

The Ophthalmologist: Life and Sight Preservation from the Cradle to the Grave

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It is a singular honour to be requested to deliver the Fifth Annual Faculty Lecture of the Nigeria National Postgraduate Medical College in Ophthalmology. As a foundation fellow of the College (1970) and past Faculty chairman, it gives me pleasure to observe the growth of the Faculty.

We pay tribute here to the many who laboured to ensure that we have a Faculty in the College and whose interest in its development has remained unshaken. But some are no more. May their souls rest in perfect peace.

As a professor in a university, often sooner rather than later, you will be called upon to deliver an 'inaugural lecture' in which you will concentrate on the highlights of your contribution up to that point in the time or developments in your discipline. I presented my inaugural lecture nineteen years ago in 1983, and it was primarily beamed at the university community.

TODAY the purpose of this lecture for the Postgraduate Medical College is different. It is partly to explain to 'Town and Gown' what Ophthalmology entails but primarily to raise funds to ensure the steady growth of the Faculty. It behoves us therefore to provide a menu which will accommodate different tastes - hosts and guests alike.

To ensure we have an even playing field (cricketers use this term, politicians too), my subject is centred round what the public wants to know.

Not infrequently during or after a social gathering or sometimes outside a supermarket, an acquaintance will walk up and remark.

"I don't see clearly. Suffer from headaches a lot. Could these be from my eyes? Do I need to wear or change my glasses?"

Needless to say I cannot answer such enquiries on the spot. Both suggestions from my acquaintance may be right. They could equally be wrong. Unfortunately, in my experience, before I can make an input a third question is sometimes asked.

"What is the difference between an ophthalmologist and an optometrist?"

I hope at least one enquirer is here today, while I try to answer these questions.

THE OPHTHALMOLOGIST

Life and sight preservation from the 'cradle to the grave' is the primary function of the ophthalmologist. That role carries enormous responsibilities. God gives life and sight in His infinite goodness. Ophthalmologists are tools in His hands to preserve both.

Sight protection begins with the baby in the womb. It is known that certain infections in the mother in the first three months of pregnancy, eg, Rubella virus (German measles) in a non-immune mother may present post nately as congenital cataracts or more severely as congenital Rubella syndrome. The syndrome manifests as congenital cataracts, microphthalmos (abnormally small eyes), abnormalities of the retinal pigment epithelium and glaucoma (or raised pressure in the eyes). Other features may include deafness and cardiac abnormalities. The congenital cataracts we see here in Nigeria, may not be unrelated to such infections.

Non-immune, non-pregnant, women can be immunised against Rubella. Immunisation is affordable. Better still, rubella immunisation may be combined with our Expanded Programme on Immunization (EPI). Rubella infection and transmission to the unborn is preventable.

TOXOPLASMOSIS

Congenital toxoplasmosis results from transplacental transmission of maternal infection before or during pregnancy. In the newborn child, the result is retinochoroiditis (inflammation of retina and choroid), hydrocephalus, microcephaly (abnormally small head) cerebral calcification, seizures, enlarged organs, jaundice, rash and fever. Most cases of ocular toxoplasmosis are congenital.

Toxoplasmosis is endemic in Nigeria. The defective vision complained of by my enquirer may have resulted from such an infection while still in the womb.

Other neonatal abnormalities of the eyes in the newborn-ophthalmia, neonatorum or congenital glau-

coma presenting as large eyes (buphthalmos) - may result in total or partial loss of vision, depending on the effectiveness or lack of treatment.

Disease states in the mother - diabetes mellitus, galactocaemia, hypoglycaemia, hypoparathyroidism - may predispose the child to congenital cataracts. Likewise, principally autosomal dominant inherited disorders - myotonic dystrophy, oxycephaly, Weil Marchesani syndrome - can be complicated by cataracts. An ophthalmologist will be required to remove the cataracts.

In all these disease states the ophthalmologist uses the interdisciplinary approach in medicine.

PRE-SCHOOL AGE

In the pre-school child (under 5 years), a squint may draw attention to defective vision or the presence of a lethal tumour-retinoblastoma. Prompt examination of the eyes under anaesthesia may reveal early tumour growth which requires surgical removal of the eye (enucleation) and radiotherapy.

