PSYCHO OPHTHALMOLOGY: THE INTERFACE BETWEEN PSYCHIATRY AND OPHTHALMOLOGY

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ABSTRACT

The practice of ophthalmology and psychiatry meet over several aspects of patient diagnosis, management & therapy. The ophthalmologists should be able to recognize signs and symptoms of psychiatric disorder. Non organic disorders could have ophthalmologic manifestations related both the afferent system and motor system related symptoms. Ophthalmologist should be aware of conditions like functional vision loss and visual field loss, voluntary nystagmus, spasm of near reflex, non-organic disturbances of eyelid function, ocular and facial sensation and psychosomatic diseases of eye.

Many of the drugs used in psychiatry may cause ophthalmological side effects. These drugs can affect retina, optic nerve, higher visual centre, cornea, lens, ocular motor system and intra ocular pressure. Thalidomide used in 1950s was known to cause congenital ocular defects. Psychological reaction and psychiatric complications are well known after cataract surgery.

Other then these problems there are psychiatric disorders which can present ophthalmologic signs and symptoms.

Keywords: Ophthalmologic manifestations, functional disorders, functional vision loss, visual field loss, black patch psychosis, flash backs.

The practice of ophthalmology and psychiatry meet over several aspects of patient diagnosis, management and therapy. The ophthalmologist may recognize signs and symptoms of psychiatric disorder including conversion disorders and provide further evidence of non physiologic visual dysfunction. On the other hand, the ophthalmic examination may reveal signs of organic disease/dysfunction in patients with well known or presumed psychiatric disorder. Some of these findings may reflect unrecognized central nervous system pathology secondary to neoplastic, infectious or degenerative diseases. Other signs like abnormalities in eye movement control may be associated with specific psychiatric disorders (e.g. schizophrenia) and could potentially aid in diagnosis and management. The ophthalmologist may identify subtle toxic effects of psychiatric therapies on the visual system even prior to overt symptomatology. Finally, many patients of various eye diseases may have psychological reactions to blindness and eye surgery, which may need to be identified and managed. Also, there are several eye diseases with unknown causes in which psychological factors are implicated in causation.

OPHTHALMOLOGICAL MANIFESTATIONS OF NON ORGANIC (FUNCTIONAL) DISEASES

The patient with non organic visual or ocular motor dysfunction is a common reason for interaction between the psychiatrist and the
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neuroophthalmologist. It is estimated that such cases constitute up to 5% of a general ophthalmologist’s practice (Kathol et al., 1983). Psychogenic or functional visual dysfunction is never a diagnosis only of exclusion of organic factors, but also presence of positive findings of stressors or psychological factors are required to make a diagnosis. Also, it is not enough to demonstrate that the patient’s responses are non physiologic. It only helps as an adjunct and acts as confirmatory evidence of a non organic disorder. Functional disorders would include disorders currently classified by the psychiatric diagnosis of somatoform disorder, conversion disorder, factitious disorders with physical symptoms or malingering.

The patient with psychogenic complaint involving visual dysfunction may present with involvement of the afferent system (visual acuity or visual field loss or hallucinations) or the efferent system (ocular motility disorders or pupillary disturbances), eyelid position and function, corneal and facial sensation. With the knowledge of neuroanatomy, neurophysiology and a working understanding of basic ophthalmology equipment, the ophthalmologist can demonstrate the integrity of the visual system.

AFFERENT SYSTEM: MONOCULAR, BINOCULAR VISION LOSS OR VISUAL FIELD LOSS

Non organic disease that affects the afferent visual pathway is the commonest form and may be characterized by monocular, or binocular decreased visual acuity, abnormal visual acuity or both (Keltner et al., 1985). Decreased visual acuity is the most common non organic disturbance in ophthalmology (Keltner et al., 1985; Weller & Wiedemann, 1989). It occurs most often in children and young adults (Murata & Takayashi, 1992) but it may be observed in patients in 6th decade of life or older (Keltner et al., 1985). It could be psychogenic or malingering. Psychogenic visual loss is more common in children with more females being affected than males (Barric et al., 1992; Nucci et al., 1993). Malingers are usually adult males involved in motor vehicle or work related accidents seeking financial gain or compensation. Patients with non organic loss of visual acuity complain of a variable loss of vision in one or both eyes that is not explainable by refractory error, a disturbance of ocular media or other evidence of retinal or optic nerve dysfunction. Abnormal visual field or an abnormal colour perception may accompany the visual loss.

