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Psychological File Review

Veteran's name: John Doe
DOB: XX/XX/XXXX
File #: XXX-XX-XXXX
Report date: X/XX/XXXX
Reviewer: Todd Finnerty, Psy.D.
Records Reviewed: I have reviewed all the records provided from the Veteran's claims file. No examination was conducted for this medical opinion; I have performed a thorough review of the Veteran's medical history.

Referral Questions: The Veteran is applying for service connection for sleep apnea secondary to PTSD with alcohol abuse.

Sleep apnea analysis: Mr. Doe experiences PTSD with alcohol abuse from his military service per his VA treatment records. The diagnostic criteria for PTSD include two separate symptoms associated with PTSD reflecting recurrent and distressing dreams as well as sleep disturbance such as difficulty falling asleep or staying asleep or restless sleep. The chronic sleep impairment from this differs from the symptoms and impairment associated with sleep apnea. Mr. Doe is also diagnosed with sleep apnea based on his 1/23/23 sleep study from Acme Sleep Medicine. Sleep apnea is, of course, a separate entity from mental health disorders, but that does not mean that one disorder cannot lead to and/or substantially aggravate the other.

The VA's own website indicates sleep apnea has a "relationship with PTSD" (*ex: https://www.research.va.gov/topics/respiratory.cfm#research8*). There is ample evidence of multiple risk factors which show an association with sleep apnea-- one of these factors— at least as likely as not on par with the others or with stronger association than the others is PTSD; we also know the direction of the relationship as by definition PTSD is caused by trauma and PTSD is not caused by sleep apnea. These associated factors (ex: weight gain) can significantly contribute to the development of sleep apnea and/or substantially aggravate sleep apnea. There is scientific evidence that PTSD can also at least as likely as not cause sleep apnea through multiple pathways. Some C&P examiners perform a cursory review of data related to sleep apnea using resources such as UpToDate; UpToDate does not reflect an adequate literature review related to the evidence associated with sleep apnea being secondary to PTSD. It is not peer-reviewed research literature, it is geared toward giving a basic overview of the characteristics of sleep apnea to non-experts, and it does not address the growing body of primary source journal articles that are specifically related to sleep apnea secondary to PTSD. Citing sources such as UpToDate—or no sources at all-- is a sure sign that the examiner lacks relevant expertise in the subject matter. Some C&P examiners attempt to describe what sleep apnea is characterized by as if this is what it is ultimately caused by, however this confuses the signs and symptoms of the sleep apnea process for sleep apnea's cause; this is an error. Factors such as sleeping position, weight gain, dentition,

and pharyngeal anatomy are symptoms of or may worsen the symptoms of the sleep apnea process but do not necessarily reflect the ultimate cause of sleep apnea. His PTSD has likely had a profound impact on the development and subsequent substantial aggravation of his sleep apnea. Pharyngeal anatomy explains only a minimal portion of the variability in measures like the Apnea-Hypopnea Index (AHI). We know it is not just an anatomical disease when surgery to alter anatomy is of minimal benefit to a large number of individuals with OSA. Individuals with OSA often likely have hyperarousal concerns– a low threshold for arousal– which is an underlying mechanism of obstructive sleep apnea. Individuals have difficulty activating the muscles and stabilizing breathing in a normal fashion. PTSD can lead to this hyperarousal. The supporting research is outlined below. It is at least as likely as not that the Veteran's sleep apnea is secondary to his PTSD.

