"Tobacco Amblyopia."

by


The following paper is a digest mainly of the chief writings of English authorities on the subject of blindness arising from the abuse of tobacco, with casual allusions to Continental authors; and I have added here and there some of the observations I was able to make upon persons suffering from this affection, while I was officiating as Clinical assistant at Moorfields Ophthalmic Hospital last year. I give these observations merely that appear relevant to the settlement of any dubious point, and I have made no attempt to append full reports, or even the statistics, of all the cases I have seen, as such a proceeding would only burden my paper with what is now almost universally admitted to be an ophthalmic entity, viz., a typical "tobacco-case." Patients thus afflicted are almost daily applicants for advice at Moorfields, and for twelve months I had the opportunity of seeing the majority of them. I am indebted, however, more particularly to Messrs. Jay, Gunn, & and
and

Nettleship, surgeons to the above institution, for cases and also for many hints and assistance in studying them, and my thanks are also due to my fellow-clinics, Messrs. J. Hutchinson, Jun., and W. H. J. Brown.

**Definition:**

Amblyopia is derived from two Greek words, ἄμβλοπός, blunt, and ὀπίς, vision, so that its literal signification is 'bluntness of vision'. "Tobacco" Amblyopia is that variety of defective sight which is due to over-indulgence in the use of tobacco.

Amblyopia, like many more medical terms of classical origin, was simply used, as a cloak to ignorance, the same meaning being expressed by the common English words 'partial blindness.' The prefixing of the word 'tobacco' improves matters to a slight extent, as, by the statement of the causal relationship of the amblyopia, the excessively wide scope of this expression is somewhat defined. Still, from a pathological point of view, the nomenclature of the disease is not at all satisfactory.

**Pharmacology:**

A very brief account of the composition of Tobacco—
Tobacco-smoke is all that is necessary for my present purpose. Dr. Mitchell Bruce in his "Materia medica and Therapeutics" says:

"Tobacco-smoke contains the very smallest traces only of nicotine, or none, but a number of volatile bodies, chiefly pyridine compounds, such as pyridin, x; pirenin, x; lutidin, x; collidin, x, which have somewhat the same action as nicotine, but less severe. Hydrocyanic and hydro-sulphurous acids, other simpler gases, creasote, &c., also occur in tobacco-smoke."

In 1880, Dr. Le Bon made some experiments on Tobacco-smoke, the results of which he published in the 'Journal de Therapeutique.' He concluded that its properties were not to be attributed solely to nicotine, but were also due to various emphysematic substances, especially an aetaloid—collidin, and to hydrocyanic acid.

Whether Dr. B. W. Richardson had himself investigated the subject or not, he wrote a quasi-scientific paper in the Social Science Review in 1863 on "Tobacco, in its relation to the Health of Individuals." He states that "the effects that result from smoking are due to different agents imbiber by the smoker, viz., Carbonic acid, Ammonia, nicotine, a volatile emphysematic substance"
"substance, and a bitter extract. The more common effects are traceable to the carbonic acid and Ammonia; theparer and more severe to the nicotine, the empyreumatic substance, and the extract."

All admit that Tabaei Folia, the leaves of Nicotiana tabacum, the tobacco-plant, when taken internally in a sufficiently large dose, will act as an acute cerebro-cardiac poison, and that it owes its poisonous properties to the alkaloid, nicotine, which is a liquid deadly poison and in small doses produces a rapidly fatal result. While there is complete unanimity as to the reality of acute tobacco, or nicotine, poisoning, there is still some divergence of opinion as to the reality of chronic tobacco-poisoning, or rather, of the eye-phenomena connected therewith. Clinical observation has satisfied me that tobacco-smoking and a variety of blindness stand in the relation of cause and effect, and the point as to whether the nicotine, the collidine, or any other ingredient of the smoke is the special agent in causing the mischief is beyond my ken.

Pathology:

"White atrophy" was said to be the condition that smoking gave rise to, when the evolution of tobacco emblyopia first began. This view of its...
pathology was strongly advocated by Wordsworth, when he propounded his views on the subject in the medical press in 1863; and, upon this point, Ernest Hart and R. A. Carter crossed swords with him, both going the length of denying the possibility of tobacco affecting the eyes.

Hutchinson, in 1864, stated in his first paper that a peculiar form of amaurosis or atrophy of the optic nerve was caused by tobacco; but, in 1876, he had altered his views of the pathological condition as he found that a large majority of his cases had recovered, while very few had become worse.

Streatfeild, in his article on Amblyopia in Quain's Dictionary of Medicine (1883), upholds the view that the disease which forms our present subject is an idiopathic 'white atrophy'. He there makes the statement that "some cases of white atrophy occur in which there has been no preceding neuritis", and he instances tobacco amblyopia as an example. The fallacy here is so evident that it is almost superfluous alluding to it. The statement is based merely upon the absence of ophthalmoscopic signs of neuritis; but we cannot dogmatically regarding the condition of the retro-bulbar portion of the optic nerve after using the ophthalmoscope with a negative result.
any more than we can give the pathological state of the optic papilla from an inspection of the cornea by focal light.

