**Fear and the DWI Field Sobriety Tests**

By Mimi Coffey

The National Highway Traffic Safety Administration (NHTSA) developed the Standardized Field Sobriety Tests (SFSTs) in a vacuum. None of the original research simulated real life testing conditions; e.g., a person performing the standardized field sobriety tests with the fear of going to jail. This missing premise negates the validity of the tests as far as the “divided attention tests.” The critical flaw of the SFSTs contributes to false convictions in the nationalized DWI testing protocol.

What is fear? Fear as a scientific term describes a behavioral, cognitive-emotional condition in which a set of biological adaptive responses activate in the presence of danger.[[1]](#endnote-1) These physiological responses are hardwired to how our brain operates.[[2]](#endnote-2) Fear is uncontrollably present in humans and animals when unpredictable, aversive events cause debilitating behavioral, cognitive and somatic effects.[[3]](#endnote-3) Fear is controlled by ancient systems in the brain, primarily the amygdala, which acts relatively independent of later emerging higher cognitions.[[4]](#endnote-4) Numerous studies have linked the amygdala with fear.[[5]](#endnote-5) Much of fear’s effects on the amygdala are subconscious with sensory information accessing the amygdala with minimal cortical processing.[[6]](#endnote-6) It is important to note that fear is not related to intelligence or its effects negated by alcohol. Alcohol reduces *anxiety* but not fear.[[7]](#endnote-7) Extreme experiments prove the symbiotic relationship of fear to the amygdala. In one, amygdala-lesioned rats approached a sedated cat, crawling over it and even nibbling on its ear.[[8]](#endnote-8) In another, Rhesus monkeys had their amygdalae removed through a bilateral temporal lobectomy resulting in no innate fear of snakes, such as avoidance or freezing—a condition is known as ‘psychic blindness’.[[9]](#endnote-9) The function of fear is to motivate organisms to manage threats that jeopardize survival through the use of coping reactions clearly focused on escaping, attacking, and freezing.[[10]](#endnote-10) Fear potentiated startle (FPS), a variant on freezing, is an instinctive response to a combination of light and noise stimuli.[[11]](#endnote-11) A person’s fear of police, at its basic element, is similar to that of rats to cats. In an experiment done by psychologists Blanchard and Blanchard, rats were exposed for 15 minutes to cats causing each to scatter into burrows and avoid open areas, in addition to curtailing non-defensive behaviors such as grooming, mounting, eating, and drinking, for the duration of the test.[[12]](#endnote-12) The ability to focus one’s attention while in fear requires first an understanding of the complex reactions occurring involuntarily in the body and beyond one’s control.

Fear activates stress. Stress hormones will dramatically alter the turnover of several classes of neurotransmitters in the prefrontal cortex of the brain.[[13]](#endnote-13) The prefrontal cortex has extensive projections from the limbic system, the mammalian part of the brain involved in emotion, which explains why strong emotions can adversely impact the quality of executive functions increasing the likelihood of imprudent or impulsive choices.[[14]](#endnote-14) A study of threatening and neutral images established that the orbitofrontal cortex, as part of the fronto-parietal network has a key role in spatial attention; attention as well as emotion are two key components compromised in fear. [[15]](#endnote-15) In particular, stress releases damaging hormones. Stress activates the hypothalamic-pituitary adrenal (HPA) axis which releases the glucocorticoids (cortisol) by the adrenal cortex and negatively interferes with brain structures central to memory.[[16]](#endnote-16) Corticotrophin (CRH) released with physical and emotional stressors, causes increased blood glucose, heart rate and blood pressure, in addition to increased tolerance of pain and *changes in motor activity*.[[17]](#endnote-17) Along with these physical reactions, an increase in glucocorticoid secretion is associated with immobilization or freezing.[[18]](#endnote-18) Advances in the understanding of neuroendocrine and neurochemical behavioral responses have come a long way since 1911 when Walter Cannon confirmed the secretion of epinephrine after an emotional response with fight-or-flight.[[19]](#endnote-19) Recently it was shown that norepinephrine and dopamine, also interactively involved in stress induced arousal, have effects on one’s prefrontal cognition as well.[[20]](#endnote-20) The problem with the body’s natural reactions as a result of fear or stress is the deficits which result in the “loss of normal mental and physical faculties” judged for intoxication in a DWI. It is a recognized fact that when the basoleteral part of the amygdala is activated (such as in fear) and glucocorticoids are released, stress-induced retrieval deficits occur.[[21]](#endnote-21) Retrieval deficits result in a negative effect on memory.