Article • Anxiety, Distress, and the Visual Process

Evan Brown, B.Optom. • Auckland, New Zealand

ABSTRACT

Anxiety, sympathetic nervous system arousal, stress, and distress are frequently encountered states of behavioural disposition in symptomatic patients presenting for visual assessment. Our unique percept of safety and threat is an individual response that lies within a continuum from mild to severe, dependent upon the helixing of multiple genetic, environmental, and psychological factors. Each individual response, the embeddedness of the response, and the frequency and length of the stimulus influence the chemical cascade affecting our central and autonomic nervous systems and the functioning of our sensory-motor process. This impacts our ability to derive meaning and direct action. This paper reviews the effects that distress and anxiety have on the visual process and the diagnostic signs that might alert a behavioural optometrist. It also proposes considerations for the treatment of visual conditions associated with or caused by sympathetic nervous system arousal.

Keywords: accommodation dysfunction, anxiety, autonomic nervous system, stress response

Introduction

Over the past century, the developing model of behavioural optometry has challenged us to think and to practice holistically. We must not confine ourselves to the optometric data points and the isolated diagnosis, but see the total person to understand their visual process. Optometrists frequently examine individuals having sustained an acquired brain injury who present with visual symptoms as part of the complex behaviour of anxiety.

What is Anxiety?

Many of us get anxious from time to time. It is a normal response to stressful situations. Our unique percept of safety and threat is an individual response that lies within a continuum from mild to severe. The response is dependent upon the helixing of our genetic character, the environment that surrounds us, our upbringing and experiences, our unique psychological disposition, and learned patterns of coping.

When someone feels anxious about a number of things on most days over a long period of time, 6 months or more, they may be diagnosed with Generalised Anxiety Disorder, or another kind of anxiety disorder, such as a phobia. This includes social phobia, when someone feels very fearful to the point that it interferes with life and function. Examples include being fearful of attending social events; of social and performance activity, such as being called on in class or starting a conversation with a peer; or fear of driving across bridges or travelling on planes. Other anxiety disorders include obsessive-compulsive disorder, panic disorder, and, for school-age children, separation anxiety disorder or selective mutism.

Prevalence of Anxiety

Anxiety is a complex behavioural condition that manifests itself in different ways for each individual, whether it be psychologically or physically. Anxiety disorders are common, usually have an early onset, run a chronic or relapsing course, cause substantial personal distress, reduce quality of life, limit academic and occupational achievement, and impose a personal and societal burden.

