MINERS' NYSTAGMUS;

By

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INTRODUCTION

The condition known as "Miners' Nyctagmus" is an occupational disease, the incidence of which is practically confined to workers in coal mines, more particularly the miners at the coal face. Cases occur also in mines where thin coal seams are present. In other occupations, alleged to be associated with Miners' Nyctagmus, have been described in the literature from time to time, but the author has failed to substantiate these allegations by more authoritative evidence.

Miners' Nyctalopia. The chief sign of the disease is a rotary nystagmus of the eyes. All other signs frequently observed, such as disturbance of visua! function, headache, and giddiness, associated with diminished vision, are practically constant factors, while hallucinations are also common. Other symptoms are necessarily referable to the eye.

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MINERS' NYSTAGMUS:

Introductory

The condition known as "Miners' Nystagmus" is an occupational disease, the incidence of which is practically confined to workers in coal-mines, more particularly the workers at the coal-face. Cases occur also in ironstone mines where thin coal seams are present. Isolated cases in other occupations, alleged to be identical with Miners' Nystagmus, have been described in the literature from time to time, but the authenticity of these cases is questioned by most authorities.

It has received the appellation of Miners' Nystagmus because the chief sign of the disease is a rotary oscillation of the eyeballs. Other signs frequently present are tremor of eyelids, head, and even of the neck and shoulders. The chief symptoms are failure of sight, especially at night or in the twilight, dazzling and dancing of the lamps. Headache, and giddiness, especially on exertion, are practically constant features, while photophobia is also common. Other symptoms not immediately referable to the eye
No condition are also seen and will be mentioned later.

There are two distinct types of the condition.

In the first nystagmus is present, often unknown to the patient, but symptoms are absent and no incapacity results. In the second symptoms are present and the workman is incapacitated. These types may be called Latent and Manifest. Two extremes are formed by cases presenting signs but no symptoms, and others again in which the picture is composed almost entirely of symptoms.

The condition has been known for about 70 years, the first mention of it in the literature being provided by Decondé in 1861. Since then the subject has acquired a vast literature.

There have been two principal theories advanced as to its causation.

1. The Position Theory - championed by Snell and Dransart - that the prime cause is working in a constrained position with the gaze directed obliquely upwards.

2. The Light Theory - first supported by Romiée and Court - that the essential cause is insufficient illumination in the working places.

Up to about 1910 the advocates of the first theory were in the majority, but since then the pendulum has swung round in favour of the Light Theory.
More recently an organismal origin has been suggested; and a chemical toxin theory, originated in 1893 by Pechdo, has been revived and developed by Robson. A psychopathic explanation for the symptoms has also been suggested. A striking fact is the difference between the early and modern views of the pathology of the disease. Snell and other early writers considered it a local condition, - a myopathy of the elevators of the eyeball. Nowadays it is regarded rather as a general neurosis with local ocular manifestations.

The size of the literature is proof of the medical interest it aroused from the first. In recent years its economic importance has been driven home.

In May 1907 the Workmen's Compensation Act (1906) was extended to Nystagmus, and the disease was included in the Third Schedule as follows: -

<table>
<thead>
<tr>
<th>Description</th>
<th>Description of Process (selected of Disease, as liable to cause the disease)</th>
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<tbody>
<tr>
<td>Nystagmus.</td>
<td>Mining.</td>
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In July 1913 the first column was altered to read: -

"The disease known as Miners' Nystagmus, whether occurring in miners or others, and whether oscillation of the eyeballs be present or not."

This was intended to cover those cases, a
considerable number according to some writers, in which complaint is made of various symptoms with a paucity of physical signs.

The number of cases certified in 1908 was 386. This figure, that is, the number of new cases receiving compensation rose steadily up to 1912, while in 1913 and 1914 there was a marked increase so that the figure of 1376 in 1912 was increased in 1914 to 2432. There have been even larger numbers since. (See table): Compensation Claims for Miners' Nystagmus collected from Home Office returns "Statistics of Compensation"

<table>
<thead>
<tr>
<th>Year</th>
<th>Old cases</th>
<th>New Cases</th>
<th>Year</th>
<th>Old cases</th>
<th>New Cases</th>
</tr>
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<tbody>
<tr>
<td>1908</td>
<td>74</td>
<td>386</td>
<td>1919</td>
<td>3,731</td>
<td>2,718</td>
</tr>
<tr>
<td>1909</td>
<td>380</td>
<td>631</td>
<td>1920</td>
<td>4,163</td>
<td>2,865</td>
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<tr>
<td>1910</td>
<td>662</td>
<td>956</td>
<td>1921</td>
<td>4,804</td>
<td>1,913</td>
</tr>
<tr>
<td>1911</td>
<td>1,144</td>
<td>1,375</td>
<td>1922</td>
<td>5,063</td>
<td>4,092</td>
</tr>
<tr>
<td>1912</td>
<td>1,319</td>
<td>1,376</td>
<td>1923</td>
<td>7,273</td>
<td>3,863</td>
</tr>
<tr>
<td>1913</td>
<td>2,419</td>
<td>2,402</td>
<td>1924</td>
<td>7,635</td>
<td>3,271</td>
</tr>
<tr>
<td>1914</td>
<td>3,218</td>
<td>2,775</td>
<td>1925</td>
<td>7,890</td>
<td>3,445</td>
</tr>
</tbody>
</table>

No returns for war years: Strike year.
The inclusion of Nystagmus in the list of Industrial Diseases under the above mentioned act has emphasised the economic aspect of the disease and drawn a great deal of attention to the condition. So much so that a Nystagmus Committee of the Medical Research Council under the Chairmanship of Dr. J. S. Haldane was appointed in 1920 to investigate the matter, and reported in 1922 and 1923. The Committee concluded that deficient light was the essential cause of the condition. The years 1922 and 1923 gave diastatic figures and they have remained high since. This fact has been rashly regarded in some quarters as disproving the light theory seeing that electric lights have been more widely adopted in recent years.

