Article ▸ Role of Optometry in the Management of Vision and Ocular Health of Patients with Mental and Emotional Disturbance: Part 1

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ABSTRACT

A subset of patients who present to optometrists for vision care have mental and emotional disturbance. This literature review seeks to gain a better understanding of the relationship between aspects of vision and ocular health and a person's mental wellbeing. Traditional management strategies have addressed ocular health and acuity symptoms related to the effects of pharmacological substances, whilst visual perceptual deficits are often overlooked. Various treatment modalities are explored further to allow optometrists more confidence in managing patients with mental and emotional disturbance.

Keywords: Anxiety, dry eye syndrome, major depressive disorder, post-traumatic stress disorder, schizophrenia, spatial awareness, Streff syndrome, Syntonic phototherapy, vestibular system, visual acuity, visual perceptual dysfunction

Introduction

As optometrists, we are responsible for providing visual and ocular health care for patients from many backgrounds. Part of our duty of care is to recognise the impact that systemic diseases and afflictions may have on the vision and ocular health of our patients. Optometrists are not generally thought of as part of the direct team for aiding in the management of patients with mental and emotional disturbance; however, vision is a strong sense. Eyes open and even closed, we use vision to gain meaning from our lives. Inefficient use of the visual process may be responsible for, or may be increasing, some of the psychological symptoms experienced by patients with mental and emotional disturbance. Enhancing the use of the visual system and offering strategies that may be useful in coping with such disturbance, and improving or eliminating the symptoms that arise, should be part of the therapy to manage these patients holistically.

Compared to the general population, patients with mental health problems will have poorer physical health and general well-being.[1,2] This likelihood increases with the severity of the mental health problem. These patients are more likely to eat poorly, to drink more alcohol, to smoke more, and to exercise less. They tend to access health services less often than required, suffering with diabetes type 2 and heart disease in greater numbers. These factors may directly affect ocular health, consequently creating visual problems and impacting overall quality of life.

This literature review examines the aspects of vision and eye health care that may be managed by optometrists in patients with mental and emotional disturbance. It will not include mental and emotional disturbances that may also be considered childhood developmental disorders (pervasive developmental disorder (PDD), autism spectrum disorder (ASD), attention deficit disorder (ADD)). The relationship between vision and psychology is considered. An overview of mental and emotional disturbance and prevalence in Australian society is discussed. Aspects of the visual process and ocular health with regard to mental and emotional disturbances are reviewed. Assessment procedures and management strategies are discussed. Three case histories are outlined where the patients had visual concerns that related to their mental and emotional status.

The Relationship Between the Visual Process and Mental and Emotional States

Forrest discusses the relationship between one's psychological state and their visual spatial map, as well as their visual imagery ability.[3] From a psychological perspective, our central and peripheral awareness can be interpreted as the relationship that our ego has with factors outside of our own being. Our ego creates our own individuality, thoughts, and feelings and impacts our figure-ground interaction, attending to specific details of our environment while integrating these details with the rest of our spatial world.

Enhancing the visual process has the ability to improve sensory-motor central-peripheral interaction, which may transfer to generalised improvement of mental and emotional function. An efficient visual system has the flexibility to move within the visual spatial map with ease and flexibility. Likewise, an efficient mental capacity should also be able to operate with flexibility between ego and other outside factors. Enhancing the visual system may lead to an improvement in mental and emotional states. Just as important, enhancing one's mental state may lead to improvement in vision.

Overview of Mental and Emotional Disturbances

There are hundreds of diagnoses for mental and emotional disturbances, many of them co-existing to some extent, and many with co-morbid factors, including other general medical conditions, primary support, social environment, and educational, occupational, housing, and economic problems. The Diagnostic and Statistical manual of Mental

Disorders defines a mental disorder as a "clinically significant behavioural or psychological syndrome or pattern that occurs in individuals and that is associated with distress or disability or with a significantly increased risk of suffering death, pain, disability, or an important loss of freedom."[4] It is also relevant to remember that the disorder does not define the person. The mental and emotional disorder is a condition that the person has, not what they are.

A physical problem may cause a mental problem, and a mental problem may cause a physical problem. Patients with chronic physical illness may suffer mental illness as a consequence. Patients with mental illness may suffer physical illness as a result of inadequate general health or personal care or issues associated with making certain choices, leading to a physical illness. A study examining the incidence of psychiatric disorders before and after traumatic brain injury (TBI) showed an increase from 52% to 65%.[5] The most common psychiatric diagnosis before TBI was substance abuse (42%), followed by major depressive disorder (17%) and anxiety (13%). It is possible that substance abuse may pose an increased risk of TBI, although this was not stated in the study. However, post-TBI, the incidence of major depression increased to 45%, and anxiety increased to 38%, while substance abuse decreased to 21%. Neuro-rehabilitative optometrists frequently see patients following TBI or other brain injury, such as that incurred with cerebral vascular incidents (CVI). These patients may present with changes in their mental and emotional stability directly due to their brain injury but also due to the impact of the injury on their general abilities, including changes in vision, socialisation, and independence.

