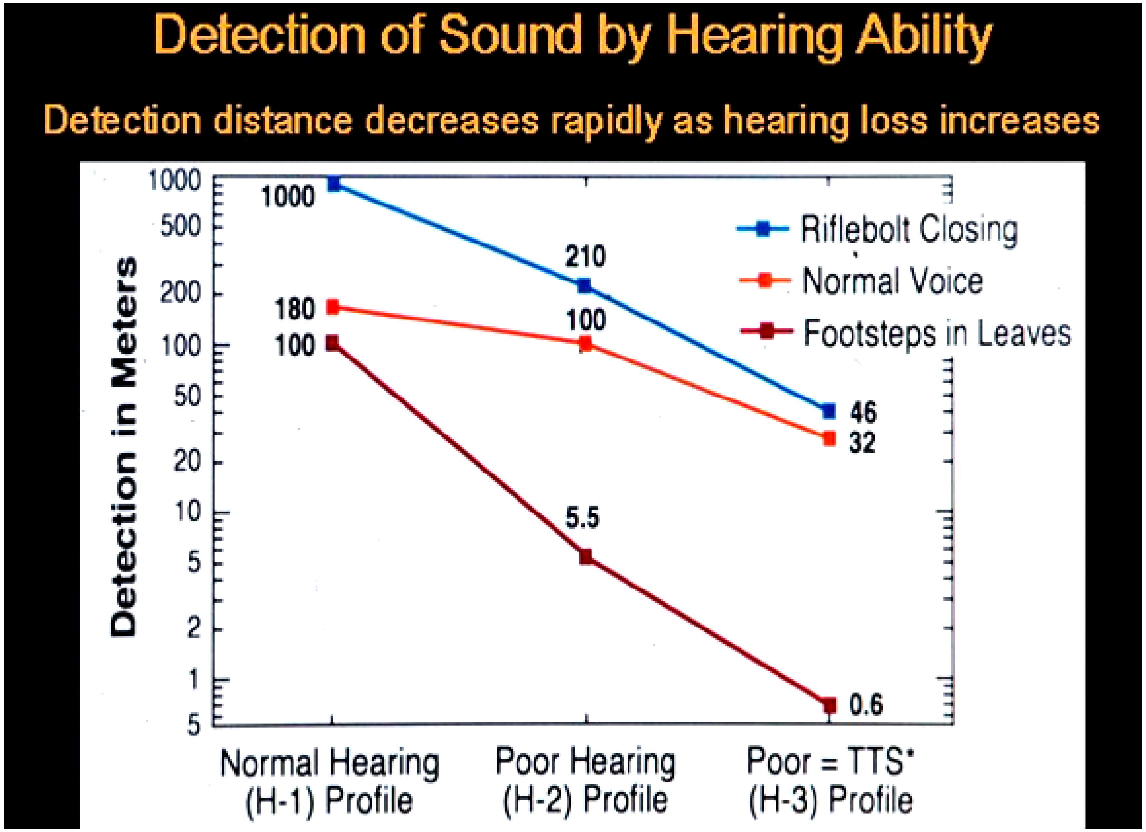


Ref: CVN 72, CVN 74 Trial data



Sound Pressure Level in dB
200
180
160
140
120
100
80
60
40
20
0
DAMAGE
WARNING
SAFE
SPL of Weapon
Attenuated Weapon (-14dB)
Time-weighted threshold for damage
Impulse threshold for damage
Tank
F-15 Fighter
Claymore Mine
Grenade Launcher
M16 Rifle
50 Cal Machine Gun
Semi-automatic Pistol
Hand Grenade
Mortar, 81mm
Howitzer
RPG 7-70-85mm
TOW Missile
M67-90mm
M40A2/A1-106mm

Military Noise Impact



(Price, Kalb & Garinther, 1989)

- Threat detection critical for survival
- Members in small groups: communication vital
- Tank crews at 25% increased risk if one member with hearing loss

Hearing loss

- Prevention
  - Close screening/Hearing Conservation Programs
  - Human Factor Engineering
  - Mechanical Protection
- Newer approaches
  - Newer audiometric techniques
  - Pharmaceutical Protection
  - Determining at risk population

Hearing Loss

- Lessons for the provider
  - Hearing loss is a significant disability not just the price of doing business
  - Hearing loss must be evaluated by audiologists and otolaryngologists
  - Hearing loss is an operational risk and a safety risk
  - Solutions include
    - Assistive listening devices
    - Hearing aids
    - Surgeries
    - Implanted devices (numerous on the market)

Mild Traumatic Brain Injury

- Mild Traumatic Brain Injury (mTBI) has been called the signature injury of modern warfare
  - Body armor allows individuals to survive more injuries
  - Personal Protective Gear can protect against other injuries but are not that effective against blunt and especially blast mTBI
  - Our enemies weapon of choice are often improvised explosive devices (IED’s)

mTBI – Frequency

- Blast accounts for 80-90% of all battlefield injuries- vast majority of these individuals suffer mTBI
- Rand Corporation suggested mTBI frequency may be as high as 19% of all those who deploy
- Accounts for approximately 1 Million ER visits a year in the civilian population
- Over 5 million Americans suffer from the effects of mTBI
- Increasingly common VA disability

(Hoffer, M.E., Balaban, Slade, Tsao, & Hoffer, B. , 2013)

(Hearing Loss 101: Stats and Figures)

VA/DoD TBI definition

Definition of Traumatic Brain Injury

- A traumatically induced structural injury and/or physiological disruption of brain function as a result of an external force* that is 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 (LOC)
- Any loss of memory for events immediately before or after the injury (post-traumatic amnesia [PTA])
- Any alteration in mental state at the time of the injury (confusion, disorientation, slowed thinking, etc.) (Alteration of consciousness/mental state [AOC])
- Neurological deficits (weakness, loss of balance, change in vision, praxis, paresis/plegia, sensory loss, aphasia, etc.) that may or may not be transient
- Intracranial lesion (U.S. Department of Veterans Affairs & U.S. Department of Defense, 2009, p. 16)

*External forces may include any of the following events: the head being struck by an object, the head striking an object, the brain undergoing an acceleration/deceleration movement without direct external trauma to the head, a foreign body penetrating the brain, forces generated from events such as a blast or explosion, or other forces yet to be defined

VA/DoD Definition of mTBI

- A physiological disruption of brain function as a result of a traumatic event as manifested by at least one of the following:
  - alteration of mental state
  - loss of consciousness (LOC)
  - loss of memory or focal neurological deficit, that may or may not be transient; but where the severity of the injury does not exceed the following:
    - post-traumatic amnesia (PTA) for greater than 24 hours
    - after the first 30 minutes Glasgow Coma Score (GCS) 13 - 15,
    - loss of consciousness is less than 30 minutes (VA &DoD, 2009, p. 17)

mTBI-causes

Blunt Head Injury

- Coup-countercoup
- Head against inside of helmet
- Frequently occur in non-operational setting

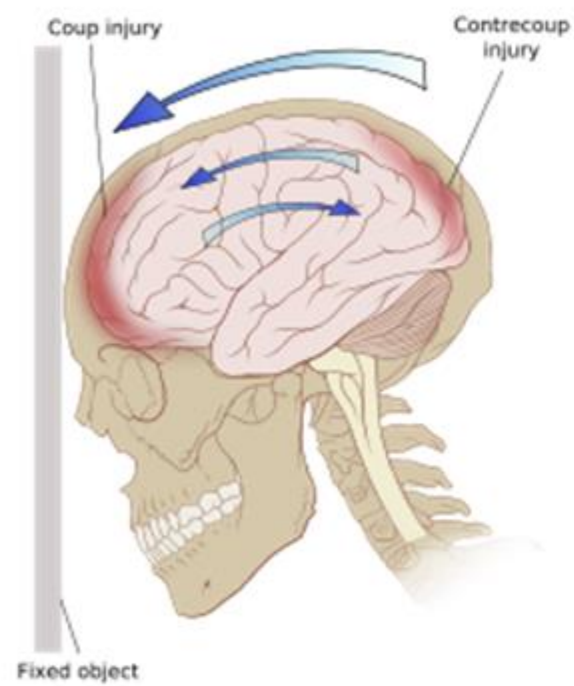
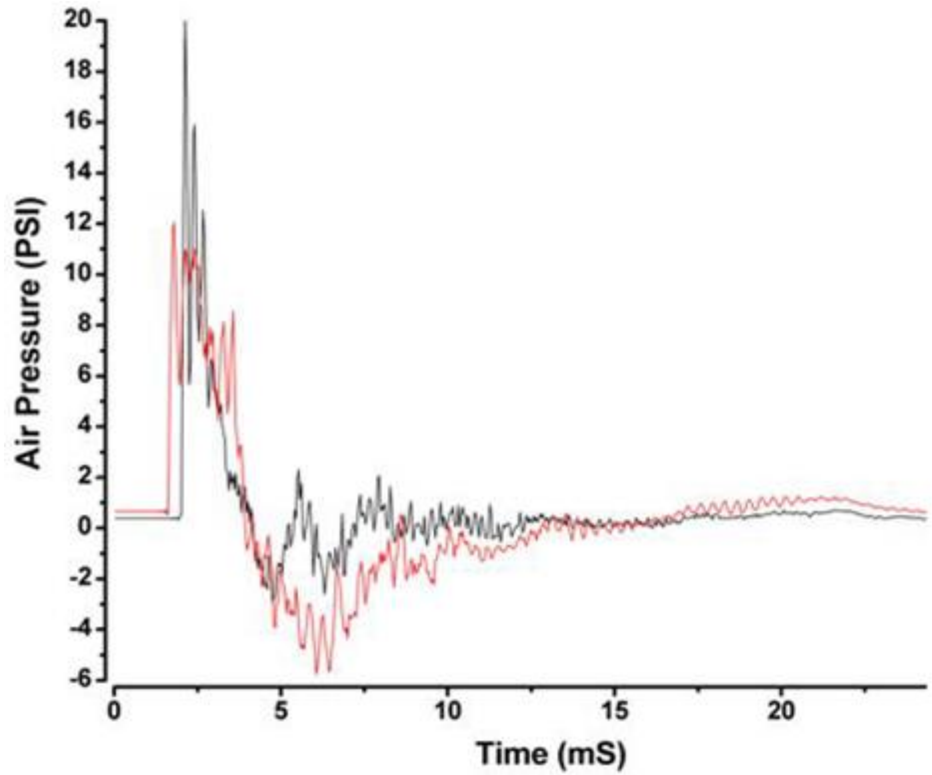