Compensation claims however are not a reliable index; and may easily indicate a spurious frequency. After inclusion in the Schedule the disease, like other compensation diseases, took a few years to reach a steady level. This seemed to be in evidence in 1911 and 1912. In 1913 the definition was so altered as to allow the admission of claims for purely psychopathic symptoms, and the result is seen in the figures for the next two years. The outbreak of war in 1914 possibly had some influence also. The records for war years are not available. In 1919 wages were high and compen-
sation still at twenty shillings per week. Then compensation pay was raised to thirty-five shillings. In 1921 the industry experienced a bad time with a drop in wages and unemployment. In 1922 there was a large rise in the number of new cases. If it is suggested that this rise is the outcome of economic fear, the suggestion cannot be lightly brushed aside. The claims for other conditions such as heat-knee have followed a similar course.

In connection with this explanation it must be remembered

(1) that a disease with psychopathic symptoms is "infectious."

(2) that from examination of large numbers of men various writers have found oscillation of the eyeballs in from 20 to 35 per cent of underground workers, and these form a vast reservoir, or, as Professor Collis puts it, a pool from which cases might be tempted by economic and other stress to rise to the fly of compensation.

Statistics indicate that the numbers of claims for a relinystagmus, accidents and other conditions are inversely proportional to the level of wages.

On the Accident (non-fatal) claims increased from just over 98,000 in 1919 and 1920 to over 155,000 in 1922 though the fatal cases for the three years were very similar 852, 769 and 765.
The effect of change of definition of the disease may also be seen in the increased number of old cases on compensation in 1914. The proportion of old to new cases has steadily risen, and this may be due to the admission of claims for psychoneurosis. In the years prior to 1907 miners with nystagmus seldom stopped work for more than a few weeks or months and then they worked on the surface. Now a considerable number of cases continue idle for years though the nystagmus and other physical signs have frequently disappeared after a few months or even weeks. In Belgium, where there has been no change in the definition neither the incidence, nor the duration of incapacity have been found to increase in that country. Compensation in Belgium is limited to six months, but hardly any cases are found unfit for work then; and in the early years the condition presented similar features here and on the continent.

Enough has been said to show that the frequency as indicated by claims for compensation is not a reliable figure. At the same time it detracts nothing from the economic importance of the condition. On the contrary, for Home Office returns for the year 1925 show that Nystagmus constituted 65 per cent of all cases of industrial disease.
The cost of the disease is immense. In the first place the worker loses the benefits of a comparatively good wage, and is forced to depend on a weekly compensation rate or to do less remunerative work. The loss of income is a calamity with disastrous results to his family. Secondly, the owner pays a great deal in Compensation and loses the profit from the work of the men affected. The amount paid in compensation for 1925 is estimated at well over £500,000. In addition the State loses the output of the affected men. The total estimated loss due to the disease was estimated by the nystagmus Committee to be £1,000,000 for the year 1920. For 1925 this figure would be nearer two than one million.

These facts enhance the importance of treatment and above all prevention.

I have been in a colliery district for some time and have observed a number of cases, some in my own practice, others outside it specially observed for the purpose of this thesis.

As the condition is of great interest and importance and the etiology still wrapped in obscurity I considered that it would be a suitable subject for a thesis.

In the ensuing pages I propose to review the subject and discuss recent developments in the light of my own experience.
Miners' Nystagmus was first described by Decondé in 1861, in a paper on Nystagmus in general, though Snell in his book (published 1892) says Dr. Gillet of Sheffield recognised the condition as early as 1855.

In 1875 (Lancet vol. i) Bell Taylor published a paper on "Miners' Nystagmus, a new Disease." He thought it analogous to writer's cramp, and attributed it to the sustained effort to see in a dim light. He says (B.M.J. 1887 vol. ii) "It is analogous to that rare condition of muscles known as auctioneer's spasm, or to a similar affection of the gastrocnemii occasionally seen in ballet dancers." "There is no central lesion; the affection is a pure myopathy."

The first authority in this country to investigate the condition closely was Simeon Snell. In the Lancet (1875 vol. ii) he points out that "it occurs chiefly if not entirely in those colliers who are obliged to work whilst lying on one side." In 1884 (Trans. Ophth. Soc:) he says that "as far as his memory and records go all his cases had worked on their sides more or less."

In his book "Miners' Nystagmus" published in 1892, after a study of 127 recorded cases he concludes
that the condition is a myopathy analogous to writers' cramp, "caused by fatigue of the Superior Rectus, Inferior Oblique and Internal and External Recti muscles induced as a consequence of the miner's work in the pit necessitating an upward and more or less oblique gaze. It occurs irrespective of illumination."

Snell claimed he was supported by Dransart in France and Nieden in Germany, and quoted a letter from Dransart (B.M.J. Aug. 8th 1891) expressing his agreement with Snell's view -- that "position is the first prime factor in the production of the disease." He seems to have changed his views somewhat and in 1913 he seems to attribute more importance to conditions of illumination than would have been acceptable to Snell. Both agree that errors of refraction are of no importance. Nieden, in a book published in 1884, (Der Nystagmus der Bergleute) writes that nystagmus is almost exclusively found in workers who have to work in a constrained position of body, head and eyes, but in a list of causes gives lack of light the first place and defects of vision second.

These three authorities were however more or less in agreement and their view, though hotly disputed by some, held general favour till 1910 or thereabouts.
it a general disease with nystagmus as one of the symptoms, and suggests the name "Miners' Neurosis." He considers it due to the fatigue which follows the disassociation of movements normally combined, flexing the head and elevating the eyes. This view has found support right up to recent times by Rutten.

In 1891 in a Report to the Derbyshire Miners' Association, Court took up the cudgels on behalf of the theory of deficient light. He quotes figures from a systematic examination of over 1000 colliers showing greater incidence in safety lamp as compared with naked light pits.