A squint may also draw attention to a refractive error. Early correction by glasses and visual exercises may prevent amblyopia (or lazy eye).

MEASLES AND MALNUTRITION

Measles is not a blinding disease in the industrialised world or even parts of in the developing world where parents take advantage of immunisation programmes and children have a balanced diet. Where malnutrition exists, and vitamin A levels are low, an attack of measles is rapidly complicated first by keratitis, followed by secondary herpetic infection which progresses to keratomalacia with corneal sloughing. This condition is worsened by the application of peppers or traditional medicine to the eyes of the convulsing child.' The drinking of the traditional concoction of 'cow urine' causes hypoglycaemia and compounds the picture with extinction of whatever vision that could have been salvaged from cortical blindness.¹ Aaby et al (1985) from analysis of European data and detailed prospective cohort studies in West Africa have shown that *overcrowding* is an important risk factor for severe measles infection, a higher infecting dose of virus, a younger age of infection, higher fatality and corneal damage.² It is therefore, clear, that various factors are at play in corneal ulceration in measles and subsequent scarring.

Here we have a scenario where the ophthalmologist cannot be indifferent to immunisation programmes and the health education of mothers at all levels. As head of the eye team, he works with those who reduce the burden of conditions which threaten vision or may lead to irreversible blindness. The community health approach is indispensable to promoting credible eye health.

Global causes of blindness in children by anatomical site

Major site of abnormality	Estimated number of blind	Per cent
Corneal scarring	500,000	33
Phthisis bulbi retina	300,000	20
Cataract	200,000	13
Optic nerve	100,000	7
Glaucoma	100,000	7
Others		3

Ocular trauma appears to be the commonest reason for surgery in children. In children the most common cause of blindness by anatomical site worldwide is corneal scarring.³ If the rest of the globe is healthy a corneal graft is inevitable.

PTOSIS / SQUINTS

It must not be assumed that every case of squinting or defective elevation of the eyelids needs surgery. In 1978 six cases with primarily ocular myasthenia were reported.⁴ Four of the six were below the age of twenty, the youngest being 2½ years. Ptosis was a constant feature. A misdiagnosis of supra-nuclear ocular palsy or internuclear ophthalmoplegia may be made in ocular myasthenia. A 'tensilon test' resolves the doubt. Treatment is medical. orthoptic or optometric ocular exercises will worsen the condition. The ophthalmologist falls back on his medical training and knowledge to make a correct diagnosis.

REFRACTIVE ERRORS

The first warning about a child not being able to see clearly usually comes from the classroom teacher: *the child can't see the black board clearly*. This may be due to a refractive error only. Having excluded any treatable eye disease, an optometrist may prescribe glasses.

A bit of dogmatism is, however, called for here. That refractive errors are a source of headaches is on the whole a *gross exaggeration*. In children under 10, it is false. Between the ages of 35-40, Africans, Indians, and Arabs tend to lose the ability to read things at close range. This may result in headaches. Caucasians tend to lose this ability to read at a close range from about 40 years. Studies show that on visual disturbance, refractive error or ametropia constitute the major cause of visual morbidity.⁵

AGES 19-29

In the Nigerian, visual blurring in one or both eyes, at the age of 19-29 is most commonly due to uveitis⁶ - an inflammation involving the iris, the ciliary body and/or the choroid. In the Caucasian, pain, watering and photophobia (light intolerance) are present in anterior uveitis. In the Nigerian and African these may be completely absent even in the presence of intense inflammatory activity in the anterior chamber. This peak period, between 19-29 years, coincides with the period

of greatest T-cell activity, as the body suddenly becomes aware of and mounts a severe reaction against a previously tolerated antigen⁷ In pregnancy, a depressed cellular immunity may explain the recurrence of ocular toxoplasmosis.⁸

Toxoplasmosis is endemic in Nigeria^{9,10} and the vast majority of posterior uveitis encountered here are presumed to be toxoplasmic in origin. Other identified aetiological factors of anterior uveitis include lepromatous leprosy¹¹⁻¹³ tuberculosis, herpes-zoster and onchocerciasis.¹⁴

The comparative rarity of acute anterior uveitis with classical symptoms may be due to the absence of HLA-27 (genetic marker) in Africans and its presence in only 4% of African Americans and altered immunological state from malaria and parasitic infections. So far visual blurring or threat to sight has been the focus, not headaches. I am not ignoring that question.

