Clinical Notes on Optic Neuritis and Atrophy of the Optic Nerve.
Clinical Notes on Optic Neuritis and Atrophy of the Optic Nerve

Having taken an interest in Ophthalmic Surgery and devoted some time to its study, I have chosen as the subject of my Thesis "Affections of the Optic Nerve which are of so much practical importance in the diagnosis and elucidation of vague and obscure cerebral disorders. This study has been the more impressed in my mind, since I have found the use of the Ophthalmoscope reveal intra-cranial disorders having a direct bearing upon the optic nerve and retina, which would have been impossible were not an every-day use of this important instrument..."
...meant made even in the ordinary routine of practice as an aid to the Clinical Study of Disease.

The appended cases which accompany my Theses are notes which I have gathered from my own observation and practice while in part studying at the Royal London Ophthalmic Hospital, Moorfields, and at the diaspore Eye & Ear Infirmary. I have not been able to follow up each case to the end as I should have liked.

The main facts, however, in the way of causation & ophthalmoscopic appearances are here indicated, which have been my aim in collecting these notes.

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M.B., C.M., Ed. 1879.
Booth's Borough Hospital, Booth, near Liverpool, Lancashire.
Cases of Optic Neuritis.
Case I.

Syphilitic Optic Neuritis

Eliza N., aged 23, married 1½ yr; four months
ago admitted into the Liverpool Eye +
Ear Infirmary on Sept. 30, 1881. Complaining
of dimness of sight.

Four years ago patient had what she de-
scribes as "genorrhea." She was at that
time pregnant and was delivered of a
child which only lived eight months.
Now had a sore throat or rash or
body (?).

Acuteemic + Stomach: has had swellings
of the legs occasionally - no sugar or
albumen in the urine - appetite fair.

Bowel regular - Teeth + Circulatory sys-
-tem good.
About six months ago she felt pains in her head, chiefly in vertex, and frontal region, and bumbled her food: these symptoms continued for some time varying in intensity. A month subsequently her sight began to fail and then gradually got worse. She went to the North Wales and was here treated for her headache for two weeks. She has been subject to headache and vomiting every morning and complained of constipation.

Her vision - right eye = \( \frac{20}{20} \) and she could read fijger 1 of the last fijger.

With her left eye, she was not able to read \( \frac{20}{200} \) but could read fijger 16.

Pupils acted regularly to light and accommo.
Case I

Syphilitic Optic Neuritis (continued)

Ophthalmoscopic Examination

R. The media were clear. Optic disc very hazy and its outline ill-defined; "woolly-striaed appearance" of disc. Vessels congested, otherwise normal.

Left Eye: Here the optic disc was scarcely visible being obscured by a cloudy swelling. The arteries appeared as mere threads, & the veins were tortuous; there were concealed in the swelling. Pelletier's edema was visible on the inner and upper quadrant overlapping the vessels.

The treatment she had on admission was:

Unquent: Hydrargyi 7f, 6 to rubbed under...
The arm fits at night, and a mixture can

trailing Potas. Solid grit and accret:

Cervix ad 3½ to be taken thence daily

Progress

Oct 4. Vision R $\frac{20}{20}$ nearly, and reads

jagers at 6 inches - Vision L $\frac{20}{30}$ barely

reads $\frac{1}{5}$ at 6 inches

Nov 15

Vision unaltered

Dec 4

Object votes of both eyes beginning
to clear up - Felt very much better.

Dec 30

Outline of disc in left eye pretty clearly defined - a tone of choroidal pig-
ment in region of disc - Patches of atrophy
choroids following course of vessels

Jan 16, 1882. In same condition.

Feb 16. Can read $\frac{1}{5}$ at 6 inches with both eyes,

and see $\frac{20}{20}$ nearly with left.
Case II.

Optic Neuritis

Kate M., 23 unmarried - House maid.

Nine months ago she noticed that her sight was failing and that she suffered from headaches & vomiting & noises in the ears.
The dimness of sight came on suddenly & gradually got worse. She also noticed that her left eye was beginning to protrude. She was admitted to the dispensary Eye & Ear Infirmary eight months ago, previous to her returning for the same complaint which was on Aug. 30, 1881. During the eight months she was under treatment she made rapid progress. She had scarlet fever when 17 years of age, followed by another attack a year or a half
afterwards. She says she had rheumatism
at the time she had the scarlet fever.
No history of syphilis - Family history good.
Her left eye is blurred - Right eye good
in this respect.

Vision

Left: \( \frac{20}{50} \) tested if 20 at 14", with difficulty.

Right: \( \frac{20}{20} \) " 14" at 14 inches.

Pupil action - no opacity of lens. Movements
of eye normal.

Ophthalmoscopic Exam:"

day:

Media clear - Outline of disc ill-defined.
Surface of disc has a "woolly appearance"
being blurred and indistinct. The arteries
are somewhat small - Veins large - no visible
pulseation in them.

Same appearances as in right - the heart's
of none not being so advanced.
Case II. (Continued)

She was ordered a mixture composed of
Hydramo 3 oz. Peper. Jodid 2Z. &
Barcit. Canthai. ad 37, Vin. mixt. daily.

Since August 30, 1827 - she was not a constant
attendant at the Hospital - I saw her again
in Dec. 16, 1829. When I found her condition
worse instead of better the following being
the notes I took at the time:

Drised hazy - vessels concealed in a patch
of exudation - vessels tortuous - outline
of discs obscured by cloudy swelling: its
edges irregular & indistinct. The Inoue
mine advanced in the pre-retinal eye (left)

Vision

left shadows: \( R = \frac{20}{200} \) read 1/8 at 14

Suffer from headache, moved in head & vomiting

In the Ophth. exam: the anterior were small & indistinct
Case III.

Optic Neuritis: Chorioiditis.

Thos. M. aged 40, admitted into the Liverpool Eye & Ear Infirmary complaining of defective vision on Sept. 13, 1884.

Noticed failure of sight seven years ago, coming on gradually, and steadily worse. Had no sore throat, or other secondary symptoms. Had smallpox 13 years ago.

Patient is married — never had any family — his wife had two miscarriages — two children dead (one after birth: the other from measles). Drinks a small amount.

Vision R 20/200 f 8 at 6 inches

d 20/200

Treatment:
Potato starch g. vii

Skt. common: arsen. 4 x

Ex aqua tur die — Blue glasses.
Case III. (Continued)

Ophthalmoscopic examination, revealed disseminated choroiditis - the optic disc hazy. Exudations in its neighborhood vessel, rather indistinct and obscured by the exudations.

Oct. 4. 1881. Improving steadily.

Vision with both eyes = 20/100.

Case IV.

Neuro-Retinitis from blow in forehead.

J. E. at 29 was thrown out of a trap on July 5, 1881, and fell on right side of his forehead - was insensible for 6 hours - vomited - had epistaxis, violent headache. When the ecchymosis of eyelids & conjunctiva
Case IV.