Nettleship has a pithy sentence or two in his paper on Tobacco Amblyopia in St. Thomas's Hospital Reports for 1879, which are worthy of quotation in this connection. He says: "There has been too great a tendency on the part of observers in this country to speak of the eye-symptoms caused by tobacco as "Amaurosis" and "Atrophy". Such terms have proved great stumbling-blocks to the general acceptance of tobacco as a common cause of failure of sight, and to the association of the well-known cases of amblyopia from central scotoma with their true cause."

Following upon the recognition of the central defect of vision, it came as a natural step in advance in the evolution of the complaint that the fibres of the optic nerve supplying the macula lutea should be thought to be those affected by the poison. Such, indeed, is Leber's theory. He considers that the central scotoma is due to a partial neuritis or atrophy of the optic nerve, or, it may be, an anomalous blood-supply. There is, in fact, inflammation of the optic nerve travelling from behind forwards, and this...
retro-bulbar neuritis affects the superficial laterally-situated fibres on the temporal side, which simply bend over into the retina (Hickel) and run outwards in a horizontal direction towards the macula.

Samelson of Cologne, at the meeting of the International Medical Congress in 1881, gave the results of a microscopic examination he had made of the optic nerve in a case of central amблиopia, and the conclusions he deduced therefrom were:

1. That central amблиopia is a retro-bulbar neuritis.
2. That the inflammation is primary, and causes a descending pressure atrophy from contraction.
3. That it is localized at the canalis opticus.
4. Bundles supplying the macula lutea are alone affected in typical cases.
5. These bundles decussate and appear finally on the lateral aspect of the nerve.

At the meeting of the British Medical Association at Cardiff last year the tobacco question was well-treaded out. The uncertainty of our knowledge of the pathological changes in this affection is evidenced by the variety of views expressed at this gathering. Dr. Benson opened the discussion on "the Causes of Atrophy of the Optic nerve other than glaucomatous," and the view he expressed is probably the correct one, that
atrophy of the nerve-trunk is probably always, if sufficient time be allowed, associated with atrophic changes in the disc. Under 'atrophy' Benson includes both acute and chronic inflammatory conditions, which is, to say the least of it, a somewhat liberal rendering of a well-defined pathological term.

Riles (Manchester) and Simon Snell (Sheffield) do not consider it proved that true nerve-atrophy is ever caused by tobacco; while Henry Power gives it as his opinion, based upon experience, that tobacco-amaurosis rarely goes on to complete atrophy.

On the other hand, Dr. Emyo-Jones (Manchester) had no doubt whatever that atrophy was caused by tobacco, and Bendelacki Newcomen was surprised to hear that there still remained doubts in some minds as to the effect of tobacco-smoking on the optic nerve.

Messrs. Edgar Browne and Marcus Dunn held views more in consonance with present-day pathology, and supported the view that the toxic action of tobacco should be included amongst the causes of a "bilateral retro-bulbar neuritis."

The comparatively recent investigations of Rettleship, Edmunds, and Lawford into the pathology of central amblyopia, published in the Ophthalmological Society's
Society's Transactions (Vol. III), practically bear out the views of the subject we have already alluded to as having been based upon theoretical considerations alone.

The question of 'atrophy or no atrophy' is certainly a difficult one to answer, and one that requires a greater experience and a more prolonged observation of cases than I am able to bring to bear upon it at present. If, however, theoretical considerations and the tendency of modern pathological investigations combine to make us admit the existence of a retro-bulbar neuritis as the cause of central amblyopia, I think that 'atrophy' as a possible result in some cases must follow as a corollary. Any further elucidation of this point must be postponed till we discuss the question of prognosis.

**Causes:**

As the name implies, the cause of this variety of amblyopia is presumed to be the administration of tobacco in some form or other, and the defective vision is merely one of the symptoms of the chronic poisonous action of the drug. The discoverer of tobacco amblyopia was MacKenzie of Glasgow. In his treatise on Diseases of the Eye published
published in 1854, he drew attention to the probable connection between certain toxic agents and obscure forms of amblyopia. He says—"Other poisonous substances"—besides belladonna, stramonium, 

etc. "applied to the body in small quantities every day, or several times every day, are probably productive of a similar effect, only that they act more slowly. Tobacco may be justly signalised as a poison of this sort. . . . One of the best proofs of tobacco being a cause of amaurosis is in the great improvement in vision—sometimes complete restoration—which ensues on giving up the use of this poison." He then quotes a case in support of his assertion.

