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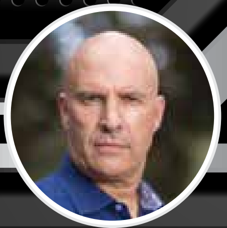

BY JON BECKER AND
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LOW-LEVEL BLAST EXPOSURE AND TRAUMATIC BRAIN INJURIES: IT IS TIME TO START PAYING ATTENTION

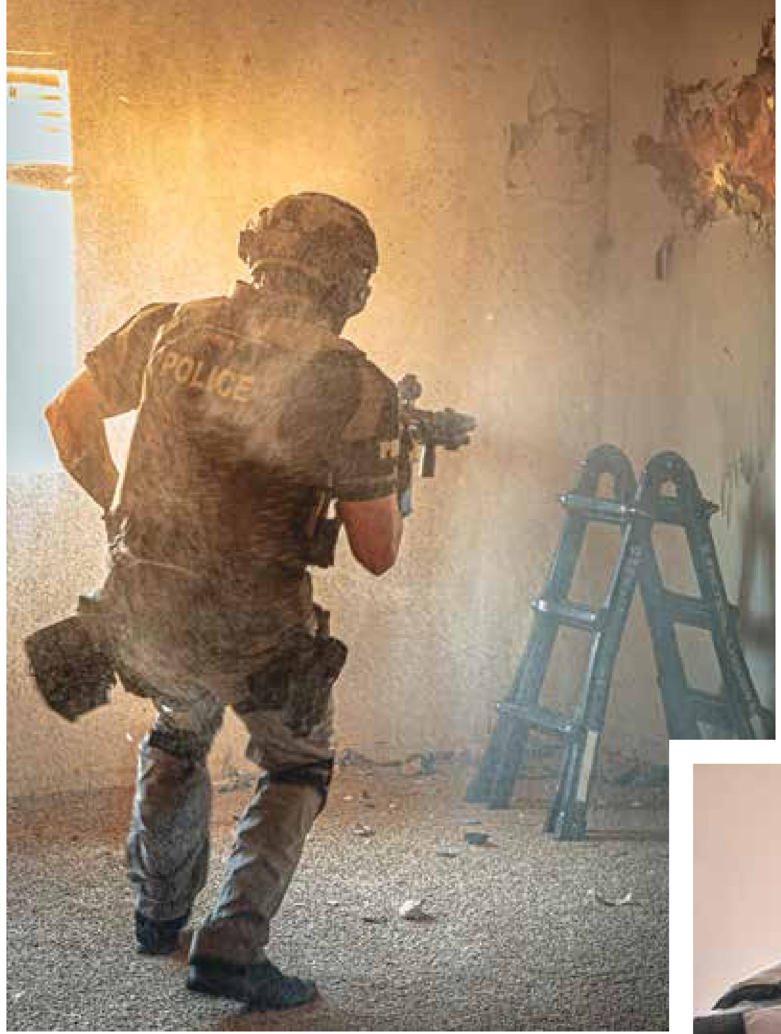
PART ONE

At a military science meeting in early 2008, representatives from the United States, Canada and New Zealand discovered that each country had received separate anecdotal reports of warfighters who experienced a constellation of symptoms associated with exposures to repetitive low-level blast effects (RLLBE), which they informally labeled “breacher’s brain.”[1] This public acknowledgment of what was previously a known but unrecognized phenomenon began a body of research and data collection efforts to understand how RLLBE leads to bodily injury, including brain injury. As this research has progressed, an ever-growing amount of data continues to cause concern about the long-term effects of blast exposure and mild traumatic brain injury (mTBI).

Over the past two years, we have been working on a series for The Debrief about the effects of blast exposure on the brains of tactical officers. In our research, we have spoken to many of the world’s top experts on this topic to gain insight into the problem and how we might prevent potential harm in the future. Although we hope to launch this episode early next season, the data we found and the arguments made by the experts we have spoken to make a compelling case for sharing this information now rather than waiting for the episode to launch. The issue of low-level blast exposure and TBI is profound, and teams must begin educating themselves about their risks and take preventative actions to reduce, mitigate and recover from unnecessary injury to their members. This is the first in a two-part series that will explore this topic as well as steps you can take now to help prevent unnecessary damage to your team.

