Numerous research studies provide evidence that repetitive blast exposures harm the brain.

I. INTRODUCTION
Trauma from blast exposure has become an increasing component of modern warfare. Blast injuries result from the sudden onset of shock waves generated by an explosion. This paper will first review scientific evidence that repetitive blast exposure harms the brain. It will underscore the vital need for blast pressure monitoring in military and tactical law enforcement personnel. Next, it will describe a recently available technology that enables this surveillance. Finally, it will emphasize the far-reaching benefits of blast pressure monitoring in protecting the health of military and tactical law enforcement personnel.

II. EVIDENCE THAT REPETITIVE BLAST EXPOSURE HARMS THE BRAIN
The signature injury of modern warfare is mild traumatic brain injury (mTBI). Blast exposure is the leading cause of mTBI for US forces deployed to Afghanistan and Iraq in Operation Enduring Freedom (OEF), Operation Iraqi Freedom (OIF), and Operation New Dawn (OND). The estimated prevalence of mTBI among returning service members is quite high, ranging from 15.2% to 22.8%. Acute symptoms of mTBI include headaches, dizziness, fatigue, irritability, confusion, memory problems, and sleep disturbances. In some cases, blast exposure can harm the brain without producing any obvious acute symptoms. Blast-related symptoms can surface much later in such cases.

Research provides evidence that blast exposure damages the structure of the brain. In one study, Yeoh and colleagues reported evidence that blast overpressure disrupts the highly protective blood-brain barrier in rats. In a separate study, Meabon and colleagues found evidence that blast exposure in mice damages the blood-brain barrier and interferes with the brain’s expression of a protein called tau. Using a swine model of blast-induced TBI, Ahmed and colleagues found evidence that blast exposure damages brain cells, compromises the permeability of blood vessels, and causes brain inflammation. In a postmortem study of veterans exposed to blast and/or concussive injury, Goldstein and colleagues found evidence of a tau protein-linked degenerative brain disease known as chronic traumatic encephalopathy (CTE). Veterans’ brain pathology was similar to that of athletes with a history of repetitive concussive injury. In another postmortem study, Shively and colleagues compared the brains of blast-exposed military personnel with the brains of civilians who had no reported history of blast exposure (including cases with no TBI, cases with non-blast TBI, and cases with opioid abuse). In the brains of the blast-exposed military personnel, they found a unique pattern of damage called interface astroglial scarring. The investigators concluded that this pattern of damage may be specific to blast and explain the persistent neuropsychiatric symptoms of blast TBI.

In a study of 134 veterans, Robinson and colleagues found that close-range blast exposure was associated with altered brain connectivity, even when the blast exposure did not result in concussion symptoms at the time. In a study of 52 OEF/OIF veterans, Bazarain and colleagues found evidence that the severity of posttraumatic stress disorder (PTSD) is related to the severity of combat stress and structural brain changes, but not to a clinical diagnosis of mTBI. They concluded that blast exposure may induce subclinical brain injury and contribute to the onset of PTSD in a combat environment.

Furthermore, many studies have provided evidence that the effects of blast exposure on the brain are cumulative and long-lasting. In a study of 27,169 U.S. Army Special Operations Command (USASOC) personnel, Kontos and colleagues found that those with a history of blast-related mTBI were at greater risk of reporting PTSD symptoms than those with no mTBI history. In a survey study, Carr and colleagues reported that repeated low-level occupational exposure to blast was associated with symptomatology similar to concussion. The number and severity of symptoms increased with history of blast exposure, and symptoms interfered with daily function. In a longitudinal study, Mac Donald and colleagues found evidence that in patients with concussive blast TBI, symptoms worsen, rather than resolve, over time. From the 1-year to 5-year follow-up evaluations, 36 out of 50 patients with concussive blast TBI (72%) showed a decline in global outcome, compared with only 5 out of 44 combat-deployed control participants (11%). In addition, the group with concussive blast TBI showed a worsening of symptoms of PTSD and depression over this time.

Meabon and colleagues found that in combat veterans with blast-related mTBI, the number of blast exposures correlated with symptoms of dizziness, loss of balance, and poor coordination. In addition, they found that an increase in the number of blast exposures was associated with lower glucose metabolism in the part of the brain known as the cerebellum. In a study of 80 USASOC personnel, Kontos and colleagues reported evidence that a history of blast-related mTBI exacerbates the initial symptoms of a subsequent mTBI. Trotter and colleagues, in a study of 249 veterans, found evidence that blast exposure accelerates the brain’s aging process by reducing the integrity of the brain’s white matter tissue, even in the absence of acute symptoms of TBI. In a recent article, Elder and colleagues reviewed substantial evidence that blast-related TBI is pathophysiologically distinct from non-blast TBI and that low-level blast has long-term effects on the brain.

III. THE CRITICAL NEED FOR BLAST PRESSURE MONITORING
Scientific studies have provided overwhelming evidence that blast exposure damages the brain, with long-lasting, adverse effects. However, our knowledge of the cumulative effects of blast exposure is severely limited by a lack of blast exposure monitoring. To estimate blast exposure, studies typically rely on self-report and semi-structured interviews of military personnel. Without monitoring blast pressure in individuals and evaluating the relationship between blast pressure levels and acute/chronic injury, we will never fully understand the
Blast exposure is an occupational hazard that injures the brain.\(^\text{17}\) Fractional anisotropy (FA), radial diffusivity (RD), and the apparent diffusion coefficient (ADC) are measures of water diffusion in the brain that provide information about the integrity of the brain’s white matter tissue. The data provide evidence that blast exposure accelerates the brain’s aging process by reducing the integrity of the brain’s white matter tissue. (The image in this figure, available at: https://academic.oup.com/brain/article/138/8/2278/330320/Military-blast-exposure-ageing-and-white-matter, is the original image from Figure 2 of Reference 17. Reference 17 is distributed under the terms of the Creative Commons Attribution 4.0 International License at http://creativecommons.org/licenses/by/4.0/.)

**FIGURE 1.** Regions of the human brain show a significant interaction between blast exposure and age on diffusion measures. Using a method called diffusion tensor imaging (DTI), researchers found that regions throughout the human brain’s white matter tissue (presented in different views in A-F) showed a significant interaction between blast exposure and age on diffusion measures.\(^\text{17}\) Fractional anisotropy (FA), radial diffusivity (RD), and the apparent diffusion coefficient (ADC) are measures of water diffusion in the brain that provide information about the integrity of the brain’s white matter tissue. The data provide evidence that blast exposure accelerates the brain’s aging process by reducing the integrity of the brain’s white matter tissue. (The image in this figure, available at: https://academic.oup.com/brain/article/138/8/2278/330320/Military-blast-exposure-ageing-and-white-matter, is the original image from Figure 2 of Reference 17. Reference 17 is distributed under the terms of the Creative Commons Attribution 4.0 International License at http://creativecommons.org/licenses/by/4.0/.)

**VI. REFERENCES**


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