

Part I: The Physiology of Stress: Auditory and Visual

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INTRODUCTION

The tactical world is by definition a high stress environment. In his book “On Combat”, Lieutenant Colonel David Grossman notes that the greatest fear of most human beings is aggression from other human beings. Whether it’s force-on-force simulation training or real combat, the tactical environment is defined by human aggression, aimed either at innocent civilians or at the tactical operators themselves. Add to this that the tactical operator is part of a close-knit team, and that he or she is under a high degree of pressure to perform as part of the team. And it only gets worse: the physical environment itself piles on other unique stressors including temperature extremes, low-light conditions, complex and cluttered surroundings, hostile animals, insects, and booby traps, and even the potential of biological, chemical, or radiological hazards. Finally, and particularly in the LE world, there is the ever-present fear of being sued or sanctioned if you do something wrong. Or even if you don’t.

Understanding how the human body responds to stress – the “physiology of stress” – is critical to performing under the barrage of hostilities that make up the tactical world. Some of these stressors are external, and some are internal. We’ve all seen two people react very differently to the same external stress. The difference can only be accounted for by the internal processing between the two. This lecture provides a framework for understanding this “internal processing” of stress so that the operator can train in such a way to mitigate some of the bad effects, and even take advantage of some of the good effects, of the physiologic stress response.

Looked at from the point of view of a physiologist, the tactical operator is a highly trained and conditioned athlete who is placed into a rapidly changing force environment. What happens during a room entry or an active shooter scenario or a security detail encounter is really the sum total of a series of feedback loops that play out within his nervous system. The operator perceives threats or changes in his environment, and then responds to that input according to his conditioning, to his departments’ SOP’s, and to his teammates’ actions. At first glance, this all looks like a video game. There is an important difference, though: the gamer is not afraid of being killed or crippled for life, of being sued or fired, or of having innocent blood on his hands if he fails to perform. All of these stressors insure that what happens to the vision, hearing, heart rate, blood pressure, fine motor coordination, and mental operations is very different between a SWAT officer operating in the worst building in the worst part of town on the one

hand, and a teenager sitting in front of a video screen with a Red Bull and a joystick on the other hand.

The first of these two lectures is on the physiology of stress, and the second is on performance enhancement. Both will borrow heavily from what we know about the science of autonomic physiology, but also refer as often as possible to real world threats and responses. The first lecture will deal with three subjects: 1) the overall stress response; or how the body's autonomic nervous system reacts to stress in general; and then, in detail; 2) how the operator's hearing may be affected by stress ("auditory exclusion"); and 3) how the operator's vision may be affected by stress ("tunnel vision" and/or loss of visual acuity).

Part I: THE STRESS RESPONSE and THE AUTONOMIC NERVOUS SYSTEM

The nervous system can be divided into somatic ("voluntary") and autonomic (involuntary, or "automatic") divisions. The autonomic nervous system (ANS) is, in turn, divided into sympathetic ("fight or flight") and parasympathetic ("rest and digest") systems. The detailed anatomy of the two systems isn't important to the operator, but the physiology is. Physiology is simply the study of *function*.

As a general rule, whatever the sympathetic nervous system does, the parasympathetic system does exactly the opposite. For example, high sympathetic activity increases heart rate, blood pressure, cardiac output, and respiration, and it also dilates the pupils. Conversely, parasympathetic activity decreases heart rate, blood pressure, cardiac output, respiration, and it constricts the pupils.

The designation of "fight or flight" as the goal of the sympathetic nervous system is actually quite accurate: in a life-or-death scenario where the organism has to either overcome or flee from danger, it makes sense that cardiac output will need to be high in order to increase oxygen delivery to the skeletal muscles, brain, and heart. This is done at the expense of blood flow to less-essential organs like the skin and gut. It also makes sense that the pupils will dilate in order to allow greater retinal image quality and greater peripheral vision. Similarly, it makes sense that glucose will be mobilized into the blood from the body's carbohydrate stores and made immediately available to the brain, heart, and muscles.

In the same way, the designation of "rest and digest" is an apt description of the goal of the parasympathetic nervous system. When there is no danger at hand, and when the hunt is over, it makes more sense to channel the body's resources and energy into digesting the recently captured prey, and to conserve movement and energy while replenishing energy stores.

The body is more complicated than this description, alone, however. Many physiologic functions require constant input from both sympathetic and parasympathetic activity. For example, even the seemingly simple act of urinating requires a precisely timed sequence of

sympathetic and parasympathetic nerve traffic to the bladder and to the urinary sphincters and other muscles surrounding it. When this symphony of nerve traffic doesn't work precisely, the result is either incontinence or urinary retention. You can get a sense of how finely tuned this simple act is by witnessing how difficult it can be to pee into a cup when you're nervous and your conscious nervous system is interfering with the ANS.

It's also important to realize that extreme stress can activate *both* sympathetic *and* parasympathetic tone – although under stress the majority will be sympathetic. One widely described but not often discussed ramification of parasympathetic tone under stress is urinary and fecal incontinence. (Take a look at the anatomy slide. The rectum, bladder, and their sphincters are heavily invested with parasympathetic nerves). This reflexive action happens more than any individual combatant will likely admit to, but it is entirely involuntary, and has very little to do with conditioning, courage, or even experience. The most experienced operator I know, who has been in hundreds of life-and-death encounters, routinely stops eating the day before and empties his bowels before any planned contact. He does this because he knows that battle incontinence is real, and that it cannot be easily prevented or controlled even by the toughest minds and bodies. "Autonomic" means just that: you can rarely fight it.

The sympathetic nervous system is distributed widely throughout the body, but I'll focus on the adrenal glands, since they release most of the hormones that make up the stress response. It's important to know that the sympathetic nervous system achieves its effects by two separate mechanisms: 1) neural; and 2) humoral ("hormonal"). The sympathetic nerves innervate the heart, part of the lungs, the blood vessels, the sweat glands, and many other end-organs. At the point of contact with these targets, the sympathetic nerves release the neurotransmitter norepinephrine ("NE"). NE traverses a very small gap before binding to receptors on the target organ. In the case of the adrenal gland, the sympathetic nerves that converge on its interior release both NE and epinephrine ("adrenaline") into the blood stream. Essentially, the same chemical (like NE) is called a "neurotransmitter" when it's released into a small gap between nerve and target organ, and it's called a "hormone" when it's released into the blood stream and has to travel a longer distance before reaching its target organ. It is the same chemical, and it has the same effect, though, regardless of what it's called or how distant it is from its target when released.