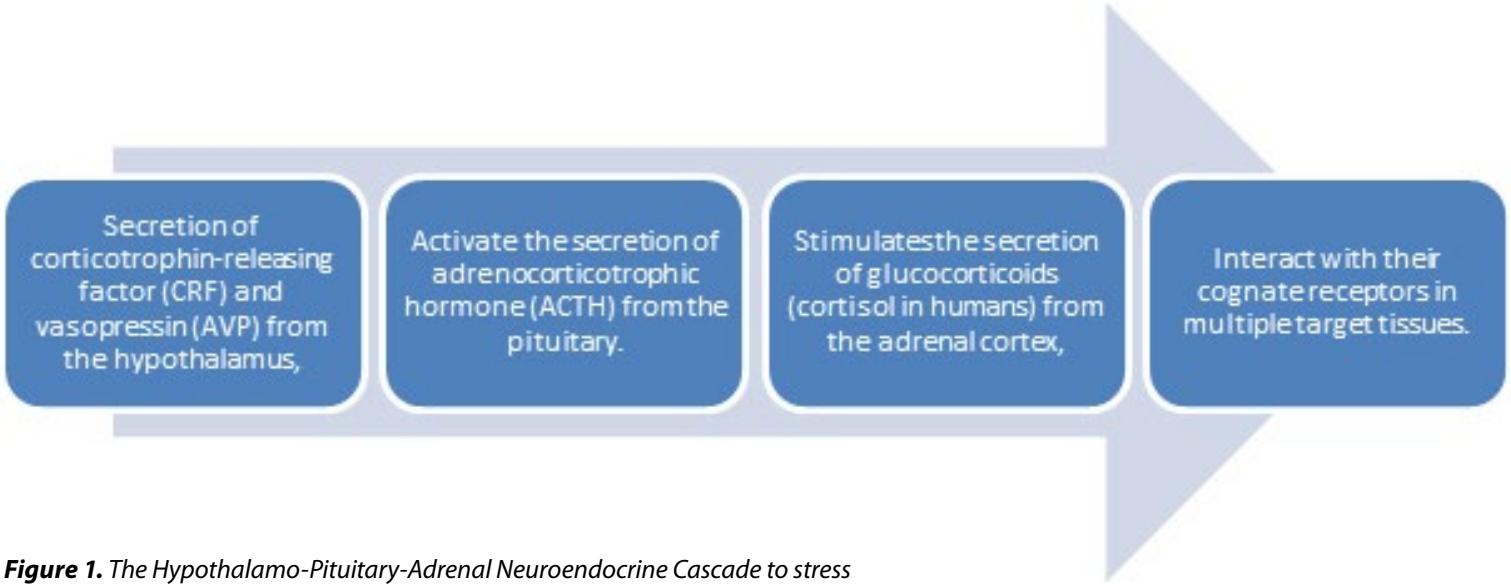

Figure 1. *The Hypothalamo-Pituitary-Adrenal Neuroendocrine Cascade to stress*

If you feel worried from time to time, you are not alone! Lifetime community prevalence of anxiety disorders reaches between 16.6 and 28.8% worldwide.[1] Based on diagnostic interview data from the National Comorbidity Study Replication (NCS-R), the prevalence of any anxiety disorder in adults is higher for females (23.4%) than for males (14.3%). In adolescences, gender remains significant, with a prevalence in females of 38.0% and in males of 26.1%.

Neuro-endocrinology of Anxiety

On an individual level, it is difficult to predict who will become troubled by symptoms of anxiety.[2] Several studies suggest that adverse early life experiences program our brain function and behaviour into adulthood.[3,4] These experiences alter neuro-endocrine function of the hypothalamo-pituitary-adrenal (HPA) axis.[5,6] This model proposes that epigenetic mechanisms program HPA activity,[7] leaving an individual more reactive and vulnerable to perceived and environmental stressors. Placing this in context with what we know about primitive reflex integration, specifically the moro reflex and the fear paralysis reflex, the known mechanism of epigenetics paints the picture of a vicious cycle of behavioural programming for those individuals living with elevated and reactive stress responses.

The HPA axis is a neuro-endocrine reactive cascade to stress. Ambient or consciously perceived stress results in secretion of corticotrophin-releasing factor (CRF) and vasopressin (AVP) from the hypothalamus, which in turn activates the secretion of adrenocorticotrophic hormone (ACTH) from the pituitary. This finally stimulates the secretion of glucocorticoids (cortisol in humans) from the adrenal cortex, which then interact with their cognate receptors in multiple target tissues throughout the body (Figure 1).

Glucocorticoids have widespread regulatory roles as part of the stress response, both in peripheral functions such as immunity and metabolism as well as in the central nervous system (CNS). In the CNS, glucocorticoids moderate neuronal survival, neurogenesis, long-term potentiation, and dendritic growth and atrophy in complex anatomical structures extensively implicated in psychopathology, particularly the hippocampus and amygdala of those individuals living with anxiety (Figure 2).[8] Expression of the glucocorticoid receptor in humans appears to be regulated by maternal care via epigenetic modification.[9,10] In addition, epigenetic regulation of the FK506 binding protein 5 gene (an important regulator of the stress hormone system) mediates the interaction between genes and childhood trauma.[11,12]

