TOBACCO AMBLYOPIA.*

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TOBACCO amblyopia, or partial blindness arising from the use of tobacco, may be regarded as a special subject in so far as it is usually looked upon as an eye disease; but it has a wide general interest as a common and important example of the action of a toxic substance upon the nervous system, especially the visual nerve mechanism, and also from the sociological and economical point of view. The potential scope of a discussion on tobacco amblyopia is therefore quite extensive so that in the present instance it will be necessary to limit our survey as far as possible to features of general interest.

Tobacco was introduced into Great Britain about the end of the fifteenth century. It rapidly acquired both friends and enemies, and it is no exaggeration to say that no drug has ever attracted more public attention. Both in poetry and prose, the most fulsome and extravagant adulation was lavished upon it, and on the other side no extreme of abuse or prejudice was spared. As a drug it was used for every possible complaint and administered in many astonishing ways. The controversy lasted for about three hundred years, but little emerged in connection with its action beyond that if used immoderately it caused gastric and cardiac disturbance. A noxious influence on the offspring of the smoker and dimness of vision are also mentioned. A strongly urged objection was that it caused excessive salivation and spitting and in this way deprived the body of a necessary fluid. It is interesting, at this stage, to note that the refinements of civilisation have considerably diminished the spitting habit, while at the same time the consumption of tobacco has increased.

In the literature of these days most of the references to tobacco poisoning are to acute tobacco poisoning, in which it was usually impossible to examine the vision as the patient was prostrated or comatose at the time. In any case no serious defect of vision appears to have remained. The first references to tobacco amblyopia were made in Germany at the end of the eighteenth century, and in Scotland in 1832 by Mackenzie in the first edition of his *Diseases of the Eye*. Later, in the

* Read at a Meeting of the Medico-Chirurgical Society of Edinburgh on 1st May 1935.

fourth edition, in 1854, he stated that cases of tobacco amblyopia might be met with any day in any ophthalmic clinic. He also drew attention to the greater susceptibility of weak or ill persons as compared to the strong and healthy.

The recognition of tobacco as a cause of amblyopia developed more slowly in England. In December 1856 Dr Samuel Solly, during the course of a clinical lecture on paralysis, made some remarks about the dangers of tobacco which gave rise to an enthusiastic controversy, " The Great Tobacco Question—Is Smoking Harmful ? " which largely filled the correspondence columns of *The Lancet* during 1857. Every aspect of the subject was discussed except that of the effects of tobacco on vision, and it was not until 1863 that Jonathan Hutchison showed the connection between tobacco and impairment of vision. In the following years the subject received a moderate amount of attention, until in 1887 a special meeting of the Ophthalmological Society of the United Kingdom was held to discuss the problem of toxic amblyopia whether due to tobacco, alcohol, or any other agent. From this report certain features emerged quite definitely :—

1. The amblyopia was found to be due to tobacco.

2. No case of amblyopia with central scotoma due to alcohol or any other toxic agent was reported. Some of the members strongly emphasised the opinion that alcohol was not a direct causal agent.

3. Depression of health from any cause was found to be an accessory influence. Diabetes, excessive alcoholism, dyspepsia, ill-health, and mental worry were referred to as predisposing factors.

4. It is evident from the report that the accurate clinical examination and analysis of the visual defect was as yet undeveloped. The presence of a central scotoma for colour was established, but its precise character was not closely scrutinised.

In spite of the absence of any evidence in this report, text-books written in English often refer to alcohol as if it were an actual or possible direct cause of visual defect indistinguishable from that due to tobacco.

We may now examine the question as it presents itself to us at the present time. The ganglion cells of the retina, or the fibres of the optic nerve which pass from these cells, are

extremely susceptible to the action of toxic substances. These toxins may be grouped in several ways. Some affect the peripheral cells or fibres and cause peripheral visual loss; others—the majority—affect the central cells or fibres and cause a central visual defect or central scotoma. Many years ago Uhthoff noted that toxins which caused central defects also caused peripheral neuritis, and that tobacco was an exception to this as peripheral neuritis was not observed. The toxins therefore exhibit a definite selectivity in a somewhat similar way to that in which the diphtheria toxin acts on the palatal motor nerves or on the third nerve, or lead acts on the musculospiral nerve.

Toxins which affect vision may also be divided into exogenic, such as arsenic, felix mas, quinine, and others: or endogenic, such as those which occur in diabetes, pregnancy, or perhaps excessive hæmorrhage; and again deficiency diseases, such as pellagra or beri-beri, have been reported as causes of visual defect of a similar type. Thus the optic nerve or its ganglion cells seems to be a sort of touchstone for toxins of certain kinds, and it is interesting to note that the group affecting the central elements is very much larger than that affecting the peripheral elements, that is, the tobacco group is larger than the quinine group. It is also interesting to note that it is the visual elements distal to the chiasma which are susceptible; as far as is known there is no toxic amblyopia above this level although the nerve fibres, as far as the external geniculate body, are the same. This is perhaps an argument in favour of the view that it is the ganglion cells alone that are concerned in all or nearly all toxic amblyopia. Atrophied nerve fibres have been traced as far as the external geniculate ganglion. Another feature of interest is the reaction of the nerve elements to alterations in the constitution of the toxins. Quinine causes peripheral blindness, but if it is altered to ethylhydrocuprein it produces central scotoma; and inorganic pentavalent arsenic compounds produce central scotoma, while organic trivalent benzol-ring arsenic compounds, *e.g.* arsenobenzol, produce as a rule peripheral blindness becoming total. Acetylarsan, a pentavalent preparation, is reported to cause peripheral defects rather than central scotoma, if improperly given or if the health is bad. Lastly one may refer to the difference between the action of toxins in regard to dosage and duration of administration. As already pointed out, no cases of acute

tobacco blindness have been reported, while quinine blindness, for example, is usually the result of one or a few doses. There does not seem to be any chronic quinine amblyopia. Similarly an extraordinary massive consumption of methylated spirit may cause blindness, but ordinary chronic methylated spirit drinking does not appear to produce any harmful effect.