GLAUCOMA

The adult glaucomas comprise:

1. Primary open angle glaucoma (POAG)
2. Primary angle closure glaucoma (PACG)
3. Secondary glaucomas, from a host of causes: neovascular, uveitic, lens related and traumatic, constitute the second cause of blindness next to cataracts worldwide.

Among Africans and Europeans, POAG is the commonest form of eye disease,¹⁵ PACG in Asians may present with pain, redness and visual blurring. In Nigeria, while cataracts are still the number one cause of blindness, POAG is number two¹⁶ - and specifically according to a study conducted in the former Mid-West State* of Nigeria, primary open angle glaucoma was responsible for bilateral blindness, next to cataract.¹⁷

Even though the complexities, frustration in definition, case detection, management and criteria were the focus of a faculty lecture in the year 2000,¹⁸ it would be a disservice to this community and audience if some aspects of this irreversible, symptomless cause of blindness were not discussed. The poor vision complained of that could not be improved by glasses, may be precisely due to this disease. To recall the questions put to me:

"Why can't I see clearly? Will glasses help?"
If the disease is POAG: Answer: No.

"Are the eyes the source of the headaches?"
Answer: No.

The worst is a brow ache in some, or when a drug like pilocarpine is used in its treatment, produces similar symptoms.

POAG involves a spectrum of disorders typified by a characteristic optic neuropathy and field loss in eyes with open drainage angles.¹⁹

1. Raised intraocular pressure is a major risk factor. Each eye is assigned a 'target pressure' at which the ophthalmologist anticipates damage to the optic nerve.
2. Age - prevalence increases rapidly after the age of 40 years.
3. Race - black populations have a rate 4 to 8 times higher than Caucasian populations.
4. Kinship - immediate relations. Genetic transmission is responsible for only about 5% in POAG. One gene, Myocilin/TIGR (trabecular meshwork inducible chromosome 1q 21-q31) has been identified.
5. High blood pressure, diabetes and myopia are risk factors.
6. Alcohol and smoking - evidence is still conflicting. Nevertheless be warned: alcohol and tobacco can damage vision irreversibly. From the above, it is clear that the aetiology of POAG is multifactorial. Treatment is either *medical* or *surgical*.

MEDICAL TREATMENT OF POAG

- a. Once a day, the use of B-blocker or pilocarpine will ensure greater compliance.
- b. Prostaglandin analogues increase uveo-scleral outflow; latanoprost (xalatan) is the most effective drug and has low incidence of ocular or systemic side effects.
- c. Topical carbonic anhydrase inhibitor-Dorzolamide is less effective than oral acetazolamide: Brinzolamide has fewer topical side effects.
- d. Since this is a neuropathy where intraocular pressure and independent mechanisms are at play, agents that improve ocular blood flow or are neuroprotective become relevant. It is possible in the future, that biochemical and genetic manipulation may provide us with a medical trabeculectomy.

SURGICAL TREATMENT OF POAG

Surgery in the form of a trabeculectomy remains the most common and effective surgical procedure and treatment.²⁰ In the African where the disease is aggressive and patient compliance with drug therapy unreliable and expensive, trabeculectomy is the treatment of choice. The use of antimetabolites: 5-fluorouracil FU, or Mitomycin C improves surgical success rate.

One consolation: In the very elderly patient, with or without minimal lens changes, the rate of progress of

* The Midwest State is now divided into two states, Edo and Delta.

the disease is slow enough to justify continued drug therapy. He will likely die before the disease blinds him.

SICKLE CELL DISORDER

In sickle cell disorder, vision is threatened or may be completely lost through central retinal artery occlusion, vitreous haemorrhage or retinal detachment – depending on the form inherited: homozygous SS or heterogenous SC disease, and its mode of manifestation: non-proliferative or proliferative. In the proliferative form, retinal detachment readily occurs. Where neovascular glaucoma becomes a feature, headache with visual loss is established.