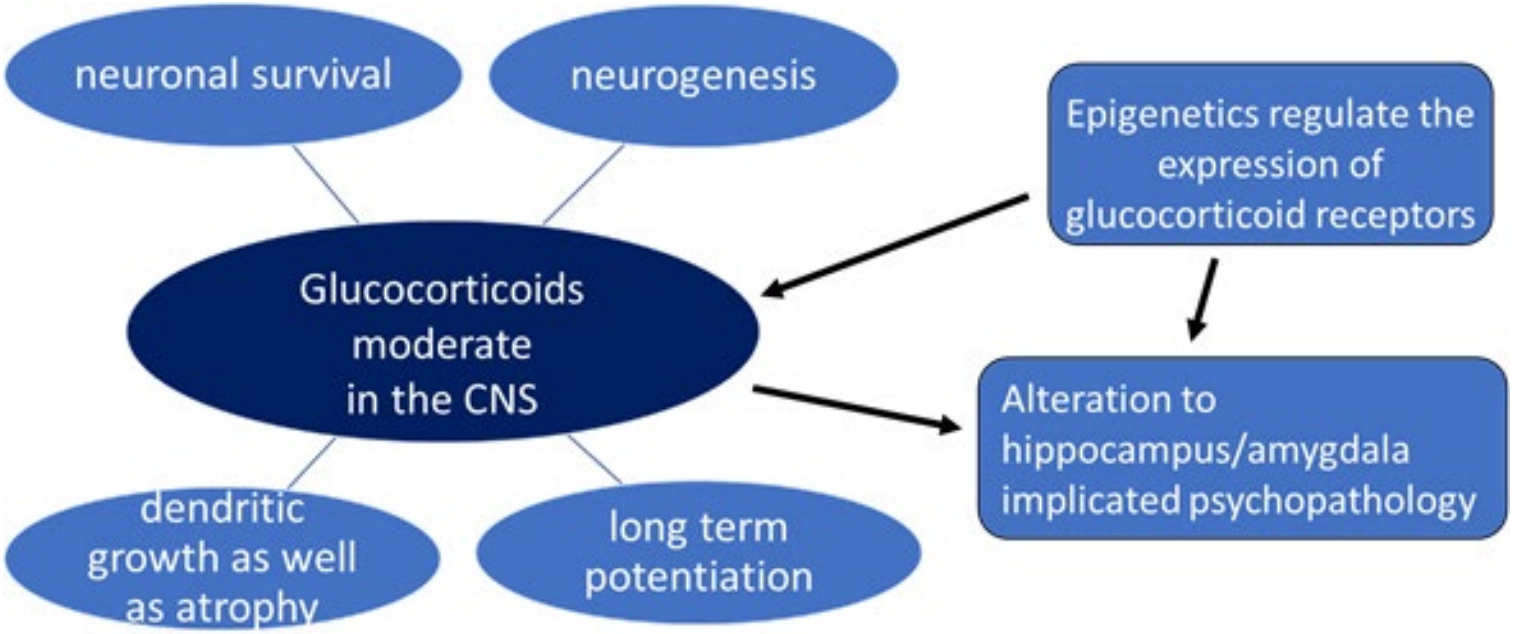

Figure 2. Upregulated stress response can alter both structure and function of the central nervous system through higher levels of glucocorticoids and changes in neuroendocrine receptors.

So, what is the stress response?

Claude Bernard[13] wrote that "the maintenance of life is critically dependent on keeping our internal milieu constant in the face of a changing environment." Canon[14] called this homeostasis. The stress response is a neural and hormonal pattern of behaviour that, for humans and mammals, maximises the availability of energy in the skeletal muscles and the brain and activates and migrates cells of the immune system to battle stations.[15]

Seyle[16] in 1956 proposed the General Adaptation Syndrome, describing a 2-stage reaction to stress, whether physical, psychogenic, or combined.

1. Specific response: Inflammation, allergy, and metabolic changes at the site of stimulation. 'Pro-body' responses to limit damage and promote repair.
2. Generalized ('anti-body') response: Anti-inflammatory response not limited to the target tissue but affecting nearly every tissue in the body.

Our individual stress response emerges from the helixing of 3 components:

1. Genetic endowment (Nature)
2. Unique experiences (Nurture), creating a platform of prior response patterns during development, with maturity from reflexive behaviour patterns being a factor
3. Individual apperception of stimuli: conscious, emotionally influenced responses to stimuli built from prior experience, nurture, and beliefs

Based on a perceived threat, we invoke coping mechanisms or behavioural schemata. A coping mechanism can be thought of as an appropriate response of the stress system to a stressor. It is a crucial prerequisite for a sense of well-being, adequate performance of tasks, and positive social interactions. An inappropriate response impairs growth and development and may account for a number of endocrine, metabolic, autoimmune, and psychiatric disorders.[17]

Personality determines the type, strength, and length of a stress reaction. Notably, aggressive personalities associate with norepinephrine release, and anxious personalities associate with epinephrine release.

Distress

Distress can be described as chronic activation of the body's stress response. This results in exhaustion if the initial alarm response results in action and repair and the resistance phase results in adaptation and vigilance. Under aversive conditions where we are unable to cope, we tend to engage a vigilance response known as sympathetic nervous system arousal. This generally results in active inhibition of movement and the shunting of blood away from the periphery.[18]

While various circumstances tend to result in predictive patterns of stress response, studies show that individual differences occur secondary to the same situation. Some individuals will show stress responses associated with active coping, while others tend to show stress responses more associated with active vigilance.[19,20]

Because many of us tend to over-think things, we may elicit persistent stress responses to a broad range of adverse living and working conditions. Individual differences persist; the main trend is that personality determines the type, strength, and length of a stress reaction.