Subsided in the fourth day, he found he could not see into the right eye, so that he was brought up to the dispensary Eye & Ear Surgery - There was complete subconjunctival ecchymosis around the right eye - There was no wound of face, head - He could not read 20 - Central vision and inner field quite lost - Birth of the f.p.h. - Thalmoscope the optic disc and retina 4 - Tender upwards just past the macula - Later was seen very swollen, of a milk - white colour - Veins engorged - Extensive diplopia - Ring in and out partially hidden - Macula is blood-red, and apparently enlarged to four times its normal size, being of a triangular shape, apex inward.
Case IV. (Continued)

No hemorrhage - Physiological cup of disc not involved - left eye read J.

Nov. 6, 1881

Optic disc white - remains of hq. mental displacement - arteries small - veins irregularly narrowed - macula upon presents "washed out" looking patches in a deeper red ground - cannot read J. 20 - no perception of colours - only perceives light in central field - no central vision - muscles all act normally - tension normal - pupils unequal -
Optic Neuritis
(after a fall)

J. F., aged 36, admitted to Liverpool Eye Infirmary
complaining of failure of sight, on Dec. 2, 1891.

On 2nd August he fell from a height (18 ft.)
and fractured frontal bone of the right eye; became insensitive six hours after
admission to the Liverpool Sculthorpe Hospital.

He noticed failure of sight in right eye
three weeks after this & could only distin-
guish daylight.

History of syphilis eight years ago. Rather
intermediate in his habits.

On admission he could not see over 20
his dig; stereoscopic if 11 ft.?
Case V (continued)

Mr. E.

Vision R = \( \frac{20}{200} \); L = \( \frac{20}{30} \)

Ophthalmoscopic Exam. (Right Eye)

Media Clear - Fundus congested - Iris, pale, white - vessels rather tortuous - Arteries, thin and veins congested.

She left up with the exception of some old chorioiditis was normal.

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Case VI

Optic Neuritis, after, in all probability, an apoplectic stroke. Haemorrhage.

Alex. E., came to the Boston Brough Hospital on August 29, 1881, complaining of loss of sight. He is unmarried, at 41, and by occupation a fireman on board a steamer.

He came home from sea seven weeks previous.
To his consulting one, after which time he complained of having no feeling in his arm, and felt quite dizzy afterwards. During this time, he was under medical treatment, and slightly improved. Four weeks subsequently, he was exposed to wet and cold and noticed his face falling to one side and objects appearing double. He attributes all these symptoms to a stroke he received shortly after arriving from sea.

Had gonorrhea ten years ago, it has been an intermitten liver for the last twenty years. He never smoked. Had hallucination six months ago.

Visua with both eyes not 20/20 but 3/9.

Both pupils were somewhat inactive, the left one being smaller than the right.
Case VI, Continued

No opacity of lens - Media clear -

Ophthalmoscopic Exam* (both eyes)

Findus Congested - Patches of atrophied

Choroid in neighborhood of disc - Outline

of disc irregular + blurred, especially in

the left eye - Lenticir, somewhat small.

Blinks not much altered.

Co-ordinating movements were a little

abnormal - The gait, more of a paraplegic

nature yet not having the atonic character

altogether - Patellar reflex diminished -

Heart sounds full - Urine normal -

He was ordered a mixture containing

Iron +Zymaphenine - Patient never made

his appearance since -
Case VII

Fractured base of skull

Optic Neuritis

John J., joiner, Oct 26 was brought to the Bottoe Borough Hospital in an unconscious state having sustained a fracture of the base of the skull, while engaged in working on the roof of a house, from which he fell, a distance of 40 ft. on January 20, 1882.

On March 3, I examined his eye, and found the disc very irregular and cloudy with straight ill-defined margins: the retina were congested - the veins showed curvature, and were enlarged - anterior cuneus: what diminished in calibre - the neuritis appeared to be more advanced in the right eye. Vision could not well be tested. Right pupil was smaller than left islagery. Both internal recti muscles were paralyzed.
Case VIII.

**Optic Neuritis**

Mr. D, aged 42, coal porter, came to the Royal London Ophthalmic Hospital in May 1884, complaining of defective vision in both eyes.

He noticed the sight in his left eye failing him a week previously; it came on suddenly, and was prevented from following his usual employment. The day after this, he noticed that his right-eye was becoming affected. He also complained of supra-orbital pain and of his eye aching much. Digestive system good.

No ataxic symptoms. Had been a great drinker and smoker. General appearance poor and weakly. No history of Syphilis could be elicited. His could his vision be examined. Though his ignorance of the Alphabet, he could
Great fumes as at a considerable distance.
(about 3 feet from eye) - While walking he
was obliged to keep his head low down; he saw
poorly until he got about.

Ophthalmoscopic Examination

Retina somewhat congested, and particularly
the veins. Choroidal pigment diffused over
fundus - optic discs congested; margins ill-
defined, surface fulvous, and blurred.
Veins much enlarged - arterioles small (1).

He was treated with a mixture containing

\[
\begin{align*}
\text{Potato:} & \quad \text{Berberis} \quad \text{g} \\
\text{Ferrum} & \quad \text{comp. ac.} \quad \text{gr} \\
\text{Potato:} & \quad \text{Iodid} \quad \text{gr} \\
\text{Syr.} & \quad \text{curantii} \quad \text{sp.}
\end{align*}
\]

\[\text{Aequam ad } 3\text{j}. \quad \text{H.t.}\]

I did not see the patient since
Cases of Atrophy.
Extreme Atrophy of Optic discs.
Paralysis of left internal rectus muscle.

F.O., aged 31, labourer, married, was admitted to the Royal London Ophthalmic Hospital, Moorfields, complaining of loss of sight in both eyes.

Loss of sight commenced 2 years ago, by blurring left eye after taking an emetic. He noticed gradual complete failure of sight in this eye after this happened. The sight in the right eye failed him nine years ago. He was a painter for 23 years, and had dead Colic, & Must-Drop. Has been a healthy, strong man otherwise - never drunk or smoked much - no history of Syphilis - never had any family - appetito good - Bmrbs regular - never suffered from headaches or vomiting.

Papilla: reflex normal

With left eye can't distinguish light from darkness - with right eye he can count fingers when held close to the eye, & can see pretty well to go about in his town of residence (window).

Ophthalmoscopic exam revealed atrophy of both optic papilla, more advanced in the left eye, its internal rectus muscle being paralyzed. He was treated with the plan of Sodio of Potassium to the half ounce of water, it was taken as an in-patient.
June 3, 1849

Case II.

H. A. Seaman, aged 35, came to the R. D. O. H. complaining of total loss of vision in right eye, and partial loss of vision in the left.

Three years ago while steering a yacht in the Mediterranean he was seized with an attack of sun-stroke, fell down, and suddenly became insensible. On arrival at Woolwich he became somewhat better. From the time he had the attack till arrival at Woolwich he was laid up, being nearly insensible most of the voyage. He was admitted into the Union Hospital at Portsmouth having been here under treatment for the sun-stroke. When he first had the attack he could see a little with the left eye, but not with the right. He has now lost his sight in the right eye.