Forrest discusses the use of the word emotion and its relation to psychological states.[3] Initially referred to as an intense state of mind, mental agitation, or disturbance, it has now come to be used with milder affective states of feeling and mood. He describes an emotional state as being a state of action or motor, with the changes being those physiological changes that occur within the body.

Prevalence within the Australian Population

The Australian Government Bureau of Statistics states that 45% of Australians aged 16-85 have experienced a mental health issue over their lifetime.[6] Anxiety disorders have affected 14.4% of adults, with women having a higher incidence (17.9%) than men (10.8%). Depression has affected 20% of the population, with 6% experiencing a major depressive incident. Schizophrenia affects 1 in 100 adults, with a ratio of 3:2 of males to females. Mental illness is the third leading cause of disability in Australia, affecting 27% of the population. Major depression results in more days of work lost to illness than any other illness in Australia. The incidence of mental and emotional dysfunction appears to change with age. Twenty-seven percent of 18- to 24-year-olds have a diagnosis of mental or emotional dysfunction, but only 6.1% aged over 65 have a similar diagnosis. The incidence of mental dysfunction is higher in areas that are more socially disadvantaged: up to 20% in these areas, but only 8% in more advantaged socioeconomic areas. The incidence of mental and emotional dysfunction appears to have increased. In 1995, there was a 5.9% incidence, while in 2004, there was an 11% incidence. This may be due to the willingness of individuals to visit health professionals, as well as an increase in awareness and diagnosis on the part of health professionals.

These figures mean with certainty that we will see patients in an optometric setting who are suffering or who have suffered with mental or emotional dysfunction. They may have chronic mental illness or have experienced only one episode. They may or may not willingly discuss their mental health issues and disclose medications. Regardless, investigation of our patients' vision and ocular health should encompass a battery of tests which allow us to consider the possibilities that visual problems may interfere with a person's overall sense of emotional well being.

Characteristics of Mental and Emotional Disturbances

Mental and emotional disturbances fall into different categories. Those most common in adults are affective disorders (such as anxiety, depression, and mood disorder) and psychotic disorders. Anxiety disorders include panic disorder, obsessive-compulsive disorder, post-traumatic stress disorder, acute stress disorder, and generalised anxiety disorder. Mood disorders include major depressive disorder and bipolar disorder. Psychotic disorders include schizophrenia. Many common disorders usually first diagnosed in early childhood include PDD, autism and Asperger's syndrome (ASD), ADD, behaviour disorders, and tic disorders. This literature review will not include mental and emotional disturbances that may also be considered as childhood developmental disorders (PDD, ASD, ADD).

Anxiety disorders are characterised by at least 6 months of persistent and excessive anxiety and worry. Panic disorders show symptoms associated with increased sympathetic nervous system action such as palpitations, sweating, trembling, shortness of breath, chest pain, nausea and abdominal distress, dizziness, derealisation, fear of losing control, fear of dying, paraesthesia (numbness), and chills or hot flushes. Obsessive-compulsive disorder is characterised by obsessions that cause anxiety and compulsions that help to relieve the anxiety. Post-traumatic stress disorder is re-experiencing a traumatic event, with flashbacks or nightmares resulting in increased sympathetic arousal, whereas acute stress disorder comprises similar symptoms experienced directly after a traumatic event.

Mood disorders are divided into two distinct areas. Major depression is characterised by one or more major depressive episodes lasting more than 2 weeks and a less-normal response to a life event such as the death of a spouse. There is a distinct

loss of interest or pleasure in life, accompanied by difficulties with appetite, sleep, energy levels, feelings of worthlessness or guilt, difficulty concentrating, self-blame, and possible thoughts of death or suicide.

Bipolar disorder shows one or more mixed or manic episodes, most often accompanied by a major depressive episode. The manic phase is characterised by an elevated, expansive, or irritable mood lasting more than a week. This may include inflated self-esteem with grandiose delusions, decreased need for sleep, increased speech and flow of ideas, distractibility, and excessive involvement in goal-directed activities or pleasure-seeking activities with no regard for negative outcomes.

Schizophrenia is characterised by episodes of psychosis with delusions and hallucinations. There is dysfunction with interpersonal relations, work or education, and self-care. Patients may display inappropriate emotions (disorganised type), anhedonia (lack of pleasure), social withdrawal, difficulty concentrating, depersonalisation, derealisation, motor abnormalities (including left/right confusion and poor motor skills), and a lack of insight into their condition, which predisposes treatment noncompliance. Neurophysiological findings include slow reaction times and abnormalities in eye tracking, either as a symptom of schizophrenia or medication-induced.

The childhood disorders commonly labelled as PDD and ADD may be considered more developmental rather than mental disorders, as filling in the developmental gaps may provide significant improvements in symptoms. They are characterised by impairment in social interaction, communication, and stereotyped behaviours. Attention-deficit/hyperactivity disorder is characterised by significant symptoms of inattention, disruptive behaviour, and hyperactivity with impulsiveness. Tic disorders, including Tourette's disorder, show an onset before 18 years of age, with multiple motor and vocal disorders (although not always concurrently) that occur many times per day nearly every day, for a period of more than a year, without a break of more than 3 months. They cause impairment in functioning within social and occupational areas.