Neuro-physiology of Anxiety and Emotion

Our autonomic nervous system (ANS) is all about safety. ANS activity in emotion has been actively researched for over a century since Walter Cannon[14] in 1915 first discussed how as one nervous system responds as a matter of survival in preparation for action, the other drops biochemically in activity, proposing the fundamental principles of homeostasis, internal equilibrium, and the resource of energy in the body.

Research done over the last 50 years has invalidated the view that the sympathetic division of the ANS functions in an all-or-none fashion. Rather, each organ and tissue is innervated by distinct sympathetic and parasympathetic pathways, with very little or no cross-talk between them.[21,22] Pools of sympathetic neurons can be selectively engaged such that individual systemic circuits or other effector units are independently activated.[23] Current understanding describes a sympatho-adreno-medullary system consisting of two parts, a direct-nervous system that executes precise, rapid, and often highly differentiated adjustments, and an adrenomedullary hormonal system that independently modifies metabolic functions. In some emergency situations resulting in generalised activation of the sympatho-adrenal system, the two parts may mutually support each other.

Polyvagal Theory

Anxiety has been almost unanimously characterized by sympathetic activation and vagal deactivation, a pattern of reciprocal inhibition.[24] Polyvagal theory[25,26] offers a model for understanding how the vagus nerve, one part of the ANS, connects the brain, heart, and viscera with our human ability to relate to and communicate with each other.

As humans, we frequently detect threat as an ambient behaviour before we consciously think about it. Porges[25] coined the term neuroception to describe this innate, unconscious awareness through the ANS of influences: in the body, in the environment, and in interactions with people.

The vagus nerve (CNX) has two distinct branches:

1. Vegetative vagus: The phylogenetically older branch originating in the dorsal motor nucleus (DMX branch)
2. Smart vagus: The newer branch, originating in the nucleus ambiguus (NA branch).

Both branches provide inhibitory input into the heart via the parasympathetic nervous system (PNS). The vegetative vagus functions to suppress metabolic demands under conditions of danger and is involved with the release

of dopamine (calm), serotonin (happy), and cortisol (stress). The smart vagus dynamically regulates cardiac output, behaviourally mediated either by sustained attention and/or social engagement. This results in vagal heart rate deceleration; by fight/flight response this is associated with near complete vagal withdrawal, which facilitates large increases in cardiac output by the sympathetic nervous system (SNS) that is no longer opposed by inhibitory vagal influences.

Polyvagal theory proposes the mammalian ANS to be phylogenetically hierarchical, with response to threat dictated by the newest neural structures initially. Thus, if vagally mediated social adaptive behaviours are ineffective in coping with a stimulus, response strategies shift to fight/flight behaviours mediated by the phylogenetically older SNS. If fight/flight strategies fail, immobilization (freeze) behaviours are initiated, mediated by the vegetative vagus.

Emotion regulation and social affiliation are considered emergent properties of the regulatory functions served by the smart vagus. The smart vagus modulates breathing, hearing, expression through facial muscles, and vocal tone patterns, allowing us to relate to and communicate with each other even in times of increased stress, where we are coping. Deployment of the newer vagal system suppresses the robust emotional reactions that characterise fight/flight responding, a requisite for the emergence of complex social behaviour. Functional deficiency of the smart vagus place individuals at risk for emotional liability. Consistent with this, attenuated vagal tone has been observed among anxious and panic disordered groups,[27] and excessive vagal reactivity to various challenges is seen in children who are shy or angrily reactive.[28,29]

Polyvagal theory provides context for understanding emotion dysregulation as failure of the smart vagal system in situations where an individual is not adaptive, resulting in deployment of the SNS-mediated fight/flight behaviours, trait anxiety, and the withdrawal behaviours of anxiety and panic disorder.

Several authors have noted that the preschool years represent a critical period during which the developing noradrenergic, serotinergic, and dopaminergic systems that govern behavioural control are vulnerable to long-term changes in functioning.[30,31] Exposure to intense and chronic stressors during the developmental years has long-lasting neurobiological effects and puts one at increased risk of anxiety.[32] Polyvagal theory suggests a neurobiological mechanism whereby lack of developed executive control over emotional response patterns is reflected in deficient vagal modulation of SNS-mediated responses. The prefrontal cortex connects to the smart vagus through the amygdala, creating a neuroanatomical pathway for understanding how the development of executive functioning is intertwined with emotional regulation.