Family History: Good. Had gonorrhoea twelve years ago—never drank or smoked much. Had in his occupation been exposed to varieties of temperature.

Vision Left Eye: Can see letters on board at 20 feet distant but can't read them, the letters running one into the other. Can see clock at 18 ft. but can't distinguish the figures. Can count fingers at 24 inches from eye. Says he sees better in the dark and shade than in light.

Right Eye: Distinguishes light from darkness, sees objects when placed close to his eye, but faintly. Perception of vision very bad. Can't count fingers.
Case II

Continued.

**Ophthalmoscopic Examination.**

Action somewhat congested - Discs large, flat, palish grey (nearly white). Arteries, small - veins large - tendency of disc margins is still definite. The vessels seem to lose themselves as they enter the disc, which is bounded by a zone of pigment.

He was treated with:

- Fuchs fluid q.d.
- Ferri Arsenae Cit. gr. 6, and
- Sulf.: calomel. 1/2 gr. t.d.s.

The pupils in both eyes were sluggish - there were no ataxic symptoms.

This was in all probability a case of atrophy consequent to neuritis.

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**Case III**

June 1889. - R.H. H.

G. S. at 39, complained of loss of sight in the right eye.

Seven weeks previously he suffered from pains across the brow and across head - lost his right gradually eight months ago.

General health good - no history of syphilis.

No ataxic symptoms - Slight a great deal.

**Ophthalmoscopically.** The disc was found in a condition of white atrophy (right eye).

- Arteries, reduced; vessels large.
- Vision: Right 20/20; left 20/20 faintly.

Advised by Dr. Perkin,\footnote{Truch, M.D. N.Y. 1777.}
The following are two cases, where extreme atrophy of both sides was found: the patient being sisters.


And one brother blind - used to suffer from fits.

D. G. Oct 12: in same condition as H. G.

Both these two patients were of a very dark complexion - their appearance was dull and stupid, so that very little or nothing could be elicited from them to aid me in the history of the cases.

The atrophies here are probably secondary to some atrophic condition of the brain.

The cases are also of extreme rarity in patients so young. I therefore intently studied - the beyond some years ago(?).
Case VI.

A. J. aged 14; school-girl, came to the Liverpool Eye Infirmary on September 29, 1881, complaining of loss of sight. She never had any illness. Has always enjoyed good health; never sustained a blow on the head; nor has she suffered from any cerebral symptoms save occasionally from giddiness. Suffered from headache, now and then.

No other sister all healthy. Other family history good.

Dark complexion and somewhat pale. About three weeks ago patient suddenly noticed that she could not see with the left eye. She had had no pain or any other sensation to draw her attention to the eyes.

Ophthalmoscopic Examination (left eye)
Optic Papilla quite white. Vessels near Optic Disc situated.
Vision: L. = Bright light

Treatment:

Mercurial Inunction (right & morning). Oxide of Potassium & Stramonium internally.

November 15, 1881.

Case VII

E. J. admitted in July 1881, complaining of her sight failing and of pain in the head. (Emerald Eye Infirmary)

53 years of age; single. The child also bad of it, the remaining five died during infancy.

She noticed her sight failing suddenly the morning after rising from bed in her left eye - a fortnight subsequently, the right became affected.

Vision on admission:

\[ R = \frac{20}{20} \; d = \text{oakmoss} \]

Ordered a mixture containing, Sulphate of Iron, Sulphate of Magnesia, & Infusion of Onion Juice daily.

She complained of frontal headache - no history of sickness: the family, however, had a tendency to tuberculous disease - no swelling of legs - circulatory system good - time normal.

She was not seen again till January 10, 1882, when she was wrote:

Vision:

\[ R \; not \; \frac{20}{20} \]

Can count fingers at fifteen feet distant; d is same condition.

Complaints of headache occasionally.

Defect of vision towards periphery of field.

Ophthalmoscopic examination revealed telangiectasis of optic discs, definitely secondary to remote bilateral lesions; atrophy being more advanced in left eye.

Prescribed Syphoeme 1/2.
Case VIII.


W. H., age 38, married; sailor; four children living. Complains of dimness of vision in the right eye. He also says he could see nothing with the left eye until the exception of distinguishing light from darkness.

Vision in left eye was deficient since childhood.

Noticed suddenly that his vision in the right eye was deficient; this happened in the month of April.

There was no cough, but chewed a good deal of tobacco (almost eating it).

Had a chance of quinsy a few years ago.

Temperate in drink, otherwise healthy.

His wife had two miscarriages.

Patient, reflex height, exophthalmos.

Gait normal — no headache or vomiting.

Pupils, dilated, contracting to light and accommodation — media clear.

Vision on admission

\[ R = \frac{20}{200} \text{ year old eye at 12 inches.} \]

\[ D = \text{conal finger rule.} \]

Admitted Dec. 1881. 901.

December 12.

Vision

\[ R = \frac{20}{100} \text{ of 16 at 12 inches.} \]

\[ D = \text{conal fingers.} \]

Ophthalmoscopic Examination.

Both optic discs Adam's peak, the left one advanced than right - well marked.
Case VIII. Continued

Atrophic excavation in both — veins diminished; arteries diminished in caliber and tortuous in their course — the atrophy is of the grey variety, here, so I believe for many years, the result of tobacco chewing.

Case IX.

Dec. 20: 1887. Left Eye Defective

Q.B., age 43, single,ammered, complained of defective sight.

Failure of vision came on gradually 18 months ago: the left eye being attached six months previous to the right — this steady, got worse.

Syphilis while a young lad - otherwise healthy.

Used to smoke six months ago (about two nunes a week) no headache or vomiting.

Circulatory & Digestive Systems, good — urine normal

Vision

R + L — Finger family.

Ophthalmoscopic Examination:

Thyroidea = Helminths — atrophy of optic disks.

Treatment:

Otom: Iodic 0.1 gr

Peri o Duo Oint. Tinct. gr. 10
Case X.

Feb 10 1882. Discharged by G. Limerick.

**Atrophy of Optic Nerve.**

Locomotor Ataxia.

W. J., pensioned army officer, at 45, married, was admitted complaining of his sight failing.

Has two children alive and healthy. One died of Scarlet Fever. His wife had six miscarriages. Had syphilis fifteen years ago & urachal structure - Malarial Fever in India eighteen years ago - Seven months ago while reading, his sight was impaired, & his has gradually deteriorated - said he could see better with right than left eye.

Had been subjected to variations of temperature - once white abroad.

Suffers from headache, chiefly frontal - has sickness or vomiting - never had fits of any kind.

Appetite good - Circulatory System fair.

Is very unsteady in his gait - Sways to and fro when he stands with his eyes shut - has characteristic ataxic walk (heels first to ground first). Patellar Reflex absent - sensation somewhat impaired - no lightning or other pains. Complained of, naked, shooting pains in legs occasionally.

