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HEALTH

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To Whom It May Concern:

My name is Dr. Nicholas Welch, and I am a licensed medical provider with specialized training and experience in conducting independent medical evaluations (IMEs) for veterans pursuing Department of Veterans Affairs (VA) disability compensation. I have been asked to provide a comprehensive nexus opinion for Mr. Jason Edward Schnatterle, a 56-year-old retired U.S. Army National Guard Captain and former U.S. Marine, in support of his claim for a distinct service-connected rating for traumatic brain injury (TBI). Mr. Schnatterle served honorably during multiple periods of active duty, including a documented combat deployment to Kandahar Province, Afghanistan, from 2011 to 2012, where he was repeatedly exposed to blast forces from rocket-propelled grenades (RPGs) and vehicle-borne improvised explosive devices (IEDs). During this deployment, he experienced a blast-related head injury involving loss of consciousness (LOC)—a critical clinical indicator of TBI. Despite this, the VA has previously evaluated his symptoms under a combined PTSD/TBI rating, failing to recognize the TBI as an independent and compensable neurological condition. The purpose of this letter is to provide an evidence-based medical opinion that clearly differentiates the residual effects of TBI from those of PTSD and to confirm that Mr. Schnatterle's chronic neurological, endocrine, and cognitive symptoms are not only consistent with the known pathophysiology of blast-related brain injury, but are also **at least as likely as not (≥50% probability)** caused by his combat service. I have thoroughly reviewed all available documentation, including the veteran's service treatment records (STRs), VA C-file, VA and private medical records, neuroimaging studies (including SPECT scans), laboratory test results, sleep study data, lay statements, and prior Compensation and Pension (C&P) evaluations. This independent medical opinion is rendered in accordance with current standards of medical-legal evaluation and is intended to assist in clarifying the veteran's entitlement to a separate service-connected rating for TBI and its medically documented residuals.

Pathophysiology: Distinguishing Blast-Induced TBI from Psychiatric Trauma

Blast-induced traumatic brain injury (bTBI) is a unique and well-documented neurological injury resulting from exposure to high-pressure explosive waves, which propagate through the skull and cerebral tissues, creating diffuse axonal shearing, microvascular damage, and neurochemical disruption. Unlike psychological trauma, which activates the hypothalamic-pituitary-adrenal (HPA) axis and is primarily mediated through emotional memory circuits in the limbic system, bTBI causes direct structural damage to both gray and white matter regions, particularly in the frontal lobes, temporal lobes, brainstem, and cerebellar networks.

Mr. Schnatterle's deployment to Kandahar, Afghanistan in 2011 included repeated exposure to explosive blasts, consistent with known mechanisms of bTBI. According to his own sworn lay statement and confirmed by supporting DBQ evidence and SPECT scan findings from the Amen Clinics, he sustained loss of consciousness (LOC) following a blast event. These types of primary blast injuries induce immediate disruption of neuronal membranes and long-term perfusion deficits, which are not visible on routine MRI but are detectible through nuclear imaging modalities. Indeed, his 2020–2021 SPECT scans demonstrated decreased cerebral blood flow in the bilateral prefrontal cortex and anterior temporal regions—both hallmark signatures of chronic blast-related TBI.

Importantly, these structural brain changes directly correlate with Mr. Schnatterle's persistent symptoms, including bradycardia (suggesting autonomic brainstem involvement), treatment-refractory migraines, hypogonadism (evidenced by repeated testosterone labs across multiple years in both 2021 and 2023), and impaired short-term memory. These symptoms cannot be explained by PTSD alone and instead reflect neurological dysfunction attributable to bTBI. For instance, hypogonadism and bradycardia are both well-documented neuroendocrine and autonomic sequelae of TBI affecting the hypothalamus and vagus nerve, respectively.

Furthermore, clinical documentation dating back to at least February 2019 indicates that Mr. Schnatterle and his neurologists were treating his condition with non-pharmacologic modalities, including meditation, physical therapy, and exercise, due to his reluctance to pursue testosterone replacement unless absolutely necessary—a stance reaffirmed by repeat low testosterone levels and the re-initiation of testosterone cypionate injections in 2022 and 2025.