The objective of this article

It is important to start with a disclaimer or, more precisely, a qualification about the objectives of this article. This area is far from being currently settled science. It may be decades from being completely understood. While it has been the subject of mainstream news stories and technical journal articles and is the basis for a new bill pending in Congress, it must be kept in context. In many ways, this period for blast injury and TBI feels like the 1960s and 1970s for cigarette smoking research. There is a significant amount of anecdotal evidence and negative data surrounding the issue. The evidence includes a rapidly growing body of computer modeling, scientific and medical research that demonstrates both causal effects in animals along with strong relationships between blast exposure and TBI in humans. It is rapidly becoming a cause célèbres for news outlets, veterans’ groups and members of Congress. Yet, like in the 1960s and 1970s, when no definitive medical data was showing that smoking causes cancer, there is also no definitive medical research



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that demonstrates direct causation between blast exposure and TBI in humans. This is due, in part, to the fact that 1) you cannot deliberately expose humans to harmful or potentially harmful blasts in a scientific or medical research study; 2) the signature injury, astroglia interface scarring, is microscopic; and 3) the signature injury is not visible with current neuroimaging methodologies. Thus, taking everything in context is essential to avoiding the temptation to rush to judgment since we don't know exactly who, why, what, when or where the injury will emerge from blast exposure.

Does that mean we should ignore the issue until it's proven? Absolutely not! Does that mean we should curtail the use of explosives? Of course not. Energetic materials are an essential component of tactical work. They save lives, and we are not arguing for their elimination. That said, it also doesn't make sense to take unnecessary chances or expose team members to unnecessary damage if it can be avoided. Certainly, no one would argue that blast exposure is good for you or recommend unnecessary exposure. The objective of this column is to make members aware of the evolving science and to recommend changes that can reduce exposure and risk without compromising operational readiness.

Blast overpressure and mild traumatic brain injury

When an explosion occurs, a high-pressure blast wave is created, which is a sudden and intense increase in air pressure that rapidly expands away from the source. As the energy from the explosion moves through the air, it creates a pressure front, or wave of high pressure, known as blast overpressure.[2,3] It is this overpressure which passes through the skull that appears to cause damage to the brain.[4]

Traumatic Brain Injury (TBI) is a form of brain damage caused by external forces like impacts or pressure waves that can lead to short- and long-term adverse side effects.[5] Blast-related TBI stems from exposure to overpressure produced by explosive breaching, diversionary devices, shotgun breaching, large caliber weapons, etc.[6,7] Blast-related TBI is uncommon in civilians; it is more common in professions like the military and tactical law enforcement, where repeated exposures to blasts is common.[8,9]

Beginning in World War I, medical professionals noted that soldiers were dramatically affected when they experienced blast waves on a large scale.[10] Soldiers near an explosion and the resulting shockwave arrived at field hospitals with little to no physical signs of trauma.[11] However, they would suffer subsequent symptoms such as headaches, dizziness, balance problems, nausea, sensitivity to light and sound, vision changes and impulsivity. A British doctor named Charles Myers coined the term "shell shock" in 1915 to describe the array of symptoms experienced by soldiers because of explosion-related shock waves. The question the medical profession faced after identifying the symptoms was whether being "shell shocked" was the result of physical or psychological injury.[12]

After World War I, the medical profession understood blast-related TBI to be an issue because of the perceived effects it had on the brain. However, TBI was not explicitly defined until much later, other than referencing someone being "shell shocked." During World War II and the Vietnam War, the debate around TBI was ongoing, but the issue came to the forefront during the Iraq and Afghanistan wars. These two wars ushered in a renewed interest in the research because doctors found that 10% to 20% of veterans returning from Iraq and Afghanistan were experiencing TBI. While most of these were attributable to exposures to IEDs, not all were caused by large-scale blasts.[13]

TBI is categorized as mild, moderate and severe, depending on the immediate effects of the blast on consciousness and the severity of the injury. Blast waves cause diverse levels of injury because overpressure is the highest closest to the source of the explosion and diminishes with distance.[14,15,16] Many factors contribute to the significance of blast-related trauma; some of those factors include intensity, duration and the environment where the blasts take place. For example, blast waves reflect off walls and other structures like an echo, causing the potential for multiple exposures to a person from one blast. Proximity to the explosion and safety equipment worn by personnel also are significant factors.[17]