Visual Acuity

As the severity of a person's mental health problem increases, their access to appropriate eye care decreases.[1] Studies assessing habitual visual acuity of psychiatric inpatients report 65% of patients with impaired distance visual acuity of less than 6/9. Half of the patients who had previously worn glasses or contact lenses no longer wore them because they had been lost. Of the patients who were aware of the vision problem and reported it themselves, 88% showed impaired visual acuity.[1] A study based in Hong Kong showed 75% of the sample of mental health patients with impaired visual acuity, 39% of those being due to myopia.[7] Although this is consistent with the general population, these visual problems are not as well addressed within the population of patients with mental health dysfunction.

Patients with schizophrenia in particular have increased risk of visual impairment for both far and near visual acuity, whereas patients with affective disorders do not differ from the general population in terms of decreased visual acuity.[8] There is a five-fold increased risk of decreased distance vision and a six-fold increased risk of decreased near vision with schizophrenia.[8]

Although decreased visual acuity is most commonly from refractive causes, it may also arise from ocular pathology.[8] The risks of macular degenerative changes and cataract are increased by many co-morbidities in mental health patients, such as smoking, the metabolic syndrome of hyperglycaemia (diabetes) and hyperlipidaemia, and hypertension (often due to increased body mass[1,2,8]), increased affinity to bright light and possibly medication side effects.[2]

Spatial Awareness, Empathy, Memory, and Mental Imagery

Schizophrenia (SZ) has been associated with deficits in allocentrically referenced spatial awareness (the ability to understand, to organise, and to manipulate space between objects where we remain still); whereas, egocentric spatial awareness (the ability to understand, to organise, and to manipulate space when using ourselves as a reference point moving around within that space) remains intact.[9,10] Additionally, SZ patients show deficits with explicit memory, whereas implicit memory remains intact. Explicit memory is that which we consciously work to remember. It is our declarative memory and is used to remember information, where things are, and things that are important to us. Implicit memory is that which we use without real conscious thought: the song we sing a few hours later after hearing it on the radio, or an activity that we have been taught some time ago and remember without thinking about it, such as swinging a tennis racket.

SZ patients also lack empathetic abilities (feeling the same emotions as another person); rather, they show better ability for sympathy (acknowledging the feelings of someone else). Thirioux et al.[11] showed that the most affected patients with higher negative symptoms (lacking in empathy and emotion) showed deficits in empathetic or allocentric concepts. The ability to use mental imagery and to translate this to one another's visual spatial memory capacity is greater in SZ patients when egocentric references are made, relating one's position to others.

Empathy is a major part of social interaction. It is difficult to make the connections that lead to fulfilling social interactions without it. This in itself may increase the impact that mental illness has on an individual, as they become further withdrawn from others. SZ patients show poor recognition of facial emotional expressions and their feelings (as is also the case with individuals on the autism spectrum). Paranoid symptoms may also be associated with a lack of

understanding of the feelings and reactions of others, an over-reaction to action or thoughts. In contrast, empathy may also lead to depression if the individual is overly concerned with the feelings of others.

These various aspects may all inter-relate if we think about the terms empathy, memory, and spatial awareness with regard to egocentricity and allocentricity. All of these aspects are deficient in SZ when the patient cannot relate their own ego to the situation. This begs the question as to whether therapeutic training of skills of spatial orientation and mental imagery from a more allocentric point of view would be useful in these patients.

Visual Perceptual Dysfunction

A study by Flach and Kaplan et al. (a psychiatrist and an optometrist, respectively) evaluated visual perceptual dysfunction in both patients with SZ and those with affective disorders.[12,13] They regarded accommodation and convergence dysfunction as signs of visual perceptual dysfunction. Both groups showed deficient findings of binocular vision and depth perception (amplitude of accommodation; esophoria or exophoria; suppression during vergences and base out and base in fusional ranges) compared to a control group.

Esophoria and exophoria findings were significantly increased in both SZ (69%) and affective disorder (74%) patients compared to 32% of controls, although only at near distances. Reduced near-point fusion occurred twice as much in patients with affective disorders (65%) compared to controls (27%). Vergence suppression (where one eye breaks without the patient noting diplopia) occurred nearly 6 times more in SZ patients (35-58%) and 4 times more in those with affective disorder (30-35%) compared to controls (5-11%). A crossed disorganised left apex of the Van Orden star was found in 46% of SZ patients and 17% of patients with affective disorder, compared to 7% of controls.