The light theory had previously been put forward by Romié in Belgium from 1878 and in a later paper (1908 Bull de la Soc belge d'Ophth. No 25) he gives figures showing a fall of 50% below 1891 figures following improvement of lamps. He was the first investigation to establish the actual incidence of miners' nystagmus by systematic examination of large numbers of men as they came from work. He thinks insufficient light the only cause, nystagmus resulting from excessive accommodation for a long time in bad light.

Pechdo in 1893 suggested poisoning by inhalation of gases given off by coal but this has not found
much support, though developed recently by Robson. Christie Reid in 1906 (Brain 29,3) points out that in the miner there is as a result of dim light imperfect fixation, and this coupled with frequent disturbances of position produces the disease.

In 1907 Peters suggested a labyrinthine origin for the disease. Since 1907 as a result of its inclusion in the third Schedule of Industrial Diseases, a great deal of attention has been focussed upon the subject and the light theory has been more generally adopted.

Elworthy (B.M.J. Nov: 7th 1910) lays more emphasis on the quality of the light, and thinks exhaustion of the eyes from working in blackness without any colour relief causes incoordination and spasm.

Llewellyn (Miners' Nystagmus 1912) gives the result of several years study. His photometric measurements of the light at the coal face have shown how very deficient is the illumination with which the collier works. He strongly supports the light theory.

Shufflebotham (Lancet 1914. July 4) in his Milroy lectures gives an account of the disease, and gives as causes deficient light, cramped position, refractive errors, and ocular injuries.

Ohm of Westphalia considers the cause to be
lack of light and other conditions which bring about too strong and too infrequent innervation of the labyrinth, the nystagmus being a disorder of tone. Stassen of Belgium after mentioning various subsidiary factors thinks bad conditions of illumination the essential cause.

Martin (1920) thinks it is comparable to the anxiety neuroses of soldiers.

Anderson (1920) thinks errors of refraction, especially astigmatism, of chief importance.

In 1920 the Medical Research Council appointed a committee to investigate the matter, and they reported in 1922 and 1923. They were unanimous in finding deficient illumination to blame.

In the first report they conclude:

(1) The essential factor in the production of Miners' Nystagmus is deficient illumination. Other factors such as position during work, accidents, alcoholism, infections, malnutrition, hereditary predisposition, and errors of refraction are of secondary importance only, while depth of workings, thickness of seams, and the ordinary gaseous impurities in mine air have no direct influence on the disease."
The deficient illumination is due to the low illuminating power of the safety lamps in general use and the conditions in which they have to be used, such as their distance from the working place, and the dusty atmosphere.

Workers at the coal face are more affected than other underground workers, and this appears to be due to the unrelieved blackness of the coal and the greater need for accurate vision.

Distinct signs of nystagmus are present in a large proportion of coal miners, though only in a small proportion do the symptoms ever become so severe as to cause even temporary incapacity from work underground.

They recommend improvement of illumination either by greatly increasing the candle power of the safety lamps as ordinarily used, or by the use of an electric lamp capable of being fitted on the cap or belt so that the light shines on the spot where the worker wants it.

A separate section of the report by W.H.R. Rivers deals with the psychoneurotic factors of the disease as studied by H.W. Eddison. He considers many of the symptoms are not directly due to the eye condition. He distinguishes two main
varieties "one beginning with nystagmus and the symp-
toms consequent upon it, upon which an anxiety state
and abnormal fears supervene; and a second group in
which the psychoneurotic symptoms are primary or at
least more prominent than those that can be directly
ascribed to the state of the eyes." In this group
he includes those cases with lid spasm.
Fear is one factor concerned in the production of the
psychoneurotic features. In addition the nystagmus
both produces an anxiety state and acts as a nucleus
round which other symptoms tend to centre. Rivers
thinks that the increased frequency of disability is
to some extent due to compensation by its action on
the psychical and psychoneurotic aspects of the dis-
order, and compares the situation to that presented
by pensions in relation to psychoneuroses of war.
He thinks increased illumination will "diminish both
the prevalence of Nystagmus in the strict sense, and
its psychoneurotic accompaniments, since the defect
of illumination acts as a condition which enhances
the psychoneurotic aspect of nystagmus by increasing
the element of danger, real or imaginary."

Eddison emphasises the psychoneurotic aspect
in the Journal of Mental Science, April 1926, and
the psychoneurotic explanation of the symptoms was

supported by several speakers in a discussion on the subject at the Royal Society of Medicine. (Brit. Journal. Ophth. Nov: 1925)

In the Second report of the Nystagmus Committee the question of incapacity is dealt with and a contrast drawn between the duration of disability in this country and in Belgium, where compensation ceases at the end of six months apparently with advantage to the workers. Pooley in a separate section of the report deals with errors of refraction and concludes that "errors of refraction in themselves have no effect whatever either on the incidence of Miners' Nystagmus or on the age at which incapacity commences from Miners' Nystagmus.

The findings of the Nystagmus Committee have been criticized and hotly disputed by a few writers.

In 1924 Robson (Proc: South Wales Inst. of Eng. vol. XXXIX Nos. 2 & 4.) suggested that nystagmus is due to some toxic gas—probably carbon monoxide, inhaled in small amounts over long periods. He does not bring much evidence to support it.

Freeland Fergus of Glasgow in a number of papers (Lancet, May 23rd 1925 and Trans. Int. Mining Eng: vol. LXXI part 2) claims that the case for the
light theory is "not proven," and suggests without any appreciable evidence to support his view that the condition is more like an infection in several respects and may be either microorganismal, or due in some way to the products of microorganismal activity in bygone centuries when the coal was laid down.

Percival (B.M.J. May 5th, 1923 and March 22nd 1924) does not accept the light theory. He still attributes most importance to position at work, and thinks heredity must play some part from the in-breeding so common in pit villages.

Haldane and Llewellyn in a joint paper (Trans: Inst: Mining Engineers vol. LXXIII part 2) reply to their critics.