A clue that patient’s visual loss is non organic can be obtained from the patient’s history and observing the way the patient behaves during history taking. Patients who are truly blind in both eyes tend to look directly at the person with whom they are speaking, whereas patients who have psychogenic blindness often look in some other direction. Also, these patients claiming complete blindness may wear sunglasses even though they do not have photophobia and the external appearance of the eyes is perfectly normal (Miller, 1995).

There are several tests which can establish the organic nature of monocular visual loss. One set of tests work on the principle of ‘fogging’ in case of monocular visual loss. Here using the phoropter, the patient views the eye chart with both eyes open, the “good” eye is subtly fogged using concave lenses of increasing strength so that any useful binocular vision must be a result of “bad” eye function. Another way is making the patient wear glasses with one red and other green lens while viewing a chart with alternate green and red letter. Here the function and acuity of each eye can be assessed individually. Similarly, the patient can be given polarized glasses with different axes in each lens and asked to read a polaroid eye chart with some letters perceptible only to one eye or the other. This manoeuvre takes advantage of the fact that during binocular vision it is difficult to separate out what each eye sees. Stereopsis is a binocular function requiring good vision and good fusion bilaterally. The degree of stereopsis on a set of standardized test has been correlated with the minimum visual acuity required in each eye.
(Newman, 1993).

Also, in a patient claiming profound monocular visual loss, the absence of relative afferent pupillary defect (Marcus-Gunn or swinging flashlight sign) makes functional visual loss extremely likely (Newman, 1993). More difficult are the cases of binocular visual loss. Here the patient is asked to read the eye chart from bottom upwards, beginning with 20/10 line. The examiner allows significant time on each line encouraging the patient and explaining the patient that they should be able to read the chart. Frequently by the time the patient reaches the 20/20 or 20/25 lines, good vision is established. Similarly, lenses that when combined are equivalent of plain glass can be placed over the patient’s refraction while the examiner suggests that these will magnify the letters on the chart. Another feature is that functional patients frequently claim the same visual acuity when the distance from the eye chart is halved. The physiology of vision is such that if patient sees the 20/100 line at 20 feet, they should be able to see the 20/50 line at least at 10 feet.

Other less qualitative manoeuvres can be used in functional patients with severe bilateral vision loss. In the mirror test, a large mirror is rotated back and forth in a vertical axis in front of the patient. It is very difficult for a seeing patient to avoid following this moving image. Similarly, the optokinetic drum will elicit appropriate fast and slow phases of nystagmus in eyes that have at least 20/200 vision (Newman, 1993).

Additional findings which confirm nonphysiologic tendencies include failure to direct the eyes to look at their own hand and inability to touch the tips of the two index fingers together when so instructed (tests of proprioception easily passed by a truly blind person) (Miller, 1973; Thompson, 1985).

VISUAL FIELD LOSS

The most common function all visual field complaint is that of concentric loss of peripheral vision, like "tunnel vision". The field may be constricted to 5-10 degree centrally, yet the patient has no difficulty manoeuvring around objects in periphery. The classic configuration on tangent screen testing is increased (Miller, 1973; Thompson, 1985). Visual fields performed kinetically on Goldmann perimeter may show similar constriction with non physiological overlap of isopters (i.e. patient claims to see smaller less bright objects at the same place as the larger and brighter test objects). Alternatively, the functional patient may plot out a continuous spiral or a jagged inconsistent star pattern. Poor testing parameters and inconsistent responses do not differentiate between the patient with non physiologic visual dysfunction and the organic patient unable to adequately perform this test.