The reactions of the optic nerve to toxins may be summarised as follows :—

1. The special susceptibility of the retinal ganglion cells and possibly the optic nerve fibres. In addition there is the relative susceptibility or " idiosyncrasy " of individuals.
2. The immunity of other parts of the visual pathway.
3. Precipitation or aggravation of the symptoms by ill-health of any kind.
4. The cumulative effect of the toxic substance.
5. The establishment of tolerance in varying degrees.
6. The selective affinities of different toxic substances for different groups of visual elements.
7. The variation in this selective affinity by altering the constitution of the toxin.
8. The difference between acute and chronic poisoning in respect of certain substances.

It is evident that the whole subject of the effect of toxins on the optic nerve and the retinal ganglion cells is one of great interest and appears to afford fruitful ground for further research.

Tobacco belongs to the group of poisons which affects the central nerve elements. Amblyopia is only one of the symptoms produced by excessive absorption of the drug : the gastric and cardiac effects have already been mentioned. The toxic substance in tobacco is nicotine, which may be absorbed by smoking, chewing, snuffing, or eating tobacco, so that the actual toxin is not necessarily a distillation product. As we have seen, acute tobacco poisoning does not appear to produce special effects upon the optic nerve, and a certain degree of tolerance is usually soon established. Most of us can remember the period of initiation. Tobacco amblyopia is therefore a symptom of chronic tobacco poisoning and therefore many tobacco amblyopes, if carefully examined, exhibit other signs of intoxication. The different forms in which tobacco is used influences the results. Thus it is usually necessary for an

enormous number of cigarettes to be smoked before amblyopia is produced, while a much smaller number of cigarettes will produce definite gastric or cardiac disturbance. On the other hand, if a pipe is smoked or if chewing is practised, the gastric and cardiac symptoms may be very slight while amblyopia is pronounced. The method of absorption of the nicotine is probably partly directly through the mucous membrane and partly by solution in the saliva and nasal mucus which are subsequently swallowed. In this respect the connection with spitting is interesting, and the question arises as to whether the old habit of spitting, which seems to have been regarded as a necessary accompaniment of smoking, was not to some extent helpful as a preventive of excessive absorption and subsequent amblyopia.

In Edinburgh and in some other parts of Great Britain tobacco amblyopia is relatively common. The statistics of the Royal Infirmary, Edinburgh, show that about eighty-four cases are seen there every year. The total number of cases in Edinburgh and district must be considerably larger, as cases are seen in other clinics and in private practice, and some are warned by opticians or by their friends and do not get included in statistics. At a rough estimate, possibly about one in ten thousand of the population is affected.

Tobacco Amblyopia, 1913 to 1934.

Total patients	183,715
Tobacco amblyopia	1856 (1·01 per cent.)
Year of highest percentage	1915 (1·55 ,,)
Year of lowest percentage	1921 (0·46 ,,)
Highest month (average)	April (9·3)
Lowest month (average)	December (4·2)
Highest age	84
Lowest age	21
Average age	54·2
Female cases	8
Highest consumption	9 oz. weekly
Lowest consumption	½ oz. weekly
Average consumption	3-3½ oz. weekly
Occupations	1386
Outdoor	655
Indoor	731

The proportion of tobacco amblyopia cases to all cases of eye trouble attending the out-patient department of the

Infirmary is about 1 per cent. It has been as low as 0·45 per cent. and as high as 1·55 per cent.; in the year 1916 it was as high as 2·13 per cent. in one clinic, an exceptionally high figure. The average percentage over the last twenty-two