Smoking is the most common procedure that the tobacco-worshipper resorts to in order to obtain the comfort of the "fragrant weed"; but the less aesthetic or more disgusting, modes of administration by chewing or snuffing have also their votaries. When it was first mooted that tobacco was the cause of a form of amblyopia, a great argument on the negative side was the comparative infrequency of the complaint compared with the universal prevalence of the habit that was supposed to be its cause. In this country, Wordsworth was the first to draw attention to the subject. In the Medical Times and Gazette of April 4th, 1868,
he reported a case of "Amaurosis occurring in a young man, an immoderate smoker of tobacco." The patient smoked the regulation $3.6 per diem that the inebriate lower-class smoker usually pleads guilty to. He had partial atrophy in both eyes. Wordsworth stated his conviction that the complaint was incurable, and our knowledge of to-day would support his views to this extent—that, when the neuritis arising from chronic nicotine poisoning has proceeded to such an extent that the ophthalmoscopic appearances are those of atrophy of the Optic Nerve, we are not likely to have any improvement. A. B. Barker entered the list against the new idea, and pooh-poohed Wordsworth for promulgating it. The point at issue, however, being whether "white atrophy" was or was not caused by tobacco smoking. Later on in the same year Wordsworth published a further report of cases, and this evoked the scorn of Ernest Hart, whose letters to the Lancet give us an indication of what the general state of knowledge regarding the disease was at that period. His argument against tobacco being a cause of amblyopia is based upon the fact that he had made ophthalmoscopic examinations in a certain number of smokers; and, the results being 
\[\text{negative}\]
negative as regards the presence of "white atrophy,"
he concludes with the fallacious non-sequitur that
tobacco-smoking does not affect vision. He also
reasons (?) against tobacco being a cause of white
atrophy from the fact that two patients who had
optic atrophy and smoked, got no better when they
abstained from the pipe. In replying to Hart's
criticism, Wordsworth quoted confirmatory evidence
of his views from Siebel's article in L'Union
Medicale of the same year, and gave it as his
firm belief that tobacco is largely concerned in the
production of amaurosis, and that the change in
the nervous structure of the eye is ultimately one of
atrophy.

In 1879, Brudenell Carter still denied the
reality of tobacco amaurosis, being confirmed in
his views of the subject by a negative experience
of the disease during a long residence amongst such
inveterate smokers as the Turks.

Idiosyncrasy and the variability in the strength
of manufactured tobaccos are sufficient answers to
those who would argue in the lines of Messrs. Hart
and Carter. The "first pipe" does not affect every
individual to the same degree, nor do all smokers
become inured to the habit with equal celerity & comfort.

\[ \text{End of text} \]
Not only is there a general idiosyncrasy, however—
a variability in the action of tobaccos upon different
individuals, but there is also a great variability in
the extent to which it affects the same person at different
times or under different circumstances. That is to say,
we have an inherited idiosyncrasy, and an idiosyncrasy
that is engendered by the surroundings. Speaking
from my own personal experience, I was able, when
surgeon on board a ship in 1881, to smoke five or
six times as much as I am able to do now that
I am confined to the non-too-profusey-oxygenated
atmosphere of the East End of London.

The quality of the tobacco smoked is a very
important point to take into consideration in studying
the causation. "Shag" is usually the variety patronised
by those patients whose condition is diagnosed at Moor-
fields as amaurosis from tobacco. Brudenell Carter's
argument from his experience in Turkey is, therefore,
comparatively worthless. The same may probably be
said of surgeon. Major Hogg's statement regarding
New York in the Lancet of 27th November, 1880. He
considers that "the question as to the effects of tobacco
upon vision still remains an open one"; and one of
his arguments is that "in New York, where people
eternally smoke, instances of amaurosis are comparatively
rare."
It has been denied that tobacco can cause amblyopia because of the supposed immunity of workers in tobacco factories from any such affection. Balegowstky, however, has stated that such factory hands do suffer from loss of sight, and the mydriasis with which I have noticed such operatives to be at times affected must alter their near vision at least. Dr. Davidson, certifying factory surgeon in the city of London, first drew my attention to this point, and, as I have been associated with him in his work for over a year, I have had ample opportunity of confirming it. Absorption may not take place to any great extent through the thick epidermis of the hands; but, in such a factory, the workers are constantly breathing an atmosphere that is simply loaded with the short glandular hairs and fine particles of the leaves. A complete, if somewhat uncomfortable, demonstration of this fact will be obtained if the visitor, on entering a tobacco factory where the ventilation is not very perfect, take a full inspiration per os.

Dr. Syl, in the New York Medical Journal for April, 1880, gives some statistics on this subject. Out of 102 tobacco workers whom he examined, 88 had normal vision. "In 2 only could it (the defective sight) be fairly attributed to this, no positive proof existing, however, that
that it was so. He states his belief that the influence of tobacco is comparatively slight, but, at the same time, admits that either idiosyncrasy may be essential to its action, or that constant contact may act as a preventative.

Mr. Shears, formerly resident medical officer in the bye Infirmary at Liverpool, came to the same conclusion as Guty from observations he made in Gutes. Coke's factory in that city. It is a good general rule that statistics regarding the banefulness of an occupation should be based upon the personal observation of the statistician, and not upon the more or less unreliable answers of the employer or employees. The application of this to the action of tobacco upon the eye renders the observations practically worthless, unless each operative were examined prior to commencing work in the factory, and after being at work in the tobacco-impregnated atmosphere for a certain number of years. To be absolutely reliable, the cases should be those of healthy young persons, preferably females, who had never smoked and were teetotallers. While I have made casual observations on this subject, I have certainly shirked the Herenluten task of compiling statistics that would exclude the coefficient of error so completely as to permit of their publication. One point has, however, struck me very forcibly during
my weekly visitations to such factories, and that is the great frequency with which they change their "young persons." This, I take it, is due to the fact that, while idiosyncrasy and custom aid the large majority of those who try this occupation in continuing at it, a certain number of the more susceptible, or, in other words, those who would be most liable to suffer from chronic tobacco-poisoning, are "weedeed out" and forced to give it up for something else. As we will never have the power to compel such individuals to continue at the occupation against their will, the action of tobacco upon those employed in its manufacture is an insolvable problem.