HIV/AIDS

Since the first reported case of HIV/AIDS in Nigeria in 1986, the number had soared to 1.26 million in the year 2000.²¹ The presence of the HIV virus in tears, cornea and the anterior chamber (AC) calls for caution in the sterilization techniques of the eye surgeon especially during corneal grafting. Herpes zoster ophthalmicus and squamous cell carcinoma of the conjunctiva are the obvious external ocular manifestations of HIV/AIDS in the African. Besides, the combination of low grade papilloedema and headache in the HIV positive patient, even in the absence of fever and neck stiffness, should alert the ophthalmologist to the possibility of cryptococcal meningitis. This African feature may be due to the prevalence of *Cryptococcus neoformans* in the environment. Neuro-ophthalmological manifestations include, papilloedema, retrobulbar neuritis visual field defect, cortical blindness and oculomotor nerve palsies.²²

ENVIRONMENTAL

Where people contract onchocerciasis in Africa, the usual form of infection is the parasite, *Ochocerca volvulus* which is transmitted by the fierce biting fly *Simulium damnosum*. The adult worms of the parasite may live 9-15 years in the skin. The female worm produces microfilaria which invade the skin and the eyes. Disfiguring skin lesions are a feature, but in the eyes, the cornea, anterior chamber, choroid and retina, and the optic nerve are affected. The clinical features are snowflake or fluffy keratitis depending on the load of infection. If high, a sclerosing keratitis may result. Iridocyclitis may result from anterior chamber invasion with secondary glaucoma and resultant headache. Vision is further compromised by choroidoretinal lesion, optic neuritis or papillitis; blindness results.

Factors, which predispose to infection are:

- i. Fishing and setting up fishing camps on sand banks.
- ii. Farming next to infested fast flowing river banks.

- iii. Social functions at infested riversides.

Large-scale chemotherapy with Ivermectin to destroy the microfilaria, and aerial spraying with DDT for vector elimination for several miles are measures used to control the disease.

Onchocerciasis may progress insidiously over several decades. Night blindness and narrowing of the visual field occur at an early stage of the disease. Serious impairment of vision often develops more rapidly in later stages of the disease. Nigeria is the most endemic country in the world for onchocerciasis.²³

NUTRITION AND EYE DISEASE

The visual defect complained of by the patient may be attributed to diet. Various forms of processed cassava form the main diet of a sizeable proportion of our Nigerian population. Monekosso and Wilson (1966),²⁴ Osuntokun (1968),²⁵ and Williams and Osuntokun (1969)²⁶ concluded that chronic cyanide intoxication from hydrolysis of cyanogenic glycosides present in the roots of the manioc or cassava plant were the most important factor in the pathogenesis of tropical ataxic neuropathy. Visual impairment is a feature. Glasses will not correct this. Ogun State with a high intake of cassava prepared in the form of *pupupuru* is endemic for this disease. Edo State is also a focus of the tropical amblyopia and tropical ataxic neuropathy syndrome.²⁷ It is relevant to observe that the more common mode of visual impairment from chronic cyanide intoxication is damage to the relatively unprotected macula and peripheral retinal receptors within the vitreous.^{28,29}

CATARACTS

Cataracts were the subject of the guest lecture in the year 2001 by Prof. O. Osuntokun. Simply put, cataracts are an opacity of the lens of the eye, which may cause impaired vision in its early stages and reversible blindness. Cataracts are the commonest cause of blindness worldwide.

Cataracts do not usually cause headache except rarely from abnormal swelling (intumescence) and an ensuing glaucoma. Senile cataracts in old age are the commonest types seen.

Ophthalmologists round the country are being retrained to offer cataract surgery with intraocular lens implant, so that the patient may see immediately and not spend more than 24 hours in hospital. In essence, this is old age without tears.

MACULA DEGENERATION

“Why am I not seeing clearly with my glasses”?

The macula is the most sensitive part of the eye for seeing clearly. In Nigerians macular degeneration may occur early (between 19 and 29 years) and it causes

defective vision, not headache. Among Caucasians, age-related macula degeneration occurs above 65. Why it occurs so early in the Nigerian is not clear (could it be the result of chronic chloroquine ingestion?). This is an area for further research.