Back to the adrenal glands. Their name means, literally, "on top of the renals (kidneys)". The adrenals are a pair of triangular glands that sit above each kidney. They are divided into an outer area called the "cortex" that secretes steroid hormones like cortisol and aldosterone, and an inner area called the "medulla" that secretes epinephrine ("adrenaline") and norepinephrine ("noradrenaline"). In fact, the adrenal medulla is essentially a very large sympathetic nerve ending, or ganglion, that empties its neurotransmitters directly into the blood stream.

When we use the term adrenal "stress response", we're really referring to the adrenal medulla, and not to the cortex. It's important to know, however, that the cortex also gets activated under stress and dumps cortisol into the blood. As a general rule, cortisol does you no good under acute stress. It tends to undo much of what the sympathetic nervous system is trying to

do as part of its “fight or flight” response. For example, cortisol de-mobilizes glucose and other energy stores, it impairs immunity, and it can even cause mental confusion at high doses. Blood cortisol levels are used as a marker in human and animal studies for chronic stress. As one example, scientists who study baboon colonies have found that the alpha male has greater chronic stress than the beta (second-in-command) male, based on chronically higher levels of cortisol. Although the actions of cortisol are many and complex, it is fair to think of it as an unintended byproduct of the adrenal stress response.

The other important steroid hormone secreted by the adrenal cortex is aldosterone. In contrast to cortisol, aldosterone has a more obvious beneficial effect under acute stress: it causes the kidneys to retain salt and water; and therefore, is partially protective in the face of hemorrhage and loss of blood volume.

The remainder of this discussion of the adrenal glands will deal only with the adrenal medulla, and its hormones: epinephrine and norepinephrine. These are the real “fight or flight” hormones, and they accomplish the sympathetic responses listed at the beginning of this section: increased heart rate and contractility, increased blood pressure, increased cardiac output and delivery of nutrients and oxygen to the skeletal muscles, increased respiration, mobilization of glucose into the blood from carbohydrate stores, pupil dilation, and central nervous system arousal.

At the same time that the adrenal medullas are excreting epinephrine and norepinephrine into the blood stream, other sympathetic nerves that directly innervate target organs are firing at an increased rate. This dual combination of neural and humoral (hormonal) sympathetic activation accomplishes the stress response.

One dramatic example of how fast, and how potent, the autonomic system is may be seen from the diving reflex. Here, a parasympathetic response is triggered from immersing the face into ice water. [Don't do this at home!] Within seconds of immersion, the heart comes to complete standstill. In the slides presented in class, the subject is fully conscious. This is because it takes some time before cardiac arrest causes loss of consciousness since the brain has several minutes of oxygen and glucose stores. In spite of being able to hear his own heart rate grind to a halt (his ears are above the surface and the heart rate monitor is making an audible sound with each heart beat before his cardiac arrest), and in spite of the sympathetic activation from the pain and stress of this exercise, his parasympathetically-mediated cold water diving reflex overwhelms the increased sympathetic tone from whatever stress response he has from pain, and his heart stops. This is a very reproducible reflex; and, in fact, has been used to treat dangerously rapid heart rates. The diving reflex has adaptive values to sea mammals, where it doesn't stop the heart, but instead just slows it down to preserve oxygen, and diverts blood flow from non-essential organs like the gut and the skin to deliver it preferentially to the heart, brain, and skeletal muscle. The human diving reflex is stronger, more dramatic, and less adaptive to the stresses that we face. Nonetheless, it demonstrates how powerful the ANS is, and how it can overwhelm conscious control.

Another important point about the adrenal stress response is how to measure it. As the diving reflex demonstrates, heart rate alone is not always a reliable indicator. Better yet is to actually measure circulating epinephrine. This can be a tricky business, though: epinephrine is degraded in the blood soon after it is released, so it must be collected and measured quite rapidly – almost in real time. That’s why you don’t hear about many studies in the tactical world that report circulating epinephrine levels in operators. The Moss study presented in the next slide is one of the few that demonstrates actual epinephrine levels in humans during stress. These samples were obtained from volunteer anesthesiology residents who agreed to have a central venous line placed in their jugular vein before delivering a public lecture. The fear of public speaking can be far worse for some people than even the fear of human aggression. At it’s peak, and in the subject who showed the most dramatic elevation of epinephrine, the blood concentration was the equivalent to giving 2 micrograms of epinephrine IV. Remember this level, because it becomes relevant when we examine the claims that “auditory exclusion” is the result of circulating epinephrine closing down blood supply to the inner ear.

Can the overall stress response be modified through training? “Stress inoculation” exposes trainees to repetitive stress and teaches them to control their own autonomic responses to pain, danger, or other stressors. If we can accept heart rate, pupil diameter, rapid involuntary eye movements (“nystagmus”) and involuntary eyelid movement (one of the “micro-tells” that interrogators look for) as legitimate measures of the stress response, then the answer is yes. Although there is great inter-subject variability in responses to stress inoculation, in general, a willing subject can be taught to dramatically dampen his stress response – even to intense pain.

Even civilians – particularly when they don’t anticipate pain or being shot, but are in a stressed environment and have already mounted a stress response - can fail to recognize that they have been shot or feel pain once they have recognized the injury. The common denominators appear to be: 1) they have a stress response already in place because of a combat environment; 2) they have no anticipation of being shot; and 3) bullet wounds are more likely to go unnoticed than machete or edged weapons wounds, in part because the assailant and the assault are visible to the civilian victim.

1 The Stress Response - Conclusions



1. The stress response is mediated by the autonomic nervous system (ANS), which has two components: the Sympathetic Nervous System (“Fight or Flight”), and the Parasympathetic Nervous System (“Rest and Digest”).
2. The ANS uses two mechanisms to produce the stress response: Neural (mediated by nerves), and humoral (hormonal). The neural response is faster (e.g., diving reflex).
3. The stress response interferes with fine motor coordination, judgment, and has variable effects on memory. Adrenal medullary response (epinephrine, norepinephrine) can help to consolidate memory. The adrenal cortical response (cortisol), on the other hand, can cause a temporary memory deficit. The stress response can also produce a loss of bowel and bladder control.
4. “Gross motor” tools (e.g., Glock pistols), and gross motor skills (e.g. SPEAR or CQC combatives) can partially compensate for the stress – induced loss of fine motor coordination. So can “Stress Inoculation” training.