Movements of eye normal - no photic
Insult somewhat small, contracting to accommodation but not to light (Colquhoun Robertson's phenomenon)

Vision
Cannot distinguish the letters on the board - Sees to count fingers with right eye at a distance of 10 ft - With left eye counts fingers at 2 feet barely.

d = Heads 1 1/2 g at binocular - R. reads J ½ at 6 inches

Ophthalmoscopic Examination

Disc White - Outline irregular - arteries small - Patches of choroidal atrophy to be seen in the apparent meter side of the disc following the course of vessels. He was treated with Silverate of Potassium (Sulphate) and the condition has cleared up considerably.

Case XI

Feb. 17, 1882. Hospital Eye Dept.

Mary W., 51, was admitted complaining of dimness of sight in the left eye.

Family history unimportant - has had fits of any kind.

Two years ago the sight in her left eye was becoming affected, it has since got worse and she has been in the habit of using her right eye.

Before cataract she used to suffer from headaches, no sickness or vomiting. Suffer from headache,
Case XI. Continued

Headaches occasionally now since attack.
She noticed her sight getting worse while engaged in sewing. When she could see better towards the side of left eye.

Visia

\[
R = \frac{20}{40} \text{ at } 6 \text{ inches}
\]

f = fingers, seen better when held toward outer side of her field.

Optic atrophy.

Both optic discs are atrophied; more especially the left one, anterior, reduced to
Mercedes. Atrophy last & marked in right eye. (Contraction of field toward inner
side (left eye).

Treatment

Sy.: Tur. Sodiurns ups.
Potas.: Spirit. quinqu.

Small blister to nape of neck -

March 10.

Visia

\[
R = \frac{20}{30} \text{ at } 20 \text{ cm.}
\]

fingers when held toward outer side of
her field.

Field toward temporal side lost in right
eye: dots of field in left eye.

This is in my opinion a case of atrophy
due to meningitis affecting the chord-
ma & spreading along the optic nerve line,

to the intra-ocular termination of the nerve.
Case XII  
Mar: 1882  Mr. J.

64 yrs; alt. 6 ft; Cabinet Maker; failing sight eighteen months - no fever. Healthy looking. Gait unsteady (shut striker ground first), no swaying to and fro with eyes closed, standing. Pupils very small - do not act to light on accommodation. Patellar Reflex normal.

Father of eleven children, but of last four two were miscarriages, the last two died in infancy. From whooping cough. Drank very little - smoked 2 oz. tobacco per week. Never consulted a medical man before.

Vision in both eye, not 20/200.

Other signs - large white cornea.

A mixture containing Ricinoleic of Mercury to be taken twice daily, was ordered.

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Case XIII  
December 1884  Mr. J.

47 yrs; alt. 30. Skip steward - unconscious for nine days: bit his tongue. Had Sunstroke in India 8 years ago - no specific history. Feeling of numbness in legs & arms - no ataxia.

Knee reflexes normal. Drank a great deal. Failing sight soon after he had the attack of Sunstroke, worse during 1880.

Pupils equal and circular.

Fundus clear - eyes opaque - damage.


1/20; f.16; Colour perception normal.
Remarks.
Remarks

The study of Optic Neuritis and Atrophy of the Optic Nerve is one of great clinical significance, but which, however, is at present hid in a cloud of obscurity, and regarding which a great amount of controversy and discussion has of late arisen at the various Clinical Societies.

We to a certain extent have grasped the disease from an etiological-point of view, yet still there is open a wide field for further research & investigations into its true nature.

No clinical observer can dispute the great interest attaching to it, and the intracranial mischief, whether tumour or inflammatory growth, it manifests by means of Helmholtz’s wonderful discovery the Ophthalmoscope, an invaluable instrument in a physician’s armamentarium.

While admitting this, my experience has taught me how unreliable the signs of Optic Neuritis are in the diagnosis of diseases remote from the eye or even having their origin within the cranium. I have examined cases where evident and pathognomonic traces of Neuritis were to be met with, and where the affection seems to have originated idiosyncratically, a hypothesis substituting the unknown cause the more reliable symptoms; the result of or accompanying the disease being from the patient’s own history altogether absent.

Case III (Optic Neuritis) illustrates partly what I mean, where there was nothing in the history to account for her neuritis but Scarlet Fever & Rheumatism, though however
In this particular instance she suffered from head-ache and vomiting.

In Case VI (among the cases of atrophy of the optic nerve) I was rather puzzled to find out what the atrophy was due to, since there appeared to be a history of sudden failure of sight three weeks before I saw her, no cerebral symptoms present, except giddiness occasionally. Transient attacks of headache, which, however, were very slight. Family history good, and the patient herself had always enjoyed good health.

Examination of the eye revealed an atrophic disc. I can only suppose this to have been due to some previous inflammation of the brain, which probably from some extraneous or mental exertion had occurred, that the process of inflammation had spread along the optic nerve fibre, so affected the nerve, either by pressure, or that the atrophy was consequent to a previous neuritis; this is the only accountable cause I can form for myself unless some disorder of the blood was the foundation of the mischief.

Another remarkable feature in some of the cases of neuritis that I have examined has been the small degree of impairment of vision in proportion to the extent of the affection.

In this respect I have been misled, and had been under the opposite impression for some time till I found it necessary to use the ophthalmoscope in all cases.
notwithstanding the good perception of vision present.

The acuity of vision has been good and the neuritis has passed off with or without treatment.

I regret I have not taken notes of cases where there was little or no impairment of vision, though its acuity may be unaffected even with a considerable degree of neuritis. In more intense cases, however, sight being impaired or lost altogether.

I have also learnt that good perception of vision does not negate the presence of neuritis, that the affection may pass away under treatment leaving the sight good, even when there has been complete blindness.

Case I illustrates the good results produced under treatment. In this patient, a female, I suspected specific disease, & although she stated she had never had any sore throat or rash on her body, the fact of her being pregnant & miscarried, together with her general appearance, left no doubt in my mind as to her having been attacked with Syphilis.

The notes of the case show that on admission the vision in her right eye was good, but in the left eye it was seriously damaged as she could not see 20/200.

She suffered also from headache, chiefly frontal, and by dint of her feet. This lasted six months before admission, the failure
of light commencing a month subsequent to the headache.

Ophthalmoscopic examination of left eye showed that the optic disc was concealed in a cloudy swelling, as well as the vessels. Having ascertained or suspected Syphilis, I gave myself the benefit of the doubt, and put her at once under specific treatment — ten grain doses of Oxyd of Potassium, twice daily of Unguentum Hydrargyrum, to be rubbed into the.arm pits right and morning.

The marvellous effects of this course of treatment are indicated in my notes, where the discs gradually cleared up, their outline being pretty evident, the vision gradually improving, until when seen on February 6th the could see 20 with the bad eye (left) nearly.

I have seen her since and examined her discs which though not altogether free from traces of neuritis still had a clear outline, and the tendinitis had disappeared. Her vision was quite normal.