Next in importance to the question as to the place occupied by tobacco in the causation of amblyopia comes the argument as to the effect of alcohol. Hutchinson in reporting in the Medical Times and Gazette of Sept. 4th, 1869, "a case of tobacco amaurosis ending in blindness" gave it as his opinion that the injurious influence of tobacco is to some extent counteracted by alcohol. As has been well pointed out by Dr. Berry in his article on "Central Amblyopia" in the Moorfields Ophthalmic Hospital Reports (Vol. X) we must make a distinction between alcoholic stimulation and alcoholism (chronic) — between alcohol as a medicine and alcohol as a poison. In the
the former capacity, its stimulant action may antagonise
the sedative effect of the tobacco; but the chronic alcoholic,
instead of being in a stimulated condition, is rather constantly
in need of one. That tobacco is a sufficient cause by
itself is proved by the occurrence of the disease in teetotallers.
Stanford Morton, in the British Medical Journal of 9th
September, 1879, mentions the case of a teetotaller suffering
from central amblyopia, who recovered his vision simply
by abstaining from tobacco and using an eye wash as a placebo. Nettlefish also reports the case of a man
who recovered his sight completely by giving up his
smoking, although he continued to drink heavily through-
on. On the other hand, alcohol seems also capable
of itself of causing central amblyopia, a point that
is insisted on by, amongst others, Prof. Ernest Fuchs,
who, in his "Causes and Prevention of Blindness" gives
it as his opinion that alcohol, as well as tobacco, is a
cause of toxic amaurosis. Mr. Nettlefish also kindly
offered to put at my disposal particulars of a case occur-
rising in a man who had never smoked; but the following
is sufficient proof of the agency of alcohol in either
aiding to produce the affection we are at present discus-
sing, or one so similar that our present means of
diagnosing by ophthalmoscope, perimetry, etc. are not
refined enough for differentiation.
"J. C. Labourer 42; accustomed to drink ten to twelve pints of beer daily, but smokes little. He is suffering from partial amblyopia; complains of everything being misty after 8 or 9 P.M. He is very tremulous - in hands, head, and tongue. There is no swelling of eyelids or feet.

R.H. 30 and 9.2.

The discs are pale and the outer margin of the right disc is rather ill-defined.

Treatment - Mist. Quiniae Catharticae three times a day; and to abstain from beer.

A week later he reported himself at Moorfields. He had almost entirely left off beer, taking only half a pint occasionally. This abstinence in his idea made him “tremble”, but the tremor was not really so marked as before. Ultimately he recovered his vision completely.

The difficulty of differentiating between the various causes is very great, and Hutchinson devoted his first paper almost entirely to the settlement of this point. It had struck him as peculiar that the disease - “idiopathic cerebral amaurosis” - should be almost exclusively confined to the male sex. Such causes as occupation, syphilis, intemperance, sexual excesses, and the like had, he considered, to be carefully excepted before the cause of giving rise to the disease could be fixed upon tobacco.
Nettleship ("Diseases of the Eye") thinks tobacco is the sole cause of tobacco amblyopia so-called, and does not consider it settled what share in producing amblyopia is to be attributed to alcohol and such causes of general exhaustion as anxiety, underfeeding, and general dissipation. He points out that the only cases that will prove anything definite as to the part played in the causation by alcohol and tobacco respectively will be those of smokers who have never tasted alcohol or of drunkards who have never smoked.

Lowe ("Manual of Ophthalmie Surgery") in speaking of white atrophy says—"This form of atrophy is usually believed to result from the excessive use of tobacco, it is apparently checked by a discontinuance of its use. It is certainly common in invertebrate smokers, but it appears to occur as the result of any lowering cause, dissipation, drink..."

The first edition of Lawton's "Diseases and Injuries of the Eye" (1869) took no notice whatever of 'Tobacco Amburoxie', and the last edition (1885) has only mentioned the disease to deny its existence. The author says—"The theory that tobacco in excess will produce a peculiar form of white atrophy of the optic nerve, has received the sanction of the late Dr Macintyre and Ritchie, and of Messrs. Wordsworth, Hutchinson, &c others."
others. My own experience at the Royal London Ophthalmic Hospital, however, leads me to dissent from this doctrine, as I do not remember ever having seen a case in which the loss of sight could be fairly attributed to tobacco only. There was always, in addition to the immoderate smoking, some other excess, such as intemperance or an undue mental strain with loss of rest. I have also met with a similar condition of progressive optic atrophy in women who drank spirits largely. The rapid improvement of the sight which followed the giving up of the spirits could leave no doubt but that alcohol was the cause. In these cases there was no tobacco element in production of the atrophy." In adhering to the somewhat ancient confusion between 'white atrophy' and 'tobacco amblyopia', the author of this book is scarcely up to date with his knowledge of the subject; but I am not surprised at this after noting the pains he takes in diagnosing and treating external diseases of the eye, and in operative work at Moorfields Eye Hospital, compared with the minimum of attention he gives to such conditions of the eye as require 'perimetric' or 'ophthalmoscopic' examination for their determination.