None of the recent views has gained much ground, and at present the balance of opinion strongly supports the light theory.
The Clinical Picture.

It may be as well before discussing the controversial points of aetiology and pathology to give a brief account of the clinical picture of the condition.

The disease is usually described as having two forms or stages, latent and manifest. In the latent form there are no subjective symptoms and no disability. The only physical sign is a rotatory nystagmus. The patient is usually quite unconscious of his condition.

The manifest cases are divided into mild, moderate and severe classes. The onset may be gradual, when the patient will say he has had trouble with his eyes for 12-18 months or even longer, or it may be dramatically sudden, especially in relation to accidents. All grades are found between these two extremes.

The first symptom the miner complains of as a rule is failure of sight, especially at dusk or in the early morning or in doing more skilled work such as notching timber. He is greatly worried by moving lights and complains that the lights dance and flash or go round and round. This dancing can be stopped as a rule by looking downwards and converging, or by
closing the eyes.

Headache is nearly always present. It may be felt over the occiput or in the temples - behind the eyes. It is made worse by stooping and exertion. If often comes on at night and is one of the most persistent symptoms often being the last to clear up. Giddiness is another constant feature. It is increased by stooping and exertion, and is often the symptom most complained of. Some cases find it troublesome in descending steps.

Night-blindness is a symptom the frequency of which is disputed.

Romée considers it very rare, while Butler gives it as present in 60% of cases, and Court in 77%.

Evidently different interpretations of the term are meant. If it merely means difficulty in seeing in dim light and in the dark, it is very common indeed, - almost constant in fact. The difficulty in "finding his sight" on descending the pit, of which the nystagmic miner complains is an expression of the same thing and is due to failure of dark adaptation. I have seen two cases who evidently "lost their sight" completely for a short while, but they came to no harm.

Photophobia is generally regarded as common. Llewellyn gives its frequency as 46%. I have seen
very little of it in my cases. Mental symptoms are common. Mental hebetude is sometimes marked, and there may be fits of depression when the nystagmic will assure you he is "thoroughly fed up with life," as one of them put it. Even bouts of hysterical weeping and expression of suicidal intentions are recorded. Sleep may be broken by headache or dreams. The characteristic physical sign is oscillation of the eyeballs. The oscillations are nearly always rotatory. They are regular and similar in the two eyes. I have only seen one exception to this (Case 18). They are increased by any exertion, stooping, confinement in the dark or elevation of the eyes, and in many cases can be elicited only with the aid of one of these methods. They can usually be stopped if the patient looks down and converges, though occasionally they persist at all times. Actual nystagmus may be absent in some cases from the beginning, examination even with the ophthalmoscope failing to detect any oscillations; and the nystagmus is often the first to disappear, it may be long before the subjective symptoms. It bears no definite relation to the amount of disability.

The rate of movement is usually given as 100–350 or more per minute. Some authors record a rate of 500 or more, but I have never succeeded in

20.
counting over 200 with accuracy. The excursion varies from the slightest flicker to coarse movements, the coarser movements accompanying the slower rates.

Lid movements are common and are of two kinds, one a blinking movement possibly of the nature of a habit spasm from blinking to protect the eyes, the other a tremor or clonic spasm. It is not due to photophobia as it is often present in the absence of the latter, and persists in the dark.

Head tremor is also present in a number of cases. Llewellyn says it is constant. I have been unable to detect it in about half my cases. The best way to elicit this sign is to flex the head strongly with one hand and ask the patient to look up at a finger of the other hand. There is sometimes also a tremor of the shoulders and hands. A rapid pulse is almost invariably present, 90 to 120 per minute or more.

In many longstanding cases, and in some cases from the very beginning, there are no physical signs related to the eye, but a variety of symptoms and signs that are evidently neurotic in origin. These are headache, sleep broken by dreams, anxiety, depression, tremor, mental irritability, tachycardia and innumerable vague sensations.

It is important to note that the disability
and symptoms are not proportional to the nystagmus, while the disability does bear a definite relation to the nervous symptoms just mentioned.

Causation and Pathology.

There are a number of noteworthy differences between Miners' Nystagmus and other forms of nystagmus acquired late in life.

In Miners' Nystagmus the oscillations are rotatory and nearly always equally marked in the two eyes. In the other forms the oscillations are usually lateral and the two eyes often present distinct differences. There may also be a greater frequency on looking in one direction. Miners' Nystagmus is often present even when the eyes are in the position of rest, while in other cases nystagmus is usually elicited only on voluntary movement of the eyes in some direction.

In Miners' Nystagmus movement is equal in rate to and from - the pendular type, while in other forms there is generally a quick component and a slow component - the rhythmical type.

There is too a noteworthy difference in the matter of symptoms. In nystagmus in general the patient is not usually conscious of movement of objects except
in recent and severe cases, as in some cases of labyrinthine disease, when vertigo is also present. There is never anything like the train of symptoms usually seen in cases of Miners' Nystagmus claiming compensation. The sign nystagmus is seen in a variety of conditions which may be briefly mentioned. First of all there is what is known as Physiological nystagmus. Instances of this are seen in railway nystagmus, in which if, after looking at the rapidly moving landscape, one fixes an object, that object seems to be moving in the opposite direction. Another example is seen in a number of normal persons in whom forcible attempts to open the closed eyelids are accompanied by oscillations of the eyeball and jerking of the lids. These can hardly be regarded as proper nystagmus. A small number of cases of Spontaneous Nystagmus are reported in the literature. (Ophthalmoscope - June 1912, p.316). In these cases the gifted individuals could induce nystagmus by some particular effort - such as extreme lateral fixation, extreme convergence or gazing into the distance. Another group of cases which are of interest in their possible bearing on the condition under discussion are those cases of Congenital or Early Infantile nystagmus developing in the first year or so of life. These arise where there is interference with vision from
any cause e.g. Ophthalmia Neonatorum or congenital cataract. They are due to imperfection in the development of fixation which occurs in the early weeks of life. The eyes of a newly born child move independently until a light or some object attracts the eyes. As the image reaches the fovea it becomes sharply defined, and the muscles and visual centres are quickly coordinated so that without conscious effort an image can be focussed on the fovea at once. All muscular movements also become coordinated. For the proper development of coordination the formation of clear images on the retina is a sine qua non. If proper retinal stimulation is absent, fixation is imperfect. There are some cases even of adult nystagmus which are apparently attributable to extreme errors of refraction. Other cases of Congenital Nystagmus are seen in Abinism in which condition there is a deficiency of pigment, general and ocular. It is believed that it is the pigment of the retinal epithelium that matters, for cases occur with albinotic heredity but no general or obvious ocular lack of pigment. The group known as Hereditary Nystagmus, in which there is nystagmus but no sign of retinal or choroidal disease are believed to belong to the pigmentary group. Nystagmus is seen also in total colour blindness, in
which vision depends on the rods alone and there is a central scotoma.