The incidence of insomnia and obstructive sleep apnea (OSA) in service members is increasing. "Between 2005 and 2019, incidence rates of OSA and insomnia increased from 11 to 333 and 6 to 272 (per 10,000), respectively." The incidence rates have "markedly increased" since 2005 [*see Moore, et. al. (2021), Incidence of Insomnia and Obstructive Sleep Apnea in Active Duty United States Military Service Members. Sleep, doi: 10.1093/sleep/zsab024*]. However, the evidence for service connection is not based on incidence rates alone. Significant associated factors that reflect both a causal relationship and substantial aggravation have been identified in the research literature and they are consistent with his mental health records. His mental health symptoms, which appear to be a response to traumatic stress from the military, have likely created a significant and substantial progression and increased severity of his sleep apnea above and beyond the natural progression of the disease. His sleep apnea aggravates his PTSD symptoms and his PTSD symptoms have substantially aggravated his sleep apnea. The course of the Veteran's sleep apnea is not inconsistent with exacerbation tied to traumatic stress and other psychiatric conditions including depression. There is scientific evidence supporting a link between his psychiatric disorders and his sleep apnea. The research literature establishes that PTSD is commonly associated with sleep apnea and that there is an arousal-based mechanism initiated by PTSD that promotes the development of sleep apnea in trauma survivors. Sharafkhaneh, et. Al. (2005), in the article Association of Psychiatric Disorders and Sleep Apnea in a Large Cohort (*SLEEP, Vol. 28, No. 11, 2005)* found that sleep apnea is associated with a higher prevalence of psychiatric comorbid conditions in Veterans Health Administration beneficiaries, including PTSD. Compared with patients not diagnosed with sleep apnea, a significantly greater prevalence ($P < .0001$) was found for posttraumatic stress disorder in patients with sleep apnea. The research supports the presence of a significant connection between PTSD and sleep apnea, including an arousal-based mechanism initiated by PTSD that promotes the development of sleep apnea in trauma survivors: This is supported by Krakow, et. al. [*see Krakow, B., Melendrez, D., Warner, T.D. et al. To Breathe, Perchance to Sleep: Sleep-Disordered Breathing and Chronic Insomnia Among Trauma Survivors. Sleep Breath 6, 189–202 (2002)*] who note that emerging evidence invites a broader comorbidity perspective, based on recent findings that post-traumatic sleep disturbance frequently manifests with the combination of insomnia and a higher-than-expected prevalence of sleep-disordered breathing (SDB). In this model of complex sleep disturbance, the underlying sleep pathophysiology interacts with PTSD and related psychiatric distress; and this relationship appears very important as demonstrated by improvement in insomnia, nightmares, and post-traumatic stress with successful SDB treatment, independent of psychiatric interventions. Continuous positive airway pressure treatment in PTSD patients with SDB reduced electroencephalographic arousals and sleep fragmentation, which are usually attributed to central nervous system or psychophysiological processes. Related findings and clinical experience suggest that other types of chronic insomnia may also be related to SDB. "We hypothesize that an arousal-based mechanism, perhaps initiated by post-traumatic stress and/or chronic insomnia, may promote the development of SDB in a trauma survivor and perhaps other patients with chronic insomnia. We discuss