1 The Stress Response – Conclusions (continued)



- 5 The ANS operates independently of conscious control, and its powerful reflexes can override conscious control. The diving reflex is a good example of this.
- 6 There are several overlaps between the ANS and conscious control, however. For example, eye *blinking* and *breathing* can both be consciously modulated, but neither depends on conscious control.
- 7 Colonel Grossman and others have advocated “combat breathing” as a means of exploiting conscious control over stress responses.
- 8 There are other, and perhaps faster, ways of exploiting the few areas of overlap between conscious and ANS control. This includes “Verbal Override” (covered in the next lecture).

AUDITORY EXCLUSION

One of the most fascinating aspects of Colonel Grossman's book "On Combat" is the recounting of many reports from both the law enforcement and the military communities of "auditory exclusion". In brief, this is the well-documented phenomenon of failing to hear loud noises, or partially suppressing loud noises, under extreme stress. Auditory exclusion takes many forms, but one of the most dramatic is the suppression of the sounds of an officer's own gunshots during his debriefing after a gunfight.

Some officers have even reported that they assumed that their gun had misfired, fired a squib round, or failed to fire at all during a gunfight, only to discover later that they had fired several rounds with no malfunctions. Similarly, soldiers after the heat of battle have reported not hearing noises as loud as artillery pieces firing in their vicinity during the battle. More disturbingly, for our purposes, are episodes when a team member or leader's shouted commands were apparently never heard by the officer or soldier even though they should have been clearly audible.

There are several possibilities at play here. Before looking at the physiology of auditory exclusion, we need to exclude the usual list of suspects for strange phenomenon: 1) Are the reports exaggerated? 2) Are they the result of faulty memory? 3) Are they limited to either very experienced or very inexperienced combatants? 4) Are they rare, outlier events?

Given the number of well-documented events, and the even larger number of personal reports that were not formally documented, but well-remembered, it appears that auditory exclusion is a real phenomenon, and cannot be written off as a rare outlier event. There does not appear to be an association with the experience level of the combatant reporting the event. As to exaggeration, although that can never be excluded because no one other than the reporting officer or soldier truly knows what they did and did not hear, it seems unlikely that so many combatants have reported separately and independently the same thing.

What about the possibility that auditory exclusion is just the result of faulty memories – i.e., he heard the shot in real time, but consciously or unconsciously suppressed the recollection of the sound later? One reason why this explanation is not entirely plausible is that auditory exclusion can happen in less stressful environments than a gunfight, and multiple observers can demonstrate very different real-time responses to the same sounds. For example, in their first few days of training, anesthesiology residents are not yet cued into the sound of the pulse oximeter, which has a frequency, or tone, that rises or falls with the blood oxygen saturation. An experienced anesthesiologist will immediately detect even a change in saturation of 1% and a correspondingly minimal frequency change. In contrast, the rookie will not appear to hear even a more dramatic change in tone, and will ask, "Did I hear what?" when asked, "Did you hear that?"

The same is true during a hospital-based cardiac arrest and resuscitation (“code”). It is not uncommon for some members of the code team to report that they never even heard an order given even when it was clearly heard by other people in the vicinity. It is likely that, at this point in your force-on-force simulation training, you or your teammates have at least once failed to “hear” or respond to, an instructor’s command to stop the scenario during the heat of simulated battle. When asked why you didn’t stop, you probably reported, ‘I’m sorry – I never even heard your command!’

These latter events are easy to chalk up to selective attention, rather than selective hearing. But what about failing to hear something as loud as your own gun firing? Or an artillery piece?

Some commentators have expressed the opinion that there might be a physiological mechanism by which the ear and its sound conducting system are physically “shut down” during a stress response. More specifically, the hypothesis has been offered that the high levels of circulating epinephrine during extreme stress constrict the blood supply to the inner ear. After all – epinephrine is known to constrict arteries and arterioles, which is why you’ve been taught to exclude epinephrine from local anesthetics injected into fingers or other appendages.

Is this hypothesis reasonable? Can our ears function like electronic hearing protection and shut down at a moment’s notice under stress?

My own answer, as a physiologist, is “no”.

Let’s take the two most plausible mechanisms for the ears to “shut down” biomechanically: 1) the acoustic reflex; and 2) circulating epinephrine constricting the modiolar artery of the inner ear.

The electronic counterpart of the acoustic reflex operates every time you use a cell phone. As soon as the phone detects your voice in the microphone, it shuts down the speaker to your ear. The idea is to prevent interfering sounds from occurring at the same time – in particular, to prevent the sound coming from the speaker (the ear piece) being misinterpreted by the phone as your own voice and amplified back to the other party. It’s not a system without flaws, however: which is why it’s so irritating when both you and the other party begin talking at the same time: you can’t hear them, not only because you are talking, but because your ear piece is shut down while you are talking, and the loop goes on and on until one of you break the cycle by remaining silent for a few seconds.

Back to our ears. The middle ear contains the three small bones that you probably memorized in school: the malleus, the incus, and the stapes. These bones, collectively called “ossicles” connect to each other and transmit sound waves from the eardrum to the inner ear. Accompanying the three bones are two muscles: the stapedius and the tensor tympani. When these muscles contract, they stiffen the ossicles and interfere with their ability to transmit sound waves. In fact, the effect on low frequencies is profound: a reduction of 30-40 decibels occurs, which is the equivalent of lowering a loud voice to a whisper. As you might guess, the

acoustic reflex serves the same function as a cell phone speaker cut-off: when you are talking, the acoustic reflex kicks in and shuts down, or reduces, a significant part of your low frequency hearing.

While this may seem like a plausible theory for auditory exclusion, it doesn't hold water. There are two reasons: 1) it doesn't reduce sound transmission enough to explain the complete absence of heard noises or voices; and 2) it isn't fast enough. The electronic hearing protection that you wear on the range takes advantage of almost speed-of-light microelectronics. When the front of a pressure wave from a gunshot reaches the microphone, it almost instantaneously shuts down the amplification. As well, the system cheats a little by introducing a slight delay in the sound amplification for voices so that the electronic trigger to shut it down during a gunshot wave has a head start (like the five second television delay for live broadcasts that enable real-time censoring).

The acoustic reflex, on the other hand, isn't nearly as fast. The "latency" (the time between stimulus and response) is 80 milliseconds. While that doesn't sound very long, compared to the time between the beginning of a gunshot sound wave and its peak pressure is less than 1 millisecond. So much for our own, biological, ear protection. The acoustic reflex works well for taming interference during conversations, but it wasn't designed for a .308.