The probability is, as is held by Graefe, Siehle, Salezowski, Sellenberg Wells, and others, that the combined influence of tobacco and alcohol have been at work in
the majority of cases of disturbed central vision that come under our notice.

A paper on "Tobacco Amblyopia" was read by Joseph Nelson M.D. at the Annual meeting of the British Medical Association at Cambridge in 1880. This gentleman holds the view of Hutchinson with regard to alcohol and tobacco being antagonistic. His conclusion is based upon the fact that, during the twenty months he had attended Arel's clinic at Vienna, he had only seen four tobacco amblyopes who were free drinkers; and, as these patients were the longest— from the onset of the disease—in seeking advice, he considers the slower invasion of the amblyopic condition was occasioned by the counteracting effect of alcohol. Unless their vision at the date when they were first seen was relatively better than the average of the non-drinkers, I would consider that their late application for advice was due to the dulling influence of the alcohol upon their general nervous system rendering them less anxious than the others about the state of their eyes. Alcohol is so universally recognised to be a preservative against post-mortem putrefactive changes that it is rather paradoxical to assert that it has a similar action against ante-mortem vital alterations, even though they be morbid degenerations.

S. Boelberg Wells
Scelberg Wells' view of the causation, to which I have already casually alluded, is as follows:

"I fully admit the fact, that the excessive use of tobacco, (but most frequently together with other causes) may produce considerable impairment of vision, and finally even atrophy of the optic nerve. But I cannot, from my own experience, accede to the doctrine, that there is anything peculiar in the form of atrophy of the optic nerve.

I believe that, in the commencement of the amblyopia of smokers and drunkards, the disturbance of sight is at first only functional. But if the cause persist, the disease passes over into an organic lesion."

Dr. Argyll Robertson in his summer course of lectures on diseases of the eye (1882) defines amblyopia and amaurosis as signifying impairment of vision due to some diseased condition of the optic nerve whether visible by the ophthalmoscope or not, and he gives the "topic" (due to poisoning of the nerve) as one of the varieties. Besides tobacco and alcohol, lead, quinine, opium, praemia, oxaluria, and diabetes are mentioned as causes.

Iler considers tobacco intoxication the cause.

I have already quoted from "Tobacco Amblyopia" by W. Shaw Sears, a paper which appeared in the British
British Medical Journal of June 21st, 1884. He alludes to the cases of atrophy we so often see where no cause can be ascertained, and suggests that some of these may be tobaccos. On the other hand, he considers that atrophy so seldom results probably because patients being, as a rule, healthy men in daily employment, they seek advice as soon as the complaint interferes with their work.

Clinical Features:

Tobacco Amblyopia was first described by Desmarres the Elder and Siehe in 1863. About this time, as we have seen, Wordsworth and Hutchinson were working at the elucidation of the facts, while Mackenzie had many years previously stated his belief in the existence of such a disease. It was in 1865, however, that Graefe drew attention to the broad distinction between progressive amblyopia with absolute limitation of the field of vision, and curable amblyopia with normal periphery but central defect in the field—central ambly prosoma. Tobacco Amblyopia was the general term used by the Germans, until Förster in 1868, and later in 1876, individualised the tobacco variety. His conclusions were:

1. The improvement on stopping of the tobacco gives the causation.
2. The periphery of the field is normal, and the amblyopia is
due to a central defect in an area reaching from the fixation point to the blind spot. In this region there may only be a colour defect.

3. Atrophy is rare.

4. The disease is in the Optic Nerve, not in the Brain.

Although Hutchinson had published some carefully compiled statistics in a series of papers, Hesteship was probably the first in this country to draw attention to the central colour scotoma, the delicacy of which as a test for toxic amaurosis had been discovered by Förster and Leber. He published a series of 14 cases in 1877 in the 'Medical Times and Gazette'.

One of his patients (Case 12) is stated to have said that "things looked black", and he describes this as probably due to the central scotoma. With the ophthalmoscope he found that the yellow spot third of each optic disc was paler than the other two thirds.

In his work on "Diseases of the Eye" (1882) the same authority describes the condition as one of progressive and equal failure without any other local symptoms, but with a general want of tone and sleeplessness. The ophthalmoscopic appearances are varied; the defect of sight is greatest in bright light; the pupils are normal or slightly sluggish; and there is a central colour scotoma which extends from the fixation point to the blind spot.
Previously to this, Rettelson had published a report of a series of 23 cases in the St. Thomas's Hospital Reports for 1879 under the title of "Notes on the Diagnosis of "Sbacceo Amblyopia". He drew attention in this paper to the following points:

1° The patient has no difficulty in walking about.
2° The sight is best in rather dull light.