As of 2014, mTBI outnumbered moderate and severe TBIs in the population of returning veterans.[18,19] Researchers have shown that mTBI can result from repeated low-level exposure to blast waves that cause sub-concussive injury.[20] A sub-concussive injury is a type of brain injury that occurs when the head experiences a forceful impact or rapid movement but not to the extent that it causes a concussion.[21] Over time, however, it is believed that the cumulative effect of multiple sub-concussive injuries can also lead to long-term neurological damage, like chronic traumatic encephalopathy (CTE), which is a degenerative brain disease found in individuals with a history of repetitive brain trauma from impact, like football players.[22] Growing evidence suggests microscopic injury occurring in the brain after blast injury is a reactive pro-

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cess called astrogliosis. Compelling postmortem studies on U.S. service members who have a history of RLLBE show accumulation of this astroglia interface scarring at the junction of white and grey matter in their brains.[23]

In a 2016 study published in *Military Medicine*, researchers found a relationship between repeated exposure to low-level blast waves and neurological alterations. The group studied were instructors and students at the U.S. Marine Corps Weapons Training Battalion Dynamic Entry School. The study focused on explosive breaching instructors because of their repeated exposure over several years. The two-week course exposed instructors and students to 40 low-level blasts. At the end of the course, the instructors demonstrated impairment on cognition tests, while the students did not. The data suggests a person's sensitivity to blast waves increases with repeated exposures.

In the context of this study, instructors received multiple exposures because they taught multiple two-week breaching classes a year. In contrast, students received only 40 exposures over two weeks. The instructors sustained impairment due to the school, and the students did not. The article points out that this occurred even though the instructors were often at greater standoff distances. This raises concerns that long-term, consistent exposure to blast environments may increase the probability of injury when re-exposed later. While this study suggested that repeated exposure to low-level blast waves negatively affects the brain, it was inconclusive because of the small sample size.[24]

In 2020, the *Journal of Neurotrauma* released a study pointing out that humans show adverse neurological effects from blast-related mTBI. The authors examined career breachers with more than 400 blast-related exposures and non-career breachers with less than 40. They discovered that the career breachers had a general increase in cortical thickness, providing evidence of structural differences in the brains of career breachers compared to the control group. The authors cautioned that scientists need to conduct more research into mTBI and that the results of their study are inconclusive. They also made no concrete claims of what adverse effects those structural differences would cause. However, the findings add to the existing knowledge that repeated low-level blast exposures impair the brain in some fashion.[25]

Unlike penetrating or severe TBI, where the symptoms are evident because there is a physical injury or immediate and obvious psychological symptoms, it is incredibly challenging to define mTBI related to blast waves because of an absence of an agreed-upon clinical description of the symptoms.[26] Some of the symptoms of mTBI are headache, fatigue, poor concentration, lethargy, depression, anxiety, irritability and insomnia.[27,28] However, many of these symptoms overlap with Post Traumatic Stress Disorder (PTSD), even in the absence of a psychological stressor.[29] As far back as World War I, doctors recognized symptoms from blast exposure that were like those found in post-concussion syndrome and PTSD.[30] However, because of the overlap in symptoms (irritability, insomnia, anxiety, memory issues, etc.), it is sometimes difficult to differentiate if one is suffering from mTBI, PTSD or both.[31]

More confounding is the fact that post-concussive syndrome and lingering mTBI symptoms will not necessarily be close in time to the blast event. They can occur quite a bit later. Complicating it more, combat veterans, military personnel and police officers can often experience repeated low-level blast wave exposures, which can have a cumulative effect. Since these exposures do not cause immediate extensive brain injury and subsequent visible symptoms, it is often challenging to diagnose mTBI.[32]

Evidence implies repeated low-level blast exposure may cause microtrauma damage that will negatively affect physical and mental well-being. Unfortunately, there is currently no definitive way for a doctor to diagnose mTBI. There is no blood test or diagnostic procedure they can use to prove mTBI definitively. As a result, a diagnosis, if one is made, is usually based on a collection of common symptoms that are difficult to attribute directly to an event from the past. This is especially true in a population like law enforcement, who may be more susceptible to mTBI because they have played contact sports, been in fights, had car crashes, etc.