What about the theory that circulating epinephrine cuts off the blood supply to the inner ear during high stress states? Again, it sounds plausible – and, in fact, this mechanism has been proposed by several people who write about tactical physiology. Unfortunately, like the acoustic reflex above, it doesn't hold water. It also has two major flaws: 1) it isn't fast enough; and 2) experiments with intravenous epinephrine do just the opposite: they increase, rather than decrease, blood flow to the inner ear.

Let's take the timing problem first. Like the acoustic reflex, the latency of a biological vasoconstriction reflex is way too slow to attenuate a sound wave that begins and ends as quickly as a gunshot. What about the idea that high stress and epinephrine causes a shutdown of the artery *before* the gunshot? That might be plausible, but it wouldn't explain why other noises and voices that occurred just before, just after, and even during the gunshot *were* heard.

The other problem comes directly from experiments on the relevant artery that supplies the inner ear (the spiral modiolar artery). When topical epinephrine is placed directly on the artery, it constricts and the blood supply to the inner ear (the cochlea) decreases. But when intravenous epinephrine is given – even at doses 10-15 x what we saw in the experiment with the anesthesia residents during public speaking – blood pressure rises more than the spiral modiolar artery constricts, and the net effect is that blood flow to the inner ear *increases*.

There are simply too many problems with the acoustic reflex and the epinephrine theories to explain auditory exclusion. So, what *is* the mechanism? The bottom line is that no one knows. My own guess is that it is a block not at the level of the ear itself, but in the neural pathways in

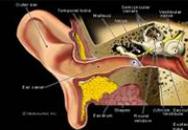
the brain that process hearing, and that are susceptible to modulation by higher brain functions like selective attention, emotions, and stress.

2 Auditory Exclusion - conclusions



1. Auditory Exclusion (A.E.) is real, but its mechanism(s) remain unknown.
2. Although there is a plausible mechanism to "shut down" the middle ear's role in sound transduction by contracting the *tympani* and *stapedius* muscles (the Acoustic Reflex), it is doubtful that this either can, or actually does, completely uncouple sound transduction. Also, the latency period for this reflex is too slow (40-80 msec) to function like electronic hearing protection. Nor does the acoustic reflex appear to significantly attenuate LRAD sound.
3. There is also a plausible, but unproven, neural mechanism for a stress-induced decrease in cochlear blood flow.
4. Circulating adrenaline (epinephrine) tends to increase, not decrease, cochlear blood flow by increasing blood pressure.

2 Auditory Exclusion – conclusions (continued)



5. More plausible is a direct neural inhibition of the cochlea under severe stress (inferior olivary nucleus-to-cochlea inhibitory pathway).
6. The exact mechanism of ear-ringing (tinnitus) is unknown, so its absence after a percussion event tells us very little about the mechanism of A.E.
7. The documented reports of A.E. are numerous, dramatic, and impressive, but they derive from post-event interviews that cannot rule out a role for selective auditory *attention* during the event and/or selective memory after the event.
8. We need to study A.E. *prospectively*. The outstanding question is whether or not a simulated tactical environment can generate enough stress to reproduce real-world A.E. If so, can we train our operators to avoid *signal* A.E.? I think so - we do it every July in anesthesia training.

VISUAL ACUITY and TRACKING

Before discussing visual acuity and tracking in the tactical environment, it's worth visiting the commonly referred to phenomenon of "tunnel vision". This is when the operator focuses on one thing in his central visual fields to the exclusion of other things in the periphery.

A recent example was described by the police officer who shot Major Hasan, bringing to a stop Hasan's killing spree at Fort Hood. Before the officer (Sergeant Todd) shot the active shooter, he confronted the assailant while he was still holding his gun – an FN Five-Seven pistol. In describing what he saw, Sergeant Todd stated that the pistol "looked like a howitzer" to him. Now, an FN Five-Seven is many things, but a howitzer it is not. In fact, the muzzle has an aperture approximately the same diameter as a .22. This is a classic example of "tunnel vision". While Sergeant Todd didn't say as much, he undoubtedly minimized or failed to notice anything other than the pistol while he was perceiving it as an exaggerated weapon.

Another example is lent by a DEVGRU operator with many CQB contacts who told me that he only experiences anything like tunnel vision when he is too far back in an entry stack to take the shot himself – at which point, and in frustration – he focuses on his buddy's gun and everything else in the room blurs.

True tunnel vision occurs in pilots and aviators during exposure to excessive +G_z (downward acceleration). The mechanism is ischemia, or lack of blood flow, to the occipital cortex at the back of the brain. This is the area of cerebral cortex that processes visual input. The area of the occipital cortex that is most susceptible to gravity-dependent ischemia is the part that processes information from the peripheral visual fields. The portion that processes central field vision is relatively spared. This same phenomenon occurs just before you faint from lack of blood pressure (which is often due to a parasympathetic reflex involving the vagus nerve – which is why we call fainting a "vasovagal" episode).

In the case of Sergeant Hasan or the SEAL, however, there is no reason to suspect that their blood pressure was low – and there is certainly no reason to suspect that they were exposed to increased G forces. Instead, their brand of tunnel vision is very likely to be a matter of selective and exaggerated attention ("fixation"), rather than interference with visual input. For that reason, although the phenomenon of tunnel vision is of tactical importance, it is not very interesting physiologically.

Visual acuity and visual tracking in the tactical environment, on the other hand, are fascinating to a physiologist – and they can be quite important operationally.

The two most important things to note about the effects of the sympathetic stress response on the eyes are: 1) it causes dilation of the pupils; and 2) it impairs the lens accommodation reflex. The latter is simply what happens when you read. Muscles that attach to the lens cause it to fatten, and thereby focus incoming light to a point closer behind the lens than when the lens is

relaxed. As you age, the condition of “presbyopia” occurs, and your accommodation reflex weakens. Reading glasses make up for the deficit.

To the younger operator, the problem with impaired accommodation occurs not because of aging and inflexible lenses, but because the stress response causes pupil dilation, and that, in turn, impairs lens accommodation. You experience the same thing when your eyes are dilated for an eye exam. Even though the amount of incoming light is increased, the complete lack of lens accommodation, as well as another problem that comes with over-dilation called “spherical aberration” makes everything blurry.

In the case of refractive surgery, the problem can be exaggerated. This is because refractive surgery often results in an intentional far-sightedness (hyperopia). People who are far-sighted focus incoming light to a theoretical point behind the retina. Lens accommodation compensates for hyperopia and brings the focus back onto the retina. Ophthalmologists over-correct their previously near-sighted patients to make them slightly far-sighted so that the surgical result is less likely to relax and go away over time. They know that the lens accommodation reflex will automatically compensate and their patients will not notice that they are slightly far-sighted.