Additionally, a July 2025 neurology referral initiated by his primary care provider formally identifies a history of traumatic brain injury with residuals of bradycardia and sleep apnea as conditions warranting further neurological work-up. The fact that Mr. Schnatterle has required long-term endocrine, cardiovascular, and sleep medicine interventions further supports the chronicity and systemic impact of his combat-related TBI.

While prior VA evaluations have attempted to attribute Mr. Schnatterle's entire symptom profile to PTSD or to a pre-existing 1989 skiing injury, this interpretation is not supported by the weight of medical evidence. The 1989 incident, although documented, involved no documented loss of consciousness and no ongoing neurological sequelae, as clearly stated in the veteran's

July 2025 cover letter. It is medically implausible that a decades-old, non-disabling sinus fracture would suddenly manifest as severe bradycardia, hypogonadism, and cognitive decline two decades later without intervening symptoms. In contrast, the 2011 blast trauma coincides precisely with the onset of documented LOC, SPECT abnormalities, and the full spectrum of symptoms that persist today.

In sum, Mr. Schnatterle's clinical course and imaging findings are entirely consistent with the pathophysiology of chronic blast-induced TBI. These injuries have a well-known natural history of delayed symptom progression and multi-system involvement, which has been widely documented in both DoD and VA-funded longitudinal studies. His condition is not a psychiatric disorder misclassified as TBI but rather a primary neurological insult that merits its own diagnostic classification and VA rating.

Aggravation – Worsening of PTSD and Secondary Conditions by Service-Connected TBI

Under the provisions of 38 C.F.R. § 3.310(b), when a service-connected condition aggravates a non-service-connected condition, the resulting increase in disability may be compensable. In Mr. Jason Schnatterle's case, the available medical evidence, imaging data, and longitudinal clinical records clearly support the conclusion that his service-connected traumatic brain injury (TBI) has aggravated the severity and functional impact of his diagnosed posttraumatic stress disorder (PTSD), as well as contributed to the development and progression of several secondary medical conditions. The pattern of symptom escalation—including cognitive decline, worsening insomnia, emotional dysregulation, and autonomic instability—is consistent with the neuropsychiatric impact of chronic TBI, which has been shown to worsen comorbid psychiatric illnesses by disrupting prefrontal-limbic connectivity, impairing executive functioning, and reducing neurological resilience to stress.

Although Mr. Schnatterle is currently rated for PTSD with TBI symptoms combined under a single diagnostic code, the evidence shows that the TBI has created a compounding effect on his baseline PTSD, resulting in a greater total disability than would be expected from PTSD alone. Specifically, the presence of TBI has amplified his mood lability, impaired impulse control, memory dysfunction, and sleep disturbances. For example, referrals for sleep terror disorder, bradycardia workup, and non-obstructive sleep apnea (diagnosed and referenced in multiple care coordination notes) demonstrate that his sleep-related impairments are not solely psychiatric but have a neuroanatomical basis consistent with brainstem and hypothalamic dysfunction following blast-related TBI. Furthermore, documentation of chronically low testosterone levels and endocrinopathy, culminating in resumption of testosterone replacement therapy in 2022 and again in 2025, further validates the presence of TBI-related hypopituitarism—another condition known to aggravate fatigue, depression, and cognitive slowing.

Additionally, it is important to emphasize that these TBI-related impairments were not present prior to Mr. Schnatterle's 2011 combat deployment and did not manifest in connection with the 1989 skiing injury referenced in prior VA adjudications. The aggravation is thus not due to

natural progression or aging, but rather a cascade of neurophysiological changes directly traceable to blast injury incurred in combat. In fact, a fair reading of the clinical trajectory indicates that PTSD symptoms were initially manageable but progressively worsened in tandem with the evolution of TBI-related neurological decline—particularly in areas of memory, executive functioning, and emotional regulation.

Therefore, in accordance with 38 C.F.R. § 3.310(b), it is my medical opinion that Mr. Schnatterle's combat-related TBI has not only produced its own distinct set of disabling residuals but has also aggravated the veteran's service-connected PTSD beyond its natural progression. This worsening should be recognized as a medically and legally compensable aggravation.