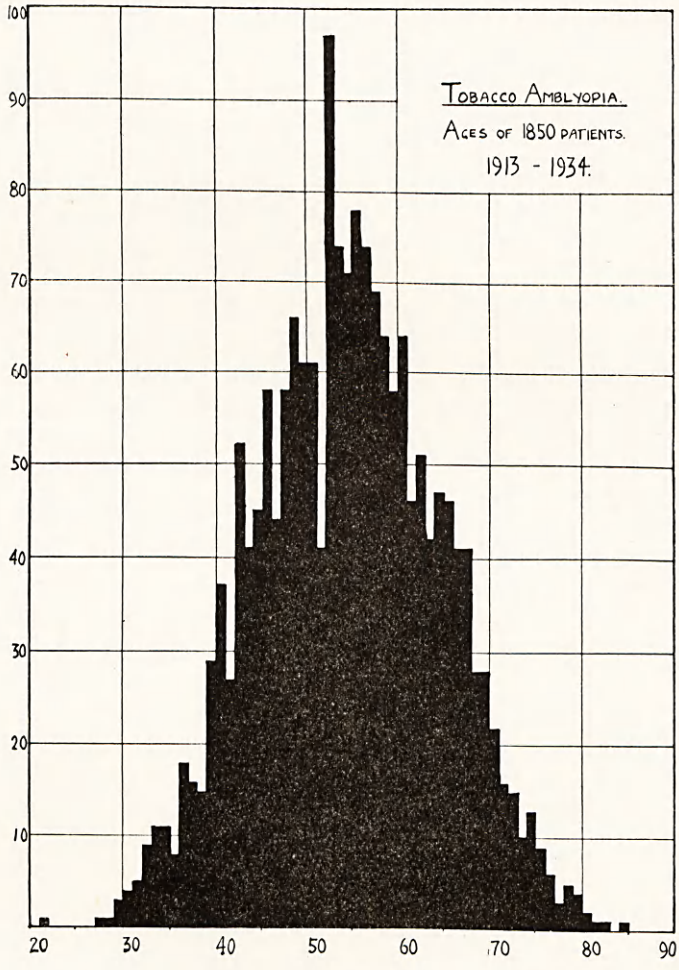
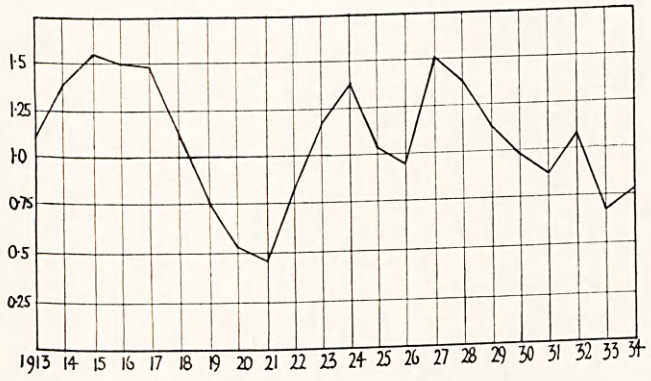


Fig. 1.

years is now just under 1 per cent. owing to a slight decline in the last five years. In the last twenty-two years there have been 1856 cases in the Infirmary, including eight females (0·4 per cent.). The ages varied from 21 to 84 years, with an enormous preponderance between 45 and 65, especially about

the age of 52. This is about the period in life when many persons find it necessary to modify their habits in the interests of their health. The occupations were very varied and were not recorded in all cases. A slight preponderance of indoor occupations was shown. Many of the patients were miners and labourers, no doubt owing to the fact that there are many miners and labourers in the area from which the patients come.

The amount of tobacco consumed varied from $\frac{1}{2}$ oz. a week to 9 oz. weekly, the average being between 3 and $3\frac{1}{2}$ oz. In five cases only $\frac{1}{2}$ oz., and in forty-four cases only 1 oz., was used weekly, and in these cases the patients were either specially susceptible or had had some illness. Only one hundred and



Tobacco Amblyopia. Annual Incidence Percentage of total eye patients.

Fig. 2.

four patients smoked 5 oz. or more, and several of the larger weekly quantities were cigarettes. It is apparently possible to smoke well over 1 lb. of cigarettes weekly without amblyopia resulting, though amblyopia may, here also, result from smaller quantities. The smallest quantity of cigarettes producing amblyopia was seventy a week. The stronger varieties of pipe tobacco are more liable to affect the eyesight. The lower figures for consumption are of interest in regard to diagnosis as they show that a consumption generally regarded as harmless may, under favourable circumstances, produce the disease.

The annual incidence is of some interest. In 1913 the number of cases was rising, and the peak was attained during the years 1915, 1916, 1917, that is to say, during the most strenuous years of the War. Before the end of the War the

number of cases had begun to fall, and this fall continued rapidly until the years 1920 and 1921 when the incidence rose again as rapidly as before until 1924. It fell to the average in 1926, but rose again in 1927, and since then has been declining. I have attempted to correlate the undulations of this graph with social conditions but without much success. It is extremely difficult to be sure that any coincident circumstances or conditions are really causally connected with the amount of tobacco amblyopia. One may note that 1921, when the amblyopia was at its lowest, was the year of the coal strike, and 1926, the point of another sharp depression, was the year of the general strike. As regards the War peak it

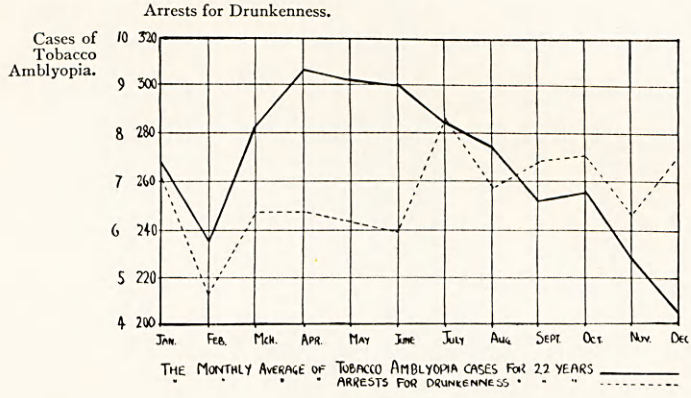


Fig. 3.

must be remembered that the tobacco amblyopia is calculated on the men left at home, and that during the years referred to they were drawing very high wages. After 1917 there were fewer men of the tobacco amblyopia age left in this country and the purchasing power of money fell, so that a drop in the number of cases is not surprising. The second peak in 1927 occurred in the boom years after the War.