[[22]](#endnote-22) The compromised memory retrieval is due to the stress hormones and neurotransmitters switching the brain into a “memory consolidation” state allowing for strong recall of the event, thereby compromising memory retrieval during the event.[[23]](#endnote-23) This makes sense as being able to recall and avoid threatening events is necessary for evolutionary survival, explaining why our brains’ circuitry is programmed in this manner. Memory retrieval is critical in a DWI investigation for tasks such as NHTSA Phase II (e.g., alphabetic and numeric countdown) exercises. Such exercises are often times requested before exiting the vehicle so that the officer may test short-term memory recall prior to testing the subject’s ability to memorize the instructions for the walk and turn and one leg stand tests. Participants in a study receiving cold pressor stress before memory retrieval recalled fewer words than the no stress control.[[24]](#endnote-24) In another stress study done in 2009, a glucocorticoid (cortisol) administered to subjects impaired their memory during a weeklong word recall experiment.[[25]](#endnote-25)

Memory is directly connected with the brain’s processing of emotion and attention. Emotion and attention can interact either by emotion modulating attentional processing or attention influencing emotional processing.[[26]](#endnote-26) In a DWI scenario, the emotion of fear precedes and interrupts task attention. Emotion functions importantly to help guide attention to emotionally valuable stimuli,[[27]](#endnote-27) none of which is beneficial in a DWI investigation. Emotions result in abnormal excitation of the nervous network, which induces changes in heart rate and secretions or interrupts the normal relationship between the peripheral nervous system and the brain.[[28]](#endnote-28) Simply put, emotions affect the central nervous system—the same system evaluated for ‘normalcy’ in a DWI. First, emotional information receives priority in neural processing.[[29]](#endnote-29) Evidence suggests that once emotional stimuli are processed visual attention is often sustained.[[30]](#endnote-30) One’s inability to ‘think straight’ while mad or ‘black out’ in fear is due to emotional information receiving privileged access to attention and awareness.[[31]](#endnote-31) To be ‘paralyzed by fear’ is due to emotions exerting their influence by modulating activity in regions involved in cognitive control.[[32]](#endnote-32) Specifically, emotional signals modulate parietal and frontal brain regions involved in attention control.[[33]](#endnote-33) This loss of attention control impedes the processing of task-relevant stimuli.[[34]](#endnote-34) When the field sobriety tests were developed, the testing subjects had no emotional detractors to interfere with their ability to process the directions. One cannot perform the tests correctly without adhering to the proper instructions such as ‘keep your arms by your side’ or ‘on the 9th step keep your lead foot planted and take a small series of steps’. Emotions are so powerful, evidence suggests, that outside of ‘top down’ mental processing, some automatic “preattentive” neural processing of emotional stimuli exists as well,[[35]](#endnote-35) (Ohman proved this in a study that had snakes hidden in pictures).[[36]](#endnote-36) Psychology literature reports several published studies that document the slowed brain processing when emotions are involved. One asked subjects to simply identify a target as a circle or square and found that when the shapes were preceded by emotional versus neutral images this slowed down the identification process.[[37]](#endnote-37) Another proved that fear conditioned cues captured subjects’ attention making it harder and slower to orient one’s self to proper locations in a spatial attention/emotion study.[[38]](#endnote-38) In another, participants found it more difficult to name colors of pictures or words when they had an emotional meaning attached to them, further proving the emotional hindrance is involuntary.[[39]](#endnote-39)

Emotion potentiates the effect of attention.[[40]](#endnote-40) Attention defined is the cognitive control involved in maintaining task rules in working memory, monitoring reward and error rates, filtering distracters, and suppressing prepotent and competitive responses.[[41]](#endnote-41) Weak attentional control increases distractibility causing attentional lapses, impulsivity and attentional fatigue.[[42]](#endnote-42) When a person loses the ability to orient attention towards relevant stimuli, sensory information can no longer be processed properly.[[43]](#endnote-43) This processing occurs in the prefrontal cortex,[[44]](#endnote-44) the same place shown to receive activation from threat-related signals.[[45]](#endnote-45) The reason why one cannot focus attention during fear is because the amygdala activates the noradrenergic system projecting into and interfering with the locus coeruleus which modulates attention.[[46]](#endnote-46) Without the effects of fear factored into the equation, DWI investigations attempt to prevent one from driving a vehicle when their central nervous system is impaired to the extent defined by the law for intoxication.