Unfortunately for the tactical operator who has had refractive surgery, maximal pupil dilation during high stress abolishes the lens accommodation reflex, and he is left with uncompensated hyperopia – i.e., visual blurring.

In the case of pupil diameter, the rule of Goldilocks holds: the optimal diameter is neither too little nor too much. A pupil at about 2.4 mm provides the best overall visual acuity in normal ambient light conditions. Importantly, there is more to vision than acuity. Accommodation to low light or bright light conditions is also important, so there is no one magical pupil diameter. The eye is a flexible, rapidly adapting organ for a reason. There is a tradeoff between the amount of light let in through the pupils and the depth of focus. A constricted pupil lets in less light, of course, and the resulting image quality is poorer. But a constricted pupil also allows for a greater depth of focus and has less “spherical aberration” at the level of the cornea, where most light focusing occurs.

The sequence of animated slides showing UBL as a target demonstrates what can happen at different pupil diameters to the operator’s ability to focus on the three aspects of a sight picture: rear sight; front sight; and target. At normal pupil diameter, all three can be in reasonable focus. As the pupil dilates modestly, the target remains in focus because the slight increase in spherical aberration is compensated for by increased retinal image quality (more light is let in). Unfortunately, at just about the distance of the front sight, matters take a turn for the worse: the combination of slight spherical aberration and decreased lens accommodation (due to the dilating pupil) can cause blurring of the front sight – the most important of the three components of a sight picture. At maximal pupil dilation – as can happen under the influence of an extreme stress response – close objects (like the rear and front sights) can blur because of absent lens accommodation and spherical aberration; and far objects (like UBL in this example) can blur because of the combination of maximal spherical aberration.

Interestingly, maximal spherical aberration even occurs along the visual axis – in other words, even light entering the eye straight on is affected. The result is the same as when your pupils are dilated with eye drops before an eye exam. You wouldn't want to enter a gunfight with such blurred vision, but that same degree of distortion can happen from a maximal stress response alone.

The other visual phenomenon that plays out in the tactical environment is visual tracking. The underlying physiology is complex, but one way of thinking about it is that the brain has both voluntary and involuntary tracking mechanisms, and the two are joined and processed by pathways that converge on two small brain areas called the superior colliculi. However it's wired, the visual tracking is capable of remarkable feats. Hitting a fastball or shooting a moving target are examples. It's important to note that even the best visual tracking system is useless to the operator unless it's coupled to the parts of the brain that control movement, or motor function. That's where the cerebellum comes in, but that's also beyond the scope of this lecture.

For the purposes of training, we should focus on the fact that part of the visual tracking system is voluntary. Not all of it, though: a lot more goes on in your visual world than you are consciously aware of, or than you can consciously control. For example, consider the example of the racecar driver. If you asked the driver what he saw during the race, he would likely report a smooth succession of images and connected events – like a video or a movie being played. That's not how the visual system works, however. Close examination of the eyes reveals that they make quick, jerking movements between objects of interest. They stay fixed on these objects of interest for about 90% of the time, and only spend 10% of their time transitioning between objects. This is called “opticokinetic” or “saccadic” movement.

Notice that I said “objects of *interest*” in the description of opticokinetic movement. That's our clue that we may have an opportunity to consciously control, or train, our visual tracking. The reason is that only higher-level brain processing (consciousness) can discriminate between what is, or is not, *interesting*.

In the next lecture, where the topic of “Physiology as a Weapon” is discussed, we'll see some ways that training can discriminate between the good guys and the bad guys. As just one example, a flash-bang tossed into the air before a room entry will cause untrained hostage takers to automatically track the flying object – to their demise. In contrast, using a technique that I call “verbal override”, we can train ourselves to develop adaptive, rather than maladaptive, conditioned responses – including visual tracking in the tactical environment.

3

Visual Acuity and Tracking - Conclusions



1. Aside from the operator's own optical correction and ambient/NVG illumination, there are two primary determinants of visual acuity: A) pupil dilation; and B) lens accommodation.
2. High stress states increase sympathetic activity, which in turn causes pupil dilation.
3. High stress states may also cause a failure of lens accommodation through the blunting of parasympathetic tone.
4. Dilated pupils transmit more light, which increases image quality.
5. However, at maximal dilation, spherical aberration becomes more important than light transmission and image quality deteriorates.
6. In the ground-based operator (as opposed to the pilot), there is no physiologic cause for "tunnel vision" at the level of the eye or the brain. "Tunnel vision" is likely to be an effect of attention, not vision.

3

Visual Acuity and Tracking – Conclusions (continued)



7. Refractive surgery often causes hyperopia (far-sightedness). Hyperopes compensate for this through lens accommodation. When pupil dilation is added, visual acuity suffers – especially for close objects like gun sights.
8. Refractive surgery results in a less rigid cornea. This may affect acuity at altitude.
9. The brain has its own visual hunting/tracking system, coordinated in part by the superior colliculus. The system moves the eyes as well as the head and neck.
10. The visual tracking system is automatic – the operator (or the subject) cannot stop it, which is why it can be used to distract bad guys.
11. The operator can bring visual tracking under useful control with the tactic of *verbal override* (more on this later).

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Part II: The Physiology of Performance in the Tactical World

James Munis, M.D., Ph.D.

INTRODUCTION

As a general rule, the body follows the instructions of the brain. This assumes, of course, that your body is physically fit, nutritionally sound, adequately rested, and adequately rehearsed (the result of repetitive training “repetitive task transfer” – what used to be called, erroneously, “muscle memory”). We’ve all seen older operators and athletes outperform their fitter or younger colleagues in certain circumstances. This can only be accounted for by a difference between their brains, not their brawn. The question of what, exactly, is going on in a more experienced or better “conditioned” brain is the subject of this lecture.

The preparation of the body to follow the brain’s instructions has been covered in other lectures on nutrition, exercise, and physical fitness. Here, we’ll look at some of the tried-and-true “mental/physical training” techniques that have been used successfully in real world tactical environments by some of our Tier-One operators. These are very simple, practical, concrete training methods that have proven themselves in the worst circumstances. But we won’t stop there. Modern physiology and neural imaging techniques are finally beginning to catch up to the study of athletic and tactical performance, and those scientific tools may shed light on what the brain is doing under stress. Physiology and neuroimaging findings may also suggest ways what we can help train the brain and perform more efficiently. The operative goal here is simply to give the good guys an additional training advantage over the bad guys.