Other patterns of visual loss are uncommon. In functional monocular hemianopia the field defect remains same as monocular or binocular field testing, whereas in patient with organic disease the good eye visual field will compensate for most of the missing bad eye field. Patients with true bitemporal hemianopia will not be able to see objects beyond the point of fixation because they will be entirely within the missing temporal fields of vision. Functional patients with bitemporal hemianopia can rarely demonstrate this. Central scotomas or arcuate defects are unlikely manifestations of functional visual loss and should prompt a careful search for true organic pathology (Newman, 1993).

THE EFFERENT SYSTEM : OCULAR MOTILITY AND ALIGNMENT DISORDERS :

Monocular Diplopia :

The efferent visual system is less often affected in non organic dysfunction. The functional patient may complain of diplopia, which is frequently monocular in nature and ocular alignment is normal. Monocular diplopia may be seen in refractive errors, some disturbance of cornea or lens. Here one image is fairly clear and the other is fuzzy. Rare examples of monocular diplopia and more often polycopia could be seen in migraine, stroke, temporoparietal or parietooccipital lesions. Here
the usually multiple images of different shapes and sizes that do not overlap, they may be of equal clarity, however, they are of different size and shapes (Newman, 1993). True monocular diplopia i.e. two separate and equal images of an object seen with one eye only is never caused by organic disease. It is most often seen in children in stressful academic, social or family situation (Keltner, 1983).

Voluntary Nystagmus:
Another occasional functional manifestation is voluntary nystagmus. It is usually characterized by bursts of high frequency horizontal oscillations that appear to be induced by a convergence effort. The nystagmus appears to be volitional and the ability to perform these movements familial (Aschoff et al., 1976; Zahn, 1977).

Convergence Insufficiency or Paralysis:
It is characterized by insufficiency or paralysis of convergence and may be associated with insufficiency or paralysis of accommodation, although either disturbance may exist independently. Patients with apparent weakness of convergence may nevertheless show normal convergence when asked to read a paragraph at length when the eyes are alternately covered. Asking the patient to perform other near tasks such as telling time by looking at his or her wrist watch, may also be associated with normal convergence (Keane, 1982).

Spasm of Near Reflex:
It is characterized by episodes of intermittent convergence, increased accommodation and meiosis. Blurred vision and diplopia are the usual complaints. On first glance it could be mistaken for unilateral or bilateral abducens nerve palsy. However, the accompanying meiosis on lateral gaze and full lateral response to oculocephalic manoeuvres usually makes the diagnosis clear. Rarely spasm of near reflex can be caused by central nervous system disease, including head trauma, encephalopathies and drug toxicity (Dagi et al., 1987). Non organic paralysis or horizontal and vertical gaze has also been reported (Miller, 1985).

FORCED DEVIATION OF EYES IN NON ORGANIC COMA AND SEIZURE
In these patients test of oculocephalic function will help identify the non organic patients. Absence of ‘dolls eye movements’ would indicate alert and intact frontal lobe functions. Oculo vestibular testing with cold water caloric test will confirm the integrity of the nervous system (Miller, 1985).

NON ORGANIC DISORDERS OF PUPILLARY SIZE AND REACTIVITY
Occasionally, especially in a medically informed, a pupillary abnormality may be self induced. Such patients may voluntarily place a topical parasympatholytic agent into one or both eyes and then present to a physician complaining of blurred vision. It is not associated with ptosis, diplopia or strabismus. The diagnosis of a pharmacologically dilated pupil is made by placing one or two drops of 1% pilocarpine in each eye. A neurologically dilated pupil (i.e. oculomotor nerve paresis, tonic pupil) will constrict maximally within 30 minutes but, a pharmacologically dilated pupil is unaffected (Miller, 1985).

NON ORGANIC DISTURBANCES OF EYELID FUNCTION
Psychogenic ptosis though rare, has been reported (Miller, 1973). Some ophthalmologists believe that blepharospasm when not associated with overt neurologic or ocular disease is always a psychogenic disturbance. Most cases occur in children and young adults and seem to be triggered by emotionally traumatic event (Cavenar et al., 1978).