The cases of Optic neuritis that have come under my own observation have generally been double. Usually, however, I have noticed that one eye has been attacked first, while the other one has become affected at a subsequent period, the failure of vision having come on suddenly or gradually, as some of the cases will show, though it is quite probable that the neuritis may have been developing itself slowly without the
patient knew her vision was becoming impaired, or without having any well-marked symptoms to direct attention to the eyes, until the neuritis had so far advanced as to render vision almost impossible.

The symptoms which I have observed and which I value as to a certain extent diagnostic of neuritis, have been transient attacks of, or persistent headache associated with or apart from vomiting, though, in other cases, there have been absent, in all probability due to a moderate intensity of inflammatory disturbance in the nerve.

The presence of tumours in the brain does I think, intensify the headache and vomiting, when concomitant with paralysis or convulsive fits.

I have also met with cases where the neuritis has been traceable to kidney disease, and where even the diagnostic symptoms of albuminuric retinitis have been absent, whilst the urine on examination was loaded with albumen.

I believe that headache and vomiting as diagnostic symptoms of neuritis are not to be relied on in some instances, and that when present, are to be regarded as of least value.

I have not paid much attention to the examination of the field of vision, though defect in this respect usually accompanies considerable change in its acuity.

Now and then, I have observed that there is an alteration in color vision, even when
perception of right is good, some colors being lost before others, such as red and green before yellow and blue.
The presence of albumen in the urine had always stimulated me to examine eyes, and in many cases, as already stated, with negative results.

Some of the Cases which appear to resemble Albuminuric Retinitis, which I have seen, are in my opinion traceable to old-standing retinal affections spreading to the optic nerve, and leaving marks of previous inflammatory process with white marks along the vessels and striation of the edge of the disc.

In the Examination of cases the direct method should always be practised, as the general outline, surface, vascularity, and condition of the blood-vessels are seen to advantage, and many irregularity or anomaly of refraction on the part of the observer corrected, while at the same time a more exact view of the parts is revealed.

Injuries of the head are a cause of optic neuritis likewise. Case VII is that of a man who sustained a fracture of the base of his skull. His vision owing to his mental state could not be accurately tested and he seemed to have visual illusions.

Feeling, however, that there might be some intraocular mischief consequent upon the injury which was also associated with symptoms of concussion of the brain, I determined to make an ophthalmoscopic examination of his eyes, which was accomplished.
With a certain amount of difficulty on account of his restlessness, on March 5th, 1872, patient having been admitted on January 20th, 1872.

I found both of the discs cloudy with striated ill-defined margins.

I expect that in this case the neuritis set in some weeks after the receipt of the injury, and that the first stage of the affection was congestion with edema, with the neuritis as the second step. In all probability due to a previous meningitis.

The injury here affected principally the frontal bone over which he used occasionally to complain of a constant headache.

There was also well marked paralytic strabismus of both eyes, indicating damage to the Sixth nerve supplying the External Recti.

Though not described in the notes of the case, I may mention that the patient was treated with iodide of Potassium in five grain doses, thrice daily, under which treatment he improved as regards the eye complication, mental condition and vision, so that he could read small type from a book at eight inches from the eye with moderate facility.

The patient, however, was discharged at his own request, and sustained another injury to his head, which caused his death.

Case IV illustrates neuritis in the right eye, consequent on a fall. The patient having fractured the frontal bone (over the right eye).

In Case IV, the injury was on the right side of the forehead, the patient having sustained a severe blow.
I put a certain amount of stress on the site of the injury, as, I believe, it is an important element in the production of neuritis, since the nearer the injury is to the eye, such as on the anterior or lateral portions of the head, the greater the risk of laceration or other damage to the nerve.

In the cases I have quoted I could not at the time I made the ophthalmoscopic examination discover any hemorrhages, though they might have been present and been absorbed.

Case VI is interesting in so far as the ocular mischief made its appearance subsequent to a stroke, with which the patient was seized. From his general appearance and gait I was inclined to think that he had hemiplegia, and that as a consequence of cerebral embolism some softening of the brain had arisen to which in part the condition of dries present was due.

The dries appeared to be congested and assumed the form which Dr. Clifford Allbutt very ably describes as "Ischemic papilla" or "Choked disc." The dries I have seen in, as a rule, cases of injury to the head have always led me to believe in the primary stage of Congestion with swelling, the true neuritic process developing, it may afterwards as a result generally of toxic metaphasic inflammation set up in the way of meningitis.
Among my cases of atrophy of the optic disc, case I gives a history of loss of sight in both eyes - the left eye being attacked before the right, and nothing to account for the state of the disc, but the Lead Coles and "brick drop" from which he suffered while a painter. I cannot account for the loss of sight in the left eye, while straining after taking an Emetic, as altogether an explanatory of the defective vision.

I consider this one of those cases where the blindness is due to the tonic effect of the lead on the nervous system and so affecting the optic nerve. The patient's occupation as a painter for 23 years might also aid me in ascertaining the cause.

Case II is that of a patient whose atrophied disc came on subsequent to an attack of Encephalo- The never had any headaches. His vision was very bad, especially in the right eye. Ophthalmoscopic examination revealed large grey and almost white disc with narrowed arteries and congested veins. Moreover, the outline of the disc was irregular and ill defined, and this last fact makes me think that here the atrophy is consecutive to a neuritis, for I consider that if the atrophy had been primary in its nature or simple, the outline of the disc would not have been indistinct, nor even would its edge have been blurred. The characteristics of consecutive atrophy or atrophy following neuritis being that the discs here have ragged edges and their outline is indistinct.
and blurred, whereas in Simple or Primary Atrophy the edges of the disc are even or clearcut and a more brilliant appearance is presented.

Case VIII is that of a sailor who complained of dimness of vision in the right eye, which failed him suddenly. In the left eye his vision was defective since childhood.

But for a history of lepsy in 18 years ago, he had been temperate in drink. He never smoked much, but chewed a good deal of tobacco, almost eating it. As headache by vomiting, I forgot to examine his field at the time the notes of the case were taken. The atrophy was of the grey kind here.

My diagnosis of atrophy being the result of the toxic effects of tobacco may be said to be a hypothetical one, for even although restriction or defect in the field of vision especially in the center ("Central Scotoma") is supposed by many observers to be one of its main symptoms, still I am inclined to lean towards the other sides of the question and agree with those who not finding any evident or proper cause for the mecllif, but excesses in Tobacco, Venery, Alcaldy attribute these as agents in the production of atrophy of the discs in an unaccountable or inexplicable manner.

Case VII presents a history of sudden failure of sight one morning after rising from bed, in her left eye with implication of the right eye a fortnight subsequently.
The complained of frontal headaches, there was an hereditary tendency to tubercular disease in the family. Her vision as the notes well show, was very bad, as she could not see $\frac{20}{20}$ with both eyes, but could count fingers at 1½ feet distant, and there was a defect of vision towards the periphery of the field.