[[47]](#endnote-47) There are two central flaws to this end result. Dr. Marcelline Burns, developer of the SFSTs, has admitted that the tests were never designed to detect impairment and one’s ability to safely operate a motor vehicle.[[48]](#endnote-48) She attempts to bridge this fatal blow by advocating her second central flaw that the field tests are designed at judging one’s ability to divide their attention.[[49]](#endnote-49) Does driving a car involve the divided attention used as an objective in the field sobriety tests? Driving often consists of monitoring external stimuli for certain classes of events (pedestrians, looming cars, etc.) with the driving and thinking representing a case of ‘simultaneous performance’.[[50]](#endnote-50) There are specifically two neural conclusions about driving: (1) perceptual monitoring occurs at the same time as central processing and (2) central processing can be interrupted quickly on the basis of detections made while scanning and monitoring the environment.[[51]](#endnote-51) Field sobriety tests under fear conditions do not fairly represent simultaneous perception monitoring and central processing. When it comes to divided attention tests many tasks interfere with each other quite drastically, although they are neither intellectually challenging nor physically incompatible.[[52]](#endnote-52) One comprehensive analysis on the topic, published in the scholarly Psychological Bulletin referred to by specialists in the field of psychology, determined “the results show that people have surprisingly severe limitations on their ability to carry out simultaneously certain cognitive processes that seem fairly trivial from a computational standpoint”.[[53]](#endnote-53) Basic science counterbalances experiments with controls. If the control studies show unsuitability for divided attention without alcohol, much less negated by the presence of fear, it is time the National Highway Traffic Safety Administration be held accountable for substandard and ineffective protocol and testing measures.

Attention at its core is simply holding information in the working memory, necessitating a basic understanding of working memory as it relates to fear. [[54]](#endnote-54) As mentioned above, memory retrieval processes are impaired with a high level of circulating glucocorticoids[[55]](#endnote-55) which occurs in concert with changes in other neurotransmitter systems.[[56]](#endnote-56) These neural systems extend from the frontal lobes into the primary cortices where attention and working memory show considerable overlap.[[57]](#endnote-57) To paraphrase, in normal cognition, memory consolidation and retrieval processes occur simultaneously; thus, a single glucocorticoid rush can alter these reactions.[[58]](#endnote-58) Specifically, stress levels of glucocorticoids influence the prefrontal cortex impairing short term memory retrieval.[[59]](#endnote-59) In one study, rats experienced foot-shock exposure for thirty minutes, resulting in a temporary memory loss.[[60]](#endnote-60) In a human study, subjects tested after glucocorticoid elevations showed the same impaired memory retention under various testing conditions,[[61]](#endnote-61) including the recall of previously learned words.[[62]](#endnote-62) The bottom line, emotionally distracting scenarios are associated with a decrease in dorsal lateral prefrontal cortex activity along with a concomitant drop in working memory performance.[[63]](#endnote-63)

Although man is the most highly developed species, we are still animals largely controlled by instincts, one of the greatest of which is fear. The processing of fear-relevant stimuli was evolutionarily optimized for survival.[[64]](#endnote-64) Our fear responses are innate, species-typical responses that are not learned or voluntary.[[65]](#endnote-65) These responses kick into full gear when we see the flashing lights behind us, hear the sirens and are approached by men and women in uniform carrying guns. Known as tonic immobility, profound temporary motor inhibitions both physically and mentally occur when we perceive ourselves to be in these constraining and dangerous situations.[[66]](#endnote-66) To disregard these natural reactions, particularly in the scenario of a DWI investigation where testing is conducted, is to ignore science which short-circuits, truth, and justice. The social sciences have recognized that people who are depressed have attentional inflexibility which is linked to impairment in cognitive control mechanisms.[[67]](#endnote-67) People with post traumatic stress disorder (PTSD) in the same light, fail to maintain and direct proper attention when faced with threatening information.[[68]](#endnote-68) Modern science has recognized that with advances in brain research, we can pinpoint some of the mechanisms at work that cause a loss of normal mental and physical faculties in emotional states. Whereas doctors have cognitive goals for their patients in these emotional states, lawyers and judges must learn to differentiate such states to prevent false convictions, particularly in the area of intoxication related offenses. There are enormous intellectual differences between the worlds of science and law in the basic premises concerning causality and certainty.[[69]](#endnote-69) It is time to close the gap.