Nystagmus may also appear in infants after the development of fixation in the condition known as Spasmus Nutans, associated with head nodding which begins and disappears before the Nystagmus. It begins usually about the fourth to the sixth month in rickety children living almost entirely in poorly lighted rooms, and frequently disappears in summer to re-appear in the following winter. It is interesting to note that Ohm found a similar condition in experimental puppies and kittens kept in a dark cellar.

Nystagmus in later life may be associated with:

1. **Labyrinthine causes.**

   These include labyrinthine stimulation in normal persons by syringing the ears with hot and cold water, rotation on a revolving stool, and passing a galvanic current through the head. The nystagmus is rhythmic, rotatory or horizontal to the side of deviation, and is not seen unless the eyes are deviated. A spontaneous rhythmic rotatory nystagmus to one side is also seen when the opposite labyrinth is destroyed. It is worse on looking to the contralateral side. It ceases if the sound labyrinth also is destroyed.

2. **Various diseased conditions of the nervous system.**

   In Disseminated Sclerosis for instance it is fre-
quent. The movements are generally horizontal, and in the early stages only elicited in extreme lateral positions of the eyes.

In Friedreich's Ataxia it is frequent though not constant. It is never spontaneous.

In Syringomyelia again it occurs with moderate frequency. In disease of the cerebellum e.g. tumour or abscess it is common. It is a rhythmical nystagmus with a coarse nystagmus on deviation to the diseased side, but a fine rapid nystagmus on looking to the contralateral side. It occurs as a rule only on lateral deviation.

Nystagmus also occurs in local lesions in the neighbourhood of Deiters' nucleus e.g. thrombosis of the posterior inferior cerebellar artery.

More rarely it occurs in other conditions such as amyotrophic lateral sclerosis, and it may occur in lesions of the peripheral nervous mechanism for ocular movements as in alcoholic neuritis. Even diseased conditions of the muscles themselves e.g. Myasthenia Gravis may be accompanied by nystagmoid jerks, but these are hardly of the nature of a true nystagmus.

It will be noted then that Miners' Nystagmus presents certain differences from other varieties of nystagmus, and all writers place it in a separate category. Shufflebotham considered Nystagmus combined with clonic spasm of the Orbicularis muscle pathognomonic of Miners' Nystagmus.
It is a condition that occurs solely among workers in coal mines, except for a few cases in ironstone mines where coal seams are present, though Snell claimed that his cases in compositors were similar. So it is evidently some condition or combination of conditions peculiar to a coal mine which underlies the production of the disease. Numerous theories have been advanced since the recognition of the condition, and I shall now give some consideration to the chief of these.

The first theory that held much sway was Snell's Theory, otherwise known as the Position Theory. According to Snell's view the disease was almost limited to a particular class of worker, those engaged in "holing" by which process the coal is undercut to the extent of several feet and then forced down by wedges from above. This is "bottom-holing." There are also "middle-holing" and "top-holing," similar processes at a different level. The condition according to Snell was due to fatigue of the elevator muscles of the eye resulting from the obliquely upward gaze the miner thus engaged was obliged to maintain. In those cases who were not "holers" he satisfied himself that there was similar cause for fatigue of the elevators, and de-
cribes one case engaged in looking after the ascent
and descent of cages at a comparatively well illuminated
pit bottom. He says "It is a myopathic disease....... a local affection. As a result of prolonged strain
in the unusual position for long and frequently
recurring periods, chronic fatigue in the ocular
muscles is brought about, and atony being induced,
oscillation of the globes is caused. All may appear
well at rest, but put the muscles implicated into
action and disordered movements become evident."
In one part of his book he expresses agreement with
Bell Taylor's view that the condition is "exactly
analogous to writers', pianists' and other professional
cramps."

Dransart, (Annales d'Oculistique 1877 vol. ii) who
agreed with Snell that position was the prime cause,
says: - "The myopathy will have its principal seat in
the Superior Rectus and the Inferior Oblique. The
pair of elevators, having an acquired feebleness,
cannot overcome their antagonists by a single effort;
they are obliged to attempt it several times with a
series of little and rapid contractions. They then
produce nystagmus, or rather the vertical oscillations.
To explain the horizontal movements recourse is had
to paresis of the Internal Recti and accommodation.
The impotence of the Internal Recti can suffice to

28.
explain the horizontal oscillation by the same mechanism as the vertical. But the accommodation contributes to increase the muscular disorder by virtue of the relations which exist between convergence and accommodation, — between the Ciliary muscle and the Internal Rectus."

Snell's chief argument for his view was the fact that the nystagmus when not present could be elicited by making the patient look obliquely upwards. The objections to his view are:—

(i) That holing in naked light pits seldom produces the disease. For instance, in the naked light pits of Somerset and Forest of Dean, with narrow seams and much bottom-holing the disease is very rare.