potential neurohormonal pathways and neuroanatomatical sites that may be involved in this proposed interaction between insomnia and SDB" per the authors. These neurobiological processes are further reviewed by Kelly, et. al. (2016) [*see Understanding Recent Insights in Sleep and Posttraumatic Stress Disorder from a Research Domain Criteria (RDoC) Framework; Curr Sleep Medicine Rep, 2016, 2:223–232*]. They note that PTSD is associated with sleep disturbances, including insomnia, nightmares, REM abnormalities and "sleep-disordered breathing" such as in sleep apnea. They note that "recent studies have expanded our knowledge of the neurobiology of trauma and sleep. In addition, intervention research has provided valuable information about how sleep treatments affect PTSD symptoms and how PTSD treatments affect sleep symptoms." Chronic activation of stress hormones (hypothalamo-pituitary adrenal axis activity) caused by PTSD is known to lead to a neural sensitization leading to upper airway dysfunction such as sleep apnea [*see van Liempt, et. al. (2013) Sympathetic activity and hypothalamo-pituitary-adrenal axis activity during sleep in post-traumatic stress disorder: A study assessing polysomnography with simultaneous blood sampling. Psychoneuroendocrinology, 38(1), 155-165; and Kritikou, et. al. (2016) Sleep apnoea and the hypothalamic-pituitary-adrenal axis in men and women: effects of continuous positive airway pressure. European Respiratory Journal, 47(2):531-540*.] PTSD causes airway instability through this hyperarousal, leading to sleep apnea concerns. a low threshold for arousal during sleep in relation to airway narrowing is viewed as one specific phenotype of OSA [*ex: see Osman AM, Carter SG, Carberry JC, Eckert DJ. Obstructive sleep apnea: current perspectives. Nat Sci Sleep. 2018 Jan 23;10:21-34 and Eckert DJ. Phenotypic approaches to obstructive sleep apnoea - New pathways for targeted therapy. Sleep Med Rev. 2018 Feb;37:45-59*]. As noted by Eckert (2018) "people develop obstructive sleep apnoea (OSA) for different reasons. The ability to understand these reasons, easily identify them in individual patients, and develop therapies that target one or more of these reasons are the keys to unlocking new approaches for the treatment of OSA. In line with this approach, recent advances in OSA pathogenesis using upper airway and respiratory phenotyping techniques have identified four key causes of OSA. A narrow or collapsible upper airway ('impaired anatomy') is the primary cause. However, the anatomical contribution to OSA varies substantially. Indeed, impairment in pharyngeal anatomy can be modest and in many patients (∼20%), pharyngeal collapsibility asleep is not different to people without OSA. Thus, non-anatomical factors or 'phenotypes' that modulate pharyngeal patency are crucial determinants of OSA for many people. These include impairment in pharyngeal dilator muscle control and function during sleep, increased propensity for awakening during airway narrowing (low respiratory arousal threshold) and respiratory control instability (high loop gain). Each phenotype is a potential therapeutic target." Individuals with a low respiratory arousal threshold-- or hyperarousal concerns-- are particularly relevant when discussing sleep apnea secondary to PTSD.

Colvonen, et. al. (2015) [*see Obstructive Sleep Apnea and Posttraumatic Stress Disorder among OEF/OIF/OND Veterans; Journal of Clinical Sleep Medicine 11(5):513-518*] found that "PTSD symptom severity increased the risk of screening positive for OSA." The authors concluded that "veterans with PTSD screen as high risk for OSA at much higher rates…" It is notable that the VA's own website implies a "relationship" between sleep apnea and PTSD and cites this study for support (*see the VA's website at https://www.research.va.gov/topics/respiratory.cfm*). The VA's own website notes "sleep apnea (pauses in breathing that occur at night) can cause excessive daytime sleepiness, trouble concentrating, high blood pressure, cardiac and pulmonary disease, and motor vehicle accidents. Relationship with PTSD—In 2016, researchers at the VA San Diego Healthcare System and the University of California found that the risk of obstructive sleep apnea among Iraq and Afghanistan Veterans increased with the severity of their PTSD symptoms. The investigators looked at 195 Iraq and Afghanistan Veterans—more than 93% were men—who had visited a VA outpatient PTSD clinic for evaluation of their symptoms. Using clinical questionnaires to evaluate both levels of PTSD and sleep apnea risk, researchers found that nearly 70% of Veterans in the study were at high risk for developing sleep apnea, and that the risk

