Thesis for the degree of
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Focal Amaurosis.

A dimness of vision caused by the action of certain poisons on the system. It is my intention to consider the subject under two heads:
A. Where the cause is Tobacco or Alcohol.
B. Where the cause is Carbon Bisulphide, Quinine, Chloral, Salicylate of Soda.

A. Tobacco or Alcoholic Amaurosis.

History. Up to 50 years ago little seems to have been known of the subject. The first mention I can find of it is an article by Dr. Bell in 1840 (De Amblyopia et Amaurosi Alcoholica). He was followed by other continental authors; but the first English literature on the subject is a paper by Jonathan Hutchinson (1863); his knowledge of the subject seems to have been scanty, but he suggests as probable causes Tobacco, Syphilis, and Sexual Excess. The same author gives another article in the Ophthalmic Hospital Report 1873 & this was followed by others by numerous observers.

In 1887, The Ophthalmological Society of the United Kingdom collected a vast amount of information on the subject from its members. But the most exhaustive & valuable work on the subject is that by Uthoff 1888. He has made a most careful study of
a very large number of cases, chiefly with reference to Chronic Alcoholism, he was able to verify his conclusions by post-mortem examinations in several cases.

This disease affects men far more frequently than women. In Germany, among women it is almost unknown, but in Russia and other parts of England it is fairly common to find women attacked. Dr. Milligan says that in Turkey this complaint is never seen. It is hard to say whether this is due to some peculiarity of race, or to the sudden forms of tobacco in use in that country, or what is most probable to the fact that by their Religious laws the Mahometans are forbidden to touch alcohol. The patients are always adults or usually over 40.

Cause. As to the immediate cause there is much difference of opinion. Continental and American authorities inclining to the view that it is chiefly caused by excess in alcohol very rarely by tobacco; whilst most English authorities agree that the chief if not the sole cause is excessive indulgence in
tobacco & that Alcohol is merely one of the depressing agents which render the frame liable to the evil influence of Tobacco. In support of the Continental view it is brought forward a number of cases in which he asserts alcohol to be the only cause in 45 alcohol with tobacco & in 23 he allows tobacco alone; but in the cases stated to be due to Alcohol alone he neglects to prove that Tobacco is excluded. One cannot help thinking that what a German would consider moderate drinking, ought by an Englishman be considered excessive. Whilst in support of the English theory it is urged that cases are sometimes met with in Total Abstainers, but these cases are rare compared with those in which alcoholic excess is admitted. I cannot, after considering lists of reported cases agree with Marcus Gunn when he says “they (total abstainers) are also probably, peculiarly apt to get the amblyopia from a comparatively small amount of poison.” The few cases reported seem to have been in so heavy smokers as where alcohol was also taken. Now in a very large proportion of cases it is admitted that the patient drinks to excess & knowing how hard it is to get an admission of excessive indulgence
in Alcohol from one who has given way to it, I am afraid we must add to the list of Alcoholics a considerable number of those who admit only moderate indulgence. Of course the dryness of the tongue caused by the excessive smoking increases the temptation to drink.

Now when one remembers the destruction that continued excess in Alcohol works on the nervous system in general; and also how frequently this excess is found in Toxic Amanuensis, it is difficult to avoid the conclusion that in these cases Alcohol is likely to be playing a considerable part in the causation of the neuritis of the Optic Nerve.

Taking all things into consideration I must arrive at the conclusion that thus form of Optic Neuritis is caused by excessive indulgence in Tobacco or Alcohol but most commonly by a combination of both.

The Tobacco may be taken by smoking, chewing or snuffing, but the case is reported by Schweinitz of a girl who had Toxic Amanuensis brought on from working in a Tobacco factory. Smoking is hence the usual method of taking this Neuritis.
The kind of tobacces is, as a rule one of
the stronger varieties, Aq, Tars, Cabanis,
but May Blossom & other lighter varieties
are sometimes answerable. Lang
tells me of a gentleman who smokes a day produced 3760* *ANNONOS.
The amount smoked varies considerably it is frequently 1/3 to 1/5 per diem. & Lang
has worked out an interesting calculation
that when a man is smoking at the rate
of 1 curl of tobacces in 3 years - at the
end of his 3rd. curl he is very liable to
get an attack of ANEMOSIS if his nervous
system receives any shock.
Of course idiosyncrasy plays a considerable
part in the amount required - as cases
are often reported in which the patient
is moderate in both tobacces & alcohol e.g.
a case recorded by Kettlest in which a tempest
man was attacked who only smoked 10
curls of tobacces in a week.
It was at one time held that those who suffer
from sea sickness were particularly liable to
get ANEMOSIS. But as my experience as
kuggen in the Mercantile Marine leads me
to think that a large majority of people
who suffer from sea sickness - as a very small
proportion of even violent smokers suffer
from ANEMOSIS I cannot see that there
is much foundation for the Theory. But it is often found that several members of a family are Amoebites, so that one is so y' others suffer from water cramps, sneezyly or some other nervous affection.