NON ORGANIC DISTURBANCES OF OCULAR AND FACIAL SENSATION
Anaesthesia or hypersensitivity of skin of
eyelids and cornea may be non-organic with the latter being associated with lacrimation, photophobia or a combination of these (Walsh & Hoyf, 1969).

**NON ORGANIC DISTURBANCES OF LACRIMATION**

Excessive secretion of tears may be non organic and may be associated with blepharospasm. Walsh & Hoyf (1969) observed a patient who produced bloody tears by depositing blood from self induced nose bleeds into conjunctival sacs.

**PSYCHOSOMATIC DISEASES OF THE EYE**

Psychological factors have been implicated in aggravating and causing certain ophthalmological disorders like glaucoma, central serous retinopathy, styes, retrobulbar neuritis and Sjogren's syndrome (Paulley & Pelur, 1989).

**OPHTHALMOLOGICAL SIDE EFFECTS OF DRUGS USED IN PSYCHIATRY**

Many drugs used in psychiatry and drugs of abuse may have action on the eyes. Various mechanisms have been described to explain the drug action, which may be as follows:

1. Absolute overdosage/relative overdosage.
2. Side effects: which are unwanted but unavoidable.
3. Hypersensitivity. It can be immediate or a delayed response.
4. Idiosyncratic reaction indicates an abnormal response unrelated to its pharmacological action occurring in a small population.
5. Intolerance - when there is a lower threshold for normal pharmacological action.

**Congenital Defects of Eyes**

Thalidomide used in 1950's as a sedative and analgesic caused microophthalmia, colobomata, ocular palsies, lacrimal anomalies and pigmentary retinopathy besides its other teratogenic effects.

**Drugs Affecting The Retina**

Phenothiazenes are known to affect the retina. Piperidine phenothiazine, thioridazine, produces pigmentary retinopathy in doses of more than 800 mg per day. Retinal arteriolar attenuation may occur, the dark adaptation, electrooculogram and electroretinogram are impaired. However, retinal toxicity has been reported in doses less than 800 mg and individual susceptibility plays a role. Pipendychlorophenothiazene was found to be highly retinotox in clinical trials and hence was withdrawn.

With aliphatic phenothiazines, like chlorpromazine, there are only isolated case reports which suggest that it could be retinotoxic. However, it has been known to cause anterior segment pigmentation including that of lens, but these do not lead to any significant visual deterioration (Hansen et al., 1997).

**Drugs Inducing Optic Neuropathy**

With some drugs like opium, morphine and disulfiram, optic neuritis has been reported only in addicts suggesting a role of nutritional factors. Other C.N.S. drugs reported to cause optic neuritis are barbiturates and tricyclic antidepressants (Hansen et al., 1997).

**Drugs Affecting Higher Visual Centres**

Stimulants like cocaine, amphetamines, cannabis, L.S.D., mescaline, pirlocrine, anticholinergics have been reported to cause visual hallucinations (Oshika, 1995).

**Drugs Affecting Ocular Motor System**

Ocular palsies have been induced by a number of drugs including barbiturates, reserpine, chloral hydrate and morphine. Ptosis will most likely be related to sympathetic blockers whereas drugs with sympathomimetic action will cause widening of palpebral fissure (Oshika, 1995).

**Drugs Affecting Cornea and Lens**

Chlorpromazine can cause pigment deposition in the cornea and lens and this effect
seems to be dose dependent. It involves granular deposits similar to those seen in skin (Oshika, 1995). It can also occur with the use of other antipsychotics. Deposition of pigments in the lens and sometimes rarely cataracts can occur. Rarely corneal oedema can also occur (Hansen et al., 1997).

**Drugs Affecting Accommodation and Intraocular Pressure**

Cycloplegia with mydriasis is a function of anticholinergic drugs. Angle closure glaucoma can occur in patients with physiologically narrow anterior chambers (Hansen et al., 1997).