increased with the severity of their PTSD symptoms. This was despite the fact that many of them did not have a high body mass index or high blood pressure, considered risk factors for sleep apnea." Zhang, et. al. (2017) [*see Prevalence of obstructive sleep apnea in patients with posttraumatic stress disorder and its impact on adherence to continuous positive airway pressure therapy: a meta analysis; Sleep Medicine, 36:125-132*] performed a metal-analysis and concluded the OSA is commonly seen in patients with PTSD. They noted that "patients with PTSD and OSA demonstrated significantly lower adherence to CPAP therapy." There is ample evidence that treating OSA improves PTSD symptoms. In Orr, et. al. (2017) [*see Treatment of OSA with CPAP is Associated with Improvement in PTSD Symptoms Among Veterans; J Clin Sleep Med, 13(1):57-63*] the authors describe "a growing body of research has suggested a link between obstructive sleep apnea and PTSD" and "prevalence estimations for OSA in PTSD patients range from 52% to 69%, with some as high as 95%." They note "treatment of OSA with PAP therapy is associated with improvement in PTSD symptoms." They note one factor is that "treatment of OSA with CPAP appears to reduce the frequency of nightmares in PTSD patients." The authors concluded that sleep apnea treatment "should be considered an important component of PTSD treatment for those with concurrent OSA." Psychiatric concerns in general have been noted to be associated with sleep apnea. Rezaeitalab, et. al. (2014) [*see The correlation of anxiety and depression with obstructive sleep apnea syndrome, Journal of Research in Medical Sciences, Mar; 19(3): 205–210*] studied individuals with obstructive sleep apnea. They found that "53.9% of the individuals had some degree of anxiety, while 46.1% demonstrated depressive symptoms. In terms of OSAS severity, this study showed that OSAS severity was associated with the frequency of anxiety, choking, and sleepiness (P : 0.001). According to polysomnographic results, we found that the majority of patients suffering from anxiety and choking (66.7% and 71.4%, respectively) had severe OSAS." Jehan, et. al. (2017) [*see Depression, Obstructive Sleep Apnea and Psychosocial Health, Sleep Med Disord, 1(3): 12*] note that "there is a co-linear relationship between" obstructive sleep apnea and depression." Patients with OSA have impaired health and their psychosocial health and daily performance also decrease. They note "because disturbed sleep can cause poor concentration, mood problems, anxiety, and MDD, these factors are also the part of poor daytime performance." Some postulate that weight gain may be a primary cause of sleep apnea, however it is interesting to note that treating sleep apnea with CPAP often actually leads to more weight gain, not less weight gain *[see de Milo and Genta (2016), Continuous Positive Airway Pressure and Weight Gain: Do We Know the Mechanisms? American Journal of Respiratory and Critical Care, 194(7): 915*]. The research evidence, and the course of the Veteran's disease, support a link between his psychiatric difficulties and sleep apnea.

Mr. Doe's report of his 1/23/23 sleep study from Acme Sleep Medicine included a BMI in the obese range; this is consistent with the obesity diagnosis in his VA treatment records. A classic but outdated view of OSA is that it is only structural, caused by obstruction of the upper airways and is predominantly found in overweight individuals. As we've seen from the research this is an oversimplification of the evidence. The Veteran has a history of possible weight issues. However, it would be an error to suggest that any weight gain occurred in a vacuum. Factors associated with his psychiatric disturbance likely significantly contribute to weight gain. The scientific literature links psychiatric disturbance to decreased activity and emotional eating which can lead to weight gain. While weight gain potentially contributing to sleep apnea could also likely be due to psychiatric disturbance, it would be an error to rely on opinions that sleep apnea was simply due to being overweight. This is not consistent with the current state of the scientific literature. In fact, neither being overweight nor the presence of structural abnormalities fully explains the presence of sleep apnea in a substantial number of cases. Being overweight is not sufficient to explain the existence of sleep apnea in many individuals. Simply suggesting that OSA was due to being overweight would reflect an overly simplistic and mechanistic view of sleep and obstructive sleep apnea which does not reflect the current state of