The monthly incidence is highest in the spring and early summer months and lowest in December but rises very steeply in January. This high early-summer incidence has been noted by others. It must be remembered that the incidence referred to here is the incidence of application for advice, not the incidence of commencement of symptoms. Symptoms began on an estimated average three to six months earlier, but it was impossible to obtain definite data. It is possible that the

over-smoking takes place in the winter and its effects become manifest in the spring.

Female cases amounted to only 8, or 0·4 per cent. Of these, five were due to pipe smoking, two to snuff, and one to cigarette smoking. One female patient, who stated that she smoked only ½ oz. of tobacco a month, had a blood pressure of 220/105 and complained of illness. It is possible that her statement is not quite reliable, and I have therefore not suggested that ½ oz. of tobacco a month is sufficient to produce amblyopia. From the statements of the few female cases it would appear that on the whole a smaller amount of tobacco is sufficient in females.

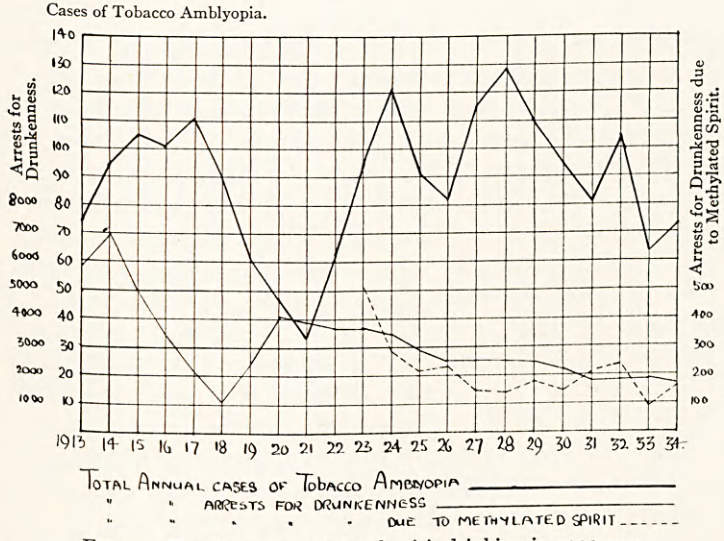


Fig. 4.—The drop in methylated spirit drinking in 1933 was due to an alteration in the basis of making arrests.

As the view is widely held that alcohol is concerned in the production of tobacco amblyopia, such statistics as were available have been collected. The graph, which in this case refers to the total annual cases of tobacco amblyopia and the total annual arrests for drunkenness, shows nothing to indicate that drunkenness is common at the same time as tobacco amblyopia, and the same is true in regard to methylated spirit. It is true that the figures are not exactly comparable, as the figures for drunkenness are for Edinburgh only, but had there been any connection between alcoholism and tobacco amblyopia a little more agreement in the graph might have been expected.

Much more convincing are the clinical reasons against any relationship between alcohol and tobacco amblyopia. The hospital patients who complain of tobacco amblyopia belong mostly to the working classes and consume relatively little alcohol. Those who consume alcohol in large quantities are not to be found in the eye department, but in the surgical or medical out-patient departments, and no cases of toxic amblyopia are ever referred from these departments. Heavy drinkers or dipsomaniacs do not develop amblyopia; and indeed it is noteworthy, when so much is heard about drug addiction, that toxic amblyopia, appearing in the eye clinic, can nearly always be traced to the same cause, namely tobacco. Even in the case of methylated spirit drinking there is no chronic amblyopia. In most towns there are individuals who soak themselves in methylated spirit without untoward results as to vision. I have been told of one in Edinburgh who has been arrested seventy-two times for drunkenness due to methylated spirit, and he has not complained of amblyopia. It is, of course, true that occasionally, though very rarely, blindness due to methylated spirit drinking may occur, but in these cases large quantities have been drunk very rapidly.

In this country at any rate there is no doubt that toxic amblyopia is due purely to tobacco, and that any influence that alcohol may have is merely that of a factor in depressing the general health. The term "tobacco-alcohol" amblyopia is therefore incorrect as far as this country is concerned. It is quite possible that in other countries alcohol may be a factor, but in these cases the disease is not due to ethyl-alcohol or to methyl-alcohol, but to toxic distillates, such as those contained in wood spirit, which have found their way through manufacture or sale into alcoholic drinks.

Tobacco amblyopia appears to be fairly common all over Europe, more common in Europe than in America, and perhaps slightly more common in Scotland than in England. A percentage of over 1 per cent. of all eye patients indicates that the disease is frequent in the country or district concerned. In eastern Europe and in tropical countries it appears to be rare, although there is a considerable tobacco consumption. According to de Schweinitz, Cubans and South Americans are relatively immune. Possibly the method of smoking mitigates the nicotine absorption. With regard to race differences, the literature provides almost no reports of tobacco

amblyopia in coloured races where large amounts of tobacco are consumed. In South Africa where much tobacco, although of a light character, is consumed, tobacco amblyopia is extremely rare either in the white or coloured population. I remember only one case in a coloured man who used to eat the "dottle" of pipes. It is probable that it is mainly the habits of the different races, rather than any physiological immunity, which causes differences in the incidence in different parts of the world.

