The enigma of low tension glaucoma has existed since von Graefe (1857) described "amaurosis with excavation" which had all the ophtalmoscopic features of chronic simple glaucoma without elevation of the intraocular pressure. It must be noted that intraocular pressure in his time was checked by digital tonometry. Von Graefe later withdrew the concept that all excavation of the optic nerve head was due to elevated intraocular pressure.

The introduction of indentation tonometry by Schiotz (1905) established the concept that optic nerve head excavation and atrophy could be present without elevation of the intraocular pressure.

The pathology of both glaucoma and low tension glaucoma was found by Schnabel to be a cavernous or lacunar atrophy of the optic nerve. It is now known that lacunar changes in the optic nerve and brain are due to disturbances of the circulation in small blood vessels. Diurnal pressure variations have been demonstrated to be responsible for some cases of low tension glaucoma.

Congenital malformation of the optic nerve head (Fuchs) and pressure on the optic nerve from tumours of the pituitary and adjacent structures were shown to lead to excavation and atrophy of the optic nerve head. It was believed that calcification of the intracranial portion of the carotid artery pressing on the optic nerve was responsible for some cases but this change has been demonstrated in up to 50% of all the people with no corresponding changes in the optic nerve head. The association of calcified carotids with small vessels feeding the optic nerve was suggested.

Eyes with a low coefficient of scleral rigidity (usually myopic eyes) were found to have higher intraocular pressure by appplanation than by indentation tonometry (Winstanley 1959) which accounted again for some patients with this anomaly (L.T.G.).

The introduction of tonography in 1950 (Grant) saw the division of low tension glaucoma into those patients with normal outflow facility and those with chronic simple glaucoma in whom the intracocular pressure was lowered by a diminution in aqueous production arising from the compensatory or atrophic changes in the ciliary body.

Low tension glaucoma was found in 0.15% of a total population study (Hollows & Graham). Small arcuate scotomata were demonstrated in patients whose highest pressures were always below 20mm of mercury (Armaly 1966). Factors other than the intracocular pressure must be incriminated in the production of visual field changes in the arcuate area. After the steroid provocative test was introduced, it appeared that 40% of patients with low tension glaucoma responded, with a high rise in the intracocular pressure (Armaly 1967).

Reduced resistance of the optic nerve to intraocular pressure due to systemic factors was suggested (Haas 1962). The presence of a low perfusion pressure or a history of transient but severe hypotensive episodes in patients with low tension glaucoma is significant. Harrington (1959) sug-
suggested that low tension glaucoma was due
to a decreased blood flow, which was pro-
duced by arteriolar sclerosis and partial
oclusion of the optic nerve vessels.

The animal experiments of Hayreh
verified that imbalance between the intra-
ocular pressure and systemic blood pressure
altered the perfusion of the ciliary circula-
tion and the circulation in the optic nerve
head. He studied the effects of lowering the
blood pressure at different levels of intra-
ocular pressure:

Systolic Ophthalmic Artery Pressure
= 0.80 x systolic B.P. -8.63 ± 3.8

Diastolic Ophthalmic Artery Pressure
= 0.80 x diastolic B.P. +6.95 ± 3.4

When the difference between the diastolic
blood pressure and the intraocular pressure
is 10mm of mercury or less, there is
very poor filling of the vessels in the optic
disc, seen only in the temporal sector on
I.V.F.

When diastolic blood pressure is lower
than intraocular pressure, there is no filling
of the vessels in the optic disc or choroid.
(The capillary circulation at the optic disc
appears to be more precariatous with a rela-
tively high systolic B.P.) This indicates that
the extent of filling of the vessels in the
optic disc, peripapillary choroid and the
remaining choroid depends upon the differ-
ence between perfusion pressure in the
ciliary circulation and the intraocular pres-
sure — the greater the difference, the
better the filling and vice-versa. From this
study low tension glaucoma represents a
group showing a fall in perfusion pressure
without a rise in the intraocular pressure,
resulting in vascular insufficiency similar to
that caused by a rise in the intraocular
pressure with normal perfusion pressure
glaucoma). Hayreh concludes that glau-
coma and low tension glaucoma are identi-
cal processes producing ischaemia of the
optic disc and peripapillary choroid. The
low perfusion may result from narrowing
of the posterior ciliary, ophthalmic, In-
trnl Carotid artery or from systemic arterial
hypotension.

In Drance's series, 93% of patients
with low tension glaucoma showed systemic
abnormalities and haemodynamic crises;
and and low blood pressure occurred statis-
tically significantly more frequently in
them. Such crises produce the clinical pic-
ture only in the elderly and practically
never in the young. One must wonder there-
fore about an association of small or large
vessel changes which occur in the elderly
and practically never in the young. As
damage occurs in only one eye after a
crisis, there must be reasons for this asym-
metry. These may be a slightiy higher intra-
ocular pressure on the damaged side of a
partial stenosis of the carotid artery, or
may be even local changes in the small
vessels feeding one optic nerve head which
cannot be recognised clinically.

Low systemic blood pressure is also
significantly more common. The blood sup-
ply in the optic nerve is favourably influ-
enced by an adequate perfusion pressure
(opthalmic artery pressure less intraocular
pressure) and adversely by local vascular
disease. The blood supply of the nerve in a
person with a low blood pressure, particu-
larly if there is in addition a partial steno-
sis of the carotid, ophthalmic, or one of the
posterior ciliary arteries, may be suscep-
tible to small changes in the intraocular
pressure which — while statistically normal
for a population — may be twice as high
as the intraocular pressure which prevailed
in the same individual when he was
younger.