The PNS, when working well, allows access to the social engagement system: our ability to self-regulate, to communicate through voice or gesture, and to show and to read emotion. Notably, many visual abilities are important to the developmental learning of the social engagement system: magnocellular processing of motion and dorsal stream regulation of fixation, binocularity, and oculomotor control. The question therefore poses itself: Can optometrists, through careful examination of the visual process of the child, detect those who would benefit from early intervention to curb the neuro-endocrine and neuro-physiological response patterns that embed reactive anxious behaviour?

The full-scope assessment provided under the holistic model of behavioural vision care will allow for examination of the anxious child to determine normal development of the abilities of neuro-muscular control for fixation, scanning eye movements, spatial awareness, and eye contact in conversation. Appropriate

development of perceptual information processing for discrimination, awareness, flexibility, attention, and sensory integration will also occur.

Anxiety, Distress, and the Visual Process

Arnold Gesell[33] wrote, "The *visual system* is more than a dioptric lens and a retinal film. It embraces enormous areas of the cerebrum; it is *deeply involved in the autonomic nervous system;* it is identified reflexively and directively with the skeletal musculature from head and hand to foot."

Skeffington[34] stated, "The total organism, the whole body (if one is careful to include intellect and emotion as part of the body) participates in vision. The relationships between the effector processes, accommodation, and convergence are learned. An instigating matrix existed as part of the species' inheritance, but this triggering was just the start from which the learning of each individual stemmed."

Our unique percept of safety and threat is an individual response that lies within a continuum from mild to severe, contingent upon the helixing of genetic, environmental, and psychological factors. Each individual response, the embeddedness of the response, and the frequency and length of the stimulus, influence the chemical cascade affecting our ANS and CNS and the functioning of our sensory-motor process, affecting our ability to derive meaning and direct action.

From a clinical standpoint, imbalance of the ANS has a defined impact upon the visual process. Individuals with ANS dysfunction often present as symptomatic to primary care ophthalmic clinicians. A cluster of related findings may well help us understand the underlying aetiology of a visual dysfunction.

What are the ocular effects of sympathetic nervous system arousal?

The ciliary body is innervated by the ANS, with the sympathetic component opposing the action of the parasympathetic.[35,36] Parasympathetic innervation of the ciliary muscle (cholinergic) increases the accommodative response, decreases pupil size, and is involved in rapid responses (within 2 seconds). Sympathetic innervation (adrenergic) of the ciliary muscle inhibits the accommodative response and dilates the pupil. Highly anxious individuals are observed to produce larger, slower pupil responses. The pupil diameter increases in response to emotional stimuli (both visual and auditory), and the responses are individual to perceived threat processing.[37] The resulting dilation reduces depth of focus, reduces depth of field, and increases light sensitivity. The maximal attenuated effect of the SNS on accommodation is considered to be –1.50 DS; it is a slow response (10-40 seconds).[38,39] Tonic accommodation (dark focus) is influenced by the level of sympathetic activity, with increased activity moving the tonic point away, and decreased activity moving it closer.[40] Increased sympathetic activity results in the PNS working hard to engage accommodation, resulting in increased convergence and an increased gradient AC/A ratio.

Likely Clinical Findings

Accommodative dysfunction is quantified by early reports of first blur or low findings on test probes into positive relative accommodation (PRA), negative relative accommodation (NRA), or both. A model of interpretation in context for the patient with anxiety: Increased SNS inhibitory action results in remote shift in tonic point, and increased parasympathetic engagement to generate accommodation places the ciliary body in a state of increased tone. Embedding the pattern results in loss of flexibility for change, along with alterations in automaticity, accuracy, and sustained ability of response.

Near esophoria: SNS inhibitory action results in a remote accommodative shift, placing accommodation behind the plane of vergence and forcing an individual to centre their near

focus response around vergence, rather than around adjustments in accommodation. The associated tendency in anxious individuals to shrink the volume of attentional space compounds this behaviour.

Accommodative lag on Near x-cyl: Increased SNS innervation shifts the tonic point away. This is measured as increased sensitivity when the periphery and the proximal awareness system are compromised by the field of view afforded by the phoropter.

High minus AC/A: SNS inhibitory action results in increased parasympathetic demand and excessive accommodative effort to maintain clarity with excessive accommodative convergence per unit of accommodation.

Restricted divergent vergence ranges (with early blur points) indicate reduced flexibility between accommodation and vergence, with excessive compensatory arousal of the vergence system for centering.