(ii) That there is in fact no upward direction of vision in "holing." So allege Court and Llewellyn. I have had no experience of "holing" myself, but Llewellyn's photographs are quite convincing. Percival the only present supporter of the position theory admits this, but does not think a greater incidence among workers in thick veins invalidates the claim of position as a causative factor. He thinks thick seams which had to be "top-holed" would be worse.

In Percival's view (B.M.J. May 5th 1923, and
March 22nd. 1924) a small percentage (4-5%) of workers are predisposed owing to inherited instability of the fixation centre. The strained position of the eyes is the exciting cause. There is more strain in "top-holing" a six foot seam than in undercutting a thin seam. He maintains that the fact that the condition is four times more common in hewers than in non-hewers shows that hewing is an essential factor in producing the disease, for both are equally subjected to deficient illumination." If this latter factor were the cause the incidence should be equal or nearly equal in the two classes.

I cannot accept his view for it would mean that in some large areas like Somerset and the Forest of Dean there were few persons with hereditary predisposition. In America again there would be no individuals with inherited instability. He does not point out exactly what the strain is that produces it in non-hewers. Regarding his argument about the greater incidence in hewers, again I cannot agree with his statement. The hewer has worse light because of the amount of dust often present, and the unrelieved blackness of the surface of the coal. The hewers use their eyes more for accurate work, and the amount of light entering his eyes may be only one tenth of that
entering the non-hewers. Haldane thinks "he might as truly say that a seamstress working on black velvet and a lady knitting a Fair Isle jumper with a common source of light are receiving the same amount of light."

Percival cites the case of the South Moor Colliery (B.M.J. July 31st 1926) where twenty-five years ago with electric lamps there was a high incidence of Miners' Nystagmus. Oil lamps were introduced and the incidence promptly diminished. His figures are not accepted by Ingram Johnson who was in charge of the cases at the time. He says (B.M.J. Oct: 2nd and Dec: 18th 1926) the electric lamps were given up because the men complained of their weight. Percival's weak point is that he gives no indication whether the electric lamp was a better or worse illuminant than the oil lamp. I hate to think what the electric lamp was like at that time for the present day are still unsatisfactory. In any case Percival does not explain the difference in incidence with the two types of lamp, for the conditions of hewing would surely be the same with both electric and oil-lamps.

(iii) The third objection to Snell's view is that the disease does not occur among such workers as plasterers and ceiling decorators. It is called, are Romics, Om... (iv) There is no hyperphoria as one would expect. (v) Nystagmus is often as easily elicited by confine-
ment in a dark room as by looking upwards. It is elicited on elevation because elevation is our weakest movement and the position of minimum stability. Perhaps the development of flying will ultimately rob us of this method of eliciting the sign.

Snell made much capital out of the case of the compositor who, working in a good light and picking type out of a box at a level above his eyes, developed a vertical nystagmus. Later writers have regarded this as a case of athetopia with nystagmoid movements of a jerky spasmodic type in a neurotic youth who had already had cramp of the fingers.

It is generally agreed now that the condition labelled Miners' Nystagmus is confined to workers in coal mines. Bell Taylor's Theory that the condition was analogous to writers' cramp is hardly satisfying, for in this condition it is only some specific movement that is affected, while in Miners' Nystagmus the oscillations may be constantly present.

Court's Theory or the Theory of Deficient Illumination was the great rival to Snell's. Other supporters of the light theory, as it is called, are Romée, Ohm, Llewellyn and Haldane.

Elworthy supports it but thinks colour as
important as light.

The Nystagmus Committee of the Medical Research Council supported the light theory unanimously.

Court examined 597 miners who used safety lamps and found 207 cases of nystagmus along with 572 miners who used naked lights and found 32 cases of nystagmus. Of the 32, 29 had previously used safety lamps.

Court found no cases of Nystagmus among 200 men who used torch lights.

Romée agrees about the importance of light but thinks excessive accommodation in poor light is the chief factor.

Llewellyn (Miners' Nystagmus, Colliery Guardian Company p.111) points out that in the dark and on looking at a uniform surface one cannot accurately fix a point and there is a tendency to movement. This tendency is increased by the fact that the perifoveal region of the retina is more sensitive to dim light than the foveal.

"The cramped attitude of the miner helps to derange the centres of equilibration. The collier requires accurate fixation to perform his work, and to obtain this he strains his eyes and uses his accommodation excessively. Errors of refraction, if present, must..."
increase the strain." "The total result is that there is, on the one hand, imperfect fixation leading to the transmission of indefinite impulses to the brain, and on the other excessive reinforcement from the higher brain; failure of coordination results and nystagmus ensues."

Haldane (Brit. Journ: Ophth: Nov: 1925) agreeing that deficient light is the essential cause explains its action as follows:—

"The great peculiarity of foveal vision with sufficient luminosity is that the impression in consciousness of an object on which the eye is fixed is a lasting one, in contrast to peripheral vision in which objects seen at first rapidly fade out of consciousness when the eye is fixed, unless there is some movement in them."

........... "With very low luminosity foveal vision loses completely its lasting character. An object, at first seen, fades rapidly out of consciousness so that it can no longer be fixed, the conscious impression being even less lasting than with peripheral vision. The result of this impossibility of fixation is that the eye wanders about, picking the object up momentarily with the fovea and losing it again. This must be a very fatiguing process, and in all probability the fatigue thus induced is the origin of the ultimate disturbance in ocular coordination which constitutes Miners' Nystagmus." "The thresh-
hold illumination for lasting foveal vision varies considerably in different persons, and this variation may be related to varying liability to Miners' Nystagmus."

Ohm (Journal Indust. Hygiene; Dec. 1926) thinks deficient light the chief cause. He points out that "darkness is often necessary to elicit nystagmus, while bright light exerts a calming influence." He also notes "the striking resemblance of Miners' Nystagmus to the "dark tremor" in children (Spasmus Nutans) and young animals." A subsidiary but none the less important factor is the position of the body and head. He thinks that if the men could work in the upright position and did not make such vigorous movements, deficient illumination would not be sufficient in most cases to cause nystagmus.