the science. It would overlook the sleep regulating functions of the brain and the impact of psychiatric disturbance on the sleep regulating functions of the brain, including arousal-based mechanisms associated with sleep apnea which have been identified as a likely causally-based phenotype of sleep apnea. In fact, the scientific literature tells us that "a substantial portion" of patients with obstructive sleep apnea are "not obese" (*Gray et. al. 2017*). The authors of a 2017 study note that "a substantial proportion of OSA patients are not obese. Non-obese patients with OSA are a challenging group to treat with existing therapies... Our data also indicate that a key nonanatomical contributor to OSA pathogenesis, a low threshold for arousal, is likely to be particularly important in the pathogenesis of OSA in non-obese patients with OSA. A greater propensity for awakening in nonobese patients with OSA may also be a physiological factor contributing, at least in part, to poor CPAP tolerance in these patients. These findings have important implications for the treatment of OSA in nonobese individuals." (*Gray, et. al., 2017*). In addition, "arousal" plays a significant role in sleep apnea [*see Gray, et. al. 2017 Obstructive Sleep Apnea without Obesity Is Common and Difficult to Treat: Evidence for a Distinct Pathophysiological Phenotype; Journal of Clinical Sleep Medicine, 13(1): 81-88*]. PTSD is linked to sleep apnea directly, but it is also linked indirectly through weight gain, which is also associated with sleep apnea. PTSD negatively impacts motivation (ex: reducing activity) and can lead to overeating. His PTSD increases the risk for weight gain and obesity, which in turn further increases the risk for obstructive sleep apnea. The Veteran's weight gain occurred in the context of PTSD. Subthreshold and threshold post-traumatic stress disorder (PTSD) are associated with binge eating symptoms in both men and women [*see Braun J, El-Gabalawy R, Sommer JL, Pietrzak RH, Mitchell K, Mota N. Trauma exposure, DSM-5 posttraumatic stress, and binge eating symptoms: results from a nationally representative sample. The Journal of Clinical Psychiatry. 2019;80(6):19m12813*]. Hoerster, et. al. (2015) noted in "PTSD and depression symptoms are associated with binge eating among US Iraq and Afghanistan veterans" [*see Eating Behaviors; Volume 17, April 2015, Pages 115-118*] that "PTSD and depression are common conditions among Iraq/Afghanistan Veterans. In the present study, PTSD and depression symptoms were associated with meeting binge eating screening criteria, identifying a possible pathway by which psychiatric conditions lead to disproportionate burden of overweight and obesity in this Veteran cohort." Dorflinger & Masheb (2018), in the research article "PTSD is associated with emotional eating among veterans seeking treatment for overweight/obesity" [*see Eating Behaviors, Volume 31, December 2018, Pages 8-11*] presented findings that "suggest that emotional eating is common among veterans reporting PTSD symptoms, and that any degree of PTSD symptom severity is associated with more frequent emotional eating." Mental health difficulties can have a significant impact on activity levels and eating behaviors, influencing weight gain. For example, it is notable that major depressive disorder in the DSM-5-TR (*pg. 183*) lists diagnostic criteria such as "markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day…" and "significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day…" and "psychomotor agitation or retardation," and "fatigue or loss of energy nearly every day" and others. PTSD (*pg. 302*) includes criteria like "markedly diminished interest or participation in significant activities). Overeating and inactivity are symptoms of mental health difficulties which lead to weight gain and obesity. The Veteran's psychiatric difficulties not only directly impacted the Veteran's sleep apnea, but also indirectly by increasing the likelihood of weight gain. This is still a direct and proximate cause.

Mr. Doe's service connected PTSD is service connected with associated alcohol abuse (self-medicating PTSD symptoms with alcohol is common). The medical literature also demonstrates that alcohol use can make sleep apnea worse. For example, a recent meta-analysis on the topic reviewed 1,266 studies and concluded "alcohol consumption is associated with worsening severity of snoring, altered sleep architecture, AHI, as well as lowest oxygen saturation among patients susceptible to snoring and

obstructive sleep apnea" [*see Burgos-Sanchez C, Jones NN, Avillion M, Gibson SJ, Patel JA, Neighbors J, Zaghi S, Camacho M. Impact of Alcohol Consumption on Snoring and Sleep Apnea: A Systematic Review and Meta-analysis. Otolaryngol Head Neck Surg. 2020 Dec;163(6):1078-1086*]. The American Academy of Sleep Medicine (an organization I am a member of), has practice guidelines related to sleep apnea. The American Academy of Sleep Medicine's practice guidelines related to obstructive sleep apnea include, as a standard of care in the field, that there be "patient education" on the impact of "alcohol avoidance" on sleep apnea. The "avoidance of alcohol" is also included as one of the "behavioral strategies" in managing sleep apnea [*for the practice guidelines see Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine (2009), Epstein LJ; Kristo D; Strollo PJ; Friedman N; Malhotra A; Patil SP; Ramar K; Rogers R; Schwab RJ; Weaver EM; Weinstein MD. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. J Clin Sleep Med 2009;5(3):263–276*]. This reflects a recognition that the alcohol use which is tied to the Veteran's mental health difficulties can substantially aggravate the Veteran's sleep apnea directly (in addition to also leading to a history of excessive caloric intake in the form of alcohol).