**Drugs Affecting Other Eye Functions**

Toxic levels of anti depressants and antipsychotics may result in gaze evoked nystagmus. Lithium toxicity can cause gaze evoked nystagmus and consequent oscilolpsia. Toxic levels of many psychotropics particularly tricyclic antidepressants and lithium can result in visual hallucination (Oshika, 1995).

**Ophthalmology and Organic Dysfunction**

An ophthalmic examination may several times provide evidence of an organic pathology. In some disorders presenting with psychiatric signs and symptoms, early recognition of these disorders may aid in timely evaluation and institution of appropriate therapy.

Neoplasms involving the central nervous system may mimic psychiatry disorders. Upto 50% of patients with intracranial mass lesions may develop psychiatric symptoms and at times these are the only manifestation. Certain paraneoplastic syndromes like limbicencephalitis in association with small cell carcinoma of lung can present just with symptoms of depression, anxiety and personality change. As the syndrome progresses, supranuclear disorders of eye movements and eye signs of cerebellar involvement can aid in making the diagnosis.

Table 1 summarizes neuro ophthalmologic findings in neurologic neoplasms and paraneoplastic disorders with psychiatric presentation (Newman, 1993).

**Strokes may occasionally mimic psychiatric**

| Organic Disorder       | Location          | Psychiatric manifestation | Neuro-ophthalmologic finding |
|------------------------|-------------------|----------------------------|------------------------------|
| Neoplasm               | Pituitary         | Depression                 | Visual field defects         |
|                        | Hypothalamus      | Behavioural changes        | Visual field defects         |
|                        | Frontal lobe      | Apathy, Depression         | Papilloedema, Optic nerve compression |
|                        | Temporal lobe     | Personality changes        |                               |
|                        | Occipital lobe    | Visual hallucinations      |                               |
| Para neoplasic         | Limbic system     | Personality changes        | Ocular motility disturbances |

**Table 1**

**NEURO-OPTHALMOLOGIC FINDINGS IN NEUROLOGIC NEOPLASMS AND PARA NEOPLASTIC DISORDERS WITH PSYCHIATRIC PRESENTATION**
TABLE 2

NEURO-OPHTHALMOLOGIC FINDINGS IN DEGENERATIVE NEUROLOGIC AND OTHER SYSTEMIC DISORDERS WITH PSYCHIATRIC PRESENTATION

| Organic Disorder                  | Location          | Psychiatric manifestation | Neuro-opthalmologic finding |
|-----------------------------------|-------------------|---------------------------|-----------------------------|
| Degenerative diseases             |                   |                           |                             |
| Alzheimers                        | Post parietal     | Depression, personality changes | Ocular motility disturbances |
|                                   | Occipital         |                           | Visual agnosia              |
|                                   |                   |                           | Visual field defects        |
| Wilson's disease                   |                   | Mania                     | K-F ring                    |
| Multiple disease                  | White matter      | Depression, Mania, Psychosis | Optic neuropathy            |
|                                   |                   |                           | Ocular motility disturbances |
| Hydrocephalus                      |                   | Personality changes       | Papilloedema, Poor upgaze, light near dissociation |
| Endocrinopathies                  | Anxiety disorder  | Behavioural changes       | Thyroid ophthalmopathy      |
| Infection                         |                   |                           | Ocular motility disturbances |
| GPI                               | Psychosis, Dementia, Mania | Argyll-Robertson pupils | |
| HIV                               | Dementia, Psychosis | Confusional state | Infectious retinopathy |
| Medication toxicity               |                   |                           | Nystagmus, accommodating insufficiency |
| (Anticonvulsants)                 |                   |                           |                             |

Disorders as tabulated. Top-of-the basilar artery infarction can present with behavioral changes. Careful ophthalmological examination may reveal ocular motility disturbances and visual field disturbances. In a rare form of stroke involving bilateral occipital cortex there is bilateral cortical blindness and its denial.

Degenerative diseases of the brain like Alzheimer's disease can present with depression and personality change and there may be associated eye signs like ocular motility disturbances, visual agnosia and visual field defects. Similarly Wilson's disease may present with mania or depression and a slit lamp examination may show Kayser-Fleischer ring.