Clinical Features.—The clinical picture of tobacco amblyopia is so characteristic that it is frequently easy to recognise the disease without an expert eye examination. The patient is usually a man between 50 and 60. In many cases he has been to an optician who has told him the nature of the trouble, or it has been suggested to him by friends, and he comes to the oculist for confirmation and advice. In other cases, if encouraged to tell his own story, he will say that his sight or his spectacles are failing, but he usually requires to be questioned with regard to the length of time during which the failure has been going on. He often makes two very characteristic statements. Firstly, that he cannot recognise the faces of his friends in the street, and secondly that there is a mist between him and everything he looks at. He also thinks that he sees rather better in the dusk. If asked about his tobacco consumption, he may indicate quite a moderate amount; but if asked when he reduced his consumption to that amount he will give a date usually a few weeks previously, the original quantity used being as a rule between 3 oz. and 5 oz. By this time, or sooner, the examiner will be conscious of a peculiar heavy odour of stale tobacco which indicates the pipe smoker, while a somewhat similar but different odour, together with stained fingers, indicates the cigarette smoker. Sometimes the patient stresses his inability to read and only mentions his disabilities in other ways when encouraged to describe his symptoms fully. He only complains of difficulty at work when the work is of such a kind that really good vision is required, such as that of a compositor or tailor. Very frequently, arterio-sclerosis in greater or less degree is present, and if questioned about his health the patient will often state that he had some illness or accident or anxiety or other cause of depressed health before the visual trouble began.

Other symptoms of tobacco poisoning, such as tremor, dyspepsia, or cardiac disorder, may or may not be present.

Age.	No. of B.P.s.	Average Systolic B.P.	Daily.
30-40	2	160	127
40-50	3	147	129
50-60	12	173	132
60-70	16	151	140
70-80	3	172	147

The vision of the patient may be severely depressed so that he can, for example, only see the top letter of the ordinary test card at a few feet distance. In other cases the vision

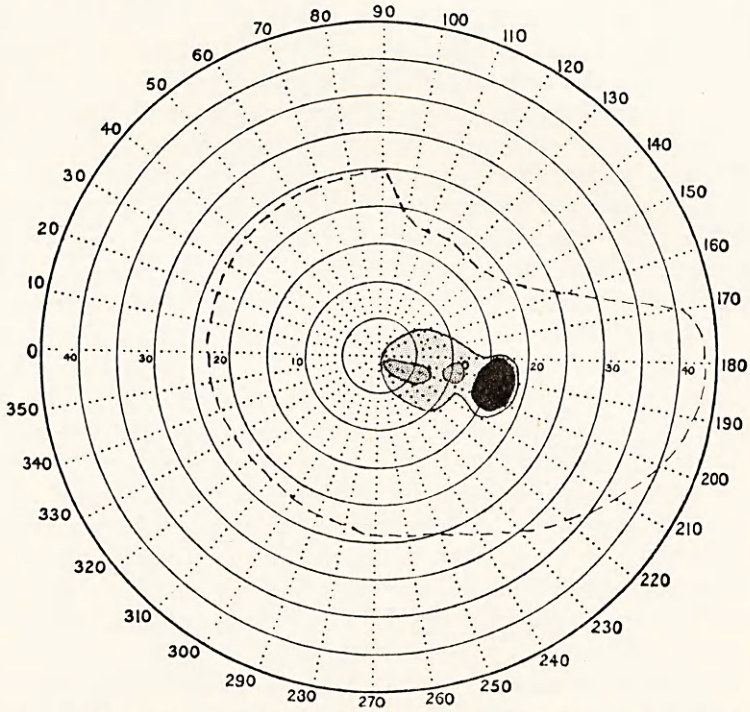


FIG. 5.—Central changes in the field of vision of the right eye in Tobacco Amblyopia. The periphery of the field was normal.

The outer line shows the outer boundary of the field for a red test object with an indentation or " breaking through " towards the scotoma. The inner boundary of the field for red is omitted. The centro-cæcal scotoma for a white test object including the blind spot and two nuclei in the usual positions is shown. Vision 6/9. (R. 1932.)

as tested by the ordinary test card may be between 6/6 and 6/18. Both eyes are always affected at the time the patient comes for advice, though the visual defect is usually worse

in one than in the other. It will be found that the visual response is worse if the contrast between the print and the paper is reduced. For example, a tobacco amblyope who can read a well-printed book in which the paper is white and the printing black cannot read a newspaper in which the paper is greyish and the printing merely a deeper shade of grey, although the letters are of the same size. This depression of the light difference sense is always met with in impairment of the visual conducting mechanism. It is well demonstrated by the use of Bjerrum's test card which has grey letters on a grey background. For this reason colour vision is defective, and red and green are the easiest colours with which to detect the failure. The presence of tobacco amblyopia in engine-drivers or pilots is, therefore, a serious matter in connection with transport by rail or by steamer.