We’ll focus on two topics: 1) selective attention; and 2) “physiology as a weapon”. Before that, however, I’ll highlight three very simple teaching and training techniques that take advantage of the known science, but that don’t depend on any detailed understanding of physiology or mechanisms.

The first technique is called “triangular instruction”. The method is straight-forward: three participants are included (A, B, and C). A teaches B something while C is observing the interaction, then B teaches C while A observes, then C teaches A while B observes. The subject matter being taught could be mental, verbal, and/or physical – it doesn’t matter. For example, it could be teaching a recipe for barbecue sauce, or it could be demonstrating knife strike zones.

To some extent, triangular instruction takes advantage of peer pressure because while B is being instructed, he knows that he is just about to be asked to instruct C the same material. It would be pretty embarrassing to have forgotten the material in the few seconds between being taught by A and being asked to teach C. The immediate repetition between observing, learning, and teaching reinforces the lesson and consolidates it in memory. Better yet, all of this is reinforced by peer pressure and the peculiar, intense, and active attention that peer pressure affords.

An example of lessons learned from triangular instruction being put to use against pirates off of the Horn of Africa will be given during lecture.

The second technique is called “scaffolding”. The analogy comes from arches – either natural arches, like those that form in the desert, or man-made arches like those in Roman aqueducts and bridges. At first glance, an arch seems like a tricky structure to build – sort of a catch-22. Each stone or part of the arch supports the rest of the structure, and it’s all held in place by a capstone. How did each of these pieces get in place and stay in place before the whole arch was assembled and self-supporting? The answer is that the arch had scaffolding, or external support, while it was being built and the scaffolding was taken away after construction. Just because you can’t see it now doesn’t mean it wasn’t present and critical during construction. In the case of a natural arch, the “scaffolding” wasn’t wood or steel, but was an earth and stone mound that was slowly eroded from the inside, leaving a hole under the forming arch.

If you observe tactical training, a similar phenomenon occurs, and if we recognize it, we can take better advantage of it. When a novice learns a new skill, it helps if he recites verbal instructions to himself during practice. The voice can be his own or his instructors; it can be said out loud or to himself. It doesn’t matter. What does matter is that he has real-time verbal cues “talking” him through the task. As his training progresses and the repetitive task transfer hones his performance, he may abandon the “talk” and just do the task automatically, without a verbal accompaniment.

Another example of this is memorization. Depending on how you learn best, you may use a variety of mental crutches to memorize something important. As you continue to repeat what you’ve memorized – “burning” it into your brain’s circuitry – you eventually abandon the crutches and find it easier just to recite the memorized material without bothering to think about the crutch.

When interviewed, some of the more experienced tactical operators report that they use very few verbal crutches, or “mantras”, as they call them. On the other hand, novices tend to make greater use of them. In the last part of this lecture, which deals with the technique of “verbal override”, the idea of a special kind of verbal scaffolding will be examined as a potentially useful device for both experienced and inexperienced operators.

The third teaching technique refers to the roles of the “feeder” and “receiver” during combat, and it is also based on what we know about the “reactionary gap”. In essence, the reactionary gap is the time it takes to respond to a stimulus or a threat. Lay people call this simply “reflexes”. Studies that have measured this interval in professional athletes, like Formula 1 racers, find that there is a certain amount of apparently genetic variation. This is no surprise. Virtually every physiological or performance function is going to vary between individuals – even those who have the same degree of training and are of the same age and physical condition. As a general rule, however, the reactionary gap for simple responses is approximately 250 milliseconds. That is, if you hold onto a fixed blade holstered close to your midline (the fastest anatomic draw position), and strike a metal plate at heart level as soon as you hear a shot timer signal, you will probably never beat 250 ms (1/4 second) between signal and plate strike. Tier-one operators average about 400 ms, and someone who can consistently hit 300 ms is outstanding. It appears that we have a neurologically hardwired performance limit. Training, practice, and concentration can pare down our actual reactionary gap to approach the theoretical limit, but not beyond.

Are there outliers, or “reactionary gap speed freaks”? Sure. Just as there are shooting freaks. No matter how much you shoot and practice, you are unlikely to outperform what Annie Oakley did 100 years ago. The same is true of the reactionary gap. Michael Schumacher in his prime or Lewis Hamilton currently will probably beat you every time – both in a performance lab and behind the wheel of a Grand Prix racer. They are not only highly trained, but highly selected freaks of nature. But that doesn’t mean you can’t learn lessons from reactionary gap studies and use those lessons to your advantage.

There are several other demonstrations of the reactionary gap. Put a wad of paper on the floor. Hold your open palm a few inches above it. Your “opponent” will hold his open palm a few inches above yours. Without warning, he’ll initiate a grab all the way around your hand and snatch the paper right out from underneath your hand before you can respond to his movement. You have every advantage of distance and trajectory (a straight, rather than circular, path). Nonetheless, he’ll beat you almost every time, just as you’ll beat him almost every time when the roles are reversed. It’s the difference between action and reaction.

Another demonstration is to hold a dollar bill vertically from the top edge while your opponent holds his thumb and forefinger in an open “pinch” around the bill from below. Without warning, you release the bill. It is very difficult for the reactor to be able to pinch his fingers closed in time to catch the bill before it falls through his fingers.

Now put this concept together with the role of the “feeder” and “receiver” during combat. In essence, the feeder initiates action and the receiver responds to it. As you might guess, the feeder has an enormous advantage. That advantage goes beyond the obvious, however. Not only does the receiver have to add a reactionary gap into his response time, but the receiver also misses out on an opportunity to learn from every action/reaction sequence. In a sparring scenario, the feeder can initiate an action and watch how the receiver responds to it. Then, without warning, the feeder can initiate a completely different action or the same action out of

sequence or at an unexpected time or coupled to a feint, or false signal, and learn from the receiver's responses. The receiver has no such opportunity to learn about his opponent.

If you consider combat from a physiological point of view, it is really a series of interlocking feedback loops between combatants – at any given snapshot in time, one is the feeder and the other is the receiver. One way to approach winning is for the receiver to turn the tables on the feeder – especially when the feeder least expects an offensive action. It is particularly important to consider whether or not a combatant is within your reactionary gap – or *visa versa*. In other words, can one combatant either initiate or react to a move within 500 ms without having to maneuver through more than one movement or action? If so, scripted CQB tactics, or any other tactic that requires conscious decision making rather than conditioned responses is simply not fast enough to protect you.