Convergence Insufficiency (CI): Parents of children with CI report significantly greater levels of adverse academic behaviours and worry about school performance compared to parents of children with normal binocular vision.[41] Borsting el al.[42] investigated behavioural and emotional characteristics of children with CI before and after treatment with office-based vergence/accommodative therapy. They showed significant mean reduction in adverse academic behaviours on the Connors 3 ADHD index and the Child Behaviour Check List for those patients experiencing difficulty with attention, anxiety, depression, having a high index for somatic complaint, or for those internalizing problems.

Early Fatigue: Functional changes in the visual process that result from a remote shift in the tonic resting point of accommodation, mean that for the organism to centre the vision process at near takes increased effort. This, along with the breakdown in schemata of the accommodation and vergence processes, will result in early fatigue.

Constriction of space: Functional field collapse is quantified with greatest sensitivity using colour field campimetry, or in some cases by frequency doubling using matrix fields.[43] In others, tubular non-specific visual field constriction failing to respect both the horizontal and vertical midlines on threshold visual fields is shown. Restricted vergence ranges often accompany this diagnostic sign.

Oculomotor function: Clinical test probes into pursuit and saccadic function often reveal a lack of freedom between the oculomotor process and the skeletal and vestibular systems. Anxious individuals more often keep the target on which they are centred directly at their midline, rotating the neck and moving the head to point the nose straight at the target, rather than moving the eyes freely for pursuits and saccades. This finding will often be associated with reduced functional fields, addition and substitution errors while reading, or on tasks such as the Developmental Eye Movement test.

Pseudomyopia, through excessive stimulation of the ciliary body by a dysregulated ANS, may be diagnosed in individuals living with anxiety.

Contrast Sensitivity Modulation: Ferneyhough et al.[44] show that anxiety modulates the attention and emotion effects on perception; specifically, contrast sensitivity is heightened in function at attended locations and impaired at unattended locations. Neurocognitive theories of anxiety and attention suggest that amygdala activity is heightened in anxiety in response to sources of potential threat. Studies investigating the role of frontal brain regions in the top-down control of emotion have shown that anxious individuals may have not only increased amygdala activity[45,46] but also decreased recruitment of frontal control regions. Imbalance between bottom-up emotional response and top-down attention could underlie the difficulty that anxious individuals have in engaging attention. Anxious

individuals in a state of increased bottom-up response will have difficulty performing visual tasks unrelated to threat, such as discriminating orientation at other spatial locations and deriving meaning from form and shape for pattern processing and recognition.

Balance and Orientation: Anxiety is highly prevalent in patients with balance disorders. While many anxious individuals have symptoms of imbalance and/or dizziness, few have a syndromal vestibular disorder.[47] Rather, many suffer from symptoms specific to space and motion, resulting in diagnostic conditions such as supermarket syndrome,[48] space phobia,[49] visual vertigo,[50] or height vertigo.[51] Redfern et al.[52] found that patients with anxiety have increased sway to sinusoidal anterior-posterior movement of the visual surround when compared with healthy controls. They are also less capable of integrating or ignoring peripheral visual motion, indicating increased visual dependence on balance and orientation.

Central-peripheral Processing Dysfunction: The difficulties that anxious individuals demonstrate in contrast sensitivity modulation, motion processing, and functional field collapse likely also correlate with reduced findings when test-probing visual processing speed, span of tachistoscopic recognition, imagery to support visual recall and working memory, dorsal stream function for anti-gravity processing resulting in sensitivity to movement, and motion. All findings support the model of central-peripheral processing dysfunction.

Attention: Corbetta[53] defines attention as "the mental ability to select stimuli, responses, memories, or thoughts that are behaviourally relevant, among the many others that are behaviourally irrelevant." Corbetta and Schulman[54] differentiated neuro-anatomically that the selection process is based upon two complimentary processing systems: top-down goal-directed selection of stimuli and responses, such as attitudes, strategies, and personality traits; and bottom-up sensitivity to stimulus salience. Emotion can enhance attention and improve perception in certain circumstances, but it can impair them in others. Anxiety modulates these effects.[55] States of anxiety can be discussed in terms of State, Trait, and Clinical. Pacheco-Unguetti et al.[56] propose that Trait anxiety creates hypervigilance in the top-down process, creating deficiencies in executive control; State anxiety increases the threat value, affecting the bottom-up system and resulting in over-functioning of the alerting (readiness to react) and orienting (selection of specific location and object information) networks. The sensory process is thrust outward, making it difficult to apply attention to focal tasks and to integrate sensory information and harder to avoid distractions or to shift attention from one task to the next.