Image Courtesy: Patrick j. Lynch

mTBI - Causes

Blast

- Feel a pressure wave which is characterized by a positive and then negative pressure
- Hollow organs thought to be the most sensitive
- Brain may actually be even more sensitive



Blast injury categories

- **Primary blast injury:** shock wave propagation through tissue
  - -***Acute acoustic trauma*** - via conductive path
  - -***Blast injury*** – Blast physics and biomaterials
- **Secondary blast injury:** shrapnel or fragments
- **Tertiary blast injury:** impact with objects in environment
- **Quaternary blast injury:** heat, electro-magnetic pulses or detonation toxins

Basic question

- Determine the difference in presenting symptoms of blast induced mild traumatic brain injury as a function of time
- Examine the diagnostic and management implications of these differences

*The following graphs and charts are from Blast exposure: Vestibular consequences and associated Characteristics Otololgy Neurotology, 2010 Feb; 31(2): 232-36.

(Hoffer, Balaban, Gottshall, Balouch, Maddox & Penta, 2010)

Materials and Methods

- Three groups of mild traumatic brain injury (mTBI) patients divided by time of presentation
  - Acute – seen in under 72 hours from blast in Iraq
  - Sub-acute – seen 4-30 days after blast at NMCSD
  - Chronic – seen 30-360 days after blast at NMCSD
- All had mTBI secondary to blast as defined by the DoD definition of TBI (April 2009)

Photo Courtesy: Michael Hoffer

Materials and Methods

Acute patients

- **History and physical**
- Dynamic Gait Index (DGI)
- Hearing test (Otogram)

Sub-acute and Chronic patients

- **History and physical**
- Rotational chair (details in paper)
- Sensory Organization test (Posturography)
- DGI
- Hearing test
- Standardized Instruments (details in paper)

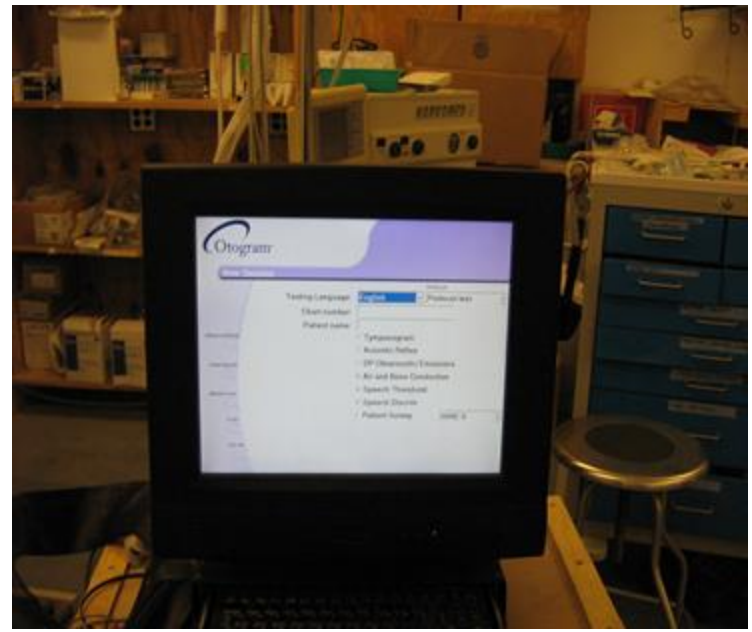


Photo Courtesy: Michael Hoffer

Results

- Groups (Median age 22)
  - Acute – 81 Individuals
  - Sub-Acute – 25 Individuals
  - Chronic – 42 Individuals

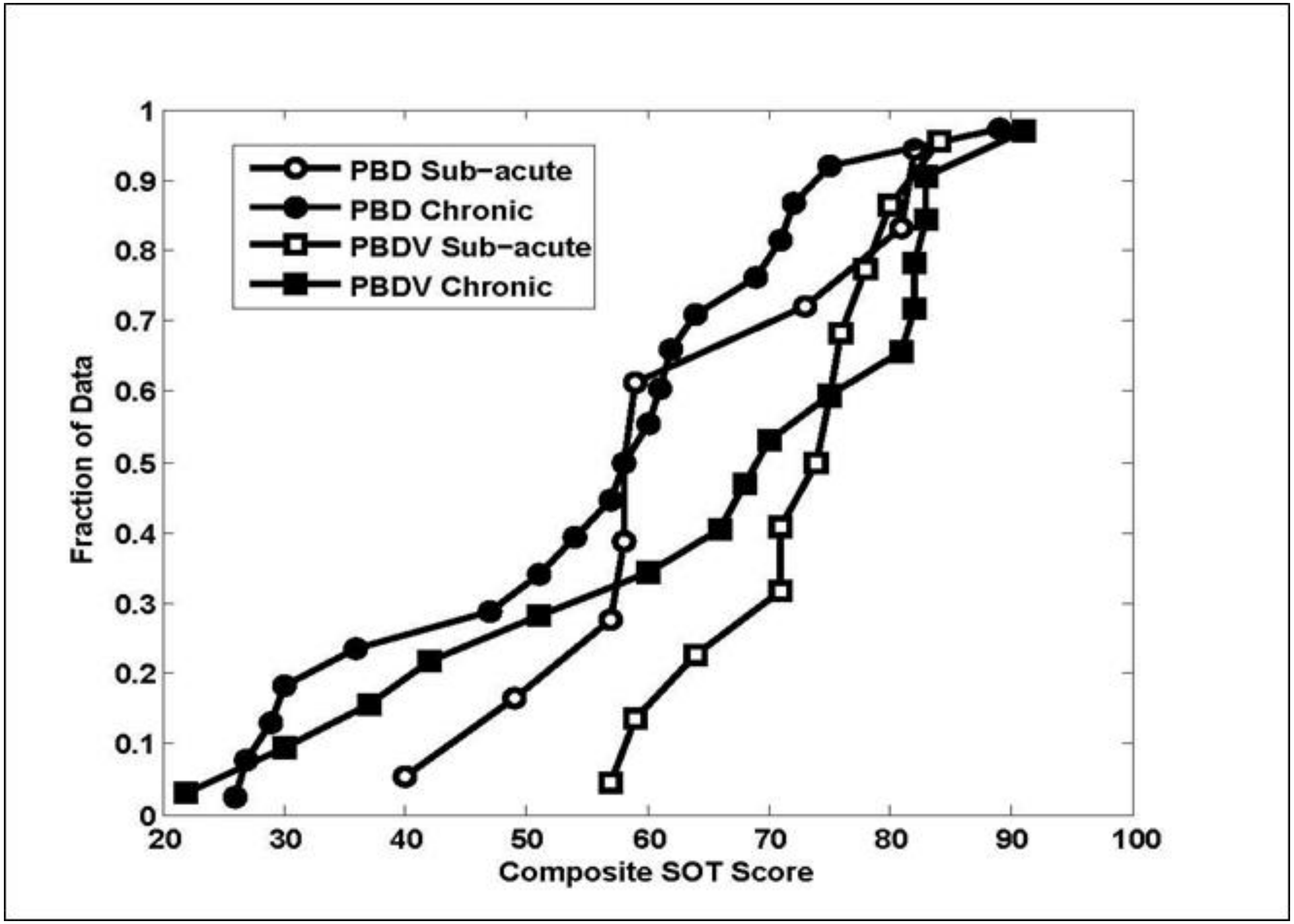
Mild Traumatic Brain Injury after Blast - Symptoms Distribution

Group	Dizziness	Vertigo	Hearing Loss	Headache	PTSD
Acute	98%*	4%*	33%*	72%	2%*
Sub-acute	76%	47%	43%	76%	20%
Chronic	84%	36%	49%	82%	44%

Patterns of Balance Disorders in Sub-acute and Chronic Blast Exposure

Entity	History	Physical Exam	Vestibular Tests
Positional Vertigo	Positional Vertigo	Nystagmus on Dix-Hallpike test or modified Dix-Hallpike test	No other abnormalities
Post-Blast Exercise Induced Dizziness	Dizziness during and right after exercise	Abnormalities in challenged gait test during exertion	No other abnormalities
Post-Blast Dizziness (PBD)	▪ Constant feeling of unsteadiness when standing and waling worse with challenging environments ▪ Constant Headache	▪ Abnormalities in challenged gait ▪ Abnormalities in tandem Romberg ▪ Abnormalities with quick head motion	▪ +/- Abnormal posturography ▪ Abnormal target acquisition, dynamic visual acuity, and gaze stabilization ▪ +/- VOR gain, phase, or symmetry abnormalities
Post-Blast Dizziness with Vertigo (PBDV)	▪ Constant feeling of unsteadiness when standing and waling worse with challenging environments ▪ Constant Headache ▪ Episodic Vertigo	▪ Abnormalities in challenged gait ▪ Abnormalities in tandem Romberg ▪ Abnormalities with quick head motion	▪ +/- Abnormal posturography ▪ Abnormal target acquisition, dynamic visual acuity, and gaze stabilization ▪ +/- VOR gain, phase, or symmetry abnormalities

Results of Posturography Testing in Sub-acute and Chronic Patients



Graph Courtesy: Hoffer ME, Balaban C, Gottshall KR, Balough BJ, Maddox MR, Penta JR. Blast exposure: Vestibular consequences and associated Characteristics. Otol Neurotol, 2010 Feb; 31(2): 232-36.