The amount of Alcohol also varies; but it often comes to several quarts of beer or stout in a day, often with ardent spirits as well. The drinking is often done on an empty stomach & one can generally find a precipitating cause such as want of food, grief, nervous shock, loss of money, an attack of illness such as diabetes &c.

**Symptoms**

The patient comes complaining of dizziness, weight which has been coming on gradually. There is a dry justy smell about him & frequently the odor of alcohol. In questioning he will admit excessive smoking, but demy recent increase in the amount, he will also often admit excess in alcohol, excusing himself by saying that he sees better after a little alcohol. He will tell you that he sees best in a dull light; one of my patients who was employed in a parcel office told me that he could not see the addresses which were written on white paper while those on brown paper he could see quite well.
There is no pain even in presence over the eye ball.

As a rule the patient is not aware of any defect in colour vision; but sometimes this is noticed. Hojes reports the case of an artist who came complaining that he could not get the brilliant effects in red to which he had been accustomed, and a patient of my own whose duty it was to match silks complained that he mixed up the reds & greens excessively. He had a typical attack & made a good recovery.

The patient again does not notice any dark spot in the centre of the field of vision, but he frequently misses an object at which he is looking. Kettle has a case of a man about to photograph a ship who suddenly found it moving from his field of vision. Another case is that of a billiard marker who could not see the red ball when he looked at it.

The failure of vision may be only a slight haziness, but it may amount to only the counting of fingers & loss of colour vision. But absolute loss of perception of light is unknown.
The Pupils are as a rule normal. Sometimes they are unequal, rarely they fail to react to light but still act to accommodation. The Tension is normal.

The media are clear except for coincidental occurrence of cataract accessory cataract.

The Perimeter gives the most important information. The Periphery of the field is as a rule normal, very rarely slightly contracted. It is in the centre that the defect lies as was first pointed out by Förster. This defect in Deutonoma is very rarely absent except in very early cases. It is first found for the colours red & green, then blue & seldom for white. In a very advanced case you may find a small central scotoma for white & all colours; outside this a ring in which white is seen but no other colour; outside this again a ring in which white & blue are seen but not red & green; and still further out a ring reaching to the Periphery in which all colours are seen; and still further out a ring, reaching to the normal periphery for each colour, in which all are seen. A case like this would be so perfect that one would rarely expect to meet with it.

As a rule the scotoma is situated around the
fixation point; more rarely it lies towards the blind spot & more rarely still to the inner side. A typical scotoma is oval involving both the fixation point & the blind spot. Poeticske holds that the position of the scotoma will diagnose between Alcoholic and Tuberculous amaurosis being central in the former & round the blind spot in the latter, but his view has not met with much support.

The size of size of scotoma varies from one up to involving the whole field for red & green. Sometimes the scotoma runs out to periphery in places as may be seen in the accompanying chart of a patient.

Right Eye

Periphery for white

Field for red.
Another curious fact in this case was that with the left eye red was at first perceived, but after gazing at the perimeter for a short time the perception of red was entirely lost and was only regained after directing the eye for some time. The return of perception was aided by darkness.

Doyne to explain a similar case postulates the theory that for perception of various colours a substance is necessary in the retina, analogous to vision purple, presumably a separate substance for each colour. He suggests that the toxic agent has the action of destroying this substance & that rest is required for its restoration. This theory is quite opposed to a rendered unnecessary by the fact that all pathological evidence goes to prove that the disease is a neuritis & not an affection of the retina. I think the phenomenon is explained by the fact often demonstrated by Physiologists that a nerve becomes 'weakened' by repeated stimulation & loses its power of conducting impressions & that this 'weakeness' is not easily produced & the recovery slower when the nerve is diseased or deprived of its nutrition; but after a short interval it will recover its power of conduction. Thus the stimulation of the image produced on the
Red by the red disc is more wearisome, the diseased fibres of the optic nerve. This also explains the fact before noted, that patients see best in a dull light. The excessive stimulation of bright light producing a weariness of the diseased nerve fibres.