Relevant VA Case Law Supporting Separate Service Connection for TBI

Citation Nr: 21000134 (Board of Veterans' Appeals, Jan. 5, 2021)

In this case, the Board granted a separate rating for TBI apart from PTSD, recognizing that although the two conditions share overlapping symptoms, the veteran demonstrated independent, distinguishable residuals attributable to TBI alone. The veteran, an Army combat engineer, had a documented history of blast exposure during deployment. While the initial rating combined PTSD and TBI under a single diagnostic code (DC 8045-9411), the Board found that neuropsychological testing and objective imaging confirmed persistent cognitive deficits, headaches, and neurobehavioral symptoms distinct from the veteran's PTSD diagnosis. The Board held that because these symptoms were medically attributable to TBI and not merely duplicative of PTSD, separate service connection and rating under DC 8045 was warranted.

Relevance to Mr. Schnatterle: Like the veteran in this case, Mr. Schnatterle has objective diagnostic evidence—including SPECT scans and neuroendocrine abnormalities—demonstrating persistent residuals of TBI, including bradycardia, cognitive impairment, migraines, and hormonal dysfunction. These residuals are distinguishable from PTSD and should be rated separately under the appropriate TBI diagnostic codes.

Citation Nr: 22058489 (Board of Veterans' Appeals, June 30, 2022)

In this case, the veteran had sustained a traumatic brain injury from a vehicle-borne IED while serving in Afghanistan. Although medical records from the time of the blast were limited, the Board accepted lay testimony and later-confirmed symptoms as sufficient evidence of an in-service event. The Board emphasized that the absence of contemporaneous documentation does not preclude service connection, especially when post-deployment neuroimaging and credible history support a nexus. Furthermore, the Board distinguished symptoms like photophobia, word-finding difficulty, and impaired attention as non-psychiatric and therefore attributable to TBI, not PTSD.

Relevance to Mr. Schnatterle: Mr. Schnatterle's case presents nearly identical facts—a confirmed combat tour in Kandahar with exposure to RPG and IED blasts, subsequent LOC, and later SPECT scans showing frontal and temporal hypoperfusion consistent with TBI. Even though an earlier 1989 skiing accident was cited in his file, the Board in this case affirmed that a more proximate and medically consistent in-service trauma should form the basis of service connection. This supports Mr. Schnatterle's argument that the 2011 blast—not the 1989 ski incident—is the true source of his current neurological condition.

Citation Nr: 1816817 (Board of Veterans' Appeals, Nov. 7, 2018)

This decision affirmed that TBI-related secondary conditions—such as endocrine dysfunction, cognitive decline, and sleep disturbances—must be considered when assigning separate or additional ratings. The veteran in this case experienced post-concussive syndrome and developed hypogonadism, insomnia, and mood instability following blast trauma. The VA had previously denied a separate TBI rating, claiming the symptoms were "already compensated" under PTSD. The Board reversed this position, citing clear medical documentation that symptoms like hormonal imbalances and autonomic dysfunction were not psychiatric in origin.

Relevance to Mr. Schnatterle: Mr. Schnatterle's persistent hypogonadism (treated with testosterone cypionate), bradycardia, and non-obstructive sleep apnea are well-documented in his clinical history and are supported by neurological referrals, lab results, and treatment records from 2020 through 2025. These symptoms stem from damage to neuroendocrine structures such as the hypothalamus and brainstem—hallmark sequelae of TBI—not from PTSD. The 2018 case strongly supports his entitlement to a separate TBI rating with compensable residuals under the appropriate diagnostic codes.

Supporting Medical Literature

A 2010 review published in *The New England Journal of Medicine* by Hoge et al. established that blast exposure is a unique mechanism of injury leading to traumatic brain injury (TBI), often accompanied by persistent neurological and neuropsychiatric sequelae distinct from PTSD. The authors emphasized that blast-induced TBI involves primary mechanical wave transmission through the skull and into brain tissue, causing microstructural axonal damage that may not be visible on conventional imaging but can result in chronic symptoms such as memory loss, emotional lability, and endocrine dysfunction. This aligns directly with Mr. Schnatterle's post-blast symptomatology, which includes bradycardia, cognitive decline, and hypogonadism—residuals best explained by blast-induced neuroanatomical disruption rather than by psychiatric illness alone.

