Introduction

Childhood blindness affects the individual, their family, and the community. [Gogate et al. 2011] Blindness also has implications for infants' development, education, and future social, marital, and economic prospects. Nearly 75% of early learning comes from vision. Early onset visual loss can have profound consequences on a child’s motor, social, emotional, and psychological development. The World Health Organization (WHO) classifies the causes of childhood blindness according to the anatomical site most affected and the underlying aetiology.

The World Health Organization’s (WHO) system for classifying blindness and low vision in children uses two methods.
1) Descriptive classification refers to the anatomical site most affected. The following categories are used:

- whole globe (e.g. anophthalmos, microphthalmos)
- cornea (e.g. corneal scarring, keratoconus)
- lens (e.g. cataract, aphakia)
- uvea (e.g. aniridia)
- retina (e.g. retinal dystrophies)
- optic nerve (e.g. atrophy)
- glaucoma
- conditions where the eye appears normal (e.g. refractive errors, cortical blindness, amblyopia).

The information necessary for this descriptive classification can be collected on every child following examination and clinical assessment.

2) Aetiological classification, classifies blindness according to underlying cause. This method uses categories based on the time of onset of the condition:

- hereditary (at conception, e.g. genetic diseases, chromosomal abnormalities)
- intrauterine (during pregnancy, e.g. due to rubella or thalidomide)
- perinatal (e.g. retinopathy of prematurity, birth injury, neonatal conjunctivitis/ophthalmia neonatorum)
• childhood (e.g. vitamin A deficiency disorders, measles, trauma)

• unknown/cannot be determined (e.g. congenital abnormalities).

Information about underlying causes of blindness, although often more difficult to collect, is more useful for planning. (Gilbert et al. 1993)

Causes of visual loss in infants can be either prenatal (i.e., occurring at the time of conception or during the intrauterine period) or postnatal (during or after birth). Prenatal causes are congenital anomalies anophthalmos, microphthalmos, and coloboma; congenital cataract, retinal dystrophies such as Leber’s congenital amaurosis, infantile glaucoma, and congenital cloudy cornea. In the prenatal period (i.e., from the 28th week of gestation through to 1-4 weeks after birth), the following conditions can occur: cortical impairment from birth asphyxia, ophthalmia neonatorum, and retinopathy of prematurity. Postnatal conditions (i.e., those acquired after birth) are unusual during infancy. [Dale et al, 2007]

Anophthalmos and microphthalmos are congenital anomalies in which the entire eyeball is entirely absent or smaller than normal from birth. [Njuguna et al, 2009] Typical complete uveal coloboma is the most common congenital anomaly in which the embryonic fissure does not close completely by the sixth week of intrauterine life. [Sitorus et al, 2009] Coloboma can cause severe visual loss if the defect involves the macula and/or optic nerve. Surveys from schools for the blind in many parts of Asia show that these congenital eye anomalies are a leading cause of blindness and severe visual impairment in children. (Gogate et al, 2009)

These are difficult to research, even in countries with the best healthcare systems and expertise. In the majority of cases, even with in-depth molecular genetic testing and detailed investigation, no cause can be identified [Shah et al, 2011]. This is because these anomalies are likely to be due to genes controlling eye development (which are largely unknown) and possibly, gene-environment interactions, reflecting similar processes elucidated for folic acid and spina bifida. Even if an environmental agent (e.g., folate) could be identified, the intervention would have to been extremely broad to have a measurable effect, be inexpensive and safe (as it would need to be given to large numbers of people) and be nonteratogenic – a difficult proposition in developing countries. [Fitzpatrick et al, 2009]