Results of Rotary Chair Testing in Sub-acute and Chronic Patients

Diagnosis	Group	No Abnormal Tests	1 Abnormal Test	2 Abnormal Tests
PBD	Sub-acute	5	0	0
	Chronic	2	6	2
PBDV	Sub-acute	3	3	0
	Chronic	2	3	3

Summary of results

- Dizziness and headaches are the dominant symptom of mTBI seen after blast
- Balance disorders can be classified in sub-acute and chronic blast exposure patients
- Objective vestibular tests tend to worsen over time

mTBI

- Prevention
  - Personal Protective Gear
  - Human Factor Engineering
  - Pharmaceutical Measures
- Important issues
  - Go/No-go Criteria for operational missions
  - Level of care determination
  - Multiple TBI over short-term or even long-term

mTBI Considerations

- Care issues
  - mTBI is a real injury pattern
  - mTBI sequelae are a safety concern
  - Care must be tailored to individual symptom pattern
  - Long term sequelae are possible and early intervention might be important
- Co-morbidities
  - PTSD is seen in many mTBI individuals
  - Individuals can have other medical issues and may ignore mTBI issues

Noise-Induced Hearing Loss

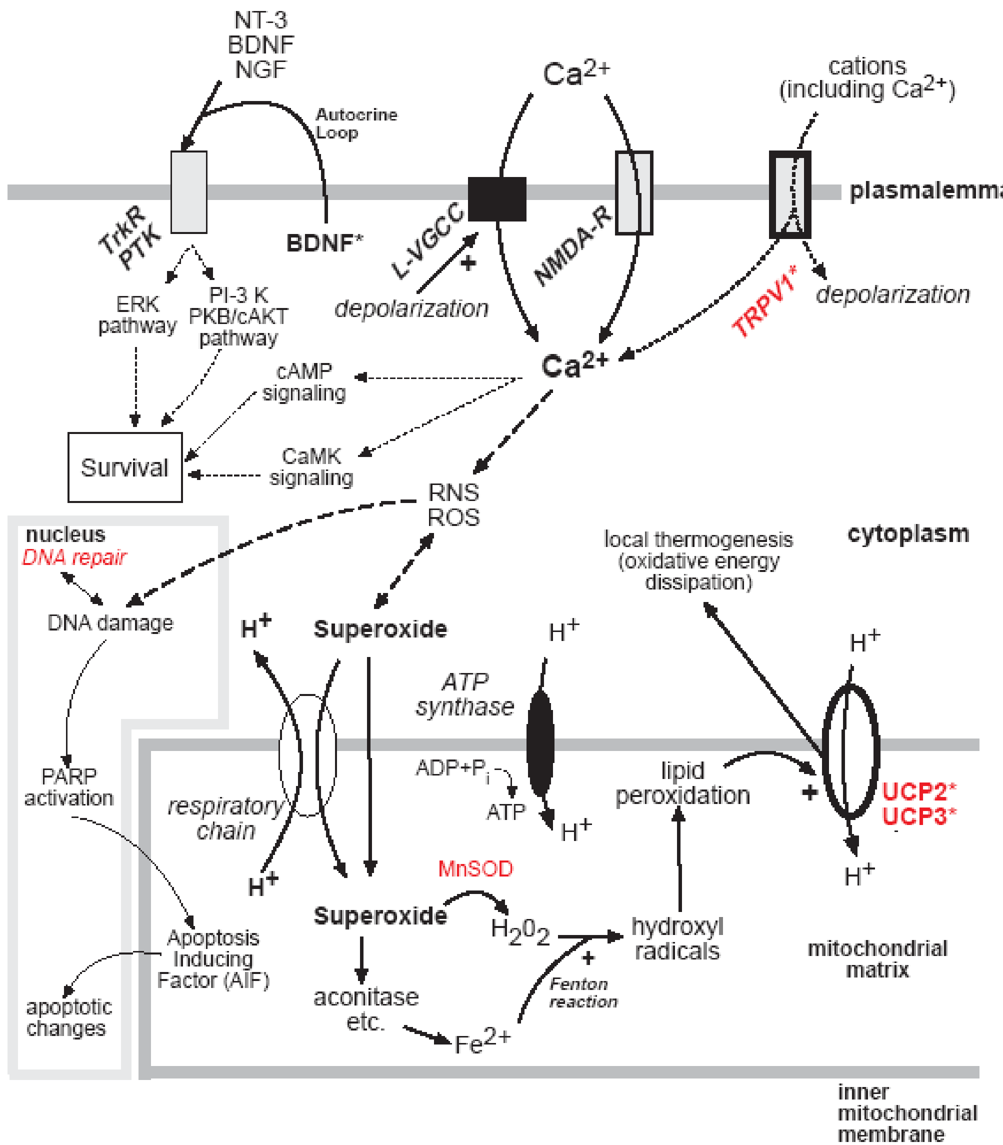
- Mechanical injury to inner ear hair cells and vasculature
- Oxidative stress
- Endolymph-perilymph homeostasis
- Interaction with ototoxins

Noise-Induced Hearing Loss

- Hair cell, ganglion cell and vascular response repertoires for ototoxins, mechanical trauma and oxidative stress exposure may contribute to tinnitus, hyperacusis and vertigo
- Trade-off between neuroprotective plus adaptive effects and symptoms

Role of Reactive Oxygen Species (ROS) in NIHL and Ototoxicity

- ROS generation implicated in aminoglycoside-induced hair cell damage
- Protective effects of antioxidant treatment
- Focuses attention on cellular responses to oxidative stress
- Aminoglycosides provide model for identifying cellular responses to ROS challenges


NT-3
BDNF
NGF
Autocrine Loop
Ca2+
cations (including Ca2+)
plasmalemma
TrkR PTK
BDNF*
L-VGCC
NMDA-R
+
depolarization
TRPV1*
depolarization
ERK pathway
PI-3 K PKB/cAKT pathway
Ca2+
cAMP signaling
CaMK signaling
Survival
RNS ROS
nucleus
DNA repair
DNA damage
cytoplasm
local thermogenesis (oxidative energy dissipation)
H+
Superoxide
H+
ATP synthase
H+
ADP+Pi
ATP
H+
lipid peroxidation
+
UCP2*
UCP3*
H+
PARP activation
respiratory chain
H+
MnSOD
Superoxide
H202
hydroxyl radicals
+
Fenton reaction
mitochondrial matrix
Apoptosis Inducing Factor (AIF)
apoptotic changes
aconitase etc.
Fe2+
inner mitochondrial membrane

Blast Traumatic Brain Injury

- **Nosology as a clinical descriptive template**
  - **Symptoms**
  - **Signs**
  - **“Biomarkers”**
- **Etiologic nosology**
  - **Elucidate longitudinal disease processes**
  - **Plan interventions appropriate to patients’ clinical trajectories**

Blast Traumatic Brain Injury: Animal Models

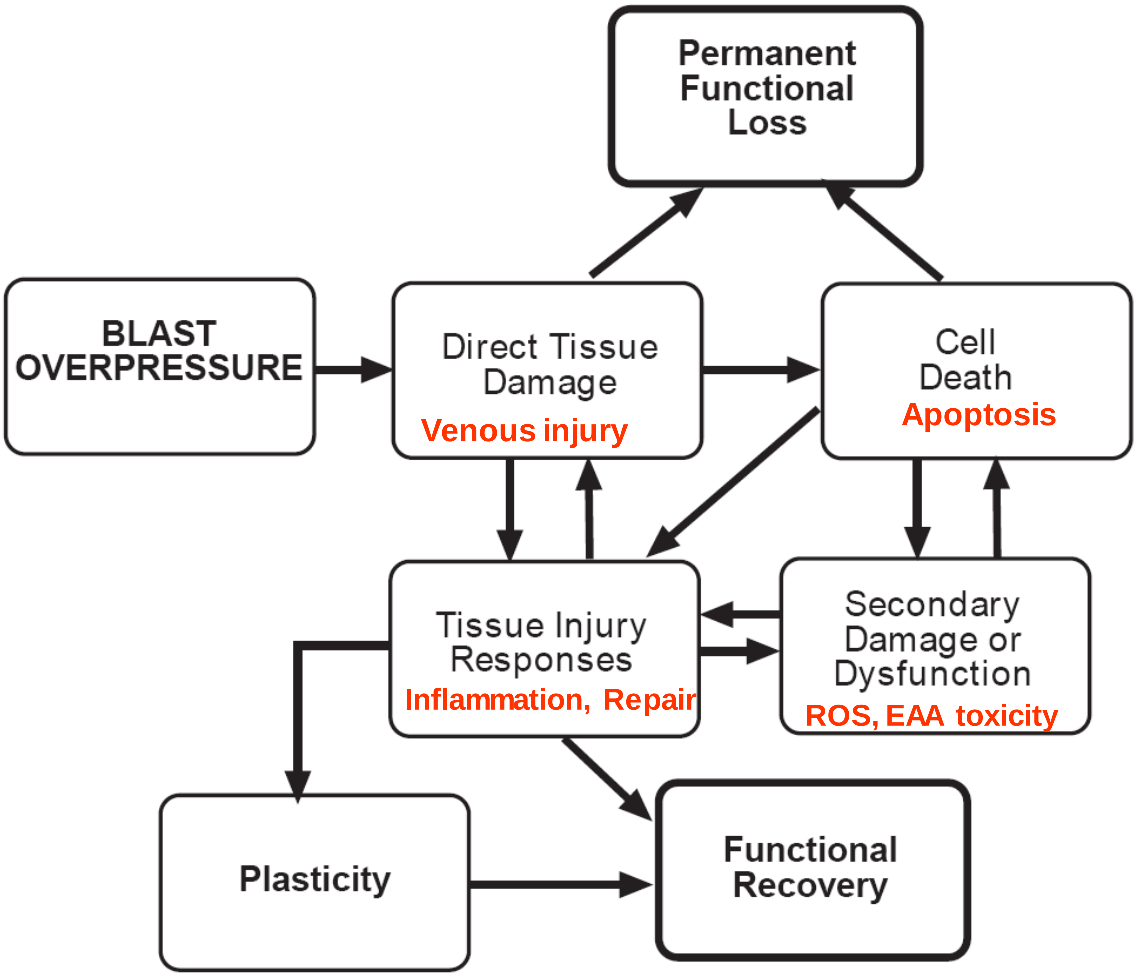
- Open field blast versus shock tube
- Range: 2.9 psi [20 kPa] - 17.5 psi [120.7 kPa] peak overpressure
  - Lower than overpressures (>17.5 psi/120 kPa) in many previous studies that have reported vascular and parenchymal injury in brains extracted from the skulls prior to histopathological assessment