Significant abnormalities were also noted during both pursuit and vergence tracking assessments. Reach, grasp, release, and re-grasp tasks were assessed in vergence tracking movements. Both the SZ and affective disorder patients showed inefficient saccadic eye movements, with head movement and loss of fixation, quitting of the task, or locking fixation at one point and then looking away. Difficulties were more noticeable in the affective disorder patients when standing rather than seated, suggesting that posture and gravity play a part in saccadic eye movements. An inability to isolate eye movements from head movement is normal in early childhood, suggesting that visual development may be immature or regressed in these patients. This also may be indicative of the time at which the visual problem and/or mental dysfunction began. A more embedded and profound problem may have begun earlier, whereas a less embedded problem may have occurred after a later-onset incident.

Another study showed higher incidence of convergence insufficiency (CI) associated with behavioural and emotional problems[14] and SZ.[15] Children aged 9 to 17 years old exhibiting a higher score on a child behaviour checklist showed a significant improvement in anxiety, depression, somatic, and internalising problems following treatment of CI.[14] SZ patients showed significantly reduced fusional reserves and reported more symptoms associated with CI.

Larger errors in saccadic localisation are found in SZ patients compared to controls.[16] This appears to be due to impaired signals between motor structures and sensory brain regions, resulting in reduced accuracy of ongoing eye movements or adjustments to an eye movement. Too much neural noise can dull sudden changes in neural activity. Excessive stimulation or hypersensitivity may result in two outcomes:[3]

- Noise – this overemphasises figure to the detriment of ground (too central, focal, field independent) or overemphasises aspects of ground to the detriment of figure (too peripheral, global, or field dependent).
- Crowding – too much information within the ground, which creates difficulty attending to the figure. Crowding may be thought of as input overload.

Efficient and accurate saccadic responses rely on balanced central/peripheral integration. The ability to attend to the target of interest within our peripheral field is what allows us to locate and to respond with an accurate saccade. Benefits to the patient may be achieved by practising vision therapy activities that work on this skill, such as Look, Ready, Touch, Back, and CP (central/peripheral saccades).

Dysfunction of saccades as a function of early-stage visual processing in patients with SZ may indicate dysfunction within the magnocellular (M) visual pathway.[17] Motion detection deficits and detection of low-contrast and low-spatial-frequency stimuli are indications that the M pathway input to the dorsal visual stream is compromised. This leads to difficulties with higher visual processing deficits, such as digit symbol recognition.

Patients with SZ also exhibit increased rapid eye movements during sleep, indicating tension and an overactive limbic system. This is relative to slow eye movements that indicate relaxation and deep sleep. They appear to have a heightened state of arousal despite exhibiting symptoms of apathy and withdrawal.[18]

Smooth pursuit eye movement dysfunction (SPEM) has been shown to be a genetic trait in patients with SZ.[17] Dysfunction is shown as saccadic intrusions and compensatory saccades that occur when the eyes lag behind the movement of the target.[19,20] Over half of patients with SZ show SPEM (52-86%) compared to 22% of other psychotic disorders, 21% of non-psychotic disorders, and only 8% of normal control groups. Forty-five percent of first-degree relatives of SZ patients show SPEM dysfunction and tend to show at least one parent with the dysfunction, whereas this is not the case in relatives of patients with bipolar or major depressive disorder. Students exhibiting SZ-type symptoms,

but not those with affective-type symptoms, also showed dysfunctional SPEM.

Anti-saccade dysfunction has also been found in patients with SZ and positively correlates to poor working memory findings.[19] Anti-saccades may also be found in patients with OCD or bipolar disorder, although less commonly. Neuroimaging suggests that such dysfunctions are due to abnormalities within the brain's frontal lobe and the connection with cortical and subcortical areas.[19]

Express saccades, small saccades that occur just before a saccade is generated, occur more frequently in patients with SZ.[19] This may relate to an unstable fixation system and possibly high incidence of distractibility. In this instance, this finding is distinguished from patients with bipolar disorder, where the incidence of express saccades is within normal limits. Interestingly, people with dyslexia also show increased express saccades, as do people with frontal brain lesions.

Using the Van Orden Star to Analyse the Visual Process

Kaplan uses the Van Orden Star as a way of analysing projected spatial behaviour.[20] If the left apex is crossed and disorganised, as reported in his analysis, does this suggest that spatial processing (which is considered the domain of the brain's right hemisphere) is also disorganised? This correlates to SZ difficulties with spatial processing and suggests that esophoria or exophoria is a result of the spatial processing deficit (accuracy in where the eyes are pointed in relation to the target within our visual space) rather than the problem in itself. When the Van Orden drawings end above the line without a definite apex, Kaplan claims that this shows a more severe problem with the 'where' part of our visual system – also known as peripheral, ambient, or magnocellular processing. When the right apex is mismatched to the left apex of the Van Orden Star, this suggests a problem with both the 'where am I?' and 'where is it?' processes. In these cases, there is a problem with both orientation within personal space and organisation of the spatial world, which may translate to more significant and longstanding psychological symptoms.

SILO or SOLI?