These two factors affecting one the optic the other the vestibular nerve are the "External Causes," which he expresses thus:-

\[ R = \text{conditions which irritate the labyrinth} \]

\[ B = \text{the illumination} \]

The stronger R is and the worse B is, the sooner will nystagmus appear.

"Internal Causes." he expresses thus:-

\[ V = \text{sensitiveness of vestibular apparatus} \]

\[ A = \text{the total efficiency of the eyes, especially the light sense, which he considers very important} \]
The greater $V$ is and the less $A$ is, the greater is the liability to nystagmus. "Under normal conditions the eye muscles receive stimuli of very small amplitude and high frequency. By longer detention of the light and by unusual irritation of the vestibular apparatus stimuli of greater amplitude and lower frequency may also serve to excite the eye muscles, resulting in an intermittent periodic stimulation of the muscles which becomes recognisable as nystagmus to the observer."

The theory of Oglesby (Brain Vol. 3) that Miners' nystagmus is due to venous congestion of the medulla finds no acceptance now and it is impossible to see how this could be produced in all cases.

Jeaffreson's Theory (B.M.J. 1887 Vol. 2. p. 834) is interesting. He says the disease is a general one of which nystagmus is one sign and suggested the name "Miners' Neurosis" because of the frequently associated nervous symptoms. He attributed some importance to position and thought the condition was due to cerebral anaemia due to pressure of the tentorial ring on the upper part of the pons when the head was thrown back. He did not consider position alone could produce it, but that nystagmus resulted when there was a "dissociation of movements normally combined" e.g. flexing
the head and keeping the eyes directed to the roof.

Jeaffreson's statements applied to an area where the miners worked in a squatting position with perhaps a tilted head, but it is not readily acceptable that the condition of cerebral anaemia does arise in the majority of cases. Besides, as will be seen later, Nystagmus is not associated with cerebral disorders.

Christie Reid (in Brain 1906 Part 3) brought forward his Equilibration Disturbance Theory. His theory is an extension of that of Gowers discussed below with regard to the pathology of the condition.

Reid postulates centres or a centre whose normal action is to maintain equilibrium of the globes in whatever position they are placed - equilibration centres. Darkness does away with the possibility of superior macular images and so fixation is difficult. The miner is subjected to disturbances of body equilibrium as in lying on his side, sitting on his haunches and stumbling in the dark. Reid points out that even in daylight slight rotation is sufficient to manifest the latent type of the disease. To explain what he calls "the beautiful rotary Nystagmus" he calls in the series of "regular, rhythmical movements with the pick swinging from either shoulder, the miner's head
revolving in consonance with these movements."

"The eyes perform compensatory movements. Fixation is difficult and hence the movements tend to escape from control and persist when the miner leaves underground."

All the above authorities with the exception of Snell, Bell Taylor and Jeaffreson are all agreed on the paramount importance of light, though they differ on other points.

More recently three other views have been put forward by some writers. One of these is the old theory of Pechdo, attributing it to the toxic action of mine gases, which has been revived by Robson.

Robson (Proc. South Wales Inst. Engineers. Vol. XXXIX. Nos. 2 & 4.) made a study of the South Wales coalfield and found that roughly the incidence of Nystagmus increased from West to East. The volatile content of the coal was found to show increase in the same direction from the Anthracite areas in the West to the Bituminous coal in the East. As no coal is volatile at ordinary temperatures he had to abandon this and to lay the blame on carbon monoxide. This is produced in minute quantities by spontaneous
combustion of coal and may also be present among occluded gases in coal. He thinks it is due to a toxaemia produced by the action of Carbon Monoxide through the circulation upon a central synapese in the midbrain or cerebellum. He mentions the case of a fireman not known to have Nystagmus who was subjected to large doses of gas. He soon developed Nystagmus and nervous disturbances viz:—tachycardia, increased reflexes, nausea, vomiting, malaise, insomnia, headache, oscillation of the eyeballs and blepharospasm.

There are very many objections to Robson's theory. In the first place some of the adjoining areas in the East show greater differences in the incidence of Nystagmus than that between the extreme East and West. Haldane and Ivan Graham have failed to detect any carbon monoxide in firedamp given off at ordinary mine temperatures, and the amount formed by oxidation is so infinitesimal that it could have no effect on man. Haldane, whose opinion on such a point should be reliable, says (Nystagmus Committee 1st Report):—"The proportion of carbon monoxide present in mine air is seldom over 0.005%, and this has no appreciable physiological action. There can be no doubt that every one of the very varied effects of acute poisoning by carbon monoxide is due to the
want of oxygen produced, owing to the fact that by combining with haemoglobin Carbon Monoxide interferes with the carriage of oxygen by the blood from the lungs."

Robson uses as his chief argument against the light theory the "fact that in spite of the improvement in illumination in recent years the incidence of nystagmus has increased." But surely it applies with much more force against his own theory. Illumination, though it has improved, is still very inadequate in most places. Ventilation on the other hand has improved immensely, and is very strictly regulated by the Mines Act. As a matter of fact our only guide to the incidence of Nystagmus is the number of claims for compensation, and, as I have shown above, this is not a reliable one.

Glaister and Logan in their book on "Gas Poisoning in Mining" describe nystagmus in association with acute Carbon Monoxide poisoning, but the nystagmus is horizontal and not at all comparable with the movements in Miners' Nystagmus. Only one case is recorded. If the condition were due to mine air one would expect it to be as common among hewers as non-hewers.

Again why is there no nystagmus in American mines? Gas is present and the electric cap lamp was introduced to take the place of the naked light. Yet
they have no nystagmus. With regard to the case Robson quotes I have seen one case identical with it except in one feature, and that is exposure to gas. There was a "blower" and a fellow-workman was "gassed." The patient (Case No. 13) however was not actually exposed to the gas, though he experienced a fright. Four days later he was in the same condition as the case quoted by Robson.