SELECTIVE ATTENTION

The easiest way for an operator to miss the boat is to focus on the wrong thing, or to fail to focus on the right thing. It doesn't take modern neuroscience techniques to reinforce this truism, but it's both interesting and instructive that the science of neuroimaging is finally catching up to the tactical world. It is now possible to correlate what we see happening with the kinds of selective attention tasks and mistakes that occur in force-on-force simulations or in the real world with specific kinds of brain activity.

Before looking at which parts of the brain are activated during which kinds of tasks, three general points should be made:

1. The conscious brain is only capable doing a small number of things really well at the same time.
2. The conscious brain is susceptible to external distractions that may push it off its game plan; and,
3. All of the above can be conditioned into tactically-relevant responses. By understanding how the brain works, and how the brain can fail us, we are in a better position to game the system to the good guys' advantage.

Let's start with a few things that the tactical world has already taken note of, and then add to it some new findings in neuroscience that fill out the picture, and that may allow us to harness attention networks in a better way.

First, the practical observations: Dr. William Lewinski of the Force Science Institute has performed studies dealing with selective attention. In a sense, he has rediscovered and

reinforced what Nideffer and Sharpe noted back in 1978 when they wrote a book on attention control training. They pointed out that the brain's limited reserve of attention was divided into external and internal targets. In other words, you can concentrate on your own body and your own thoughts (internal attention), or on people and events in your environment (external attention). Even trained law enforcement operators in Lewinski's simulation studies were not capable of fully engaging both internal and external attention at the same time. Like many other aspects of conscious brain activity, it is a "zero sum game".

Next, we have the phenomenon of distraction. In his book "On Combat", Lt. Col. David Grossman gives many examples of military and law enforcement operators being susceptible to what he calls "the little puppy dog" – that is, an external mental distraction that tugs at your pants leg and pulls your attention away from something more important.

We now know where Grossman's puppy dog, and where Nideffer, Sharpe, and Lewinski's internal and external attention areas reside in the brain, and how they interact with each other. The recent advances in functional brain imaging (fMRI, in particular) have allowed physiologists to correlate behavior with very specific regions of brain activation. More importantly, the way these regions either reinforce or suppress each other provide some clues about how to harness and condition the process so that the operator can exert conscious control of it.

To date, the neuroimaging and neuroscience studies have been performed only in non-tactical environments. The world of neuroscience is largely unaware of the tactical world, and *visa versa*. Those of us who have some familiarity with the two areas would like to bridge that gap – eventually with neuroscience and neuroimaging experiments done on real tactical operators, and in tactically relevant simulations. Until that happens, we can at least borrow from the scientific findings and interpret them for the kinds of tasks that the tactical world cares about.

Now let's add the findings from neuroimaging. It turns out that the part of the brain responsible for selective attention resides in the right cerebral hemisphere, and is dispersed among three main areas. Because each of these three areas has several names, I'll list all three areas, along with their various designations, below:

1. The Dorsal Frontoparietal, Dorsal Attention Network, or "Internal Attentional Network" area. This is the area that is active during "top-down", or goal-driven conscious processes. In my animated slides, this region is simply labeled as 'Internal' and color-coded tan.
2. The Ventral Frontoparietal, rTPJ (right Temporoparietal Junction), or "External Attentional Network". This is the area that is activated by "bottom-up", or externally-driven stimulation, and that is susceptible to distraction. In the animated slides, this region is labeled "External" and color-coded blue. It is David Grossman's puppy dog.
3. The Locus Coeruleus. This is a small, paired nucleus on the brainstem that reacts to stress or pain, and that sends norepinephrine-containing neuronal fibers to other brain

areas, including the rTPJ (“External” network, above). It is abbreviated “L.C.” in some slides, and is activated as part of the stress response that was covered in the first lecture.

How do these three areas interact? The Internal (Dorsal) area is active when the subject is thinking, or planning – that is, when his thoughts are internally-directed. The Internal area is also responsible for directing the attention of other brain areas. For example, you are more likely to find the face of a friend (or a terrorist) in a crowd if you scan a crowd with a goal-directed mission of finding the friend (or a threat) than if you just cast a lazy eye over the faces without a pre-set goal.

Similarly, when you are driving a race car, there are too many external stimuli to track all at the same time, so your Internal network consciously directs your visual attention to the next curve, for example, then to the car on your inside for a fraction of a second, then back to the curve again. While this is happening, you consciously rob attention away from the tachometer, or the spectators in the stands.

Now suppose that something surprising happens: a bird flies up from the infield. Because you weren’t expecting it, you have a startle response – almost a mini-stress response. Your heart picks up a few beats, your stomach knots. More importantly, your L.C. activates as part of the sympathetic stress response, and the L.C., in turn, activates your External network (also called the rTPJ in some of the figures from lecture) by releasing norepinephrine from the nerve fibers that innervate the External network. Now the External network activates, and, in turn, “reorients” the Internal network to command an attentional shift to the bird. In essence, the External network, like Grossman’s puppy dog, has grabbed the pants leg of the Internal network and distracted the External network from the task of driving and instead reoriented it onto the task of watching the distracting bird. And all of this takes a fraction of a second. Nerves are very, very fast.

In the next section, we’ll see what we might do, and what experts like Captain “Sully” already do, to tame the puppy dog and stay on task.

4 Selective Attention (conclusions)



1. Total working attention is fixed – it is a “zero sum game”
2. Attention is weighted toward the most important (or threatening) task at hand.
3. Attention can either be externally or internally oriented.
4. The location of some of the higher-level brain networks that handle attention have been identified.
5. Internal attention is located primarily in the dorsal (rear) right hemisphere.
6. External attention is located primarily in the ventral (front) right hemisphere.

4 Selective Attention (conclusions)



7. Planning, searching and goal-driven functions occur primarily in the internal network. During deliberate planning or searching, the internal network directs the external network's attention to the most important features of the environment – i.e., it forces the external network to stay on task.
8. Surprises in the external environment trigger activity in the external network at the same time that they trigger sympathetic activation.
9. Sympathetic activation, in turn, activates the locus coeruleus – a command and control center for our attentional shifts.
10. The locus coeruleus activates the external network at the expense of the internal network.
11. When activated, the external network re-sets the internal network to pay more attention to the new stimulus and forget the old plan (Lt. Col. David Grossman's “puppy dog” gets the operator's attention and forces him off task).