The perception of SILO (smaller in, larger out) or SOLI (smaller out, larger in) may show us the effect that our vision has on perception.[3] During vision therapy, we frequently encounter patients who experience SOLI rather than SILO. They see the image of the target as smaller but insist that it is further away. We may see them move their hand to the point in space where the image of the target really is, yet their mental logic overrides their visual perception. Forrest discusses the SOLI response as often occurring in a person with a more internalised perceptual reasoning, basing their response on past experience. They often present with more rigid attitudes and dislike change. They would prefer their current state to stay as it is.

It is interesting that there are changes in visual perception experienced by patients with mental and emotional disturbance. These include visual hallucinations in SZ, concentration impairment in depression and anxiety (one may take this as meaning inability to concentrate on the task at hand – tired and strained eyes, fatigue, and problems tracking), and spatial dysfunction in states such as claustrophobia. Flach and Kaplan also classified the severity of visual dysfunction in relation to psychiatric diagnosis.[13]

- Level 1 showed a vergence dysfunction with possible accommodation dysfunction. These patients showed orthophoria to low distance phoria and low ductions, medium to high near phoria and ductions, absence of expected near blur and medium near fusion, normal pursuit tracking, and low to medium vergence tracking dysfunction.
- Level 2 increased the above findings to spatial organisation dysfunction and also showed medium to high far phoria, medium to high ductions, and medium far fusion. These patients had begun to reduce their depth perception to a 2-dimensional world – a loss of perceptual consistency.
- Level 3 showed spatial orientation dysfunction now with loss of developmental interaction between body spatial orientation and visual spatial orientation – a serious lack of ability to determine vision and position. These patients also revealed poor pursuits while standing, midline shift and avoidance, and vergence tracking without being able to move the head or body simultaneously.

As discussed previously, patients with SZ have reduced allocentrically referenced spatial awareness but relatively intact egocentric spatial awareness. These findings of higher-level spatial orientation dysfunction correlate to these findings. Spatial visual development goes through the above stages of spatial orientation (where am I), spatial organisation (where is it), and finally vergence and accommodative flexibility. Dysfunction in these areas may show as a regression in these skills that possibly results in an associated mental and emotional disturbance. These may also be visual skills that never developed well in the first place, also impacting emotional development.

No patients with mild disorders, such as generalised anxiety, showed level 2 or 3 dysfunctions. Sixty-six percent of patients with SZ, major depressive disorder, and alcohol dependence showed significant visual dysfunction of level 2 or 3. Patients with chronic mental illness had a very high incidence of 82%, compared to only 11% of those with a recent diagnosis within 6 months. It is feasible that visual dysfunction may build on previous visual dysfunction as psychiatric episodes occur. There was also a correlation between social and occupational problems as the incidence of visual perceptual dysfunction increased. Of diagnosed patients, 12.5% were able to interact

effectively in social situations and work efficiently. However, 99% of those patients with impairments at work were unemployed or socially withdrawn also revealed level 2 or 3 visual perceptual dysfunction.

These findings show a very high incidence of visual perceptual dysfunction in patients with significant mental disturbance. A study from 2014 suggested that basic underlying visual dysfunction may be the trigger for several unconnected visual and cognitive aberrations in SZ.[21] The authors used checkerboard visually evoked potentials (VEP) and found that in 100% of their sample of 48 SZ patients, VEP was localised to dorsal (magnocellular) and ventral (parvocellular) brain areas, deviated from a normalised population. Positive symptoms, those that appear with SZ, such as hallucinations and paranoia, were more related to sensory processing defects. Negative symptoms such as emotion and empathy, which appear to be deficient in SZ, were more related to perceptual processing defects. Sixty percent of the VEP defects were found in the dorsal pathway and the right hemisphere, consistent with other studies that show magnocellular and spatial dysfunction. They were also able to establish a link between symptom severity and neural dysfunction. They stated that using VEP testing may be a way of diagnosing patients with SZ with extreme accuracy. They also noted that only sighted people have been diagnosed with SZ; no reports have been made concerning people born blind or developing blindness shortly after birth with SZ.

Mental illness was found to develop 3.1 times more frequently in children with exotropia, and there was an increased risk in children with intermittent exotropia, whereas there was no increased risk in children with esotropia compared to controls.[22] Another study showed that children with strabismus had a higher prevalence of emotional or psychotic disorders.[23] These subjects also had an increased risk of educational difficulties and tasks involving visual perception. These studies suggest that visual perception and spatial difficulties, which would include strabismus, may directly increase the risk of mental and emotional disturbances. This may be consistent with the study by Flach and Kaplan[14] and their use of the terms spatial organisation dysfunction (level 2) and spatial orientation dysfunction (level 3) occurring in psychiatric patients with visual perceptual dysfunction.

Vestibular Symptoms in Anxiety and the VOR

Recent research has investigated the effect of anxiety on depersonalisation and derealisation symptoms in patients with vestibular issues.[24] Depersonalisation is the feeling that a person's thoughts and feelings are not their own, as if that person were watching themselves from the outside. Derealisation is dissociation from one's environment. These symptoms affect twice as many men as women and are thought to be the mind's way to keep the person from experiencing severe anxiety. In vestibular patients, these symptoms were found to be more frequent and severe in patients with anxiety than those without.