His, he considers, is probably due to the dilatation of the pupils. It has been stated that this symptom is probably due to the dilatation differentiates between 'neotie' and 'alcoholie' amblyopia, Martin in 'De l'Amblyopie Neotique' giving the symptoms of the latter as 'great contraction of the pupils', and 'improvement of the vision in bright light'. These cases of symmetrical progressive amblyopia with contracted pupil are not so common in Rettelson's opinion as the others.

3° There is a central defect in the visual field, and this forms an oblong or oval patch extending from the fixing-point out towards, and often beyond, the blind spot. As green and red are perceived only in the central part of the field, the scotoma is especially marked for those colours and is symmetrical, which symmetry is anatomical, not physiological.

Martin in the M.D. thesis already alluded to comes
Comes to the following conclusions:—

1. The differential diagnosis of tobeses from alcoholic ambyopia is easy.
2. The condition of the pupil is the most important symptom.
3. In alcoholic cases progress is irregularly interrupted by relapses: in tobeses, it is slow but uninterrupted.
4. 'Tobacco' is symmetrical: 'alcohol' is not. The eyes, in the latter form of ambyopia, are either not both affected, or, at least, not simultaneously.
5. Vision in 'tobacco' cases is not better at night than during the day: in 'alcohol' it is.
6. The visual disturbances from 'tobacco' are:
   a. Binoeular ambyopia
   b. Muscular asthenopia with central scotoma
   c. Ambyopia due to both Alcohol & nicotine
   d. Here are neither hallucinations nor diplopia, which are both present in alcoholic cases.

If symmetry is so absolutely diagnostic of tobeses from alcohol, how is the following case accounted for?:

"O. E., 41; Shoemaker. Smoked 3f per day regularly for a long time until a few years ago, when he found it did him harm. This was of the strongest variety — 'Taddy's,' but since the above period he has used 'Bud's.'
Bird's eye. Only drinks one pint of stout daily. He is married and has had eight children, two of whom are dead, the one being stillborn and the other of convulsions. His vision has gradually failed for eighteen months, but the failure has been more rapid lately.

Vision:  
R. S. \[
\frac{20}{0}
\]  I 20 at 8" - 15".

L. S. \[
\frac{20}{100}
\]  I 16 at 15" - 20".

No improvement in either eye with glasses.

In each eye there was a central scotoma for colours (red and green).

He was ordered to leave off smoking, and a month later he reported that he had only smoked 3½ during that time, and that the abstinence from tobacco had done him much good. He still drank the same quantity as before. At this time the vision in the two eyes was:

R. S. \[
\frac{20}{70}
\]  and I 14 at 10".

L. S. \[
\frac{20}{40}
\]  and I 12 at 10".

The colour scotoma has apparently gone. Ultimately he recovered his vision completely, the left eye being perfect before the right.

Hirschberg, in his paper on "Tobacco Amblyopia" read at the Annual meeting of the British Medical Association at Cork in August, 1879, alluded to the fact that in England amblyopia is more frequently attributed
attributed to tobacco than on the Continent. Swanzy, in the subsequent discussion, credited the tobacco smoked—"Limerick twist", which contains a large amount of deleterious substances, with the greater frequency of the disease in Ireland. The statistics given by Hirschberg as to the amount of tobacco smoked per head would certainly lead one to expect that the disease would be more prevalent in Germany (2½ lbs.) than in France (1½ lbs.), Austria (1¾ lbs.), or England (1½ lbs.). The quality of the tobacco must be accountable for the difference in the effects.

Hirschberg is of opinion that the existence of such a disease as tobacco amaurosis is sufficiently proved by the recovery that takes place when the assimilation of tobacco is interrupted. The ophthalmoscopic appearances are, primarily, negative in character; but, afterwards, a paleness of the outer quadrant of the disc is characteristic. The impairment is binocular and there is a central colour scotoma. He is one of those who consider that there is also an amblyopia developed sometimes from the abuse, or too frequent use, of alcohol, and in this variety also there may be a complete colour scotoma, so that it is difficult to differentiate between the two. Improvement, however, takes place with abstinence from alcohol, as we have already noted. Hirschberg holds the opinion that an idiopathic
retrobulbar neuritis may exist independent of tobaee or alcohol, just as Kettleship fails to find that these two co-efficient account for every case, and therefore believes that amblyopia in some cases is hereditary.

Dr. Berry was the author of a very able paper on "Central Amblyopia," which was published in Vol. X. (1880) of the Ophthalmic Hospital Reports. He first gives the history of the specialization of the disease by Von Graefe, Leber, and others, and discusses the causation, the influence of alcohol being fully treated of. He states the occurrence of the papilloma in his opinion, usually an oval extending from the papilla to beyond the macula lutea, but it may be ring-shaped as described by Leber and Kettleship. He thinks too much has been made of the temporal half of the disc. This may be, but I have seen this change so frequently that I cannot associate it in memory from some connection with the pathological changes in the nerve that give rise to the clinical phenomena; and it is certainly a very curious coincidence, if it is nothing more, that this wedge-shaped atrophic appearance should be situated in the very part of the disc in which we would except to find it both on anatomical and physiological grounds. It is certainly very distinct from the choroidal cleft or the myopic crescent with which Berry thinks it may have been
seen at times confounded.