(Cho et al. 2013, Kaur et al. 1997, Kaur et al. 1995, Kochanek et al. 2013, Kuehn et al. 2011, Kwon et al. 2011, Readnower et al. 2010, Saljo et al. 2000, Saljo et al. 2001, Saljo, Bao, et al. 2002, Saljo, Huang, and Hansson 2003, Saljo, Jingshan, et al. 2002, Garman et al. 2011)

Pathophysiology – Blast Injury

- Shock wave effect
  - Microvascular injury
  - Shear injury in vestibular end organ
  - Oxidative cellular stress
  - Significant Release of excitatory neurotransmitters
  - Direct stimulation of apoptotic pathways

Pathophysiology – Blast Injury



Experimental Neurology 232 (2011) 280–289

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journal homepage: www.elsevier.com/locate/yexnr

A mouse model of blast-induced mild traumatic brain injury

Vardit Rubovitch [a], Meital Ten-Bosch [a], Ofer Zohar [b], Catherine R. Harrison [c], Catherine Tempel-Brami [d], Elliot Stein [e], Barry J. Hoffer [e,*], Carey D. Balaban [f,g], Shaul Schreiber [h], Wen-Ta Chiu [i], Chaim G. Pick [a]

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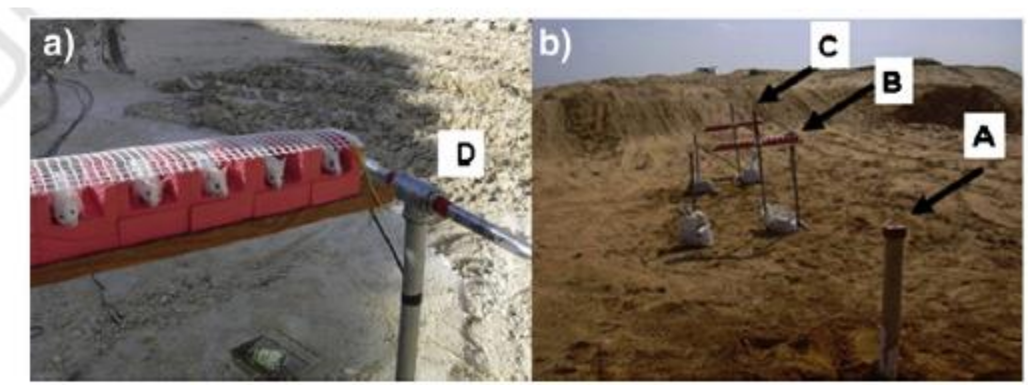


Mouse Open Field Blast Results

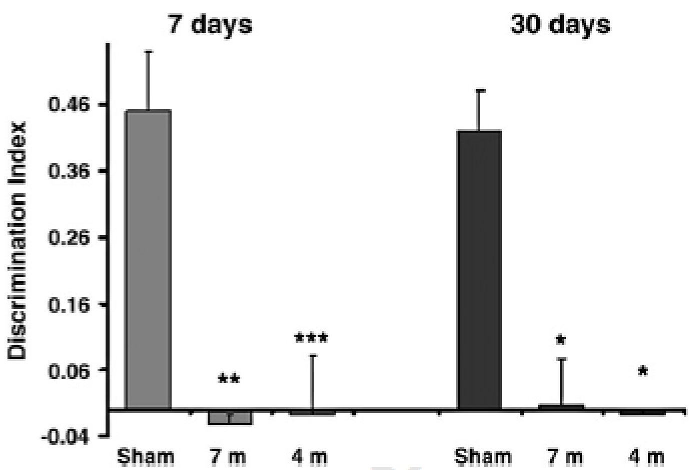


Fig. 3. The effect of blast on visual memory as assessed by the novel object recognition test. The preference for novel objects was significantly reduced in all the blast groups both at 7 days (−0.02 ± 0.01 for 7 m group, −0.008 ± 0.009 for 4 m group and 0.45 ± 0.09 for sham group) and 30 days (0.007 ± 0.07 for 7 m group, −0.008 ± 0.01 for 4 m group and 0.42 ± 0.06 for sham group). $^{*}p<0.05$, $^{**}p<0.01$ or $^{***}p<0.001$.

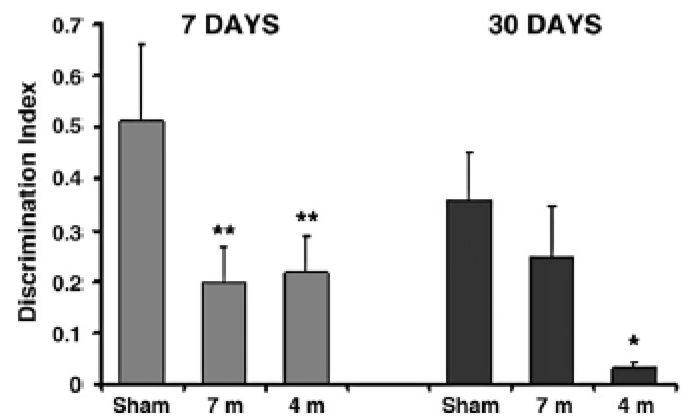


Fig. 4. The effect of blast on spatial memory as assessed by the Y-maze test. Preference for the new arm was significantly reduced in mice 7 days post blast in both groups (0.2 ± 0.07 for 7 m group and 0.22 ± 0.07 for 4 m in comparison with the sham group 0.51 ± 0.15). Similar impaired memory was found after 30 days for the 4 m group (0.033 ± 0.01 for the 4 m group and 0.361 ± 0.09 for the sham group). $^{*}p<0.05$, $^{**}p<0.01$ or $^{***}p<0.001$.

Mouse Open Field Blast Results

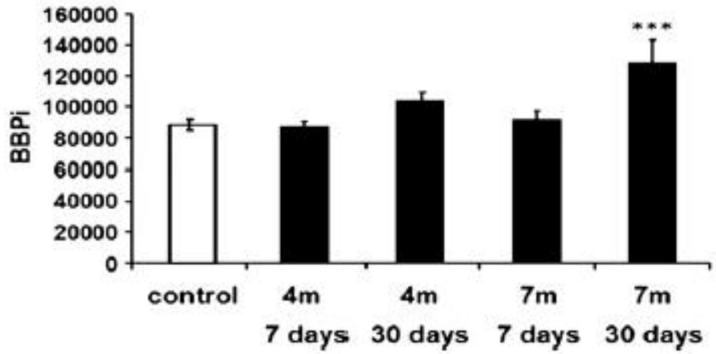
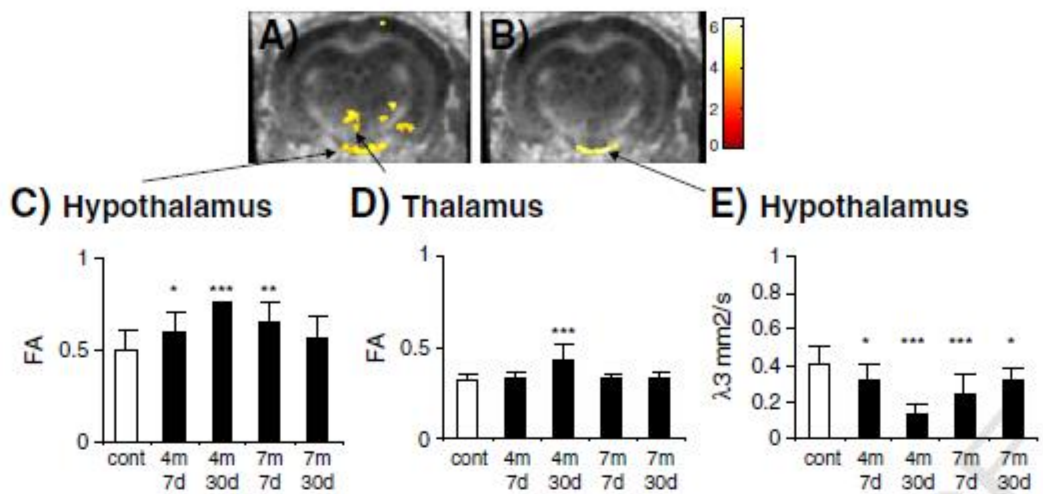
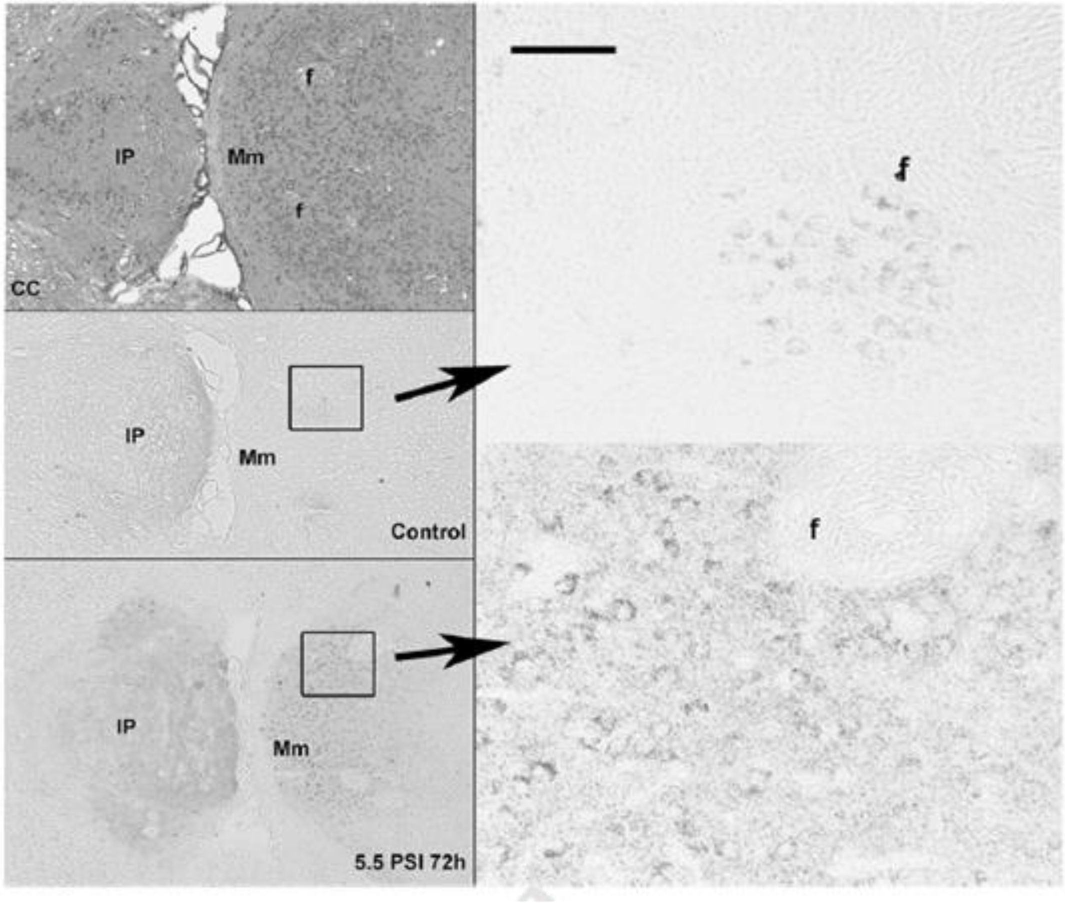