Both cheeks were atrophied. The question arises, could this have been due to a retro-ocular mischief? I think that the atrophy is secondary to a retro-ocular mischief, for we have a history of sudden failure of sight and frontal headaches. In the absence of other demonstrable or unknown cause I must conjecture the presence of some morbid process behind the eye, affecting the nerve indirectly or directly by pressure and thus producing the atrophy.

Case 5 is that of a man at 45, who complained of his sight failing. The only point in his history worthy of remark is his having had syphilis fifteen years ago with urethral structure, and of his wife having had six miscarriages. There was also a history of gradual failure of sight and of headaches chiefly frontal. He presented most of the ataxic symptoms, brought his heel to the ground first in walking, gait was unsteady, Patellar reflex was absent, shooting pains being occasionally complained of in the legs. The most characteristic symptoms in this case, however, was to be found in the condition
of the pupils which were small and presented the phenomenon first discovered by Dr. Argyle Robertson. The pupils contracted to accommodation, but not to light. The discs were in a state of white atrophy.

The condition of the pupils met with here are those associated with the group of Spinal atrophies of the Motor nerve.

Though usually a large number of spinal atrophies are of the grey variety, I cannot say that it was so here.

Charcot (Dictionnaire Encyclopédique des Sciences Méd.) says that almost all cases of primary atrophy are of the grey form, and that the subjects of them if they do not present special symptoms when seen, will do so at a future period.

Ulthoff (Archiv. Physiol.) observed that not more than one half of the cases of primary atrophy are associated with disease of the Spinal Cord.

I must admit that in the majority of cases of Locomotor Ataxia I have examined I have failed to discover any spinal lesions.

Moreover atrophy of the discs has been absent. The ocular symptoms in this case may have preceded the facial one. The atrophy of the discs occurring before any symptoms referable to the cord showed themselves.

Powers ("Medical Ophthalmoscopy") relates an extreme instance of early atrophy coming
under his own observation, in which the
atrophy of the discs was complete and
vision lost for twenty years before the
first symptoms of Ataxia showed themself
and another case in which the loss of
ight preceded for sixteen years distinct
spinal symptoms.

I quite agree with this - cases of Ataxia
having come under my observation, where
the tactual trouble was the only clue to
the disease (Ataxia), though the Characteristic
symptoms had not yet established themselves
till a considerable period after.

I think that when atrophy occurs it
so frequently an early, though a late
symptom.

Case 28 illustrates atrophied discs.
Failing sight. Eighteen months - a history
of patient's wife having had two miscarriag.
I am disposed to attribute the condition
of discs as indicative of Coenurotic Ataxia.
Is a certain measure, though the symptoms
of most report were wanting, the gait un
steady, the heel striking the ground first
on walking - puptes very small, not acting
either to light or accommodation, and the
Patellar Reflex absent - the vision was very
defective as patient could not see 20/20
of the test types.

Though always in the habit of examining
the Patellar reflex in cases of Coenurotic
Ataxia, still in many instances I have
found it normal or even slightly
Suggested, so that I am led to discuss the absence of patellar reflex, as in all cases dysphoria of Ataxia, when the atrophy of the optic nerve has established itself.

In many cases, as Westphal pointed out, it is an Early symptom of the disease (ataxia), and if searched for will be found to co-exist with atrophy of the optic nerve. I have met with cases also where the condition of pupil (Spinal myopia) and the Argyll Robertson Phenomenon was an Early symptom of the disease. It may be present along with optic neuritis and without official symptoms, or, if any very slight. That the variety of disc degeneration of the nerve usually supposed to be met with in Locomotor Ataxia, is indicative of this affection, and aids one in the diagnosis of it does not seem to me to be altogether conclusive for you may have a grey disc from other causes, such as from pressure on the nerve behind the eye.

Case 12 presents a history of gradual failure of sight. Eighteen months before admission, the left eye being attacked six months previous to the right.

He had Syphilis when a young lad; otherwise healthy; vision was reduced to counting fingers barely. Orthophthalmoscope examination revealing Choroids-Elitis and an atrophic disc.
The atrophy here is consequent on the Choroiditis Retinitis, the main element in the production of the latter being the history of Syphilis, which, if taken from the patient, and the gradual failure of sight would, no doubt, account for the primary development of the retinal mischief, the result of the Syphilitic poison, and consequent implication of the optic nerve.

Powers ("Clinical Ophthalmoscopy") says "that the recognition of this variety of Choroiditis atrophy is of considerable importance, because unless the result of Retinitis Pigmentosa, it is almost always the consequence of Syphilitic disease acquired or more frequently inherited and constitute a sign of inherited Syphilis of great importance."

In this case the disturbance of Choroid pigment was distinct and characteristic.

The patient was treated with O parachute of Potassium in five grain doses, and with Sulfate of Iron and Potassium in four grain doses, thrice daily.

The subsequent progress of the case had not taken.

Cases IV and V are interesting in that the two patients were brothers, dark-complexioned, and dull sickness in their appearance.

Both used to suffer from fits, their age being twelve and nine years respectively.

The Cases presented on Ophthalmoscopic Examination white atrophy of the discs (right and left eyes).
Though not mentioned in the notes, their vision was reduced to shadows, being somewhat dull and stupid, no history could be elicited from them to aid me. I presume we have here to deal with atrophy consequent to some morbid condition of the brain, in all probability to some previous meningitis, for we have a history of fits.

Both patients appeared strong and healthy, there was no evidence of tubercular dicesis or outward manifestation of Hydrocephalus.

I must admit to these being the first two cases I have met with, in which white atrophy of both optic papilla was present at so early an age.

Clifford Allbutt (in the Microscope in the diseases of the nervous system and of the kidneys) gives illustrative cases of atrophied dicesis in infants aged 5, 11 and 12 months, and where the atrophy was due to Hydrocephalus.

I can account for the atrophy in these cases where there was organic mischief in the brain with ventricular effusion or some other morbid product, having a direct influence on the Optic Chiasma by pressure.

The only other hypothesis I can bring forward as a cause of the atrophy in both my cases is that it might have been Hereditary.
The patient also had one brother blind. Unfortunately, the parents of these two cases were not to be found, otherwise a more correct history would have been made, and materials for a more conclusive causation found.

Dr. Wecker ("Kultur Therapeut") talks of hereditary neuritis and atrophy as sufficiently rare, and quotes an instance of a series of six patients, all members of the same family presenting themselves at his Clinique, the subjects of hereditary atrophy, and he has met with cases where there was a direct transmission from mother to son.

Now long ago the loss of vision began in my cases I could not definitely ascertain.

In Case XIX we have a history of failure of sight in the left eye two years before she came to the Hospital. She had never suffered from fits, used to suffer from headaches before Catamenia, and does so now occasionally. Her family history was good. Vision in the left eye be very imperfect; could count fingers with this eye better seen when held towards the outer side of her field. Both discs were atrophied, particularly the left, and the arteries were reduced in breadth. Notwithstanding the atrophy in the disc of her right eye, still her vision with this eye was 20; and she could read finger at six inches.