Mr. Nelson, whose paper I have already mentioned, holds with Nettleship that tobacco amblyopes see best in a subdued light, but he does not think that this improvement is due to dilatation of the pupil. He puts forward what is, to say the least of it, a very dubious alternative hypothesis, viz., that in the subdued light the cloud or veil of the object is less evident. This makes one almost ask the question whether our brain perceives most what affects our retina as a stimulus or that which prevents the same!

The affection is symmetrical, and Nelson points out that the color sense is normal for large, but defective for small objects, which simply means that some of the rays from the larger object impinge upon the peripheral unaffected portion of the retina. He discusses the question whether the scotoma begins at the fixation-point and extends to the blind-spot, or vice-versa; and gives his reasons for the opinion he holds that it begins at the blind-spot as against the opposite view of Leber, Nettleship, &c. On this point I have not made sufficiently accurate or extensive clinical observations to form an opinion; but Nettleship I am inclined to agree with him on physiological grounds, unless we predicate both a central and peripheral
peripheral neuritis to account for the symptoms.

Nelson agrees with the generality of observers with regard to the ophthalmoscopic changes, when such are present. In most cases there is hyperaemia of the disc and the margins are blurred; but sometimes the discs have pale temporal quadrants, or they are atrophic like with fewer vessels.

Mr. Bliss, in the paper already quoted, bases his diagnosis of "Tobacco Amblyopia" on finding in an excessive excessive smoker, rapid and great visual failure not remedied by glasses nor accounted for by either of obvious external changes or ophthalmoscopic appearances. The field hereunder attached was 

\[ \text{taken} \]
taken from a patient who answered the description
given by Shears, but it is evidently not a tobacco-
case, although it was thought it might be one till
the perimenter settled the point decisively. The
pathognomonic indications afforded by the perimenter
in cases of tobacco amblyopia were evidently not
sufficiently taken advantage of by this observer, as he
states at the conclusion of his paper that in only
twelve out of the forty cases he enumerates were careful
observations made with regard to a central defect in
the visual field, and, of these twelve, six cases only
had well-marked central scotomata!

Henry Jules in his "Handbook of Ophthalmic
Science and Practice" (1884) describes "Tobacco Amblyopia"
as being characterised by a diminished acuity of central vision,
of which the central colour scotoma is one of the earliest
symptoms. The failure is progressive, but the periphery
remains normal. Vision is worse in bright than in
subdued light, and it is relatively more affected for
distant than for near objects. The ophtalmoscopic
appearances he considers, are unimportant. The
insidious onset of the disease may be accompanied
by general symptoms of nervous derangement.

Dr. Argyll Robertson, in his class lectures, gives
the following as the chief diagnostic points of tobacco
amblyopia.
amblyopia:—

1. The symmetry of the affection.
2. The rapidity of the impairment.
3. The normal periphery of the field of vision.
4. The central Colour scotoma.
5. The negative ophthalmoscopic appearances.
6. The concomitant general nervous disturbance.

If a patient present himself to us with failing sight, if he be a great smoker, and if we find as external condition of the eye nor any general derangement of the system — anaemia, albuminuria, diabetes, locomotor ataxia, &c. — to account for the symptoms, attention to the points indicated in the preceding list will enable us to make a correct diagnosis.

The only other paper I will allude to is one by Hartnedge in the British Medical Journal of January 31st of this year. He divides and tabulates his twenty cases after the plan proposed and carried out by Hutchinson. The three diagnostic features he relies upon are:

1. Rapid unaccountable failure of vision.
2. Central Colour Scotoma.
3. Excessive smoking.

Diagnosis: The question of diagnosis has already been
been well ventilated under Clinical Features, and 
I have given in my adhesion to the list of differential 
points noted by Argyll Robertson. I will therefore 
content myself with giving one illustrative case, 
especially as we find it to be characterised by what 
I have already characterised as sometimes present 
without the case losing its "tobacco" nature, viz., 
slight asymmetry:

"G. W.; 42; Waterman. Sought advice at Moor-
fields in April, 1884, regarding failure of vision in 
his right eye (20/10 and J. 10). Left eye was normal.
Dr. Morton diagnosed (doubtful) retinitis, and Mr. 
As he got no better, he ceased his attendance at the 
end of a month.

At the end of September, the left eye failed. There 
was no pain in the eyes, but there was twitching. He 
is very nervous, &c. Has discus and palpitation 
after exertion. Has frequently mistaken silver and 
gold. Sees better in the evening than in the morning.
Fields have normal periphery, and there is a well-
marked central scotoma in each for red and green, 
which he calls yellow & white. This scotoma extends 
outwards, from beyond the fixation-point upwards, and 
downwards for about 15°. Blue is seen well all over
the field, as also are red & green outside the scotoma.


L. E. 20/200 & J.16 at 7.

Eyes normal : Morton says anaemia at inner edge.


L. E. 20/100 & J.14 at 8.

This improvement in four days was the result of his
having ceased smoking & taken Rup Vomica & Iron.

Jan. 22nd /85. Q. = same.

Urine - acid : no albumen nor sugar.

It is needless to give the detailed clinical notes.

Suffice it to say that, on April 9th, he had with
both eyes. Q. = 20/20 & J.1.