Correlation v. Causation — When Do We Act?

Although the science is not conclusive and has yet to establish a clear causal relationship between RLLBE and mTBI, it is important to state that the research to date has by no means been ineffective or useless. On the contrary, what the research to date has been able to establish is a strong correlation between blast exposure, especially RLLBE, and a collection of symptoms, including headaches, memory impairment, sleep disruption, poor impulse control, mood disturbance and cognitive deficit.

The more often two things are correlated, and the broader the sample set used, the greater the likelihood is that there may be a causal relationship. Put differently, although correlation does not establish causation, it can be an early indicator of a causal link between two things. That said, correlation is not causation; two things can be correlated without having a causal relationship. For example, if I turn on the radio and it starts to rain, those things are *correlated* in time, but the radio did not *cause* it to rain. The amount of data required to establish correlation is a mere fraction of that needed to show causation, especially in a case like this, where so many variables are involved. Without a great deal more research we likely will not establish a causal relationship between RLLBE and symptoms. Fortunately, research is being conducted, and the Department of Defense is taking this very seriously. Numerous well-funded studies are gradually wearing away at the mystery of mTBI. However, we are likely many years to decades away from a definite answer. Does that mean we should just wait and see? Absolutely NOT! The growing expert opinion is that there is a causal link and that acting now to prevent future damage is the only prudent course of action.

The correlation between RLLBE and mTBI is a lot like the correlation that existed in the 1950s when science first noticed that smokers seemed to have higher rates of lung cancer than nonsmokers. Although there was no "proof" that smoking caused cancer, there certainly was a great deal of evidence correlating smoking and illness. Does that mean we took action? No, it does not. The first study connecting smoking to lung cancer was released in 1950, yet it was not until 1965 that warnings even began to appear on cigarettes.

The question, of course, becomes, how long should we wait before acting on this topic? While many of the experts we spoke to urged caution in drawing large-scale conclusions or making definitive medical judgments, they all agreed that there is enough data to implement protective measures whenever possible and limit unnecessary exposure to blasts. Anyone who has ever been exposed to the effects of an explosive breach, has spent the day throwing flash-bangs or spent time shooting a high-caliber rifle has felt the effects of the low-level blast. Some effects are almost always felt, whether it's a headache, a lack of concentration, difficulty with balance, difficulty sleeping or mood disturbances. As more data emerges, it would appear wise to treat this like a flashing red warning sign and pay attention.

While recent research into blood-based TBI biomarkers has shown promise, they are not yet ready for use. Although initial research has given hope that we will eventually find a biomarker that will allow monitoring of operator safety through a simple blood test, the science is not yet developed enough for this to be possible. This leaves the medical community without an objective mTBI test, and it likely will be years to decades before there is enough data to truly establish the mechanism for injury and the causes of symptomology.[33]

This does not mean we need to freak out, stop using guns and explosive devices or lock ourselves in a plastic bubble. Explosive devices are an essential part of tactical law enforcement which cannot be eliminated. That said, numerous small changes can be implemented now with minimal effort or cost that may help prevent long-term harm.

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Taking action: Preventing damage now

By far, the most effective approach to managing mTBI and RLLBE injury is never to let it occur. While tactical officers will unavoidably be exposed to a certain amount of RLLBE throughout their career, there is certainly enough data available at this point to demonstrate that reduction of unnecessary exposure is prudent, if not essential, and recovery after injury is crucial. In this regard, the best course of action for teams is to adopt a three-pronged approach: *Reduce* the frequency of exposure, *mitigate* the effects of exposure, and *recover* from exposure prior to repetition.

Part two of this series will examine steps your team can implement in training to accomplish these objectives and reduce future damage.

Acknowledgment: *Several individuals have shared their knowledge on this subject over the past two years. Specifically, I need to thank James Engall from BAAM360, who has been a wealth of knowledge and guidance. His comments and understanding made this article better.*

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