The vestibular-ocular reflex (VOR) provides stability in vision with head movement. It detects rapid changes in head movement and accommodates with rapid changes in eye movements. The VOR gain is the relationship between head movement versus eye movements and should be 1.0. A higher VOR gain was found in patients with anxiety,[25] suggesting that patients with anxiety may have increased vestibular sensitivity. Anxiety and vestibular-visual dysfunction can work both ways. Although anxiety and depression may be the mechanism for symptoms such as floating, rocking, and spinning inside the head,[26] the possibility of anxiety and depression as a result of visual-vestibular dysfunction should also be considered. For example, elderly patients with vestibular dysfunction may feel more anxious due to the increased risk of falls. This may affect their ambulation, with notably smaller shuffling steps not always attributed to gross motor dysfunction but perhaps attributable to spatial dysfunction and dizziness. A study within a clinic specialising in dizziness found that 20% of patients with a psychogenic diagnosis with dizziness often experienced acute bouts of anxiety or panic attacks.[26]

Apart from standard oculomotor testing, the VOR can be assessed by measuring dynamic visual acuity, where visual acuity is measured as the head is moved from side to side[26] (unless contraindicated in patients with neck or spine problems). Acuity should be the same or no more than one line less than that of static acuity. Stability of fixation may be measured by head thrust, where the patient fixates a target in primary gaze and then rapidly moves the head to the left and to the right. A re-fixation saccade indicates that the VOR is unable to hold the gaze steady. Finally, a weakness in VOR may be seen if nystagmus is observed after shaking the head in downgaze 20 times. This is more easily done using high plus lenses to block fixation and to magnify the nystagmus.[26]

Optometrists may play a role in rehabilitating such patients by using low plus or lower minus lenses to increase the VOR gain, prisms (either yoked prism or small amounts of base in), and vision therapy. Vestibular therapy should be included within a vision therapy program.[27] Other recommendations, such as blinking during head movement, can eliminate the motion symptoms experienced. This dampens the effect of the saccadic mechanism, which can then be reset with fixation. Although standard vision therapy techniques may be used in this population, adding a dynamic environment to completing tasks will be beneficial to enhance visual stability.[26] Improving peripheral awareness is necessary, as this provides a lock for fusion and stability.

Vergence Therapy for Panic Attacks

Panic attacks are characterised by extreme anxiety of an anticipated threat, which may be a real or imagined threat on the part of the patient to themselves or to others. Bowan[28]

describes the use of vergence therapy using the visual-vagus connection as a possible treatment for panic attack. A panic attack stimulates the sympathetic nervous system associated with increased stress, which results in symptoms of increased heart rate, chest pain (non-cardiac in origin), tremor, increased breathing rate, difficulties with speech, nausea, and perspiration. The parasympathetic nervous system then overcompensates and can cause fainting due to a sudden decrease in blood pressure and heart rate via the vagus nerve. The vagus nerve supplies parasympathetic fibres to many organs, and the physiological responses are known to be fairly instantaneous. The Valsalva manoeuvre, which is an attempt to breathe out through a closed glottis, also activates the vagus nerve. This can effectively reset the vagal fibres to the heart, steadying both blood pressure and heart rate.

The oculo-cardiac reflex (OCR) is often discussed as a method that results in bradycardia, decreasing the heart rate. Knowledge of this reflex has been used in medicine and veterinary science. In medicine, care is taken during strabismus surgery not to overstimulate the extra-ocular muscles (EOM), as this results in bradycardia. Prolonged stimulation can result in asystole. Voluntary convergence stimulates the EOM insertions, especially those of the medial recti. Bowan implements oculo-cardiac convergence therapy, where a patient completes several cycles of near-to-far vergence changes from a distance target more than 3 metres away to a near target (for example a pen or finger target) about 10 to 15 cm away, holding fixation for about 2 seconds for each distance. Visualisation can be used to increase the feeling of depth between the two targets. This should be continued for 20 to 60 seconds, and if no relief of the panic attack symptoms has occurred, the usual conventional treatment should be utilised.

Streff Syndrome, Hysterical Amblyopia, and Conversion Disorder

Distance visual acuity is reduced in conditions such as Streff syndrome, hysterical amblyopia, and conversion disorder. Streff's non-malingering syndrome has various characteristics.[29] These include reduced visual acuity to 6/7.5 or worse, refractive error within the range of plano to +1.00, no change in distance acuity with refractive lenses, lowered school achievement over the preceding 12 months, reduced near vision to 6/12 to 6/24 (although usually less reduced than distance vision), reduced stereo acuity and colour vision, and reduced or constricted visual fields. Patients usually have no uniform psychological or personality pattern, and psychological signs and symptoms are thought to be secondary to the visual condition. Early adaptive syndrome (EAS) has also been discussed as being similar to Streff syndrome. EAS is characterised by the visual changes that occur early on in conditions of visual stress, with a slight decrease in distance visual acuity and significantly reduced near acuity.