If the field of vision is examined, a scotoma will be found extending from the blind spot to the central area—a centro-cæcal scotoma. This scotoma does not begin in the central area or specially affect it at first. It begins as an outgrowth from the nasal side of the blind spot or in the form of an isolated small defect between the blind spot and the central area. Then a more extensive defect of moderate intensity for white but definite for red or green occupies the whole area between and embracing both the blind spot and central area. It is when the scotoma begins to encroach upon the fixation area that the patient first notices any visual impairment. Characteristic features of the scotoma are the presence of one or two dense nuclei within the scotoma. These are easily detected in the early stages but in more advanced cases join together into a larger dense area occupying the centre of the defect. The scotoma is bilateral and exhibits a high degree of symmetry on the two sides. Asymmetric cases are rare and indicate that the retinal cells of one eye are affected before those of the other. As the disease develops, this scotoma becomes more and more dense, and the whole of the central part of the field of vision begins to become affected, especially upon the temporal side so that a form of bitemporal hemianopia develops.

Interesting features are that a certain amount of tolerance is induced so that patients who continue to smoke may not get worse or may even find the amblyopia varying somewhat. This is probably owing to variations in health and resisting power. Also there appears to be a cumulative effect, that is

to say, when smoking is stopped the amblyopia may get worse for a few weeks or even months before it begins to improve. This is often a source of anxiety to patients who have been advised to stop smoking and who find that their sight is not getting better. Many patients, however, are curiously uninterested, in contrast to those suffering from other forms of central defect such as Leber's disease. This is because of the steep edge of the scotoma and its more central position in the latter condition.

No changes occur in the fundus oculi except some pallor of the optic disc in very advanced cases. Changes in the appearance of the blood vessels or small hæmorrhages have been seen but are usually manifestations of arterio-sclerosis, and in any case are not connected with the tobacco.

The disease runs a chronic course becoming progressively worse, until a certain point short of complete blindness is reached. In the most advanced cases patients are always able to go about alone and do not seem to suffer, except in so far as they cannot read or see small objects distinctly. This is because they are able to see well enough for most purposes, although in a dim and misty way.

Prognosis.—The prognosis in all but the most advanced cases is good if the patient stops smoking and leads a healthy life. In severe cases, in which vision is reduced to less than 6/60, considerable improvement will occur, but in these cases complete restoration of vision is not attained. Even if smoking is not stopped, the patient will never become blind and, in some cases, some improvement of vision may occur, usually depending upon improvement in general health.

Diagnosis.—The diagnosis is based upon the clinical picture presented by the patient. There are few conditions in which the visual defect is imitated. Of these perhaps the most important is tumour in the pituitary region, whether actually connected with the pituitary body or not. Bitemporal scotomata are produced, but if carefully examined they will be found to exhibit the distinctive characters of chiasmal interference and, in addition, the general condition of the patient is not usually that of the tobacco amblyope. Disseminated sclerosis affecting the chiasma also produces bitemporal hemianopic scotomata, but here again the features of the scotomata and the clinical course of the disease enable a correct diagnosis to be made.

Other forms of chronic bilateral centro-cæcal scotoma are rare. The most common form, namely Leber's disease or hereditary optic atrophy, may sometimes cause difficulty, but here again similar differential features are present. The details of the scotoma and the clinical picture as a whole are usually sufficiently different to prevent confusion.

It has been said that a scotoma in all respects the same as that of tobacco can be produced by other agents. In extremely rare instances this may be true as regards the shape and position of the scotoma, and even the presence of dense nuclei within it; but if the onset and course of the visual defect and the general features of the case are taken into consideration, a real difficulty in diagnosis can hardly arise. In cases which are seen for the first time when the scotoma is large and dense, more difficulty may be present, but here also the true diagnosis is usually easily made. If the characteristic visual defects are present, a small consumption of tobacco, or the female sex, or a relatively low age, should not be allowed to influence the diagnosis if it is supported by the history and general condition of the patient. These statements are based on the fact that cases of mistaken diagnosis have been extremely rare. I personally have no note of any such case.

Owing to the fact that tobacco amblyopes rarely die while under the observation of ophthalmic surgeons, post-mortem material is hardly ever obtained, and little is known of the pathological anatomy of the disease. It is sufficient to say that the ganglion cells of the inner layers of the retina and fibres in the optic nerve, traceable as far as the external geniculate body, have been found atrophied. Such evidence as there is indicates that it is the cells and not the fibres which suffer in the first instance. Experiments have been done on dogs with quinine but, as far as I know, experimental chronic nicotine poisoning has not been studied in any lower animal, and human pathological material, though it cannot be rare, is almost impossible to obtain.

Treatment.—Treatment should consist in the stoppage of tobacco and measures to restore the patient to a healthy condition. It is a good practice to advise the patient to drink large quantities of water and use some laxative medicine, and any gastric or cardio-vascular disorder should receive attention. There is no specific treatment which can be directed towards the affected nerve elements. The use of

vasodilators such as sodium nitrite has been advocated upon the hypothesis that the affection is due to vascular constriction. No evidence has as yet been brought forward to show that this hypothesis is correct or that the treatment is efficacious.

Relation to Work.—The large number of cases of this disease indicates that many workmen must be rendered more or less inefficient. When the tobacco amblyopia follows an accident, questions of compensation and insurance arise, and as is usual in such cases the prolonged incapacity is regarded as due to the accident. The disease itself causes direct incapacity in the case of men whose work entails the recognition of colours, such as pilots and railwaymen and some other workers. It is not necessary to go into detail in regard to this part of the subject. It is sufficient to point out that a considerable amount of unnecessary expense and trouble is caused to any community in which tobacco amblyopia is common.