DEMYELINATING DISEASE

Poor vision that cannot be corrected by lenses may be the result of demyelination involving the optic nerve – the nerve of vision in optic neuritis. Optic neuritis is seen in Nigeria. Bilateral rather than unilateral retrobulbar neuritis (RBN) occurs more commonly, – the second and third decades of life being the periods of greatest affliction. Unilateral RBN occurs more often in females, where neuromyelitis optica is also seen.³⁰ The onset of visual loss is sudden and may precede or follow a transverse myelitis by a few days.

INTRACRANIAL TUMOURS

My patient who complained of headache and visual disturbances may have:

- Primary intracranial tumour glioma
- Meningioma
- Craniopharyngioma
- Pituitary adenoma
- Pinealoma
- Teratoma
- Colloid cyst of the third ventricle or primary intracranial Burkitt’s tumour.

These tumours can cause headache and visual disturbances if they produce a rise in intracranial pressure with resultant papilloedema. Visual defect occurs if they impinge on the visual pathway, the optic nerve, chiasma, optic radiation and occipital cortex. Field defects point to possible location of the tumours. Intracranial tuberculoma cannot be ignored in Nigeria. Gliomas form 45% of primary intracranial tumours, while meningiomas once believed to be rare in the African constitute 25%. The meningiomas may grow to enormous size and most are supratentorial.

Secondary tumours (metastatic) are most commonly from chorioncarcinoma in Nigeria (70%).³¹ The breast and kidneys account for about 12% each. This picture is very unlike what occurs in Caucasians, where the lungs and breast produce the commonest metastasising tumours to the brain. Unrecognized, death inevitably follows.

ERRONEOUS BELIEFS

There is a thoroughly erroneous belief in our culture that eye disease does not cause death. For brevity I shall give examples to the contrary.

1. **Stye:** Is an infection of the lash follicle; if neglected the infection may spread into the brain and cause death.

2. **Orbital cellulitis:** May result from infection in the adjoining nasal cavities may result from respiratory infection or ethmoidal sinusitis. This can become an emergency: if improperly treated the infection can spread into the cranial cavity where it can cause death.

3. **Retinoblastoma:** This is a childhood ocular tumour which may present just as a ‘simple squint’. When a child below 5 years is not looking straight or there is a white pupil reflex, an ophthalmologist’s examination under anaesthesia is obligatory. If retinoblastoma is detected, the eye must be removed to save the child’s life. Bilateral retinoblastoma may occur. Tuberculoma of the eye, however, may mimic retinoblastoma.³¹

4. **Drugs:**

i. *Retrolbulbar injection:* A local anaesthetic prior to eye surgery may inadvertently be injected into the optic nerve, meninges or CSF. Optometrists who are not trained for this procedure should not engage in it. Respiratory depression and seizures may occur.

ii. *Beta Blockers:* eg, Timoptol is used in the treatment of glaucoma (POAG), but severe asthmatic attacks and cardiac arrest may be precipitated with death resulting. An ophthalmologist should not prescribe beta blockers to patients who suffer from asthma or have a heart condition.

5. **Malignant melanoma:** When it occurs in Caucasians, it is a lethal tumour.

If my enquirer would like a summary of possible causes of his headache and visual loss, he should see the table in annex 1 for differential diagnosis and misleading signs. With headaches in particular where none of the above is relevant and medical history and examination draw a blank, he may well give you the answer himself: His girl friend has run away, or, he is very short of money.

EYE CARE PROFESSIONALS

The teaching hospitals and eye care centres, where you have the highest concentration of ophthalmologists, realize they cannot make any meaningful uptake of services’ available if they wait for patients (often blind) to come to them. Eighty per cent of the 120 million Nigerians live in the rural areas. That informed the now established practice to delineate catchment areas, where you take services to the people in the villages and local government areas. Preceding each ophthalmological congress of the society each year there are programmed forays into the community. The local populace through the community nurse or community eye nurse assembles those in need of eye care or surgery. Right there in the local cottage or district hospitals services are provided and the needlessly blind have their sight

restored. It is not the city dwellers alone that need eye care.