Stimulus Bound: Clinically, I like to probe individuals for their ability to be free of dependence upon a stimulus. The anti-saccade test is a simple behavioural task that utilises eye movement measures to investigate individual differences in attention control across anxiety and mood disorders.[57] Participants are asked to look away from a visual cue (to its mirror location) as quickly and as accurately as possible, with attention control indicated in (1) the ability to withhold (inhibit) reflexive saccades, and (2) the generation of volitional saccades to the correct location.[58] Anxious individuals are more likely to make erroneous eye movements on anti-saccade trials.[59] Clinically, this will often correlate with inability to clear -2.00 DS OU at 6 metres, a low ability to mobilise accommodation around the point of vergence (relative accommodation measures), voluntary convergence control, and symptoms of an inability to "see ahead" and to anticipate.

Other Likely Holistic Findings we Might Observe in Our Patients

- Round-shouldered or esomorphic posture
- Breathing slow and shallow, with hyperventilation and elevated heart rate, resulting in decreased carbon dioxide partial pressure (pCO2)
- Hyper-motion and movement overflow, resulting in fidgety fingers, hands, toes, and tongue
- Dry mouth, difficulty swallowing, gastrointestinal upset, and poor bladder control
- Early fatigue, short temperedness, cranky and irritable disposition
- For those with a "fight" behavioural disposition: unusual aggression
- For those with a "flight" behavioural disposition: evasiveness and hyperactivity
- For those with a "freeze" behavioural disposition: selective mutism, withdrawal, a tendency to ignore
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Notably, "freeze" mode is associated with vagus nerve activation, which results in low muscle tone, slowed breathing, slowed heart rate, low blood pressure (one might faint), digestion suppression, reduced bladder and bowel control, and reduced sensory processing.

Likely Clinical Profiles

Clinical experience examining the visual process of patients living with anxiety shows it to be consistent with the general bodily stress response; individual differences occur to the same situation. Some individuals will show stress responses associated with active coping, while others tend to show stress responses more associated with active vigilance. The predominant tendency is to present with dysfunction to test probes that involve the accommodative process. Often, the profile may struggle to fit the clinical profiles described by Howell[60] and Skeffington[34] involving near point stress, but have many similar characteristics.

Flax[61] associated a B2 profile as more concerned with "what is it" and the B1 profile as more concerned with "where is it" functioning better in the distance. Clinically, this profile correlation can often be seen in terms of the patient living with anxiety presenting with a primary diagnosis of accommodative insufficiency versus a CI.

Peachey[62] proposed the minimum attention model. Individuals with anxiety fit well within the model of schemata breakdown, with accommodative dysfunction, saccadic dysfunction secondary to collapsed functional fields, and magnocellular dysfunction affecting the automaticity of the visual process to derive meaning and to direct action. As a result, these patients present with difficulties in executive functioning, attention, information processing, and sports performance.

Streff syndrome was first described in 1962 by Dr. John Streff as non-malingering syndrome. In 1962, Dr. Streff and Dr. Richard Apell[63] expanded the concept to add early adaptive syndrome as a precursor to Streff syndrome. Streff syndrome often involves reduced or blurred distance and near vision, reduced stereopsis, large accommodative lag on dynamic retinoscopy, and a reduced visual field. There is a general correlation with the individual being in a perceived environment of stress and resultant anxiety. Observed changes in the threshold and functional fields fail to observe the vertical or horizontal meridians, tending to be tubular or spiral. Patel[43] showed that Streff syndrome may be associated with a dysfunction in the magnocellular pathway of the retinal ganglion cells, whose function is to provide information about the location and orientation of objects (where), information processed primarily through the dorsal stream. The dorsal action stream generates an egocentric coordinate map for motor planning, important for the function of how to direct eye movements and in binocular depth perception.[64]

Management and Treatment

Optometrists have a role to play as primary care practitioners in differentially diagnosing presenting conditions and helping patients understand the complexity of the underlying diagnoses that result. The tool box of the behavioural care practitioner allows for a more holistic approach. Careful examination and consideration will enable us to avoid falling into the trap of simply diagnosing accommodative dysfunction, instead recognising the profile of presenting symptoms that allows the optometrist to open a discussion about how the patient is feeling and coping with the pressures of life.