The finding of small haemorrhages on
the optic disc are usually transient and
easily missed. Such haemorrhages always
accompany ischaemic optic neuropathy
(Foulds 1969) but in these latter patients
infarction is usually total or subtotal, and
the patients present themselves because of
central visual loss or, more often, an altitu-
dinal field loss. The patients with glau-
coma or low tension glaucoma have no com-
plaints. In both conditions the haemorr-
hages probably indicate small episodes of
infarction or vascular insufficiency to optic
tissue. After such haemorrhages,
notching of the involved neuroretinal rim
has been described (Begg & Drance 1971)
and occurs some two to three months after
the haemorrhage has disappeared. The
optic nerve after the usual ischaemic optic
neuropathy becomes atrophic but rarely
cupped.
There are probably many mechanisms by which an optic nerve head may become cupped and atrophic. Some ways have been observed on groups of patients:

(a) One had a pre-existing large cup which was followed by atrophy of a segment of the neuroretinal rim after a local vascular occlusion;
(b) Notching of the neuroretinal rim after the appearance of small haemorrhage on the disc with the appearance of a corresponding nerve fibre bundle defect, and considered to be a small optic nerve infarction.
(c) A third mechanism may be inferred; a man with deep cupping of both discs developed optic atrophy on one disc after a massive gastro-intestinal bleed.

The presence of cupping in an atrophic disc does not necessarily differentiate an acute from a chronic vascular impairment.

Low tension glaucoma does not always develop progressive field defects. The lack of progression in almost all cases who had a Lumo-dynamic crisis — 99% in Drance's series — and the progression in those who did not have such a crisis's (70%) is an important finding and can be valuable in estimating prognosis. The presence of low blood pressure, vascular disease, high myopia and diabetes make slow progression of field defects likely.

**Mechanical Factors:**

(Phillips, Tomlinson, Leighton.)

The high cup/disc ratio with which a high axial length of the eyeball is associated as well as ocular tension may explain the unduly high prevalence of myopic eyes in both open angle glaucoma and low tension glaucoma.

Eyes with low tension glaucoma are significantly more myopic and have a greater axial length than open angle glaucoma. For pathological cupping and glaucomatous field defects to occur in large eyeballs, the ocular tension needs to be only slightly raised. In small eyeballs the critical level of ocular tension for the development of field defects needs to be higher to offset the lower susceptibility of the optic disc.

The difference in axial length, which would tend to make low tension glaucoma eyes relatively more myopic than open angle glaucoma is neutralised by a significantly greater vertical corneal radius in eyes with low tension glaucoma.

A significantly greater length of the vitreous body in low tension glaucoma more than open angle glaucoma indicates that the posterior segment of the eye is bigger and would tend to have a higher cup/disc ratio and therefore more susceptible to glaucomatous cupping, with the result that the ocular tension needs to be only slightly raised to produce cupping. Sjogren (1946) had observed that deeper excavation of the optic disc is found in low tension glaucoma relative to open angle glaucoma.

The retinal nerve fibres in an eye with a greater axial length and a high cup/disc ratio and probably a large disc may have less 'slack', especially at the disc, than in a normal eye; so that quite a small degree of cupping may stretch them or kink them over the edge of the disc or the cribriform plate. That there seems to be more 'slack' at the upper half of the disc may explain why the lower field is normally spared until late in the disease; the papillo-macular bundle may suffer less damage because it passes through the cribriform plate quite peripherally. Another related factor may be the tendency for the blood supply of the inferior half of the disc to be less well developed than the upper half because the foetal fissure is located in an inferior position.

The large disc which exists in the eye with a greater length may be a factor in making it more susceptible to pathological cupping than the normal or small disc. The 'force' tending to bow the disc backwards depends primarily on the pressure x area (a 10% increase in diameter will produce a 21% increase in area.) The bowing is the result of (a) bending and (b) stretching, the effect of the area being greater on the former.

**Treatment**

The treatment of low tension glaucoma is directed towards reduction in the ocular
tension by medical or surgical methods. One must bear in mind that it may be more beneficial to reduce the intraocular pressure from 40 to 25 than from 20 to 15. As it is more difficult to obtain a large reduction in the intraocular pressure if the initial pressure is low, the possibility of improving disc perfusion is much less.

In low tension glaucoma we have an ischaemic process of the optic nerve head which may be the result of many interacting factors. The search for a single cause or process which leads to this condition may therefore continue to be frustrating. Even the separation of low tension glaucoma from glaucoma proper is artificial and to some lacks significance. It seems unreasonable to think of two separate diseases occurring in those patients with classical low tension glaucoma in one eye and overt glaucoma in the other. Chronic simple glaucoma and low tension glaucoma are more likely to be manifestations of a disease process in which many factors assume varying importance in interfering with the perfusion of the optic nerve head. A clear recognition and understanding of the factors concerned in producing low tension glaucoma, and the significance of the intraocular pressure is an important, but by no means the only, factor leading to the ischaemia of the optic nerve head which may help to answer some of the uncertainties of chronic simple glaucoma itself.

References