1. J. J. Kim & K.M. Myers, *Fear: Psychological and Neural Aspects*, 8 International Encyclopedia of the Social & Behavioral Sciences 5428, 5428 (2001). [↑](#endnote-ref-1)
2. *See*  *Id*. at 5430. [↑](#endnote-ref-2)
3. Ruben P. Alvarez et al., *Phasic and Sustained Fear in Humans Elicits Distinct Patterns of Brain Activity*, 55 NeuroImage 389, 389 (2011). [↑](#endnote-ref-3)
4. Arne Ohman, *The Role of the Amygdala in Human Fear: Automatic Detection of Threat*, 30 Psychoneuroendocrinology 953, 954 (2005). [↑](#endnote-ref-4)
5. *See generally* Steve R. Makkar et al.*, Review: Behavioral and Neural Analysis of GABA in the Acquisition, Consolidation, Reconsolidation, and Extinction of Fear Memory*, 35 Neuropsychopharmacology 1625, 1632 (2010); Joseph LeDoux, *The Emotional Brain, Fear, and the Amygdala*, 23 Cellular and Molecular Neurobiology 727, 727 (2003); Patrik Vuilleumier, *How Brains Beware: Neural Mechanisms of Emotional Attention*, 9 Trends in Cognitive Sciences 585, 588 (2005); Ralph Adolphs, *What Does the Amygdala Contribute to Social Cognition?*, 1191 The Annals of the New York Academy of Sciences 42, 42 (2010). [↑](#endnote-ref-5)
6. *See* Ohman, *Role*, *supra* note 4 at 954; Ralph Adolphs, *Fear, Faces, and the Human Amygdala*, 18 Current Opinion in Neurobiology 166, 167 (2008). [↑](#endnote-ref-6)
7. *See*  Christine A. Moberg & John J. Curtin, *Alcohol Selectively Reduces Anxiety but not Fear: Startle Response During Unpredictable vs. Predictable Threat*, 118 Journal of Abnormal Psychology 335, 345 (2009). [↑](#endnote-ref-7)
8. Rene Misslin, *The Defense System of Fear: Behavior and Neurocircuitry*, 33 Neurophysiologie Clinique 55, 61 (2003). [↑](#endnote-ref-8)
9. *Id­*. [↑](#endnote-ref-9)
10. Arne Ohman et al., *On the Unconscious Subcortical Origin of Human Fear*, 92 Physiology & Behavior 180, 180 (2007) (freezing “is a behaviorally quiescent state that involves scanning of the environment to assess risks and opportunities”). [↑](#endnote-ref-10)
11. *See* Amanda R. de Oliveira et al., *Conditioned Fear Response is Modulated by a Combined Action of the Hypothalamic-Pituitary-Adrenal Axis and Dopamine Activity in the Basolateral Amygdala*, European Neuropsychopharmacology (forthcoming). [↑](#endnote-ref-11)
12. Misslin, *supra* note 8 at 58. [↑](#endnote-ref-12)
13. *See* Robert M. Sapolsky, *The Frontal Cortex and the Criminal Justice Syste*m, 359 Philosophical Transactions of the Royal Society B 1787, 1792 (2004). [↑](#endnote-ref-13)
14. *Id*. [↑](#endnote-ref-14)
15. *See* Jorge L. Armony & Raymond J. Dolan, *Modulation of Spatial Attention by Fear-Conditioned Stimuli: An Event-Related fMRI Study*, 40 Neuropsychologia 817, 824 (2002). [↑](#endnote-ref-15)
16. Tom Smeets, *Acute Stress Impairs Memory Retrieval Independent of Time of Day*, 36 Psychoneuroendoctrinology 495, 495 (2011). [↑](#endnote-ref-16)
17. *See* T.M. O’Connor et al., *The Stress Response and the Hypothalamic-Pituitary-Adrenal Axis: From Molecule to Melancholia*, 93 Quarterly Journal of Medicine 323, 328 (2000). [↑](#endnote-ref-17)