Discussion.

Dr E. H. Cameron said—Tobacco amblyopia is very uncommon in private practice. It is a hospital disease, and the question is, Why is that so? Is it on account of the kind of tobacco smoked by hospital patients, and if so, what is the deciding factor that is present in that kind of tobacco which is absent in the better brands?

I was much interested in the question of the incidence of tobacco amblyopia in other countries. I remember once discussing this question with Professor Van der Hoeve of Leyden, and I was surprised to hear him say that tobacco amblyopia was uncommon in Holland, though in that country tobacco is extensively used in the form of cheroots. Why should there be a great deal of tobacco amblyopia here and so little in Holland? I once discussed with Professor Barger and Professor Clark the possibility of finding out what is present chemically in tobacco which poisons the optic nerve or retina, and we came to the conclusion that the investigation would be a very difficult one. Numerous experiments on monkeys would probably be necessary, and the impossibility of carrying out any functional test would be a drawback. The question of alcoholism is also of interest. Like Dr Traquair, I can hardly remember any case in which alcohol alone produced amblyopia; but I can remember one patient, whom I saw in private practice, who had all the symptoms of advanced tobacco amblyopia, although he said that he was a non-smoker. He was a dipsomaniac who drank port wine, but he was not in a sound mental state and thus no great reliance could be placed on his statements.

Professor Bramwell said—The selective action of various poisons, using this term in its widest sense, is an extraordinarily attractive

problem. Why should a particular " poison " pick out a particular organ, tissue, or tract? Dr Traquair has told us that an individual may develop a tobacco amblyopia although he is what some of us would regard as a comparatively light smoker. There must, I take it, consequently be some particular predisposition or some additional factor or factors. One meets with many cases of polyneuritis nowadays in which the cause is obscure or in which more than one cause is acting. On the other hand, I gather, from what Dr Traquair says, that the character of the scotoma in tobacco amblyopia is distinctive. There appears to be no familial predisposition. Is there any evidence to suggest that excessive use of the eyes or exposure to a bright light may be determining factors?

Dr R. A. Fleming said—With regard to the incidence in the different months, we are told by Dr Traquair that in March and April there are more cases of tobacco amblyopia than in the later months of the year. Is it possible that in those months, when one has that curious " springish " feeling, when one's tissues are beginning to grow again, one may absorb more of the nicotine, or whatever causes the tobacco amblyopia? In regard to the summer months, if any of you live where I go in August and September, you would find smoking an absolute necessity because of the midges and other viciously biting flies. Smoking is one of the few ways in which it seems to be possible to keep these creatures in check. I have always been interested in tobacco amblyopia, although I do not see many cases myself. I was surprised to find that it is not in the earlier part of life, when one would expect the younger person to be more liable to be injuriously affected by tobacco, that we get the largest number of cases of tobacco amblyopia, but, from the tables shown by Dr Traquair, we have seen that the greatest number of cases occur about the age of 50. Probably, in the later part of life, Dr Traquair would assume that the individual has rendered himself more or less immune by long indulgence, but at the same time it is curious that a toxin like tobacco should not affect the younger person more.

Another thing that I learned for the first time was that in hot countries apparently the natives can consume a lot of tobacco without ill-effect. Would one not expect that the glaring light of the sun, say in India, which produces so many cases of cataract, would make those ganglion cells of sight suffer specially from the toxin?

I should like to say a word about methylated spirit drinking, of which I happen, indirectly, to have seen quite a lot. The more sophisticated so-called methylated spirit drinker really drinks commercial spirit. I am not chemist enough to know whether commercial spirit is very different in its effect from the other kind, but it produces intoxication which lasts one to two days, and may even prove fatal. The result of this very powerful spirit on the mucous membrane, one can imagine, would be to allow the nicotine in, say,

chewing tobacco, to be more easily absorbed. I should like lastly to know Dr Traquair's opinion as to whether arterio-spasm, or angio-spasm, may not have some effect in producing a proportion of these cases, where one has a permanent (if I can venture to call it permanent) interference with vision cells and vision fibres. I know that when one begins to inhale tobacco one frequently gets a good deal of palpitation, and we know that in investigating the fundus in certain patients, who are suffering from quinine amblyopia and a few cases of tobacco amblyopia—particularly in older patients—you get an alteration in colour in part of the fundus. That alteration, I think I am right in saying, is said to be due to angio-spasm.

Dr W. Ritchie Russell said—I was interested in Dr Traquair's paper, particularly from the point of view of the general health in the development of tobacco amblyopia. It is interesting to compare this condition with diseases elsewhere in the nervous system. There is increasing experimental evidence to show that the chemical changes associated with the passage of efferent nerve impulses are quite different from those concerned with afferent conduction. It has also been shown that in certain experimentally produced deficiency diseases, the afferent nervous system is affected exclusively. Diseases such as subacute combined degeneration of the cord, polyneuritis of pregnancy, and polyneuritis of alcoholism, appear to be associated with a specific degeneration of the afferent nervous system, and it is quite possible that these diseases are associated with a type of deficiency disease. It is quite possible that the afferent conduction of vision employs a chemical mechanism similar to that of the afferent conduction in the spinal cord and peripheral nerves. Thus while alcoholic polyneuritis and tobacco amblyopia are principally caused by direct poisoning, a deficiency factor may also act in both diseases.