Fig. 6. Blast effect on BBB permeability index (BBPi). Permeability increased after blast-explosion at 30 days at 7 m compared with control group (F (4,40) = 5.9, $P<0.001$). *** indicates significant difference between the 7 m 30 days post-blast group compared to the control group, $p<0.001$.



Mouse Open Field Blast Results

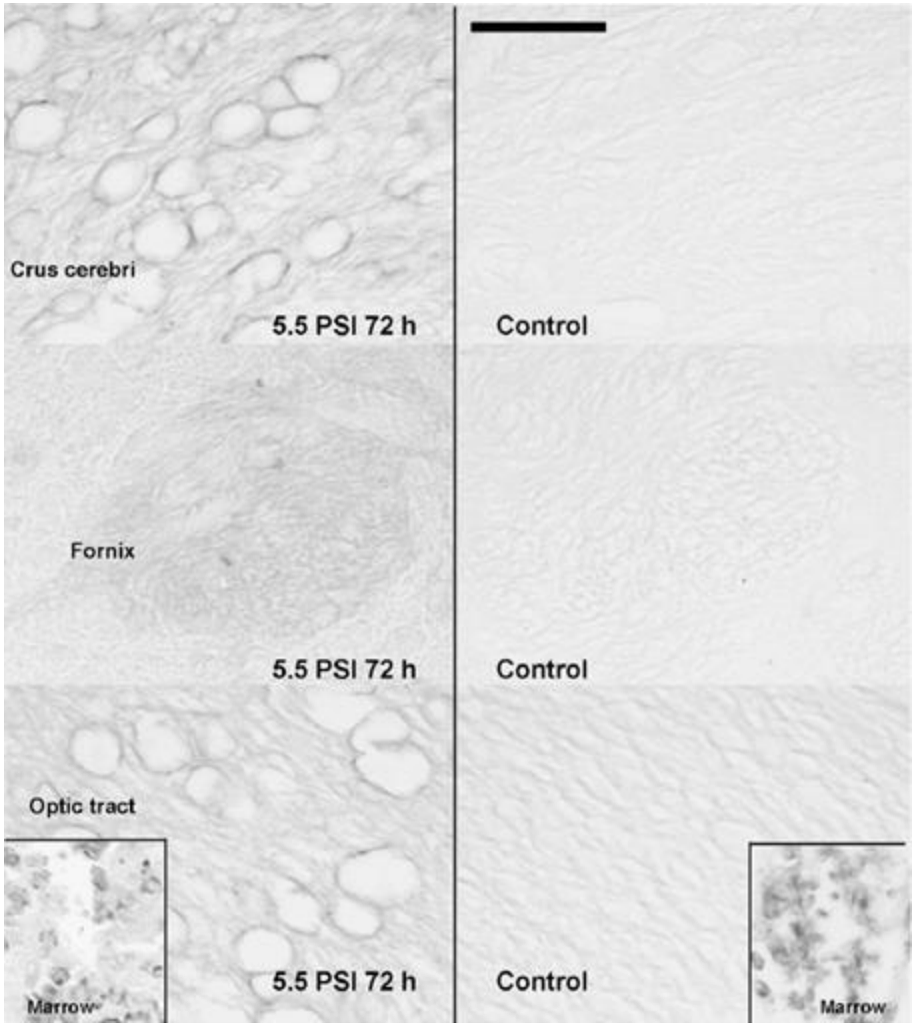
MnSOD2 upregulation in regions showing FA changes



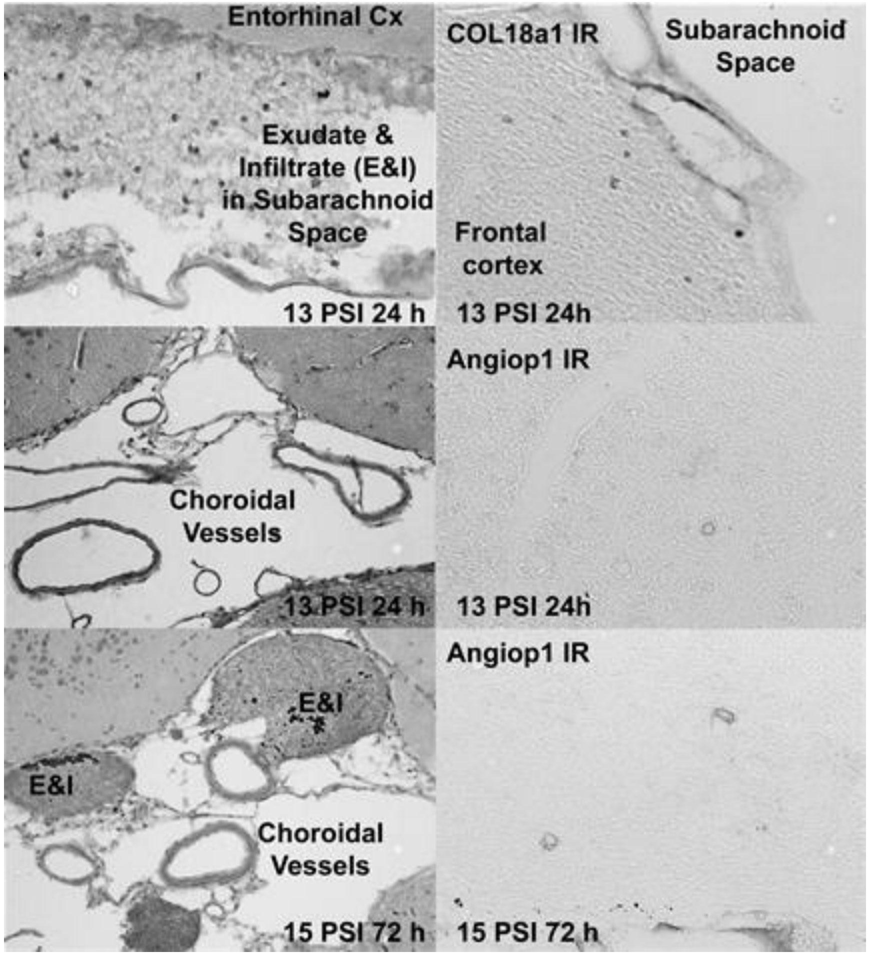
Mouse Open Field Blast Results

C-X-C motif chemokine receptor 3 (CXCR3) upregulation around blood vessels in fiber tracts