18. Thierry Steimer, *The Biology of Fear-and Anxiety-Related Behaviors*, 4 Dialogues in Clinical Neuroscience 231, 234 (2002). [↑](#endnote-ref-18)
19. *See* R. McCarty, *Fight-or-Flight Response*, in 2 Encyclopedia of Stress 62, 62 (2d ed. 2007). [↑](#endnote-ref-19)
20. *See* Anreas Boehringer et al., *A Combination of High Stress-Induced Tense and Energetic Arousal Compensates for Imparing Effects of Stress on Memory Retrieval in Men*, 13 Stress 444, 451 (2010). [↑](#endnote-ref-20)
21. *See* Smeets, *supra* note 16 at 500. [↑](#endnote-ref-21)
22. *See* Marie-France Marin et al., *Modulatory Effects of Stress on Reactivated Emotional Memories*, 35 Psychoneuroendorinology 1388, 1388 (2010). [↑](#endnote-ref-22)
23. Benno Roozendaal*, Stress and Memory: Opposing Effects of Glucocorticoids on Memory Consolidation and Memory Retrieval*, 78 Neurobiology of Learning and Memory 578, 590 (2002). [↑](#endnote-ref-23)
24. *See* Smeets, *supra* note 16 at 495. [↑](#endnote-ref-24)
25. *See* Marin, *supra* note 22 at 1389. [↑](#endnote-ref-25)
26. Armony & Dolan, *supra* note 15 at 817. [↑](#endnote-ref-26)
27. *See* N. Fragopanagos & J.G. Taylor, *Modelling the Interaction of Attention and Emotion*, 69 Neurocomputing 1977, 1982 (2006). [↑](#endnote-ref-27)
28. *See* Steimer, *supra* note 18 at 231. [↑](#endnote-ref-28)
29. *See* Marie T. Banich et al., *Cognitive Control Mechanisms, Emotion & Memory: A Neural Perspective with Implications for Psychopathology*, 33 Neuroscience & Biobehavioral Reviews 613, 614 (2009). [↑](#endnote-ref-29)
30. *See* Greg Hajcak et al., *The Dynamic Allocation of Attention to Emotion: Simultaneous and Independent Evidence from the Late Positive Potential and Steady State Visual Evoked Potentials*, Biological Psychology (2011), *available at* http://www.sciencedirect.com/science/article/pii/S0301051111002973. [↑](#endnote-ref-30)
31. *See* Vuilleumier, *How Brains Beware*, *supra* note 5 at *5*86. [↑](#endnote-ref-31)
32. *See* Banich et al., *supra* note 29 at 620. [↑](#endnote-ref-32)
33. Vuilleumier, *How Brains Beware*, *supra* note 5 at 591. [↑](#endnote-ref-33)
34. *See* Hajcak et al., *supra* note 30. [↑](#endnote-ref-34)
35. Patrik Vuilleumier et al., *Effects of Attention and Emotion on Face Processing in the Human Brain: An Event-Related fMRI Study*, 30 Neuron 829, 829 (2001). [↑](#endnote-ref-35)
36. *See* Ohman et al., *Unconscious,* *supra* note 10 at 182. [↑](#endnote-ref-36)
37. *See* Hajcak et al., *supra* note 30. [↑](#endnote-ref-37)
38. *See* John G. Taylor & Nickolaos F. Fragopanagos, *The Interaction of Attention and Emotion*, 18 Neural Networks 353, 358 (2005). [↑](#endnote-ref-38)
39. *See* Vuilleumier, *How Brains Beware*, *supra* note 5 at 585. [↑](#endnote-ref-39)
40. Ohman et al., *Unconscious*, *supra* note 10 at 183. [↑](#endnote-ref-40)