Annexed are the "fields" in this case taken on Jan. 19th:

---

Diagram:

- Right Eye: Scotoma for green, vision not reduced.
- Left Eye: Scotoma for green, vision not reduced.
Prognosis:

This part of our subject has been already fully treated of when discussing the question of the occurrence of atrophy as the result of tobacco-poisoning, so that very little remains to be said.

The alteration in Hutchinson's views has been noted. Nettleship believes that recovery of sight is the rule in cases which come under treatment at an early stage, and where the cause is done away with. In the more chronic cases, or where already the whole disc is pale, a moderate improvement, or even an arrest of progress is all we can expect; but "complete blindness seldom, if ever, occurs." Nelson says that recovery may follow provided the patient gives up or reduces his tobacco. Stieglitz holds that complete blindness never occurs. In the discussion on Stieglitz's paper, S. E. Walker of Liverpool maintained that complete blindness frequently results. In Juler's opinion the prognosis is favourable in proportion to the rapidity of failure and the shortness of the duration of the disease. Argyll Robertson considers the prognosis is favourable, total blindness seldom resulting. My own opinion conforms to this. The prognosis I consider to be decidedly good, with a qualification as to the cause being done away with at an early stage of the disease, or when, according to Stieglitz well, the
disturbance in the optic nerve is either merely functional or very little organic alteration has taken place. While, however, predicated the rarity of atrophy as a result, I admit its possibility. As it has been well put, there are those who would rather smoke than see."

Treatment:

If our view of the etiology of the complaint be correct, avoidance of the cause is a sine qua non to the permanence of a successful course of treatment. Hutchinson says "immediate and complete abstinence," and in Nettleship's series of 14 cases (1877) abstinence from tobacco was followed either by recovery or arrest of the disease. In 1879 (series of 23 cases) he states that absolute withdrawal of tobacco is essential, but Shears quotes him as stating that mere reduction of the quantity is sufficient. G. E. Walker is of opinion that mineralization and the local installation of sulphate of styrchnia into the eye aid abstinence from the exciting cause. Argyll Robertson joins the administration of styrchnia and iron with avoidance of the cause. Jules is very emphatic in laying down the law that abstinence, total and unconditional, from both alcohol and tobacco is essential to successful treatment. General tonic
treatment he also considers helpful. My own views of the subject are that a patient with tobacco amblyopia may recover by simply moderating the quantity or modifying the quality of the tobacco consumed, so that there will be more likelihood of his doing so if he abstain completely from the drug, that the administration of a serin and blood tonic, such astrychmin & iron, probably act as an auxiliary though it is not by any means an essential.

The following cases are illustrative of the above assertion. In the first one, reduction of the amount of tobacco consumed sufficed to effect improvement; in the other, quinine was alone prescribed and the results were just as satisfactory as if the patient had been mercurialized (according to Walter), or had undergone a course of hypodermics of strychnia. W. C.; 50; upholsterer. Consulted Mr. Dunn in June, 1885. Symmetrical failure without pain for eight months. Vision best in the evening. No mistaken coins. Has smoked 3 2/3 daily of "cut Cavendish." Also chews. Central corneal scotoma for green red. Knee reflexes normal. Fundus and media normal. O. (Both) = 20/200.

Although he had only reduced his tobacco to under 3p per day, this patient's vision was improved in
five months to 20 and 1/16. Whether entire abstinence for the same period would not have cured him completely remains a matter of doubt; the probability is that it would.

The second case is:—


Vision (Both) = 20/70 and 1/20.

Smoked 3½ of shag daily. He was advised to give up smoking which he did completely. A mixture was ordered containing Quinine Sulph. = gr. 1; thrice a day.

A month later = 20/70 and 1/16.

6½ months after first attendance, his vision was:

R. E. = 20/20 and 1/1.

L. E. = 20/30 and 1/1.

Six months after — Vision Normal.

His fields were taken, and although the hemianopic was normal and the scotoma for green had disappeared, there appeared to be a central defect for red remaining."
Books and Papers Consulted:

- Mitchell-Bruce's Materia Medica.
- Garrod's
- The National Dispensatory (Stille and Mauzch).
- Notes of Dr. Argyll Robertson's Lectures on Diseases of the Eye.
- Quain's Dictionary of Medicine ('Amblyopia').
- Royal Medical & Chirurgical Soc. Trans. for 1867.
- Prof. Brainger Stewart's Introduction to Diseases of the Nervous System.
- Mackenzie's, Lawson's, Juler's, Settle's, and Lowe's Text-Books of "Diseases of the Eye."
- Galezowski's Amacrines et Amblyopies Oculaires.
- Ophthalmological Society's Transactions - Vol. 3, 8, 110.
- International Medical Congress' Trans. - Vol. III.
- St. Thomas's Hospital Reports for 1879.
- Rankin's Abstract for 1863 (Vol. II) and 1865 (Vol. III).
- Lancet for 1863 (Vol. II).
- British Medical Journal for 1879, 1880 (Vol. II), 1884 (Vol. I), and 1886 (Vol. I).
- Braithwaite's Retrospect for 1880 (Vol. I).
disturbance in the optic nerve is either merely functional