

above factors, I developed in March 1965 a stapedectomy technique which has so far proved very satisfactory.

After removing the stapes, the oval window is closed by means of a vein graft taken from the dorsum of the foot. The gap between incus and vein is bridged by means of a spring made of stainless steel wire which could, in theory, attenuate a loud stimulus.

This spring prosthesis has now been used in over 40 cases in Malta and, except for one case which showed no improvement, all the others show marked improvement, normal or above-normal hearing being the rule. No "dead" labyrinths have so far occurred.

The spring prosthesis is also being used in the United Kingdom and in the United States of America, and, in fact, it is being produced commercially by a well known firm of surgical manufacturers.

Reports from various centres show that the spring prosthesis does all that is claimed for it, and what is more important, is that to date no sensory-neural changes in the labyrinth have been observed following its use.

Summary

The causes of sensory-neural changes in the labyrinth following stapedectomy operations are discussed. A new technique is described and its advantages outlined.

OCULAR TOXOPLASMOSIS: A report on a case discovered in Malta

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Before the discovery of toxoplasmic retino-choroiditis, all cases of fundus diseases in infants and young children were diagnosed as foetal chorioretinal infection of unknown origin, or congenital developmental anomaly of the retina and choroid

or chorioretinal birth injury.

Thanks to clinical and laboratory examinations, a good number of these cases can now be diagnosed as due to infection by toxoplasma. No case of ocular toxoplasmosis has ever been reported in Malta. The aim of this paper is to report such a one.

Toxoplasma is a protozoan parasite possessing a cytoplasm and a distinct nuclear chromatin. Its size may vary from 4 to 7 microns in length and 2 to 4 microns in width. These organisms may be present either as free parasites in the tissues or within fixed tissue cells, mononuclear phagocytes, eosinophiles and polymorphonuclear leucocytes. It is an intracellular parasite, reproducing itself in all tissue cells. Multiplication is rapid within cells, finally resulting in rupture of the cell wall. The infection may remain latent for a long period. It is poorly resistant to drying and freezing.

Toxoplasma is pathogenic in a large number of mammals and birds. Propagation can happen by the transfer of infected tissues and fluids by a variety of routes: by mouth or by conjunctival, intramuscular or intracerebral inoculation.

It is not easy to explain the mode of infection of ocular congenital Toxoplasmosis. It is thought that the protozoon reaches the ocular globe not only through the blood, but rather through neurotropism and direct propagation from the brain towards the eye via the cerebro-spinal fluid and the meningeal coverings of the optic nerve. A milk-borne infection cannot be excluded.

No evident sign of toxoplasmic infection is present in the pregnant mother because of a natural or acquired resistance to the infection.

Clinical manifestations

Infection may occur at any period of life and may or may not give rise to symptoms. The clinical findings depend upon the age at which infection happens and upon the dosage of toxoplasma received.

When the infection occurs in foetal or early infantile life, there is always the

presence of signs and symptoms and the results are widespread. In the congenital form, the infection acquired from the mother is usually at an advanced stage when the child is born. On the other hand, the infant may be born with the early signs of Toxoplasmosis when infection happens in late uterine life.

The most important ocular signs are chorioretinitis, papilloedema and optic atrophy as a result of papilloedema and chorioretinitis. Other ocular signs are microphthalmia, iridocyclitis and cataract. Among the general symptoms are to be mentioned: convulsions, hydrocephalus, cerebral calcification and microcephalus. Calcifications are the result of the precipitation of calcium in the periventricular region and in other parts of the brain, following upon encephalitis with destruction of brain tissue. Hydrocephalus is caused by the inflammation followed by obstruction of the foramen of Monroe and the region around the, aqueduct of Sylvius.

Toxoplasma has an elective tropism for the nervous tissue, brain and retina which no doubt offer favourable conditions for its proliferation. Poor diffusion of serum antibody into the brain substance and retina have also been suggested as an explanation. It has a particular virulence for embryonic tissues, as shown by relative frequency and particular seriousness of the infection in the foetus and in the infant.

The retina is the tissue initially attacked. The infection extends secondarily to the choroid. The choroid may be the first tissue to be attacked. In the majority of cases, the macular area is the site of election of the choroidoretinitis. It is usually bilateral and symmetrical. In one third of the cases, there is only one focus. It is present on one side only in 1 out of every 5 cases. It is generally agreed that choroidoretinitis in toxoplasmosis is typical and characteristic. In healed choroidoretinitis, the focus is usually large and irregular. There is a marked central atrophic area, with a pigmented border. Clumps of pigment may cover the atrophic central area.

If the infection occurs at an early stage of foetal life, pronounced changes occur in the eyes and microphthalmos may result.

A presumptive diagnosis of congenital toxoplasmosis can be made if the characteristic findings of cerebral calcifications, chorioretinitis and hydrocephalus are found. The diagnosis is confirmed, if the serological findings are sufficiently positive.

Case report

On the 21st January 1966, a girl aged 11 years was referred to the ophthalmic out-patients department for defective vision.

The patient had a history of squint when very young. Vision R.E. unaided 6/18. Vision L.E. unaided 6/60. Examination of the fundi after pupillary dilatation by homatropine showed no abnormal changes in the right eye.

The left fundus showed a square shaped cicatricial chorioretinal focus, situated on the temporal part of the posterior pole about 4 disc diameters from the optic papilla and measuring 4×4 D.D. The surface of the focus was covered by small clumps of pigment, surrounding a central atrophic area. Some sinuous choroidal vessels were visible at the bottom of the focus. The retinal vessels were normal. The temporal half of the optic disc showed a marked pallor.

Vision in the right eye could be improved to 6/9 with glasses. The vision of the left eye could not be improved.

Radiological examination of the cranium did not reveal any calcifications.

Physical examination did not show any abnormal changes. Two tests for cytoplasm modifying antibodies (the Sabin and Feldman test) carried out a fifteen days' interval were positive at 1/16 which suggested a past infection or a chronic infection of long duration.

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