The ophthalmologist cannot, however, carry the burden of eye care alone. He needs trained staff at various levels. This informed the commencement of two pivotal programmes in the University of Benin and also the University of Benin Teaching Hospital - namely:

1. OPTOMETRY - In the University - The first such programme in Africa.
2. OPHTHALMIC NURSING PROGRAMME - Initiated in 1980 and the first in West Africa, this programme provides training for all states of the federation.

The Optometry Programme was set up in 1973, and was the first in Africa. It was conceived by Professor Ray Wright, of the Department of Physics, to be a joint venture between the physics department (in the Faculty of Science) and the Department of Ophthalmology in the Faculty of Medicine, my humble self being the ophthalmologist. Professor T. Bello Osagie was then, first Dean School of Medicine, UNIBEN and later first Provost of the College of Medical Sciences. The course had a modest beginning with an initial intake of six students. As a federal university and with firm university support that course has blossomed to produce optometrists for country. Two other state owned universities - Abia and Imo - now offer the course. Today there are one thousand one hundred optometrists (1,100) in Nigeria.

The degree, 'doctor in optometry' is a science-based first degree. It trains students in visual science where they engage in correcting vision by glasses, contact lenses or providing low visual aids. They are not medical personnel and are not trained to operate. To avoid difficulties in differentiation I suggested in my inaugural lecture in 1983 that an Ophthalmic Council be established to regulate the practice of optometry. Decree No 34 of 1989 established the OPTOMETRISTS AND DISPENSING OPTICIANS BOARD OF NIGERIA. Optometrists who proceed to operate on cataracts etc. are running foul of the law of the land, their professional training and ethics and may be putting the lives of their victims at risk.

If an optometrist wishes to become an ophthalmologist and be duly licensed he needs to apply to train in medicine (five years only) and thereafter, if qualified, to embark on postgraduate training for four to five years to become an ophthalmologist. To go from optometry to ophthalmology, the optometrist needs about ten years of extra training in medicine after his 'doctor of optometry' science degree.

Nigeria needs optometrists. The profession also provides employment for many who may have ended up with a science degree in physics. If their proper role in the hierarchy of eye care is adhered to they can earn

a decent living and also provide the populace with some form of primary eye care.³³ Surgery, however, is beyond their training, and they must refer patients who need total eye care to an ophthalmologist.

There are 71 optometry centres in Lagos alone, bringing none forcibly the uneven distribution of trained personnel in all grades of eye care.

I hope my enquirer now has an insight into the difference between an optometrist and an ophthalmologist. The optometrist is a science graduate. The ophthalmologist is a medically qualified person with postgraduate training in eye care.

BLINDNESS IN NIGERIA

When we consider the status of blindness in Nigeria³⁴ (annexures 2-4) the following alarming statistics stare us in the face:

In 2001:

- 1.6 million were estimated to be blind
- 4.8 million with low vision
- 6.4 million national burden of visual impairment
- 2.7 million by the year 2020 (without intervention) will be blind

The estimated manpower requirement to meet our needs³⁴ are:

- One ophthalmologist per 500,000 population
- One optometrist per 500,000 population
- One ophthalmic nurse per 100,000 population
- One primary eye care worker per 2,500 population

Mr. David Yorston³⁵ of Christian Blind Mission International (CBMI), based on his experience in Africa and elsewhere, but now practicing at Morefields Eye Hospital, London commenting on national prevention of blindness programmes stated:

Eye care professionals include not only ophthalmologists but also paramedical eye workers, eye nurses, optometrists and orthoptists: All of us are involved in prevention of blindness.

Prevention of blindness on a national scale is bound to be a political issue. Sadly, care for blind people is frequently hampered by rivalry between different eye professionals, government departments and NGOs. If we spent as much energy fighting blindness as we expend on fighting each other, we could achieve the goal of Vision 2020 by 2015.