The visual signs of hysterical amblyopia are very similar to Streff's non-malingering syndrome, but the cause is thought to be psychological or malingering in origin, with the visual signs being secondary in nature. Colour vision is usually normal in hysterical amblyopia but reduced in Streff syndrome. In hysterical amblyopia, there is an immediate improvement from decreased distance acuity to normal 6/6 acuity with the low hyperopic refractive correction, and subsequently, normal acuity can be achieved with plano lenses. Hysterical amblyopia was found in many cases to be the cause of visual dysfunction in soldiers after World War II.

Whereas a psychological consultation may be required for a patient with hysterical amblyopia, Streff syndrome is managed by low plus therapeutic lenses to reduce visual stress at near. Vision training should also be prescribed to rebalance the autonomic nervous system and to establish a better awareness of the visual process. The visual signs and symptoms should be resolved fairly easily without need for psychological consult. Differential diagnosis between the two conditions may be made by assessing dynamic near retinoscopy. Patients with Streff syndrome show a lag of accommodation of +0.75 or more or an unstable lag, whereas those with hysterical amblyopia may show accommodative spasm or paralysis.

Frequency doubling technology has been used to examine visual fields in patients with Streff syndrome.[16] Studies showed a general and repeatable depression of the peripheral fields in a tubular pattern. This is thought to be due to a deficit in the magnocellular (M) pathway, and the improvement in symptoms with low plus therapeutic lenses is perhaps due to stimulation of ambient vision through the M pathway.

In any case of reduced acuity and field defect, it is important to ensure that the cause is not pathological. A neuro-optometric assessment should include pinhole acuity; pupil assessment with swinging flashlight test used to diagnose optic nerve disease; macula, optic nerve head, and retinal assessment; and colour vision assessment. Visual field assessment may show tubular fields but should not have a neurological appearance such as hemianopia or quadrantanopia.

The condition of conversion disorder shows overlapping symptoms and epidemiological factors of both hysterical amblyopia and Streff syndrome. The mean age of occurrence is 8.93 years, with 70% of cases being female.[30] It is thought to occur after a psychological conflict, with studies reporting major depressive disorder, personality disorders, borderline histrionic personality disorder, and anxiety and depression as a result of childhood abuse. Signs appear to be neurological, although they are not, and include physical problems such as poor co-ordination and paralysis and sensory problems such as visual disturbances, deafness, and loss of sense of touch. The physical and sensory problems are involuntary and are not caused by malingering.

Visual disturbances in conversion disorder include a decrease in monocular or binocular visual acuity, with improvements shown in 90% of cases after 1-3 months with appropriate discussion, psychological therapy, optical lens therapy, and use of medication. Visual field defects may be either monocular or binocular, presenting as hemianopia, bitemporal or binasal defects, ring scotoma, tubular constriction, and spiral or star fields. Near vision disturbances show insufficiency of accommodation. Convergence spasm with miosis, which mimics abduction palsy, and a deterioration in handwriting may also be seen. Intermittent diplopia, perhaps seen with ocular motility dysfunction, is usually seen with other complaints that would normally be associated with a neurological cause, such as seizures, paralysis, vertigo, pain, and blurred vision.[30] A neurological consultation must rule out pathology before an optometric or psychiatric assessment takes place.

Blepharospasm, tics, and ptosis are also seen in conversion disorder, and these should be differentiated from myasthenia gravis. Unusually, psychiatric distress has been reported to occur before the onset of myasthenia gravis.[30]

If optometric treatment is required once ocular pathology has been ruled out, appropriate treatment can include the use of therapeutic or compensatory lenses, prisms, and vision therapy. Prisms may be used to redirect light into areas of field loss, with the base of the prism towards the area of loss. Fresnel prisms can be used as a temporary measure, and they may be used on several parts of the lens to expand tubular fields.[30] This may be an appropriate situation in which to implement Syntonic phototherapy. Psychological treatment should be considered in these cases, and the use of pharmacological agents may also be warranted.

Syntonic Phototherapy

Syntonic phototherapy is the use of coloured light therapy to create balance within the visual system. Rather than the colour being the treatment, the energy content of the light frequency may stimulate brain biochemistry via the retina-hypothalamus brain connection. Syntonic therapy may work by altering the timing of circadian rhythms.[31] Poor health and moods may occur if the timing of the various rhythms is out of sync.

The alpha-omega filter, ruby, is an emotional stabiliser. It can be used in cases of emotional fatigue and visual stress and for balancing the sympathetic and parasympathetic nervous systems in patients with emotional disturbances.[32] This is useful for patients with emotional exhaustion, mood swings, negative emotional affect, and poor coping ability, especially in children.[31] Typical symptoms include photophobia, transient blurred vision, asthenopia, abnormal fatigue, headache, dizziness, frustration, allergies, asthma, and fluid retention. Optometric findings include pupil release, low breaks and recoveries on ductions (especially adductions), and exophoria due to fatigue.