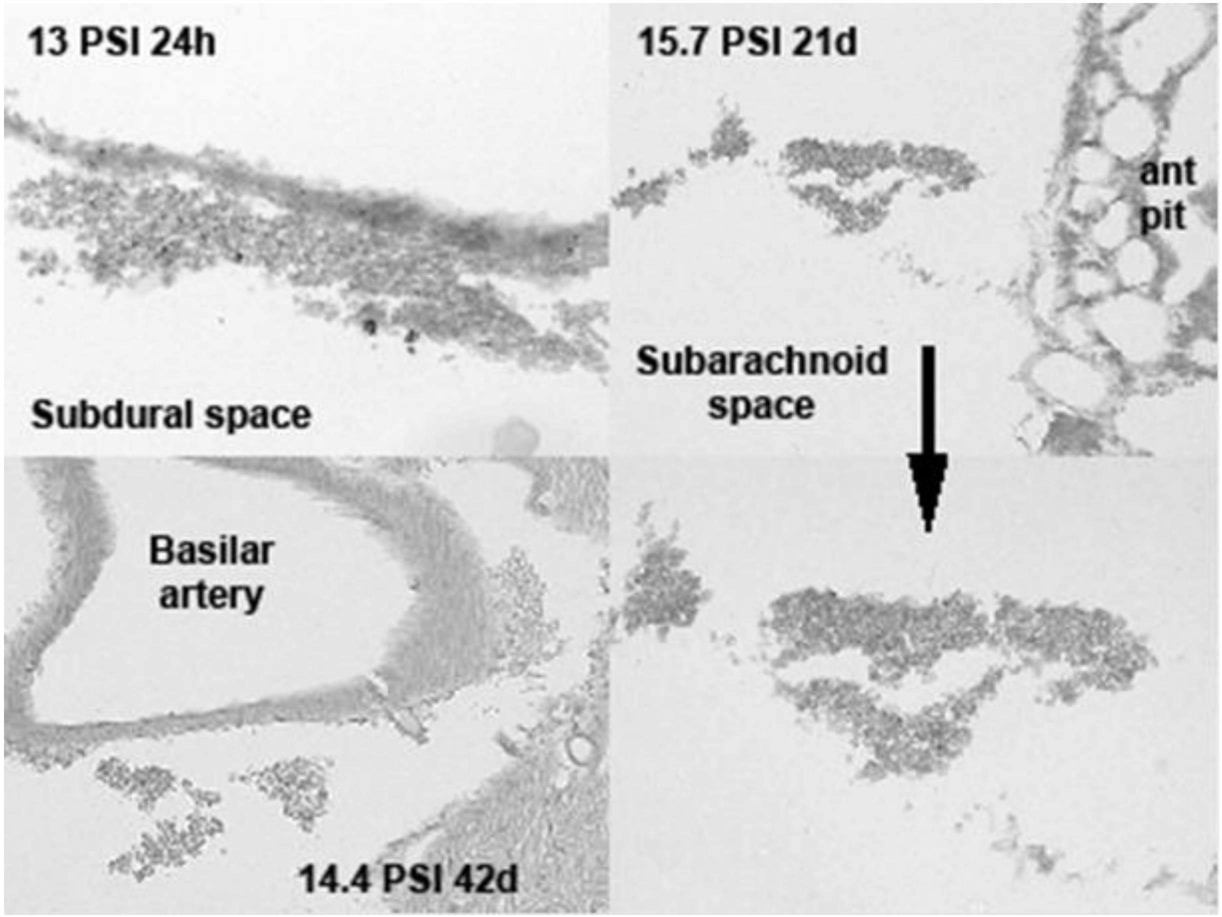
CXCR3 implicated in vascular remodeling and autoimmune disorders (e.g., thyroid disease and multiple sclerosis)



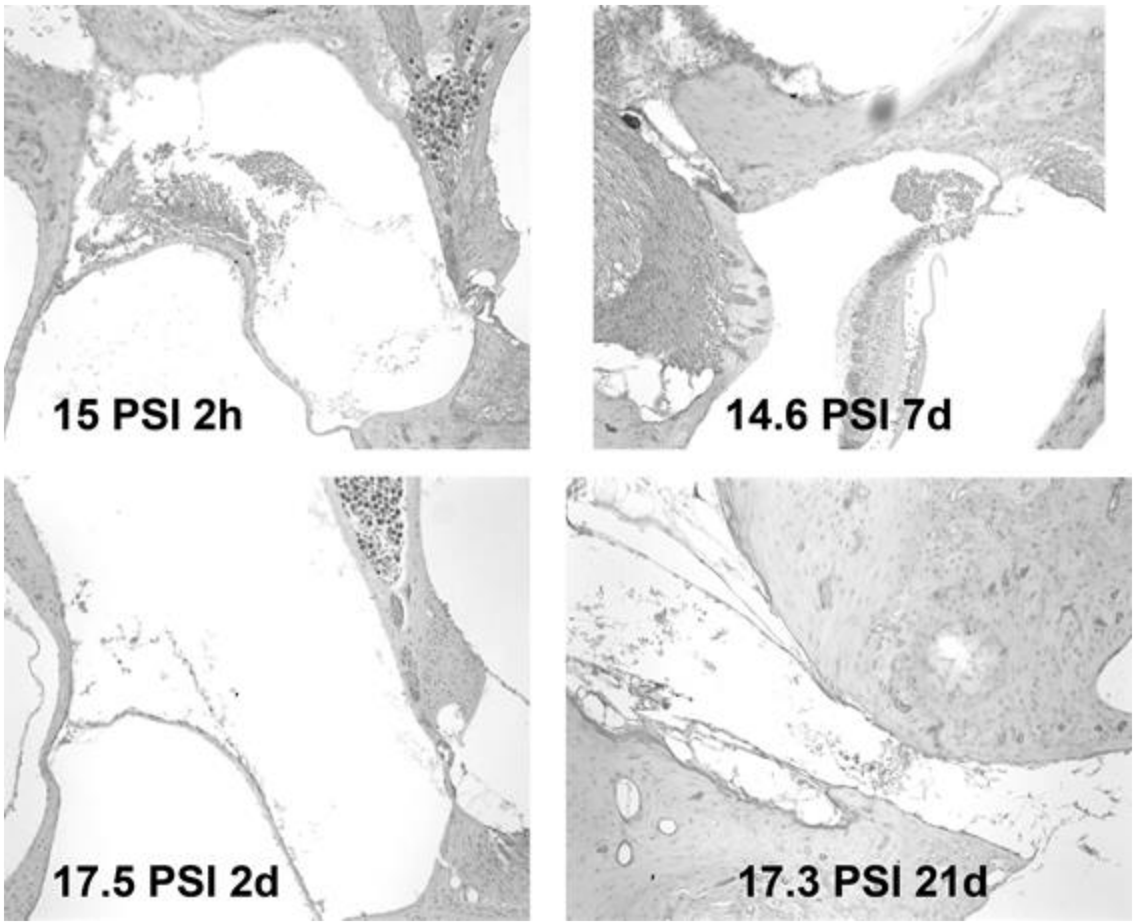
Histopathology: Rat Head



Histopathology: Rat Head



Histopathology: Rat Head
3/15 rats <8 psi; 9/17 at 13-17.5 psi

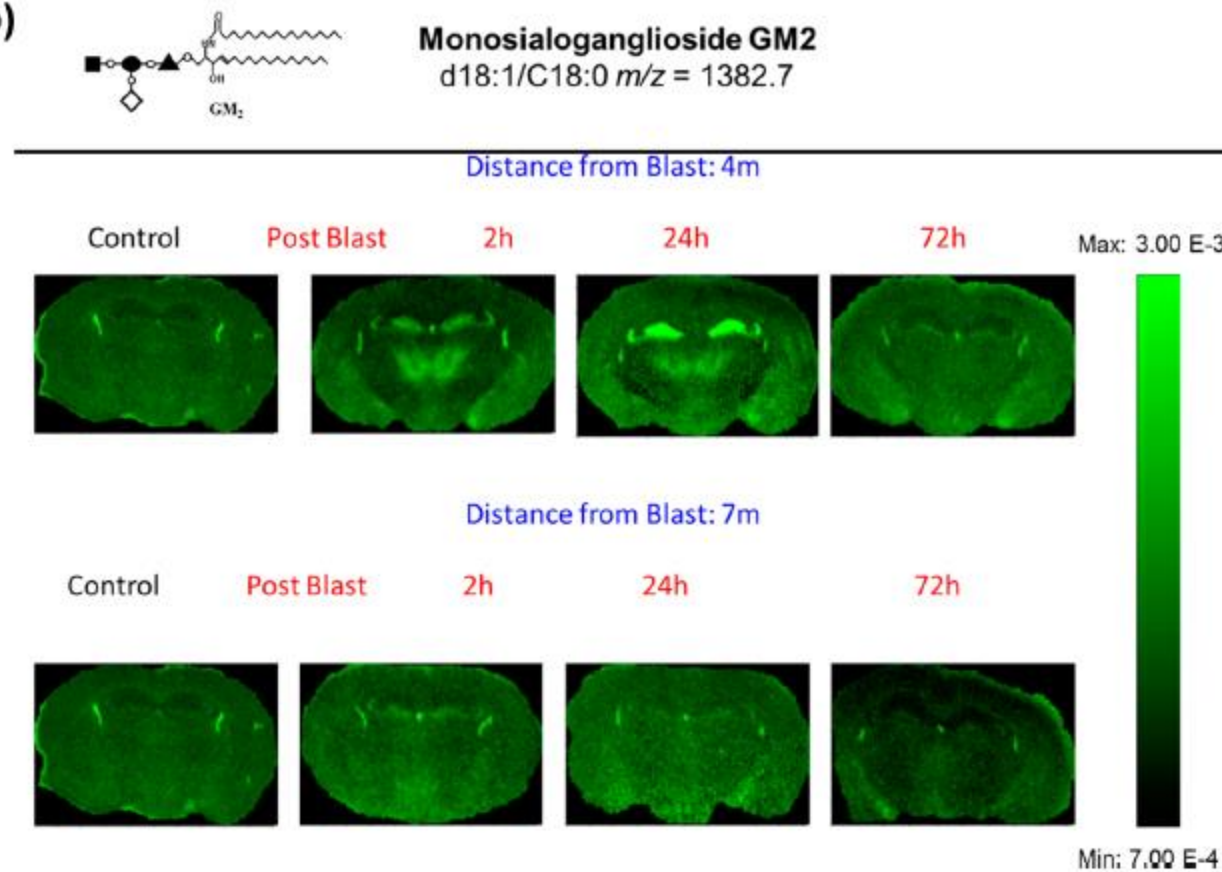


Gangliosides and ceramides change in a mouse model of blast induced traumatic brain injury

*The following slides, graphs and charts are from the article authored by:

Woods, A. S., Colsch, B., Jackson, S. N., Post, J., Baldwin, K., Roux, A., ... & Balaban, C. 2013). Gangliosides and ceramides change in a mouse model of blast induced traumatic brain injury. *ACS chemical neuroscience*, *4*(4), 594-600.