Another curious phenomenon that I have noted is that in an advanced case a red disc of 10 mm. square will not be perceived when stationary, but if this same disc be flashed across the field the red is at once perceived. To explain this I would advance the following. In order that a visual impression may be recognized it is necessary that a stimulus of sufficient strength should reach the brain; to effect this there must be a sufficiently violent impression made on the nerve endings in the retina, and also a sufficiently sensitive conduction medium to convey the stimulus through the optic nerve to the brain. Apparently different colours have different power of stimulating the retinal nerve endings; red and green yellow, blue and white best. Now if the nerve fibres are diseased and their conducting power consequently diminished, a larger number will be required to convey to the brain a sufficient stimulus to cause sensation. This must be done.
this, a larger number of nerve endings must be stimulated simultaneously. This may be done by forming on the retina the image of a larger object; or the same result may be arrived at by flashing an object, before invisible, across the field so as to give an almost simultaneous stimulus to a larger number of nerve endings on the retina. Thus allows the stimulus to be conveyed to the brain, as a single impression, by so large a number of the weakened fibres that its force is not lost.

The Sezotama is often only relative even as regards colours: e.g. it is often found that a crimson red is called pink or salmon over a large part of even over the whole section.

As a rule the disease is symmetrical both eyes being affected at the same time & to reach the same extent. But cases are sometimes met with in which this is not so. e.g. One of my own cases had 0.25. came complaining of failure of sight in R eye for 4 yrs. He smokes 9/8 of shag daily & has done so for 20 years. He drinks 50 6 pints of beer daily with a little rum occasionally. R eye v = 0.8. Left v = 0.6 no pain in either eye, pupil & tension normal. There is a large section for red in R eye.
but in the left nothing can be found. The K eye is slightly blurred at the edges.
2 weeks later there was no perceptible change. But at the end of 7 weeks patient returned saying that for 20-3 days he had noticed a failure in left eye — The K eye is numbness before with acetone for white as well as colons.
left eye V = 4.2 — there is acetone for red — the disc is blurred at edges. Patient admits that he has smoked a little & that he has continued drinking as before. A week later, during which he had been more temperate there was slight improvement R V = 4.2 0.6 7.
6 the acetone are smaller. Unfortunately after this the patient was lost sight of.
below I give the chart of this case.

Left Field on Feb 2nd —
no acetone

Right eye

Jan 20, Feb 2, 1858

Field for acetone in red
Field for white normal
The Ophthalmoscope as a rule detects a change in the temporal part of the disc, usually a palor, sometimes a blurring of the whole disc. Uthoff points out that in many cases he has observed this change in the disc before any visual defect has been noticed. Sometimes this change is to be found in the retina seldom is anything abnormal to be seen. Hemorrhages from retinal vessels are sometimes reported, but they are probably accidental accompaniments rather than integral parts of the disease.

To aid the diagnosis Power suggests the inhalation of Amyl Nitrite; in cases of toxic amaurosis this gives a temporary sudden improvement in vision, but in other forms of optic atrophy it has no effect. The same change is observed sometimes with alcohol or large doses of atropine. In the case of Amyl or Alcohol I think the improvement is accounted for by increased blood supply to the optic nerve while the atropine produces increased irritability and allows a temporary improvement in the conduction of the nerve.
Prognosis. If the patient will stop taking the injurious agents & if the case hasn't been allowed to go too far a good result may be expected; but the recovery is often tedious sometimes lasting over 15 months. Often the restoration of vision is perfect; but in many cases there is a slight permanent impairment of vision rarely one finds a case in which one can get no improvement in spite of the utmost care in treatment.

A few cases are reported in which a marked improvement of vision has taken place even though there has been no diminution in the injurious drugs. If the case be dealt with early & promptly a speedy cure may be expected; for example, C.W. came to me complaining of diminution of vision & loss of ability to perform finer muscular movements with his hands. He drank excessively though not to drunkenness, he was a heavy smoker & for the last 3 weeks had smoked even more heavily than usual. He stopped all alcohol & reduced his smoking to a cigarette a day; in the day. He was treated first with Iodide of Potassium & later with Stypheinae & Phosphoric Acid & in less than 3 weeks he had completely recovered. As the patient recovered the lesion-seats
smaller & smaller & often leaving the fixation point is last seen at one or other side of it. The following chart from case 7 A.D. 1647 in this case there is an unusual contraction at the periphery.

Second attacks of toxic amaurosis after recovery from the first have been observed, but are very rare.

**Treatment:**
Discontinuance of Tobacco & Alcohol must be insisted on - Nourishing diet, fresh air, avoidance of causes of worry & depression, if necessary tonics. Different authorities advise various drugs like Vinca, arsphenamin, Picro phosgen, Bismuth & cinn. but most
cases will do well without drugs if the general health be attended to.