The scientific literature supports a direct connection between sleep apnea and arousal associated with psychiatric disturbance, including PTSD. In addition, past VBA decisions have supported this interpretation. The scientific evidence and the Veteran's medical records support a finding that the evidence for sleep apnea being secondary to his PTSD is at least in equipoise. What is established as quite likely is a bidirectional relationship between his psychiatric disturbance and his sleep apnea where each substantially aggravates the other, and that his sleep apnea was initially directly and proximately caused by his psychiatric difficulties. There is research evidence supporting a direct cause for sleep apnea associated with the arousal related to psychiatric disturbance. In this regard, based on the mechanism of hyper-arousal associated with both his psychiatric disturbance and sleep apnea, it is at least as likely as not that his sleep apnea is secondary to his service-connected psychiatric disturbance. Countless VBA decisions have been decided favorably based on this scientific literature, including, but not limited to: *Citation Nr: 19133904 (Docket NO. 18-05 0350 5/1/2019; Citation Nr: 19164013 (Docket NO. 15-12 470) 8/19/19; Citation Nr: 19136285 (Docket NO. 18-01 222) 5/10/19; Citation Nr: 20011070 (Docket NO. 19-05 577) 2/10/2020; Citation Nr: 19155477 (Docket NO. 18-28 822) 7/18/19; Citation Nr: 19107744 (Docket NO. 16-51 926) 2/1/19; Citation Nr: 19103608 (Docket NO. 17-05 487) 1/15/19; Citation Nr. 19144930 (Docket NO. 17-49 916) 6/11/19; Citation Nr: 19141163 (DOCKET NO. 18-18 750A) 5/29/19; Citation Nr: 19110858 (Docket NO 16-53 449), 2/12/19*). This is not an exhaustive list. This interpretation of the scientific literature should be utilized in favor of the Veteran as it would be arbitrary and capricious for the VA to treat the science differently for one Veteran over another and say the science says one thing for one Veteran but says the opposite for another Veteran.

Sleep apnea conclusions: There is a link between Mr. Doe's sleep apnea and the mental health difficulties from his military service. It is at least as likely as not (a 50 percent chance or greater) that the Veteran's sleep apnea is due to the PTSD with alcohol abuse from his military service. His mental health concerns have created hyperarousal and other physiological issues including a risk for weight gain which in turn directly and proximately cause his sleep apnea while also subsequently substantially aggravating and worsening the course and severity of the disease. The Veteran's sleep apnea is at least as likely as not (a 50 percent chance or greater) substantially aggravated by his service-connected PTSD. His PTSD creates chronic sleep impairments which substantially impact the progression and treatment of his sleep apnea along with a risk for weight difficulties and treatment adherence concerns with PAP therapy which do the same. The issue is medically complex, as it requires knowledge of the interaction between multiple systems in the body. I am a psychologist with extensive training related to posttraumatic stress disorder and other psychiatric conditions; I am also trained in Behavioral Sleep Medicine and am a

member of the American Academy of Sleep Medicine and the Society of Behavioral Sleep Medicine. The research supports the presence of a significant connection between psychiatric difficulties and sleep apnea, including an arousal-based mechanism initiated by PTSD that promotes the development of sleep apnea in trauma survivors.

Rating the sleep apnea impairment: He requires the use of a breathing assistance device such as PAP therapy based on his treatment records. Sleep apnea leads to functional impairment and CPAP can help with this functional impairment, however, it is a misconception that CPAP/ PAP therapy is expected to resolve all impairments for an individual with sleep apnea; this is not consistent with the scientific evidence. In addition, the AHI is not a reliable measure for all of the functional impairment associated with sleep apnea. Some relevant information from the scientific literature includes (but is not limited to):

Obstructive sleep apnea (OSA) "impairs quality of life for numerous patients and leads to various OSA complications." The authors noted that "the role of serotonin (5-HT) in many physiological processes, studies on its connection with the circadian system, and relationship to changes in sleep architecture are insufficient to assess the interaction of this neurotransmitter with nocturnal hypoxia," but they did find that treatment with PAP therapy did lead to an increase in serotonin levels in individuals with sleep apnea [*Madaeva IM, Berdina ON, Kurashova NA, Semenova NV, Ukhinov EB, Belskikh AV, Kolesnikova LI. Sleep Apnea and Serum Serotonin Level Pre- and Post-PAP Therapy: A Preliminary Study. Neurol Ther. 2021 Dec;10(2):1095-1102. doi: 10.1007/s40120-021-00290-z. Epub 2021 Oct 20*].