Table 2 shows neuro-ophthalmologic findings in degenerative neurologic and other systemic disorders with psychiatric presentation.

Psychological Reactions to Eye Disease and Psychiatric Complications of Eye Surgery

Many abnormalities displayed by the cataract patient appear as an attempt to deny illness. Some of the patients have been reported to display disturbances of spatial recall where the patient would have made distortions in recall in such a direction as to make the surroundings of the patient resemble the patient's own home. Occasionally visual hallucinations have been reported as if to deny the fact that vision was entirely absent. Paranoid reactions and hypochondriac reactions have also been noted that have been understood as expressions of an attempt to deny blindness and helplessness.

Black Patch Psychosis

It has been known for many years that patients undergoing cataract surgery are particularly vulnerable to develop a postoperative psychosis (black patch psychosis).
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It presents with delirious reactions to surgery, to visual hallucinations, elation and delusions of grandeur and sometimes paranoid reactions. Recent developments in cataract surgery have greatly diminished the incidence of this complication. The psychotic reactions observed have been transitory in nature occurring when they were bandaged and disappeared when the bandage was removed. However, cases have been reported where disturbances did not subside on removal of bandages.

It has been hypothesized that such people may be more vulnerable to the effects of sedative medications which produce disorientation and confusion. The routine use of pre and post operative sedative drugs may also play a role in production of postoperative sedative drugs may also play a role in production of postoperative delirium. The effect of covering of both the eyes, as was being practised in the past is also another important factor in causing post operative psychosis.

Factors which have been considered as important are anxiety, physical or psychological stress of the operation itself, the postoperative pain, the limitation of mobility and the uncertainty about the outcome. Advancing age increases psychological problems because of economic insecurity, loneliness, increased helplessness due to physical and mental infirmity, as well as a diminished capacity to deal flexibly with anxiety provoking situations.

Maintaining visual contact with the environment post-operatively, early ambulation, adequate diet, early discharge from hospital are helpful in preventing postoperative reaction. The treatment of postoperative psychosis requires sedation, reassurance and if necessary physical restraint to prevent injury to the operated eye. Nowadays, with the modern advances in cataract surgery, eye need not be patched or if at all only one eye needs to be patched. In case of psychosis in one eyed patient the aluminum shield with numerous perforations can replace the black patch.

An early return to the home surroundings has been found helpful in some cases. Postoperative depressive reaction should be treated with antidepressants and supportive psychotherapy (Linn, 1962; Chaudhury et al., 1992).

PSYCHOLOGICAL REACTIONS TO BLINDNESS

Psychological reactions to blindness are disbelief, protest, depression and resolution. It is known as the 'blindness reaction' and is similar to reactions to death and dying, suggesting that adjustment to blindness involves a loss or mourning (O'Mally et al., 1989). Visual hallucinations, recurrent affective responses to dreaming and waking experiences have been noted in recently blind adults suggesting a form of "phantom limb phenomena" (Fitzgerald, 1970).

OPHTHALMOLOGICAL SIGNS AND SYMPTOMS IN PSYCHIATRIC DISORDERS

Visual hallucinations are visual perceptions that are vivid and substantial and have qualities of a percept occurring in absence of any stimulus. They can occur in various psychiatric disorders like schizophrenia, mania etc. but are more common in organic psychiatric disorders. Visual hallucinations can be elementary, simple or complex. Elementary visual hallucinations are characterised by the person seeing flashes of colours or light (often caused by irritative phenomenon of visual passages); or seeing 'stars' (as a result of damage or ischemia of retina, or migraine headache onset); or zig zag lines or circular lines (caused by ischemia of occipital poles). Complex visual hallucinations are characterised by more complicated visual images. It may occur in schizophrenia along with auditory hallucinations. It can also occur in toxic confusional states, delirium of any cause including delirium tremens of alcohol. Illusions are 'mistreadings' or misinterpretation of visual information from brain. Micropsia is a visual illusion where objects appear smaller than their size or farther away and macropsia is characterized by objects appearing larger or nearer. These can occur in temporal lobe epilepsy, migrainous vascular spasm or lesions in occipital association areas.
Charles Bonnet syndrome is a special kind of vivid complex, monomodal visual pseudo hallucination occurring usually in the elderly individual in absence of psychosis and cognitive impairment. Usually the visual experiences are vivid, episodic and picturesque with the sufferer having a full insight into its unreality. Victor and Adams (1993) have described it as an ophthalmologic hallucination occurring with partial or full sensory deprivation. It has been found that it is usually associated with some kind of cerebral pathology. There is another kind of specific visual hallucination known as L'hermitte's syndrome where hallucinations are vivid and diversified and scenes move about like an animated cartoon (Victor & Adams, 1993).