(b)

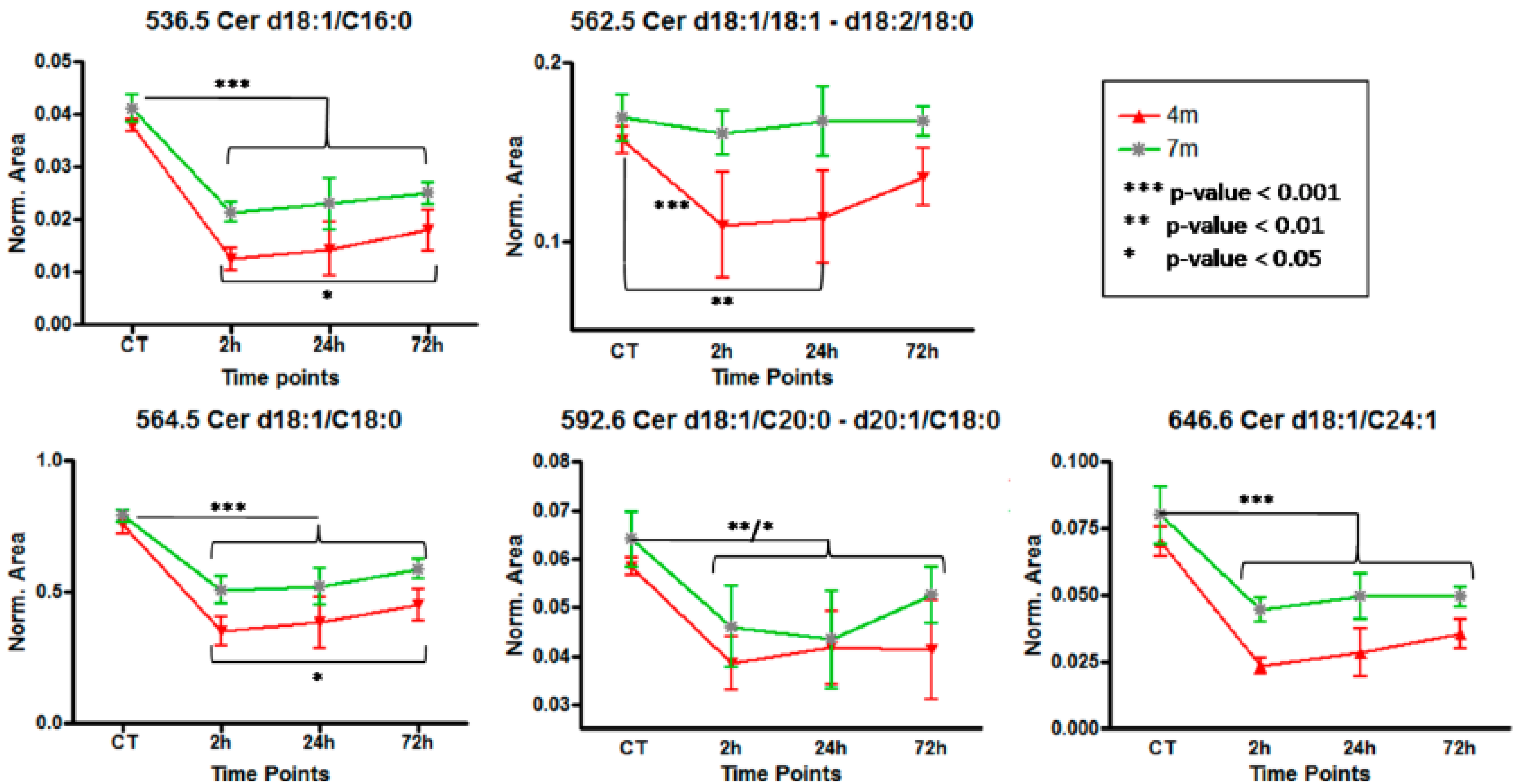


GM2
Monosialoganglioside GM2
d18:1/C18:0 m/z = 1382.7
Distance from Blast: 4m
Control
Post Blast
2h
24h
72h
Max: 3.00 E-3
Distance from Blast: 7m
Control
Post Blast
2h
24h
72h
Min: 7.00 E-4

a

	Two-Way ANOVA			Bonferroni posttests															
				Distance				Time											
				4m vs. 7m				CT vs. 2h		CT vs. 24h		CT vs. 72h		2h vs. 24h		2h vs.72h		24h vs.72h	
Ceramides	Time	Distance	Interaction	CT	2h	24h	72h	4m	7m	4m	7m	4m	7m	4m	7m	4m	7m	4m	7m
536.5 Cer d18:1/C16:0	***	**	ns	ns	ns	ns	ns	***	***	***	***	***	***	ns	ns	*	ns	ns	ns
562.5 Cer d18:1/18:1 - d18:2/18:0	*	**	ns	ns	ns	ns	ns	***	ns	**	ns	ns	ns	ns	ns	ns	ns	ns	ns
564.5 Cer d18:1/C18:0	***	**	ns	ns	ns	ns	ns	***	***	***	***	***	***	ns	ns	*	ns	ns	ns
592.6 Cer d18:1/C20:0 - d20:1/C18:0	***	*	ns	ns	ns	ns	ns	**	**	*	**	**	ns	ns	ns	ns	ns	ns	ns
646.6 Cer d18:1/C24:1	***	***	ns	ns	*	*	ns	***	***	***	***	***	***	ns	ns	ns	ns	ns	ns

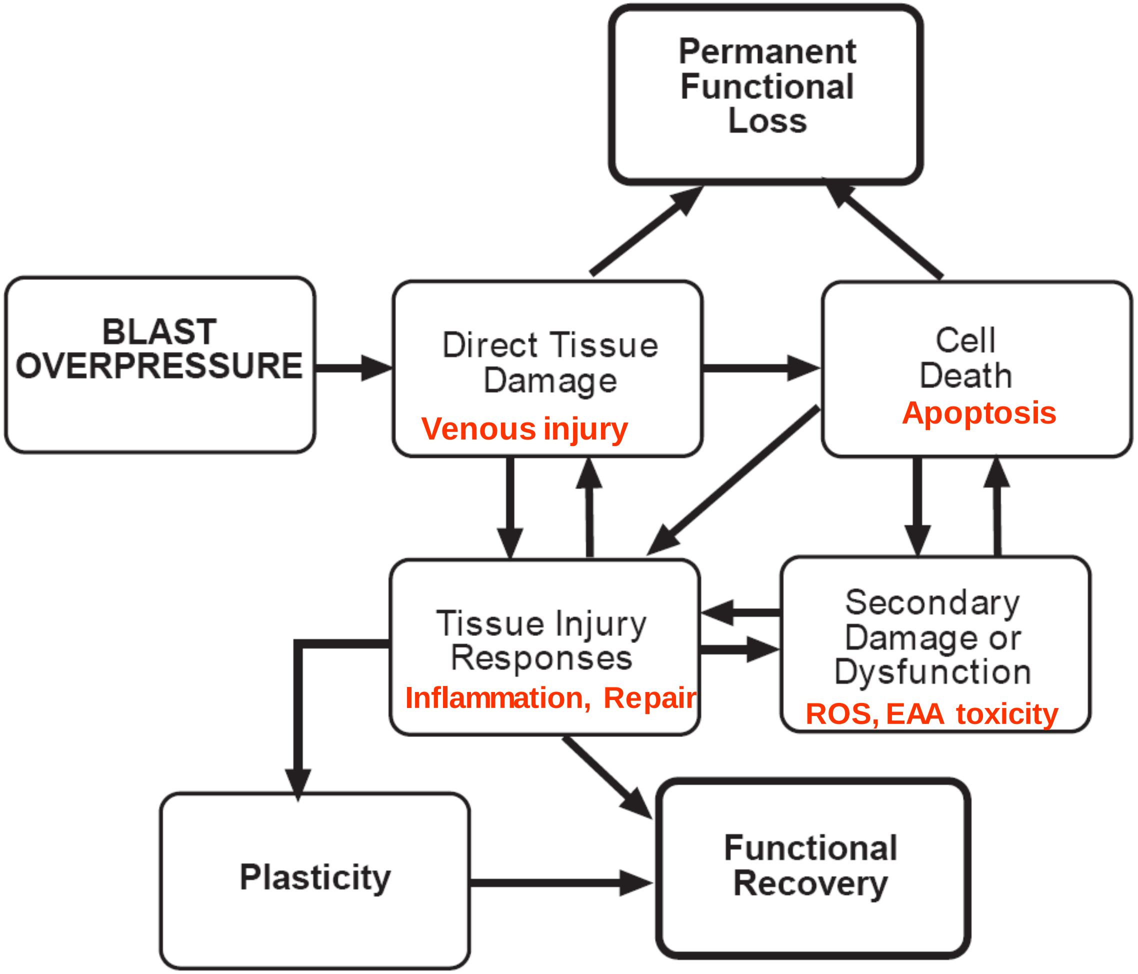
b



Blast Traumatic Brain Injury

- **Nosology as a clinical descriptive template**
  - **Symptoms**
  - **Signs**
  - **“Biomarkers”**
- **Etiologic nosology**
  - **Elucidate longitudinal disease processes**
  - **Plan interventions appropriate to patients’ clinical trajectories**

Pathophysiology – Blast Injury



Robert A Mazzoli, M.D., FACS

- Dr. Mazzoli is the Director of Education, Training, Simulation, and Readiness at the DoD-VA Vision Center of Excellence.
- Dr. Mazzoli's Army career spans 34 years of active service in both the Signal and Medical Corps. Key roles include Consultant in Ophthalmology to The Surgeon General of the Army, and Chief and Chairman, Ophthalmology at Madigan Army Medical Center.
- Dr. Mazzoli graduated from West Point and the Uniformed Services University of the Health Sciences. After an internship, his ophthalmic training was received at Brooke Army Medical Center and The Wills Eye Hospital in Philadelphia, Pa.
- His academic interests include surgical simulation in education, telemedicine and advanced technologies; military ophthalmic readiness; and regenerative medicine.
- Highlights of Dr. Mazzoli's numerous academic achievements and awards include over 40 publications and book chapters and examiner for both the American Board of Ophthalmology and American Society of Ophthalmic Plastic and Reconstructive Surgery (ASOPRS). He is a Fellow of the American Academy of Ophthalmology, the ASOPRS, the American College of Surgeons, the Society for Simulation in Healthcare, the Association for Research in Vision and Ophthalmology, and the Wills Eye Hospital Society.