The ophthalmologist, by his/her privileged position as the head of an eye care team we must emphasize elimination of 'avoidable blindness' using UNIBEN'S motto 'Knowledge for Service'

ANNEX I

DIFFERENTIAL DIAGNOSIS: HEADACHE / PAIN / VISUAL LOSS

1. (a) Chronic simple glaucoma: neuropathy, typical field changes
(b) Angle closure glaucoma
2. Migraine (teenager)
 - Unilateral headache
 - Flashing light/peripheral field loss (hemianopia)
 - Nausea, vomiting
 - Ophthalmoplegia
 - Isolated/recurrent
3. Giant cell arteritis (elderly 60 yrs)
 - Constant headache-vertex
 - Pain/tenderness over temporal artery
 - Sudden visual loss in one, after other
 - Visual loss, arthritic blockage to optic disc
4. Frontal sinusitis
 - Frontal pain tenderness
 - Supraorbital margin
5. Trigeminal neuralgia (elderly, severe pain behind eye)
 - Bouts- few minutes; increasing in frequency many times daily
 - If V₂ affected refer to ears/nose/throat ENT surgeon
 - If V₃ affected refer to dental surgeon
6. Intracranial aneurysm (posterior communicating artery)
 - Painful III n. palsy
 - (Painless III palsy with pupillary sparing- diabetes mellitus/hypertension)
7. Retrobulbar neuritis
 - Pain with visual loss
8. Benign intracranial hypertension
 - Headache, vomiting, papilloedema
 - No progressive focal signs
9. Refractive error
 - Presbyopia - headaches after close work

FALSE LOCALISING SIGNS

1. Cranial nerve palsies
 - VI n palsy on one/ both sides
 - III n palsy
 from:
 - a. stretching of nerves
 - b. compression by arteries
 - c. tentorial herniation compressing one III n at edge of tentorium
2. Midbrain symptoms (especially)
 - fixed dilated pupils produced by cerebellar vermis tumour

3. Cerebellar symptoms from tumour in frontal lobe

PRACTICAL POINTS

1. Glaucoma is the major cause of irreversible visual loss in both developed and developing countries.
2. Glaucoma surgery is considered when the risk of visual loss from progression of disease outweighs the risk of operative complications.²⁰

ANNEX II

STATUS OF BLINDNESS IN NIGERIA*

Table 1. National distribution of blindness in Nigeria by geopolitical zone

Zone	Reference population	Prevalence of blindness	Number of blindness
North West (NW)	24,715,400	1.14	281,755.56
North Central (NC)	21,732,200	2.02	438,990.44
South East (SE)	16,224,200	1.19	184,955.88
South South (SS)	18,014,600	0.62	111,690.52
South West (SW)	24,441,800	1.51	396,071.18
Total	120,000,000	1.3	1,563,438

Table 2. National burden of blindness in Nigeria

Zone	% of national population	% of national blindness	National burden of blindness
North West (NW)	20.6	18.02	0.87
North Central (NC)	18.11	28.08	1.5
North East (NE)	13.52	11.83	0.88
South East (SE)	12.39	11.32	0.91
South South (SS)	15.01	7.14	0.48
South West (SW)	20.37	23.61	1.16

* Sources of data: WHO Onchocerciasis Expert Committee Report; unpublished data from national sources; surveys using WHO simplified population-based assessment methodology; extrapolation.

Extrapolation from North West Zone:

- By 2001, 1.6 million estimated to be blind
- 4.8 million with low vision
- 6.4 million national burden of serious visual impairment
- 2.7 million by the year 2020 (without proper intervention will be blind).

Table 3. Distribution of population in Nigeria by age

Age	Population size
0 -15	54,000,000
16 - 44	51,600,000
45 - 58	8,400,000
>60	6,000,000
Total	120,000,000

Table 4. Causes of blindness: Nigeria

Zone	Number of blind from:				
	Cataract	Trachoma	Glaucoma	Oncho/ others	Total
NW	95,838	67,622	42,264	76,032	281,756
NC	226,665	71,409	32,265	108,651	438,990
NE	92,574	44,507	27,744	20,131	184,956
SE	88,574		29,109	59,287	176,974
SS	55,845	12,193	31,273	12,380	111,691
SW	184,538		11,456	73,077	369,071
Total	744,038	195,731	274,111	349,558	1,563,438
%	48	12.5	17.5	22	100

Note: Onchocerciasis is the cause of blindness in 0.2 million people (eye disease in onchocerciasis-endemic area of the forest savannah mosaic region in Nigeria. (Umeh RE, Chijioko CP, Okonkwo PO.)

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