We know that sleep apnea leads to "an increase in occupational accidents due to reduced vigilance and attention" in individuals with sleep apnea. "Such involvements were related to excessive daytime sleepiness and neurocognitive function impairments" [*see Rabelo Guimarães Mde L, Hermont AP. Sleep apnea and occupational accidents: Are oral appliances the solution? Indian J Occup Environ Med. 2014 May;18(2):39-47*]. We know that "cognitive impairments are commonly seen in patients with an OSA diagnosis" [*see Wang G, Goebel JR, Li C, Hallman HG, Gilford TM, Li W. Therapeutic effects of CPAP on cognitive impairments associated with OSA. J Neurol. 2020 Oct;267(10):2823-2828. doi: 10.1007/s00415-019-09381-2. Epub 2019 May 20. PMID: 31111204*]. "One of the major consequences of OSAS is an impact on neurocognitive functioning. Several studies have shown that OSAS has an adverse effect on inductive and deductive reasoning, attention, vigilance, learning, and memory" [*see Lal C, Strange C, Bachman D. Neurocognitive impairment in obstructive sleep apnea. Chest. 2012 Jun;141(6):1601-1610*].

Jackson, et. al. (2018) treated 110 patients with OSA with CPAP for three months and compared them to individuals in the community without OSA. "Compared to the community sample, participants with OSA were significantly sleepier, had impaired mood and quality of life, and showed decrements in neuropsychological function, specifically psychomotor function, working memory and vigilance. Some neuropsychological and mood outcomes were normalized with CPAP, but significant decrements persisted in most outcomes even in those participants with adequate device usage." The authors found that "Patients with mild to moderate OSA have significant neurobehavioral morbidity. During "gold standard" treatment, normal function was not achieved, even with adequate device usage. CPAP efficacy for improving sleepiness and neuropsychological function in this milder end of the OSA spectrum may be poor, which may affect CPAP adherence. These findings suggest that there may be neurological changes related to OSA that do not respond to CPAP treatment. [*see Jackson ML, McEvoy RD, Banks S, Barnes M. Neurobehavioral Impairment and CPAP Treatment Response in Mild-Moderate Obstructive Sleep Apneas. J Clin Sleep Med. 2018 Jan 15;14(1):47-56*].

Jiang, et, al. (2021) completed a metanalysis of multiple studies and found that sleep apnea is associated with "high risks of cognitive impairment, including Alzheimer's disease" and that CPAP only offers "partial" improvement in the cognitive problems caused by sleep apnea [*see Jiang X, Wang Z, Hu N, Yang Y, Xiong R, Fu Z. Cognition effectiveness of continuous positive airway pressure treatment in obstructive sleep apnea syndrome patients with cognitive impairment: a meta-analysis. Exp Brain Res. 2021 Dec;239(12):3537-3552*].