41. Martin Sarter & Giovanna Paolone, *Theoretical Review-Deficits in Attentional Control: Cholinergic Mechanisms and Circuitry-Based Treatment Approach*, 125 Behavioral Neuroscience 825, 825 (2011). [↑](#endnote-ref-41)
42. *Id*. [↑](#endnote-ref-42)
43. *See* Christina Lucas & Johan Lauwereyns, *Selective Working Memory Disables Inhibition of Visual Features*, 54 Experimental Psychology 256, 256 (2007). [↑](#endnote-ref-43)
44. *See* Taylor & Fragopanagos, *supra* note 38 at 353. [↑](#endnote-ref-44)
45. *See* Belinda J. Liddell et al., *A Direct Brainstem-Amygdala-Cortical ‘Alarm’ System for Subliminal Signals of Fear*, 24 NeuroImage 235, 240 (2005). [↑](#endnote-ref-45)
46. Vuilleumier, *How Brains Beware*, *supra* note 5 at 592. [↑](#endnote-ref-46)
47. *See* Tex. Penal Code § 49.01 (“not having the normal use of mental or physical faculties by reason of the introduction of alcohol, a controlled substance, a drug, a combination of two or more of those substance, or any other substance into the body; or having an alcohol concentration of 0.08 or more.”). [↑](#endnote-ref-47)
48. *See* Lori Raye Court Reporters, Examination under Oath of Marcelline Burns, 1, 39-40 (April 17, 1998). [↑](#endnote-ref-48)
49. *See* Jack Stuster & Marcelline Burns, *Validation of the Standardized Field Sobriety Test Battery at BACs Below 0.10 Percent*, (Contract No. DTNH22-95-C-05192) U.S Department of Transportation and National Highway Traffic Safety Administration, 1, 32 (August 1998). [↑](#endnote-ref-49)
50. *See* Harold Pashler, *Dual-Task Interference in Simple Tasks: Data and Theory*, 116 Psychological Bulletin 220, 238 (1994). [↑](#endnote-ref-50)
51. *See* *Id*. [↑](#endnote-ref-51)
52. *See Id* at 220. [↑](#endnote-ref-52)
53. *Id* at 241. [↑](#endnote-ref-53)
54. *See* Lucas & Lauwereyns, *supra* note 43 at 262. [↑](#endnote-ref-54)
55. *See* Roozendaal*,* supra note 23 at 591. [↑](#endnote-ref-55)
56. *See Id* at 588. [↑](#endnote-ref-56)
57. *See* Lucas & Lauwereyns, *supra* note 43 at 256. [↑](#endnote-ref-57)
58. *See* Roozendaal*, supra* note 23 at 588. [↑](#endnote-ref-58)
59. *See Id* at 587. [↑](#endnote-ref-59)
60. *Id* at 585-86 (Rats in the study were trained in a water maze to find a platform located in a specific location. Immediately after exposure to foot-shock, the rats were unable to readily identify the location of the platform previously known to each rat tested. However, after a short time elapsed following the foot-shock, rats were able to easily find the platform location again.). [↑](#endnote-ref-60)
61. *See Id* at 585. [↑](#endnote-ref-61)
62. *See Id* at 587. [↑](#endnote-ref-62)
63. Banich et al., *supra* note 29 at 618. [↑](#endnote-ref-63)
64. *See* Taylor & Fragopanagos, *supra* note 38 at 356. [↑](#endnote-ref-64)
65. *See* LeDoux, *supra* note 5 at 728. [↑](#endnote-ref-65)
66. Misslin, *supra*, at 59. [↑](#endnote-ref-66)
67. *See* Banich et al., *supra* note 29 at 620. [↑](#endnote-ref-67)
68. *See Id*. at 624. [↑](#endnote-ref-68)
69. *See* Sapolsky, *supra* note 13 at 1789. [↑](#endnote-ref-69)