The emphasis of Syntonic therapy is placed on visual fields and pupillary reactions. Functional visual field loss that is not caused by pathological concerns can result from fatigue and emotional distress. These findings are not uncommonly found in children and may relate to inefficient visual abilities and difficulties with learning and performance. Narrow visual fields may also cause binocular instability, as fusion is difficult without well-overlapped fields. Patients with Tourette's syndrome show inconsistent field loss.[31]

Excessive pupillary release, where the pupil dilates only a few seconds after constriction with a light rather than the expected 10 seconds later, is not uncommon in emotionally stressed, toxic, or traumatised patients. This often correlates in severity with functional visual field loss.[31] Pupillary dilation occurs with sympathetic nervous system activation. Pupil size increases with anxiety and alertness and during problem solving, loading, mental effort, and deep empathy.[32] Excessive pupillary release may indicate that there is imbalance between the sympathetic and parasympathetic nervous systems, with the sympathetic nervous system being on high alert due to constant stress and heightened emotional state.

Syntonic therapy may also be used to improve the sensitivity of the peripheral visual system, widening the visual fields and improving the efficiency of spatial organisation and binocular vision. Use of light therapy in Streff syndrome and stress-related visual conditions showing reduced peripheral fields can enhance the visual outcomes for these patients and is especially useful before or with vision therapy.

The success of Syntonic therapy is assessed by changes in symptoms and behaviour, including mood and coping ability, academic and athletic performance, and changes in the optometric data.

In an interesting study, perhaps in conflict with the above paragraphs, researchers stated that people with panic disorder tended to be more photophobic, showing medium to high levels of aversion to bright light, and that bright light may tend to be a trigger for a panic attack. The writers then went on to state that anxiety within depression cannot be treated with light therapy, although no further specific explanations were provided.[33]

Post Traumatic Stress Disorder (PTSD) and Vision

PTSD patients suffer with disturbing flashbacks that may interfere with their daily life and cause anxiety and depression. Blurred vision is frequently reported by patients with PTSD.[34] Emotional reactions may also cause reduced peripheral awareness and glare issues. The most common ocular side effects reported are accommodative dysfunction, conjunctivitis, diplopia, and mydriasis. As TBI is observed in 17-40% of PTSD patients, this will further compromise a fragile visual system. Memories of fear arise from the part of our brain that is controlled by the hypothalamus, which regulates bodily function as well as memory, emotion, hormonal balance, the immune system, and vision. Blurry

vision has many causes, including tear film irregularities, corneal pathology, accommodative dysfunction, refractive error, intra-ocular pressure, and retinal pathology. An emotional reaction may cause blurred vision as it affects the function of the tears, cornea, and retina, with the ciliary muscle causing accommodative dysfunction.

Various treatments for PTSD have been used over the past 20 years, including cognitive behaviour therapy and eye movement desensitisation and reprocessing (EMDR).[34] This technique involves the patient making bilateral eye movements while he imagines memories of the trauma. EMDR mimics the rapid eye movements of sleep, where the brain processes memories without reliving them, allowing the memory to be integrated in a healthier manner. The memory is no longer relived; it's just a memory. However, many studies have found the efficacy of this treatment to be no better than other re-exposure treatments.

Dry Eye Syndrome

There is an increased association of dry eye syndrome (DES) in patients with depression and PTSD. Patients seen in a veterans' affairs clinic showed an increased risk of DES.[35] Women showed a higher risk than men, with 22% of women and 19% of men showing DES. In women, this may be a normal variation, as DES prevalence increases with each decade of life. A study using a large population showed an increase of 2.5% in DES with both depression and anxiety.[36] This was also compared to the prevalence of patients exhibiting DES and rheumatoid arthritis, a systemic disease well known to be associated with DES. Results were similar amongst all groups, with estimated odds being 2.8/2.9/3.2 with anxiety, depression, and rheumatoid arthritis, respectively.

Medications to treat psychiatric conditions are possibly the cause of DES. However, in more recent years, there has been a marked increase in the use of selective serotonin reuptake inhibitors as anti-depressants rather than tricyclic anti-depressants. These newer anti-depressants are thought to contribute less to DES than the tricyclic anti-depressants.[36]

Appropriate treatment of DES is no different for these patients and includes ocular lubricants, lid scrubs, and warm compresses, as well as discussion about increasing tear film quality with supplements containing omega-3.

Conclusion

Optometrists are ideally placed to help patients enhance their use of the visual process in ways that may retrain the brain and provide freedom from the constraints of mental and emotional disturbances or the development of such a dysfunction. This can be achieved by appropriate and judicious use of lenses, prisms, vision therapy, and light therapy, as well as by understanding the mind-body connection. As the ambient or 'where' visual process appears to be more dysfunctional in such patients, vision therapy with emphasis on developing spatial awareness, central and peripheral integration, oculo-motor efficiency, fusional ranges, and visualisation will be beneficial in improving visual symptoms that may be the cause or the result of mental and emotional disturbances.

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