Pathology

The first autopsy reported is by Smeak in 1882 since that we have had others from Nettlefield, Bunge, Lawford & lately a series of cases from Vlotho.

The disease is a neuritis going on rapidly to atrophy. It first affects the fibres supplying the area of the retina between the macula & the optic nerve. These fibres where they enter the eye from a sector of the nerve to the lower part of the temporal side, extending from the sheath to the central vessels. And it is this that gives the appearance of palse of the lower part of the outer side of the disc observed by the ophthalmoscope.

In advanced cases a much larger part of the nerve becomes involved, sometimes the whole nerve; but there are always a few healthy nerve fibres scattered through the diseased nerve, and to this is due the fact that there is never complete loss of perception of light.

Vlotho describes it as a thickening & proliferation of connective tissue going on to complete disappearance of nerve structure, but with a few normal nerve fibres throughout the diseased area. Whilst
in grey atrophy normal fibres are never found in the diseased area; but the original stroma is always perceptible. So none of these cases was there any evidence of either syphilis or renal disease.

B. **Amaurosis due to other causes**

**Carbon Bismuthide**

Cases are not very frequently seen. Almost always they are in workers in rubber factories. After working for a few months the patient complains of failure of sight, hearing, smell, taste & often of general weakness & loss of sexual appetite. Sometimes the failure of vision is not noticed by the patient but on examination it is found that the fields are constricted—colours being affected most in the same order as in toxicoses amaurosis. But there is no central scotoma. Even with fields very much contracted there is often no failure of the central vision to be detected by test types. The accompanying chart from Stacke's paper will illustrate the above.
J. H. Oct 24
Vision normal
Fundus normal
Field with
Object 10mm
Square for
Each colour

The Ophthalmoscope as a rule reveals little beyond a pale of the whole disc - Exchange is found in the retina & vessels. Recovery takes place if exposure to the poison be avoided; but a second attack is not uncommon if the injurious work be resumed.

This disease is probably a neuritic affecting the nervous system in general of the failure of vision is merely an Ophthalmic symptom of the cause.
Luminine

Sometimes after excessive doses of this drug there is a sudden or at any rate very rapid loss of sight. The onset is often so sudden as to be compared to the turning out of lights, but it is usually preceded by deafness. Sometimes it is so severe that there is absolute loss of perception of light or sound or the only means of communicating with the patient is by the sense of touch.

When seen early the pupils are dilated & immobile. The ophthalmoscope reveals extreme pallor of the whole fundus with contracted thread like vessels & often the macula appears as a bright red spot.

The complete blindness persists for days, weeks or sometimes months. But when recovery begins it usually advances fastest. Colour perception is the last to be restored. Vision is often permanently impaired, a marked contraction of the field being the most frequent defect. Even when central vision is perfectly restored this contraction of the field frequently persists for a long time sometimes permanently.

The quantity of luminine required to produce this effect varies considerably. Some bodies are more sensitive to it than others.
affected by small doses; but in some cases the amount taken has been 90, 150 or even 120 grains on a day. Ocular blindness is frequently met with on the coast of true Tropical Africa when the drug is used for fever promiscuously and without medical advice.

The pathology has been carefully studied by Brunner, Ulric, and Schwendy. Numerous experiments have been made on dogs. It is usually found that there is a marked contraction often obliteration of the retinal blood vessels sometimes the vessels are blocked with clots. But there was never any neuritis found even after the dog had been blind for a month.

Amaurosis caused by Chloral, salicylate of soda, and other substances are occasionally but rarely met with.

To summarize briefly the classes of cases considered we have:
1. In Alcohol + Tobacco - a neuritis with scotoma especially for colours, but no change in periphery.
2. In Carbon Bicarbonate - a neuritis, with no scotoma but a contraction of the field especially for colours.
3. In Turmeric - a change in blood supply to the retina (but no neuritis) and absolute loss of vision.
This Thesis has been composed and written entirely by myself. My facts are obtained from personal observation of numerous cases at the Royal Ophthalmic Hospital, Moorfields - at the Middlesex Hospital in private practice. I have also made use of the writings of various authorities named in the Thesis; having consulted especially:

The Transactions of the Ophthalmological Society, 1877
The Annual of the University Medical Sciences, 1884
The Ophthalmic Review (several years)
The Ophthalmic Hospital Report (several years)
The Lancet, 1863
International Journal of Medical Sciences
And articles in several other Journals.

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