Lilliputian hallucinations are those where the hallucinated objects appear greatly reduced in size. Atropine and its derivatives used for eye drops may cause Lilliputian hallucination in sensitive individuals. It can also occur in toxic metabolic states as a reaction to a variety of drugs or even in functional psychosis (Hamilton, 1985).

Visual hallucinosis is a state where a person seems to be alert and well oriented inspite of the fact that he is having visual hallucinations. Intake of L.S.D., mescaline can cause this (Hamilton, 1985). Visual hallucinations secondary to central nervous system pathology at times can be differentiated from functional visual hallucinations. In an attempt to follow moving hallucinations, the organic patient will produce normal smooth pursuit eye movement whereas the functional patient will produce a series of saccades similar to the pattern seen when a normal patient tries to pursue an imaginary object without a true target.

Sensory Deprivation

The effects of social isolation and lack of variability in information output on certain people, like truck drivers who spend long hours driving alone, explorers on sea, prisoners and patients on respirators and experimental subjects have been reported. This syndrome has number of stages from being sleepy, then irritable and fantasising to vivid visual and auditory hallucinations. This phenomenon has been implicated in plane crashes due to 'gray out' and in traffic accidents involving trucks on long journeys over monotonous super highways. Sensory deprivation has also been used to explain black patch psychosis (Soloman & Kluman, 1975).

Flash Backs

Flashbacks are characterised by spontaneous recurrence of illusions and visual hallucinations similar to those experienced during the acute toxic state. It usually occurs in those using drugs like LSD and cannabis. It occurs months after last usage of these drugs. It is also known to occur in functional states like post traumatic stress disorder (Woody & Macfadden, 1995).

Eye Signs and Psychiatric Disorders

Smooth Pursuit Eye Movements

Diefendorf & Dodge (1908) reported that some schizophrenic patients exhibited ocular abnormalities. Their work was extended by Holzman & colleagues (1974). The results indicated that 65-80% of schizophrenics and about 45% of their first degree relatives have disturbed horizontal eye movements; that the impairment is largely independent of clinical states or antipsychotic medication; and is stable and independent of voluntary efforts to improve performance; and appears to have a strong genetic component. Less than 10% of normal subjects show similar abnormalities.

Specific optokinetic nystagmus responses may be disordered in schizophrenics compared to normals demonstrating that pursuit movement abnormality may be due to cortical dysfunction. Although the implication of such findings is unclear, it promises anatomic localization of schizophrenic disturbances (Lipton & Cabcro, 1995).

Eye Blink Rate

Eye blink rates are known to be increased by giving L-dopa which is blocked by
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dopaminergic antagonist sulpiride. From a strictly
clinical perspective, increased blinking is often
seen in neurologic examination of schizophrenic
patients on or off medication (Manschrick, 1990).

OPHTHALMOLOGY IN TREATMENT OF
PSYCHIATRIC DISORDERS

Eye Movement Desensitisation and
Reprocessing
Saccadic eye movements have been used
to treat patients with post traumatic stress
disorder. The procedure involves eliciting from
clients sequences of large magnitude, rhythmic
saccadic eye movements while holding in mind
most salient aspect of a traumatic memory. It
has been reported to produce a lasting result in
reduction of anxiety and flash backs (Shapiro,
1989; Spector & Herthwaite, 1993).

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