Kielb, et. al. (2012) also note that OSA is "associated with a number of adverse health consequences, and a growing literature focuses on its cognitive correlates." They note that "multiple studies indicate" that individuals with OSA "show impairment in attention, memory, and executive function." While CPAP was the "most effective and widely used treatment" for sleep apnea, the studies of CPAP use showed that "in general, no consistent effect of CPAP use on cognitive performance was evident." The authors noted also that "several prior reviews of the literature suggested that OSAS patients exhibit significant impairment on neuropsychological tests." They reviewed evidence that OSA patients are "are at increased risk for motor vehicle accidents." The reviewed multiple studies with mixed results and design. While some research shows some potential cognitive improvement with CPAP use, they noted that "in fact, several studies have reported that CPAP treatment has no effect on cognitive performance." The authors concluded that "cognitive deficits have long been observed in OSAS patients, and although findings in this domain remain inconsistent, evidence exists for deficits in intellectual function, memory, attention, and executive function in OSAS." Due to the mixed results of treatment studies, which may be due to variability in inclusion and exclusion criteria, study design, and duration of treatment, it is not possible to make definitive conclusions regarding the impact of CPAP treatment on cognition in OSAS. Although CPAP is a well-established, effective treatment for OSAS, it does not definitively reduce the host of cognitive deficits observed among OSAS patients. Daytime somnolence can particularly impact attention and executive functioning, including slower reaction time. Intermittent hypoxemia also can impact the performance on cognitive tests and is associated with declines in motor and processing speed, spatial abilities, mental flexibility and attention. "Some researchers argue that intermittent hypoxemia, together with sleep fragmentation, leads to prefrontal cortical degeneration, which could explain the impairment in executive function observed in patients" with OSA. Sleep fragmentation itself may be "an important mechanistic factor in the development of cognitive impairment" in sleep apnea. "For example, the number of arousals from sleep is a strong predictor of memory impairment in OSAS." Sleep fragmentation has been associated with poorer cognitive performance and reduction in neurogenesis. "Another potential mechanism that may contribute to cognitive impairment in OSAS is disruption in circadian rhythms. Researchers have reported an association between disturbed circadian rhythms (as measured by actigraphy) and severity of cognitive impairment" [*see Kielb SA, Ancoli-Israel S, Rebok GW, Spira AP. Cognition in obstructive sleep apnea-hypopnea syndrome (OSAS): current clinical knowledge and the impact of treatment. Neuromolecular Med. 2012 Sep;14(3):180-93. doi: 10.1007/s12017-012-8182-1. Epub 2012 May 9*].

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Dr. Todd Finnerty is a psychologist in private practice in Columbus, Ohio. He has significant training related to PTSD, including VA-specific training through the contractors VES and QTC. Dr. Finnerty has had the same amount of training or more training than the third-party contractors used by the VA. In the past Dr. Finnerty has performed hundreds of examinations on veterans for VA third-party contractors. Dr. Finnerty also helps make decisions on Social Security disability claims for the state of Ohio and has substantial experience in evaluating impairment. In this role Dr. Finnerty was named the 2012 "Disability Review Physician of the Year" by the National Association of Disability Examiners, Great Lakes Region and the 2010 "Consultant of the Year" by the Ohio Association of Disability Examiners. He has training in behavioral sleep medicine and is a member of the American Academy of Sleep Medicine and the Society of Behavioral Sleep Medicine. Dr. Finnerty is a forensic specialist and adheres to the American Psychological Association's Ethical Principles of Psychologists and Code of Conduct as well as the APA's Specialty Guidelines for Forensic Psychology (https://www.apa.org/practice/guidelines/forensic-psychology). These guidelines include the responsibilities of integrity, impartiality and fairness and note: "When offering expert opinions to be relied upon by a decision maker, providing forensic therapeutic services, or teaching or conducting research, forensic practitioners strive for accuracy, impartiality, fairness, and independence. Forensic practitioners recognize the adversarial nature of the legal system and strive to treat all participants and weigh all data, opinions, and rival hypotheses impartially. When conducting forensic examinations, forensic practitioners strive to be unbiased and impartial, and avoid partisan presentation of unrepresentative, incomplete, or inaccurate evidence that might mislead finders of fact. This guideline does not preclude forceful presentation of the data and reasoning upon which a conclusion or professional product is based." While it would be convenient if Veterans in need of first or second opinions on mental health related claims could seek them from treatment providers at the VA, VA policy outlined in VHA Directive 1134(2) Provision of Medical Statements and Completion of Forms by VA Health Care Providers recommends that VA mental health treatment providers not complete forms such as "mental health DBQ's" in order to "maintain the integrity of the patient-provider relationship." Therefore, both the VA and Veterans often must seek forensic specialists outside of a treatment relationship to provide opinions related to their case.