Dr L. R. H. P. Marshall said—There is one point that I think might be taken more into consideration, and that is the atmosphere in which the smoking is generally carried on. You find people can smoke more heavily in the open air without getting so much toxic absorption. It would be interesting to know if people developed amblyopia after smoking in a vitiated atmosphere—miners, for example, and people working in close rooms. That would help to explain how it is uncommon in the tropics, where the houses, etc., are much more open to the air than in this country.

Dr Malcolm Macnicol said—As the question of smoking in the tropics has come up, it might be well to remember that in India the natives all smoke through water and that would limit very much the effect of the tobacco.

Dr J. Boyd Jamieson and *Dr F. K. Kerr* also spoke.

Dr Traquair, in reply, said—In answer to Dr Cameron, I have seen quite a number of cases of tobacco amblyopia in private practice

in educated people. As regards the rarity of tobacco amblyopia in Holland, it seems to be quite common a little farther north in Denmark. It may be because of the kind of cigar and the way it is smoked—if one half is thrown away so much nicotine may not be absorbed. The condition is undoubtedly more common in certain places than in others; perhaps the reason why it appears to be more common in Scotland than in England, is because the statistics for Scotland are from industrial areas, whereas many areas in England are not so largely industrial. Undoubtedly, if a churchwarden pipe or a hubble-bubble or a clean pipe is used, it will prevent too much nicotine being absorbed by the smoker, and he is not so likely to get tobacco amblyopia—as long as he keeps well. Then in regard to experimental pathology, would it not be possible to use dogs? They might be injected or fed with nicotine or tobacco, and brought to a state of chronic intoxication, after which their retinæ and optic nerves could be examined.

I should like to know whether Dr Cameron's alcoholic patient was a non-smoker. One of the points brought out at the 1887 Convention was that the depression in health produced by excessive alcoholism was a predisposing factor. I should be inclined to attribute a scotoma, which had the characters of the tobacco scotoma, to tobacco, even if the amount of tobacco smoked is very small, rather than to some other cause.

Professor Bramwell wants to know why we put all the blame on tobacco. I quite agree that it is tobacco plus something which produces the scotoma. Why do some smokers get amblyopia while others escape? The answer must be that some have a low resisting power owing either to depressed health from illness or to personal idiosyncrasy. We recognise the amblyopia as due to tobacco because, as far as I know, no other toxic substance produces these characteristic changes in the field of vision. These seem to be typical of tobacco. Whether there is any other factor I do not know. I feel that relative over-consumption of tobacco, together with the illness, is sufficient.

With regard to the different kinds of smoking—cigarettes, cigars, and so on—so far as my experience goes, with hospital and private patients, cigarette and pipe smoking produce quite different symptoms. The person who smokes too many cigarettes may smoke an enormous number without developing tobacco amblyopia, but he or she often develops palpitation and may become a nervous wreck. Smoking a dirty pipe may produce eye symptoms without these cardiac and nervous disturbances. If cigarettes often caused tobacco amblyopia easily, there would be more of it amongst women than there is.

The comparison with disseminated sclerosis and the question of pain on movement of the eyes was mentioned by Professor Bramwell. In disseminated sclerosis there is an actual focus of inflammation in the nerve, in tobacco amblyopia there is an intoxication of the ganglion cells. In the presence of the inflammation,

movement of the nerves or pressure produces pain. In tobacco amblyopia the ganglion cells are affected by a toxin, which first partially paralyses them, and then, if absorption is continued, kills them, and there is no pain at all associated with this process. I am not sure whether Dr Russell's point can be explained in this way.

There is no evidence to show that eyes which are excessively used or excessively exposed to light are more susceptible to tobacco amblyopia. For one thing, the area of the fundus oculi which is exposed to light would be the area round about the macula, but the affected area of the retina is to one side of the macula and at first not even near the macula.

In answer to Dr Jamieson, I have never come across a case of amblyopia in a worker in a tobacco manufactory myself, but I think cases have been reported. With reference to the peak of the incidence being in spring, one must remember that it is really the peak of the patients coming to hospital. One does not know exactly how long they have had symptoms: they may have been present for five or six weeks to three or four months, or longer. Usher found the same difficulty in trying to find out the actual incidence of the amblyopia. All one can find out is the incidence of application to the clinics. I think it is probable that the patients over-smoke in winter, and in view of the fact that most people's health is a little reduced in January, February and March, the patients apply for advice in spring and early summer.

As regards angio-spasm, the narrowing of the arteries in quinine poisoning comes on after the blindness and is not the cause of it. Angio-spasm and its effects on the eye are well known, but they are not in the least like the effects of tobacco.

Dr Marshall raised the question of smoking in the open air. One cannot judge from a man's occupation where he smokes. Miners and labourers form a large proportion of the cases seen in hospital, but the miner does not smoke in the mine—he probably smokes at home. But it is also possible to get tobacco amblyopia by smoking in the open air. One case I remember quite well—a girl in the twenties, a farm labourer—who, in the cold weather, learned to smoke, after she had been shown when sitting behind a hedge how to warm her hands on the bowl of a tobacco pipe. I would recommend Dr Kerr to get a copy of *The Lancet* for 1857, where he will find both sides of the question amply and fully discussed, some writers maintaining that the country was going to the dogs on account of smoking, and others stating that a man did not deserve to be alive unless he smoked.