Management options include treating the presenting symptoms with plus lenses, yoked prisms, and selective filters to support the centring and identification processes. Syntonic light therapy can improve regulation of the ANS. Vision therapy rehabilitates the vision process, providing schemata and coping mechanisms in the centering and identification processes and in the orientation and speech-language communication processes for executive functioning and attention control. Guidance and feedback on the role of breathing (slow deep breathing activities have been found to give relief[24] and developing awareness in central-peripheral processing can be helpful schemata to build in patients living with anxiety.

A Multi-disciplinary Approach

Referral to the patient's general practitioner or local psychologist for counselling and management of anxiety disorder should be considered. Anxiety disorders are highly treatable, yet only 36.9% of those suffering receive treatment. Standard practice pathways include counselling, cognitive behaviour therapy (CBT), exposure therapy, and eye movement desensitization and reprocessing therapy.

Cognitive Behaviour Therapy (CBT)

CBT, including relaxation training, has demonstrated efficacy in several subtypes of anxiety.[65] James et al.[66] reviewed the literature and concluded:

- CBT is significantly more effective than no therapy in reducing symptoms of anxiety in children and young people
- No clear evidence indicates that one way of providing CBT is more effective than another (e.g., in a group, individually, with parents)
- CBT is no more effective than other active therapies, such as self-help books
- The small number of studies meant the review authors could not compare CBT with medication
- Only four studies looked at longer-term outcomes after CBT. No clear evidence showed maintained improvement in symptoms of anxiety among children and young people.

A form of CBT, exposure therapy, is a process for reducing fear and anxiety responses. In therapy, a person is gradually exposed to a feared situation or object, learning to become less sensitive over time. This type of therapy is particularly effective for obsessive-compulsive disorder and phobias.

Acceptance and Commitment Therapy (ACT)

This type of therapy uses strategies of acceptance and mindfulness (living in the moment and experiencing things without judgment), along with commitment and behaviour change, as a way to cope with unwanted thoughts, feelings, and sensations. ACT imparts skills to accept these experiences, to place them in a different context, to develop greater clarity about personal values, and to commit to needed behaviour change.

Dialectical Behavioural Therapy (DBT)

Integrating cognitive-behavioural techniques with concepts from Eastern meditation, DBT combines acceptance and change. DBT involves individual and group therapy to learn mindfulness, as well as skills for interpersonal effectiveness, tolerating distress, and regulating emotions.

Interpersonal Therapy (IPT)

IPT is a short-term supportive psychotherapy that addresses interpersonal issues in depression in adults, adolescents, and older adults. IPT usually involves 12 to 16 one-hour weekly sessions. The initial sessions are devoted to gathering information about the nature of a person's depression and interpersonal experience.

Eye Movement Desensitization and Reprocessing (EMDR)

Under certain conditions, eye movements appear to reduce the intensity of disturbing thoughts. A treatment known as EMDR seems to have a direct effect on the way that the brain processes information. Basically, it helps a person see disturbing material in a less distressing way.[67] EMDR appears to be similar to what occurs naturally during dreaming or REM (rapid eye movement) sleep. Scientific research has established EMDR as effective for adverse life experiences.[68] Clinicians have reported success using it to treat panic attacks and phobias, and the treatment is considered to be evidence-based practice.[69]

Medications

Antidepressants such as selective serotonin reuptake inhibitors also show efficacy in anxiety,[70] especially when generalised anxiety disorder (GAD) is comorbid with major depression, which is the case in 39% of subjects with current GAD.[71]

Referral to Practitioners who Work in ANS Regulation

Consider referral to other professionals, such as cranio-sacral therapists, reflex integration therapists, osteopaths, acupuncture practitioners, massage therapists, and anthroposophical nursing, all of whom work in autonomic nervous system regulation. Nutritionists are also helpful for advice and diagnosis around nutrition and gut dysbiosis.

Conclusion

Individually, we live with the insecurities of our daily lives. Where the demands of a situation exceed the adaptive resources of an individual, an acute stress response occurs. Optimistic individuals with good coping mechanism responses will likely benefit from such experiences. Stressors that are too strong and too persistent in individuals who are biologically vulnerable because of age, genetic, or psychosocial factors, will likely produce maladaptive strategies that produce specific visual profiles. Observant optometrists probing the visual process with a full battery will uncover profiles that indicate SNS arousal. The diagnosis, management, and treatment of these individuals will take on holistic and likely efficacious pathways if we are able to understand the underlying issues present in individuals presenting with dysfunction of the visual process in the presence of anxiety.

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Correspondence regarding this article should be emailed to Evan Brown at eb@evanbrown.co.nz.

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