There was also contraction of the field towards the inner side (left eye).
Under treatment she improved slightly. Vision in her right eye being 20, but unaltered in the left. The field towards the temporal side was lost in the right eye while it became altogether absent in the left eye.

We have here diminished acuity and defect in the field of vision limited towards one half in the right eye, while it was altogether absent in the left.

Diminution in the acuity and alteration in the field of vision are said to be symptoms usually associated with atrophy of the nerve, the former being invariably when the atrophy is more pronounced, and generally more marked in one eye than the other; one may, however, be misled unless any anomaly of refraction or defect of accommodation be properly estimated. In the case I have quoted, I ascertained that there was no fault in her refraction to account for the diminished acuity of vision. Here colour blindness was very deficient, red and blue disappearing first towards the periphery of the field, and then yellow. Patient could count fingers with left eye when held towards the outer side of the field, which became impaired subsequently.

Another peculiarity in this case, was the severe degree of impairment of vision in her right eye, which showed an atrophied papilla, not, however, in such an advanced stage as in the left eye. Could this be accounted for by it?
gradual propagation of the inflammation from one nerve to the other?
In this case, the left disc being primarily involved, the right one being secondarily implicated, the structural integrity of its nerve fibres having been to a certain degree damaged.

As stated in the notes of the case, I am disposed to attribute the atrophy of the discs to a meningitis, affecting the Optic Chiasma by pressure, and spreading along the optic nerve fibres to the intra-ocular terminations of the nerves.

That the atrophy is not primary but secondary, may also be proved by the loss of one half of the field of vision in both eyes.

Gowers ("Medical Ophthalmoscopy") says "That a loss of one half of the field of vision is met with chiefly in secondary atrophy, especially when the cause is pressure on the Chiasma, the temporal halves of the field being then usually lost.

Not having devoted much time to ascertaining defects in the field of vision, I cannot say that I am in a position to receive this doctrine as diagnostic of atrophy of the discs, whether primary or secondary, though great stress appears to be laid on the mapping of the field of vision,
as an aid to diagnosis, by many observers,
especially when the atrophy is in the
early stages.

The Conclusion.

To enable us to ascertain how
inflammatory disturbances, in set up
in the optic nerve, it is necessary
to know something of its anatomy.

Though I have spoken of inflammation
of the optic disc, as optic neuritis still
I should consider it more convenient
were the terms "Pappillitis", restricted
to inflammatory mischief, affecting the
intracranial termination of the nerve,
the optic papilla, and confine the
term optic neuritis to an inflammation
of the trunk of the nerve.

This nomenclature, however, is
inimportant, for by optic neuritis, in
the present acceptance of the term,
it is also understood that the optic
disc or papilla is affected.

It is important to remember that in
connection with the trunk of the optic
nerve, there exists a double sheath
which closely invests the nerve, the
inner one being delicate and
continuous with the perineurium of the
brain, the outer sheath, thicker, and
fibrous, uniting in front with the
dura mater, and is continuous at the
optic foramen with the dural mate.

The space within the outer sheath
of the nerve is known as the "vaginal canal" and is continuous with the sub-arachnoid space around the brain. That in the optic disc (or the pia-mater ending of the nerve) we have blood vessels, chiefly capillaries, nerve fibres which spread out on the retina, these fibres being more dense towards the macular area and more numerous on the nasal side; and that we have also in the optic disc supporting connective tissue.

The practical utility of all this is proved by some of the theories which have been brought forward as causes of optic neuritis and atrophy.

Schmidt says that in some cases of neuritis, especially when due to increased intra-cranial pressure, there is distension of the sheath of the nerve by effusion of fluid into it, sometimes this fluid passing into lymph spaces in the lamina cribrosa.

Hughlings Jackson says that though distension of the optic sheath is certainly very frequent in cases of optic neuritis, it is not, however, as has been alleged, invariable either in cases of cerebral tumour, with optic neuritis, or in conditions of increased intra-cranial pressure.

Clifford Allbutt describes the condition of discs ("choked disc") of Vaquez, not with in optic neuritis due to intra-cranial pressure, where there is considerable swelling with homogeneous
and vascular distension, as consequent on obstruction to the return of blood from the eye by compressing the cavernous sinus.

In cases of atrophy of the optic nerve, we have here direct damage brought to bear on the nerve, causing it to undergo wasting and degeneration, by interfering with its nutrition, and with the capillary which confer on the disc its normal rosy tint.

Regarding the stages of neuritis my experience has taught me to believe in

1. First stage of Congestion with Edema, where one meets with a condition of increased redness, and cloudiness, obscuring the edge of the disc, this being more markedly noticed in the direct, while the edge of the disc is perceptible to indirect examination.

2. Second stage — the true neuritis — characterised by the disappearance of the edge of the disc, even to indirect examination, its edge being blurred and outline indistinct.

White lines and spots are also usually found, particularly in cases in which the changes are slight. The swelling and obscuration of the disc may affect all parts, or may be more marked on the nasal, than on the temporal side of the disc.
in this stage, sometimes, on the surface of the prominence or swelling, or on the least changed parts of the disc, the arteries present little change, in the slightest degrees of herinitis, though they are a little concealed at their emergence; the veins may or may not be congested.

As the herinitis proceeds the swelling increases, and is often so great, that the swelling itself is with difficulty seen; the veins as they curve down the sides of the swelling are concealed, just beyond its edge by the adjacent retina; they now begin to present some enlargement.

Extravasations of blood appear on the swelling, often concealing the vessels.

There is a stage of herinitis, where strangulation takes place; where the arteries are much narrowed and reduced to threads; where the swell on the disc is considerable; and where extensive hemorrhoises appear, the veins being greatly distended. Light during this stage fails entirely.

The signs of most value in the diagnosis of optic herinitis are:

1. Irregularity and indistinctness of edge of disc.
2. Swelling, associated with increased redness of the disc.
Prognosis and Treatment of Optic Neuritis.
In some cases, I have met with elevations of the edge of the disc also.

Prognosis of Optic Neuritis.
This will depend upon the causes which give rise to the impairment of vision.
It is better in the slightest degrees of headache and in proportion to the chronicity of cause and dependence on causes which can undergo treatment.

It certainly is worse when there is reason to suspect retrobulbar mischief, worse in proportion to the intensity of the changes and when ophthalmoscopic examination reveals evidence of Compression taking place in the disc.

It is better in Syphilis than in acute cases, and better in these than in cases of disease in other forms.

Causes.
The most common causes of optic neuritis are associated with diseases of the brain, prominent among which is Tumour.

Highly go Jackson says that besides the nature, size, nor seat of the tumour appears to exercise much influence on the occurrence of neuritis.

Next to tumour, the most frequent cause is Meningitis, then abscess of the brain, Hyphaloid disease, and softening of the brain from vascular obstructions.
Other causes are albuminuria, lead poisoning, certain pelvic diseases, anemia, and certain other morbid states of the blood.

It may also occur without any obvious exciting cause, or from disturbances of menstruation, exposure to cold or excess in tobacco, venereal, or alcoholic.