PHYSIOLOGY as a WEAPON

Understanding the sequence of events and the interactions between the three brain areas outlined above, an overall strategy can be formed to prevent distraction and stay on task. As a practical matter, and without knowing what the underlying physiology shows, Colonel Grossman has already taken a big step forward with his advice to use a “tactical breathing” exercise to overcome some of the side-effects of the stress response.

From the point of view of a physiologist, Grossman’s breathing strategy works because it takes advantage of one of the few areas of overlap between conscious and autonomic control of our body. These areas of overlap include eye blinking and breathing. To some extent, we have some conscious control of our heart rate (for example, snipers and marksmen can be taught to slow their heart rates and entrain their shooting to it). For the most part, though, the easiest way to exert conscious control over the autonomic nervous system is to control the pattern and rate of breathing. By doing this, some other areas of the autonomic stress response are also brought to heel. It is also likely that the tactical breathing technique of Grossman works simply, and ironically, by distracting the combatant from the distraction of stress, and causing him to focus on something less fear-arousing than the fight.

A study of the neural networks involved in tactical-like task performance, as well as a study of what people do to rescue themselves from a stress response combine to suggest another strategy that may be effective in the tactical environment. I’ll call this “verbal override”.

Two non-tactical illustrations may help to clarify what I mean by this:

In 2002, the comedian Jay Leno was invited to be the first North American to drive the Mercedes-McLaren SLR supercar at a test track at the Idiada Proving Grounds in Spain. The car was still in development, was hugely expensive, and it was surrounded by a small army of very serious Mercedes engineers. Needless to say, the pressure was on Leno to perform, but also to bring the car back safely from his >200 mph test run. At one point, he pushed the envelope too far and the car spun out of control. The natural, and physiological, thing to do at that point would be to panic. And the autonomic stress response would be there to aid and abet the panic by activating the entire sympathetic nervous system and by dumping epinephrine into the blood stream.

What Leno did involved an amazing bit of self-control and self-discipline. He remembered what he had been taught as a teenager about recovering from a spin, and he repeated the formula to himself at the same time that he followed his own instructions with his hands and his eyes. That formula is to “look where you want to go; steer where you want to go”. It worked. He recovered from the spin, and didn’t pack-in the prototype supercar.

A more dramatic example comes from Captain Chesley “Sully” Sullenberger’s now famous landing of US Airways Flight 1549 on the Hudson River on January 15, 2009. Almost everyone knows the details. What is not widely known, however, was a cryptic comment that Captain

Sully made while he was revisiting the route with a reporter in a helicopter. He stated that while he was landing, he had to simultaneously keep his eye on the river and on his instruments. Any dip of a wing or deviation from horizontal could cause the aircraft to cartwheel on impact with the water and kill everyone on board.

Remarkably enough, Captain Sully reported none of the usual features of the stress response that we covered in the first lecture: no auditory exclusion; no tunnel vision; and no time distortion. What he *did* report, though, was a continuous verbal interaction between himself and: 1) ATC; 2) his crew; 3) the passengers; and, most importantly, 4) *himself*. In fact, while in the helicopter weeks later, he related to the reporter that he maintained the plane's attitude by repeating to himself the verbal directions to look in a sequential loop, back-and-forth between the "horizon", "instruments", "horizon", "instruments". "horizon", "instruments" until he safely landed the plane.

To a physiologist, what Jay Leno and Captain Sully did was this: The external stimulation (threat) was huge, and the internal effect (stress response) was impossible to prevent initially. The L.C. was activated as part of the sympathetic response; and this, in turn, activated the rTPJ (External network); which, in turn, reoriented the Internal network to redirect valuable attention away from the task at hand and toward the variety of stimuli for panic (e.g., the rushing walls at the race track; the rushing Hudson River; the panic in the voices of the passengers or crew). Both Jay Leno and Captain Sully overcame this automatic response by: 1) consciously referring back to training; and 2) talking themselves through the steps to recovery.

Just as Grossman's breathing exercise serves to push away his distracting puppy, so, too, does the mental activity associated with verbal commands – either aloud or internalized. In essence, the verbal activity not only projected up onto the screen of consciousness just the right tactic at the right time, but it also suppressed the External network and kicked the puppy dog away to allow the Internal network to regain control and reorient attention back to the best plan: the very same instructions that they were rehearsing mentally. In other words, the "verbal override" killed two birds with one stone: 1) it provided the right plan at the right time; and 2) it suppressed the L.C. and External network (rTPJ)'s distraction.

What about the tactical operator? One of the greatest risks in a fight to devote too much attention to the wrong thing (either to panic or its symptoms itself, or to a single weapon or assailant) for too long, and to miss other threats, or to freeze up in the middle of an otherwise well-executed plan. Locked attention ("perseverating") also prevents the operator or combatant from following through with the recurrent feedback loops that make up a fight. This allows the opponent to work within the operator's reactionary gap rather than *visa versa*.

Verbal override accomplishes the same thing that tactical breathing does, but it also adds a front-and-center attentional shift to an instructional set that can save the day (in other words, during verbal override, attention is shifted to a plan for winning, but during tactical breathing, attention is shifted only to a plan for breathing). Finally, verbal override makes use of what we now understand about the way the brain distributes and shifts attentional focus.

Specific examples relevant to tactical tasks will be presented during lecture. To summarize the basics of verbal override, however:

1. Training before the fight is paramount. Leno had his old high school instructions to fall back on. Captain Sully had his previous training as a simulation instructor to fall back on. The verbal “script” used in verbal override has to make sense and be a time-proven and effective tactic for the situation at hand. This is the result of effort, work, sweat, and realistic training.
2. The operator needs to recognize that his stress response is happening, and/or recognize that he has just entered a rapidly changing, complex environment.
3. Instead of either freezing, or “winging it”, the operator talks himself through the sequence (either out loud or to himself – depending on the context). It is important that the script take into account contingencies. For example; once one threat is recognized and dealt with, the script must call for an immediate scan for additional threats. The right script actually makes the operator more, not less flexible and responsive to changing threats



5

Physiology as a Weapon (Conclusions)

1. You *cannot* overcome the initial autonomic (SNS) response to stress.
2. Your brain will *automatically* activate the external (ventral) attention network in response to surprises: either visual or auditory.
3. However, you *can* consciously shift between the external and internal attention areas.
4. How? Using breathing control techniques, or ...
5. Using a **VERBAL OVERRIDE**
6. The sympathetic nervous system (SNS) response doesn't have to be your enemy.
7. SNS activity can facilitate *learning* and *memory*.
8. Don't worry - SNS activation will automatically and reflexively shift your attention to new outside threats as they find you ... or as you find them.

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