Combat Eye Injuries

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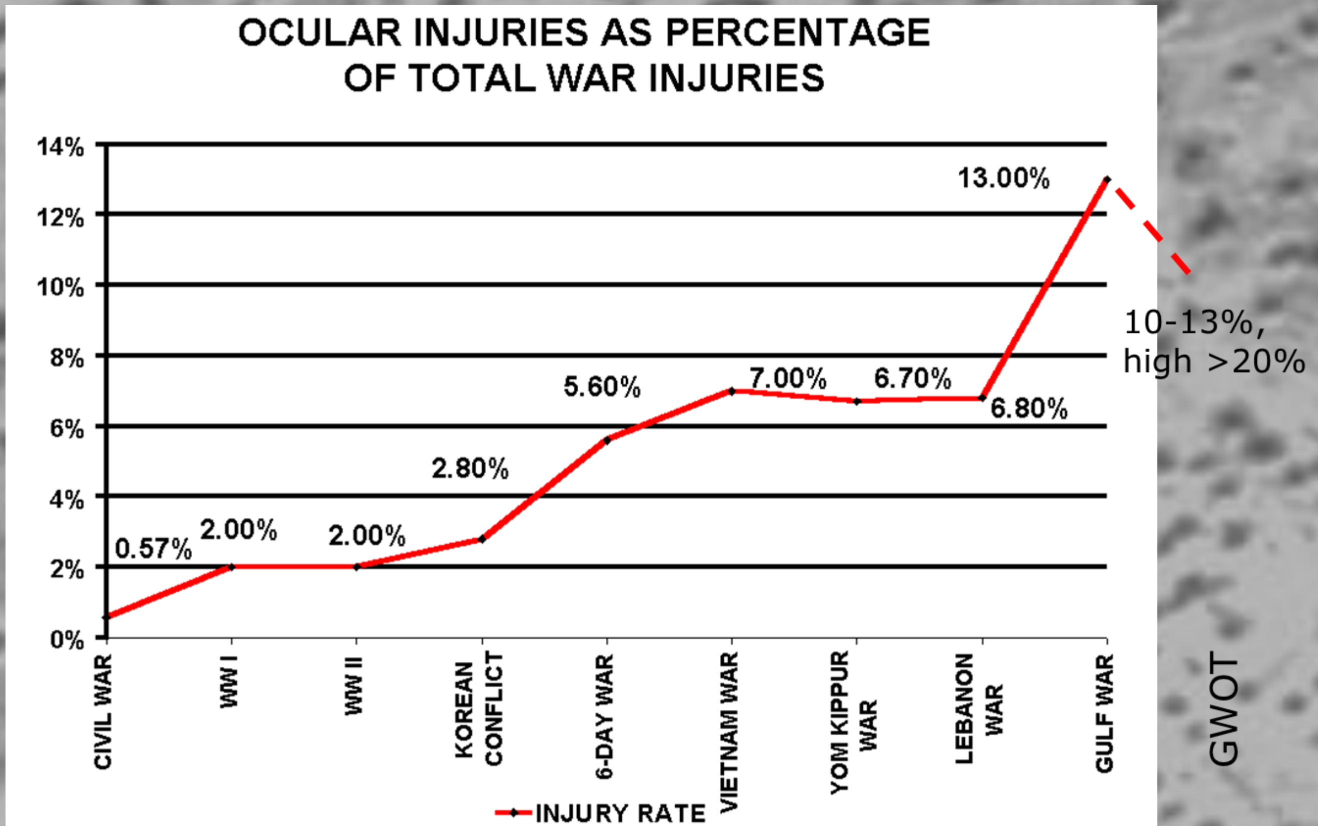
Disclaimer

- The opinions and assertions are purely those of the author and do not represent those of the Departments of Defense or Veterans Affairs
- The author has no financial interest in any product mentioned
- Presentation includes clinical photographs

Historic Rates of Eye Injuries



(Weichel, Colyer, Ludlow, Bower & Eisman, 2008)

Why the increase?

- Combat is visual
  - Can't see, can't fight
  - Reluctance to interfere with vision
  - Eye is increasingly vulnerable in modern combat
- Incidence of injuries 20-50x higher than expected by surface area
  - 0.27% of BSA
  - 4% of face
- Increased survival rates (95%)
  - Decreased KIA, body armor

Public domain: DoD archives

Causes of Eye Injuries in Combat

- Henry II of France; 1559
  - Died of eye/ brain injury in joust
  - Accurately predicted by Nostradamus
  - Similar casualty in 2007
- Civil War; eye/ head injuries lethal
  - Black powder (low energy)
    - Non-Friedlander physics
  - Shrapnel shell (1780s)
  - Minie ball
  - Large fragments

Photo Credit: Getty Images

Photo Credit: Katie Zezima

Public domain
http://en.wikipedia.org/wiki/Mini%C3%A9_ball#mediaviewer/File:Minie_Balls.jpg

Causes of Eye Injuries in WW1

- Cordite (High Energy)
  - Friedlander physics
- Close proximity, high explosive
  - Shrapnel shell, trench mortar
  - Armor, grenades
- Small fragments
- Head, neck, eye injuries
  - "The Trinity"
- Gas

soldiers, blinded by tear gas during the Battle of Estaires, 10 April 1918

Public domain http://en.wikipedia.org/wiki/World_War_I#mediaviewer/File:British_55th_Division_gas_casualties_10_April_1918.jpg

Causes of Eye Injuries GWOT

- Close proximity high explosives
  - Friedlander physics
  - Complex, reflected waves
- IED
  - Artillery, mines, car, suicide, fertilizer
- Shrapnel, fragmentation
- Head + eye injuries: >30%
- Conventional injuries

IEDs found in Baghdad.

Public domain - http://en.wikipedia.org/wiki/Improvised_explosive_device#mediaviewer/File:IED_Baghdad_from_munitions.jpg

Blast Injury Effects

- Primary: Blast overpressure
  - HE: Friedlander waveform
  - Supersonic
- Secondary: Blast wind
  - Shrapnel, debris
- Tertiary: translocation
  - Objects or people
- Quarternary: Burn, collapse, crush
- Quinary: Environmental toxins

Photographs were made by Edgerton,, Germeahausen and Grier,, Inc. (EG&G) for the Atomic Energy Commission (AEC) -- now DOE.

Blast Eye Injuries: Primary

"…absence of outward signs is not necessarily an indication of lack of intraocular lesions, and moreover, of extensive ones…" LTC GE DeSchweinitz AJO 1919

- *Concussive/ Contusive
  - *Hyphema, hemorrhage
  - *Commotio retinae
  - *Macular hole
  - *Visual field loss
  - *Hypotony
  - Angle recession
  - Endothelial cell loss
- *Rupture
- *Delamination
  - Retinal detachment
  - Choroidal rupture
  - PVR (proliferating chorioretinitis)
  - Cyclodialysis
- *TON
- *Transitory astigmatism

**documented in WW1*

Blast Eye Injuries: 2º, 3º, 4º

- *Laceration, penetration, perforation, crush
- *Avulsion; ON avulsion, lids
- *Burn, contaminants (dirt, mud, bone)
  - *Burn not just from blast: aviation, secondary fires
- *Fracture
- *TBI (often, symptoms only)
  - Accommodative dysfunction
  - Photophobia

documented in WW1

Photo Credit: DoD image
http://www.photonics.com/Article.aspx?AID=48445

Eye Protection:
Long ago and far, far away…

- *…inasmuch as roughly 50% of all eye wounds are produced by tiny fragments, there should be some practical method of prevention*
  - *V Morax, F Moreau (1916)*
- *The number of men who have become…blind as a result of wounds is very large. While the humanitarian interest in the question is, of course, uppermost, the economic side is not to be lost sight of.*
  - *SH Wadhams (1921)*

Eyewear Used During LASER Experiment

"Military laser experiment" by US Air Force - This Image was released by the United States Air Force with the ID 090809-F-5527s-0001 http://en.wikipedia.org/wiki/Eye_protection#mediaviewer/File:Military_laser_experiment.jpg

Eye Protection: Current

- Polycarbonate
- Anti-ballistic
- Spectacles
- Goggles
- Commercial designs
- Blast?


Photo Credit: Thomas H. Mader, MD, FACS

Photo courtesy of Col (R) MC US Army Thomas H. Mader, MD, FACS


Photo Credit: Cmdr. Tanya B. Sinclair, NC US. Navy


http://www.army.mil/article/121595/Eye_injuries_avoidable_with_use_of_eye_protection/

Conclusion

- Eye injuries are common for a variety of reasons
- Particularly in High Explosive (HE) environment (combat or terrorism)
- Visible anatomic injuries
- Subclinical anatomic injuries
- Invisible injuries (symptoms only)
- Protection is best
- Awareness of potential injury
  - Referral

Photo Credit: DoD image
http://www.photonics.com/Article.aspx?AID=48445

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Questions?

- Submit questions via the Q&A Pod located on the screen.
- The Q&A Pod is monitored and questions will be forwarded to our presenter for response.
- We will respond to as many questions as time permits.

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Next DCoE Psychological Health Webinar:

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Aug. 28, 2014

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