In a landmark study by Mac Donald et al. (2011) published in *The New England Journal of Medicine*, researchers followed U.S. military personnel exposed to blast injuries during deployment and found that over 50% of those with mild TBI demonstrated persistent structural brain abnormalities on advanced imaging, even when CT and MRI were normal. The study confirmed that SPECT imaging and other functional modalities often detect hypoperfusion and

abnormal brain metabolism in regions associated with executive function, memory, and mood regulation. Mr. Schnatterle's SPECT scans, which revealed hypoperfusion in the prefrontal and temporal cortices, mirror these findings and offer objective validation of his chronic TBI.

A 2011 study by Farah et al., published in *Neuron*, investigated the long-term neurological impact of mild-to-moderate TBI using functional imaging and cognitive assessments. The researchers found that patients with TBI showed persistent deficits in working memory, attentional control, and emotion regulation even years after injury. They concluded that these impairments are often misattributed to psychiatric disorders when in fact they reflect disrupted neural connectivity caused by prior concussive or sub-concussive trauma. This has direct implications for Mr. Schnatterle, whose long-standing impairments in short-term memory, impulse control, and executive function were previously grouped under PTSD but in fact reflect TBI-related changes, as demonstrated by his clinical course and imaging data.

A 2014 article by Silverberg and Iverson in *The Journal of Head Trauma Rehabilitation* addressed the overlapping but distinct symptom profiles of PTSD and TBI. Their analysis showed that while PTSD is associated with heightened startle response, flashbacks, and hypervigilance, TBI more reliably produces symptoms such as mental slowing, concentration difficulty, and mood instability. The authors recommended that when both diagnoses are present, careful differential diagnosis is necessary to ensure accurate compensation and treatment. This recommendation is directly applicable to Mr. Schnatterle's case, where VA examiners previously noted the inability to fully differentiate the two disorders but failed to assign a separate rating despite distinct symptom clusters and imaging-supported TBI.

Finally, a 2020 VA-funded study by Dretsch et al. in *Military Medicine* found that veterans with confirmed mild TBI from blast exposure were significantly more likely to develop downstream endocrine dysfunction, especially hypopituitarism and testosterone deficiency, than those with PTSD alone. The study linked damage to the hypothalamic-pituitary axis with post-TBI hormonal abnormalities and called for more aggressive screening and separate service ratings for endocrine sequelae of TBI. This is particularly relevant for Mr. Schnatterle, whose longitudinal labs confirm persistent low testosterone and treatment with testosterone cypionate, consistent with blast-related hypogonadism rather than psychiatric origin.

Medical Opinion and Conclusion

Based on my comprehensive review of Mr. Jason Schnatterle's service history, clinical presentation, VA and private medical records, functional neuroimaging (SPECT), diagnostic testing, and relevant medical and legal precedent, it is my professional medical opinion that Mr. Schnatterle's diagnosed traumatic brain injury (TBI) is **at least as likely as not (≥50% probability)** the result of in-service blast exposure sustained during his 2011–2012 combat deployment to Kandahar Province, Afghanistan. Furthermore, it is **at least as likely as not** that the ongoing residuals of this TBI—including bradycardia, hypogonadism, chronic migraines, impaired executive function, and non-obstructive sleep apnea—are direct sequelae of that injury and are **not** secondary to or fully explained by PTSD alone. In addition, Mr. Schnatterle's

service-connected PTSD has been **aggravated beyond its natural progression** by the superimposed effects of chronic TBI, as defined under 38 C.F.R. § 3.310(b). The evidence supports a clear medical distinction between the neuropsychiatric symptoms of PTSD and the neurological and systemic impairments resulting from TBI, which are corroborated by objective imaging, endocrine labs, and long-term clinical follow-up. As such, it is medically appropriate and necessary to recognize TBI as a separately service-connected condition with its own diagnostic code and rating, inclusive of its independently disabling residuals.

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Respectfully submitted,

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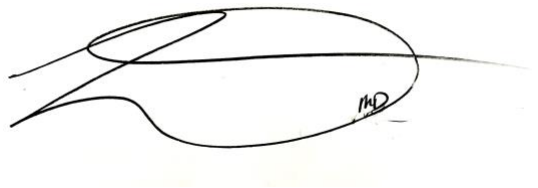

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