As a rule the neuritis is double, though now and then it is single in cerebral disease.

Another cause is Bright's disease.

Duration.

The duration of optic neuritis varies very much in different cases.

The most trifling cases are those which depend upon a cerebral condition which soon subsides, such as a transient attack of meningitis, or leptomeningeal disease, which is influenced by treatment before the neuritis attains any great degree of intensity.

The cerebral disease may progress in some cases, while the neuritis rapidly subsides; again the course of neuritis may be so chronic, that months may pass away without any change in the condition of disease, being evident.

Treatment.

The treatment of optic neuritis must depend upon the intra-cranial mischief or other affection causing it.
In most of my cases I used Soda of Potassium, where I suspected Specific disease, and as shown in Cases good results were produced under this treatment.

I have always given myself the benefit of the doubt in some cases, even though the disease has not proved to be Syphilis in its nature, and administered Soda of Potassium in gradually increasing doses with good effect.

Should the disease be traceable to other causes, such as Scurfula or Anemia, then tonic treatment is indicated.

Hughlings Jackson believes that Soda of Potassium is sometimes useful when there is no Syphilis.

Prunecut of the dilated nerve sheath, as advocated by Dr. Power and Dr. Brookes, is not I think followed by improvement, when there is a supposition that the neuritis is consequent on this disturbance.

I have seen the operation performed twice at Moorfields Hospital with unsatisfactory results, though I dare say the operation demands further trial in a larger number of cases in order to prove its efficiency.

There is one variety of neuritis in which swelling of the discus may be produced by the prolonged use of the Eye, in some cases of hypermetropia, as pointed out by Mr. Cooper. I must however admit to not having come into contact with any.
Regarding Atrophy of the disc little may be said, its diagnosis being in the majority of cases evident.

We have 1. Pallor and 2. excavation as our guides, the latter being characterized by its surface being depressed in proportion to the width of the nerve trunk.

We must however distinguish pathological pallor from normal variations in the tint of colour in the disc.

We have the grey and white varieties of atrophy to deal with also.

The retinal vessels in some cases of progressive atrophy become reduced in size in others they do not.

In the grey atrophy the vessels do not appear to undergo much change, though they are occasionally narrowed.

In cases where one suspects some retrovascular mischief, the vessels are reduced in size. Sometimes the atrophy is part of some inflammatory changes which have taken place in the nerve fibre and ganglion cell layers of the retina.

The atrophy leaves the edge of the disc very distinct and sharp; this I have noticed being characteristic of simple atrophy whereas in consecutive atrophy where there have been signs of previous keratitis, the walls with irregularity of the veins, ill defined edges, white marks along the vessels, and veiling of the lamina cribrosa.
Prognosis and Treatment of Atrophy.
Prognosis

The prognosis of atrophy of the optic nerve on whatever cause it may depend, is in my opinion always unfavourable in proportion to the actual damage of nerve fibres present, and to the degree to which the causes influencing the disease are under control, especially when the atrophy is associated with degenerative changes in the nervous system elsewhere. I have, however, met with cases where the damage to the nerve fibre has not altogether abolished vision, yet the further progress of the disease has been arrested, improvement to some degree having been obtained.

The prognosis in simple primary atrophy, which has a great tendency to degeneration, is the least favourable.

As in Optic neuritis, the prognosis varies according to the manner in which the affection of sight is impaired.

Some observers regard considerable contraction of the field of vision as very grave, lesserened acuity of vision being of less significance.

Atrophy having been once well pronounced I cannot say that I have observed restoration of vision even from any remediable, by other measures; for a diseased eye that has lost all its normal tint can never regain its tincture, so that useful vision is scarcely ever recovered.

Clifford Allbutt has, however, observed cases, where the Ophthalmoscope has
revealed white and even glistening atrophy in eyes which could read ordinary types with ease.

Causcs.
The causes of Atrophy may be:
1. Primary and 2. Secondary.
Primary Atrophy often appears without any known cause, being sometimes hereditary.
As before stated, a considerable number of primary atrophies are associated with affection of the Spinal Cord (Spinal Atrophy), as in cases of locomotor ataxia, the atrophy here being in most cases of the grey variety.
The Secondary Causes are those attributable to inflammatory growth or tumours, which by pressure damage the nerve fibres and so affects their structural integrity, and in atrophy the whole nerve is implicated.
The atrophy may be secondary to a hereditary pressure of tumours, benign growths,
narrowing of the optic foramen, or internal hydrocephalus where the distended third ventricle compresses the optic chiasma.
Though meningitis commonly produces optic neuritis, it is said by some to cause blindness in rare cases, and atrophy without intra-ocular inflammation by pressure without inflammatory invasion.
Meningitis is another cause, also bloody or by injury to the head, which by damaging the nerve directly, may produce
atrophy. Other causes, such as excesses in Tobacco, Venery, or Alcohol are supposed to produce it, probably by a gradual process, the poison finding its way to the nerve and so destroying it.

Lastly affections of the Retina as stated in one of my cases (Choroiditic Atrophy) are causes, though in these the affection is not as a rule complete.

I come now to the last consideration in connection with this subject the Treatment of Atrophy of the Optic Nerve.

Though my clinical notes are not meant to describe the treatment of these cases, my observations of their progress while under supposed remediable measures, have not as yet convinced me that there is any therapeutic agent likely to arrest the mischief altogether.

It is quite possible to attack atrophy while in its Early stages, and arrest the further progress of the disease, so as not to render vision altogether nil. Still whatever course is pursued in these cases, improvement, if any, is slight.

The therapeutic agents at our command are nerve tonics such as Strophanic, and Iodide of Potassium as well as the use of the continuous galvanic current.

Gunn (Royal London Ophthalmic Hospital Reports Vol V Part 2 1881) illustrates a series of cases in which he tried the Continuous Electrical
current. He used Weiss' Continuous Current battery of 25 cells, fitted in the ordinary way, the positive pole (Anode) was applied over the closed lids of one eye, and the negative pole (Cathode) to the opposite eye or temple, the power was gradually increased, beginning with 5 or 7 cells, until a distinct evaporation of a flash of light was experienced by the patient, in making or breaking the circuit, the pole being moved to different sides of the head.

Of eighteen cases he records, six improved under treatment; four others received doubtful benefit; and eight did not improve at all, or grew worse.

Dr. Wecker quotes cases where improvement took place by the use of the continuous current and subcutaneous injections of Strychnin.

This treatment, which, in the hands of some appears to be beneficial, still demands further trial, to prove its value.

If the atrophy, moreover, is due to a condition which is a certain measure given way to treatment, then some good may be done, as for example when dephtilic, scrofulous, or associated with excesses, or with disorders of the blood, in all these cases Strychnin and Iron may do good.

I have given Sodide of Potassium but cannot attribute any satisfactory results to the use of this drug.
Liq. Straphium has in some cases proved beneficial. If there is a nervous temperament as the foundation of the neuralgia, the treatment which one might have recourse to, along with local galvanism, would be that directed to the general health, nervine tonics being indicated.