Fergus has within recent years brought forward another suggestion. He maintains that the case for light is "not proven." He has no confidence in the Nystagmus Committee because he considers they treated the disease too much as a local eye condition and did not give other symptoms, such as tachycardia, sufficient prominence. Fergus does not consider mere deficiency of light could cause such symptoms as tachycardia and tremor of head and arms. Some of his arguments are decidedly weak. For instance he thinks (Glasgow Med: Journ: March 1926.) the absence of diplopia condemns alike the Position, Gaseous and Light theories because it would be unlikely for both sides to be affected to exactly the same degree. Yet on a later page he himself postulates the existence of a common centre for control of ocular equilibrium in the neighbourhood of Deiter's nucleus. He admits that
the condition is almost unknown in naked light pits, but refuses to blame the illumination because

(1) The American Miners use safety lamps owing to presence of gas, and still have no nystagmus

(2) He knows a number of nystagmics who could continue their work with an oil lamp, but were unable to stand an electric lamp.

(3) Some particular pit has experienced no appreciable reduction in the number of cases receiving compensation, although electric lamps have been introduced.

The first argument is easily met by pointing out that the American Miner works with a comparatively excellent light because he uses an electric cap lamp so that the source of light is near the coal face and directed to the very spot the worker wants it. It seems to indicate a lamentable want of knowledge of underground conditions that he should cite the American mines as supporting him against the light theory, when the American condition of illumination is the very one the Nystagmus Committee recommended as the best method of preventing Nystagmus.

As for the second point it is easy to realise that the hard glare of an electric lamp might cause discomfort to an actual nystagmic if it shines into...
his eyes, for sensitiveness to bright light is one symptom of the condition. One might equally well say that, because a Gargantuan feast does not tend to promote comfort in a man exposed to prolonged starvation, the cause of his condition is not lack of food.

In his third argument Fergus gives no indication that the change to electric lamps resulted in improved illumination. He takes it for granted it did have that effect. He, in common with some other writers, seems to think that the mere name "electric" confers on a lamp a high power of illumination. Many electric lamps, especially in the hands of an inefficient lampman, are much inferior to a good oil lamp.

Fergus maintains that the miner does not use his macular vision for his work because he has known cases of extreme myopia with a punctum proximum of four inches who were still efficient colliers. The fact that they were efficient without macular vision does not imply that they would not be better with good macular vision. I have myself tried hewing coal and am quite convinced that all my strokes were aimed at some particular point - even if that point was not struck with consistency. By removing my spectacles (with lenses of -3.25 D) I convinced myself that I should make an infinitely better coal-getter with my
spectacles, provided I could keep them reasonably free from dust. I questioned several hewers, and they agreed that their strokes were aimed at some particular point.

Fergus gives 107 cases in a table. Only one came from a naked-light pit, the remainder having worked with safety lamps—oil or electric. He suggests, without exactly championing it as a theory, that Miners' Nystagmus might be due to microorganic life or the products of microorganismal activities in bye-gone centuries, because

(1) There are outbreaks of the nature of epidemics in individual collieries, while neighbouring pits under identical conditions are free from it.

(2) The paroxysmal attacks sometimes seen with intervals of well-being resemble ague.

(3) The difference between the oldest and the youngest affected seems against a physical agency such as light, but is suggestive of exposure and relative immunity to some noxious agent.

No one however has recorded any elevation of temperature in Miners' Nystagmus, and no collier has ever infected any member of his family with the disease.

If it were due to some organismal agency, against which some might possess immunity as suggested by Fergus, we should not expect the incidence to be greatest at the comparatively advanced age of forty and over. The susceptibles should long ago have been
infected. Those who had escaped thus far would probably owe their escape to their immunity and continue free from it. Besides the cases do not conform closely enough to any definite course to fit in with the theory of infection. "Neurotic" signs and says:

"If the blame on organisms of bygone centuries is to put the matter outside the limits of proof or disproof. There is nothing constructive in Fergus' papers. What evidence there is is negative."

To lay the blame on organisms of bygone centuries is to put the matter outside the limits of proof or disproof. There is nothing constructive in Fergus' papers. What evidence there is is negative. A third view recently put forward is that the condition is purely a psychoneurosis. A number of writers have expressed this view notably Eddison (Journal Ment. Science. April 1926) and M. Culpin (Brit. Journ. Ophth. Nov. 1925.) Culpin maintains that the primary contrast between naked light and safety-lamp pits is one of danger and not of illumination." He says "the frequent identity of the mode of onset of Miners' Nystagmus with that of "shell-shock" clearly points to the common factor - the threat to
personal safety." He is referring here to those cases which develop dramatically after a trivial accident. He points out also the identity of the non-ocular symptoms with those following war strain. He quotes a case who manifested obvious "neurotic" signs and says:— "If he had been a business man he would probably have been treated for nervous exhaustion; an accident would have been followed by traumatic neurasthenia; if a soldier up the line he would have had 'shell-shock', whilst if he stayed at the base he would have developed that "nystagmus" of the heart known as D.A.H.; if such a patient were a woman she would suffer from spasmodic dysmenorrhoea and a nervous breakdown at the menopause. The patient was a man and finally suffered from an occupational cramp. He will stand for one type who will acquire whatever form of psychoneurosis is offered by the environment, and I venture to suggest that if he had been a miner he would now be receiving compensation for Miners' Nystagmus."

Eddison writes similarly. "The danger element determines the outbreak of neurosis, and eye-strain the predominantly ocular form." "The Nystagmus is a tremor, equivalent to the tremors of the hands and legs in war neuroses."
The above are the most important views that have been put forward to explain the causation of Miners' Nystagmus.

With regard to the pathology of nystagmus there is not much known about the exact manner of its production.

In the earlier part of this section I gave some types of nystagmus and the various conditions in which nystagmus was found.

I shall now consider theories as to the pathology of nystagmus in general and Miners' Nystagmus in particular.

Probably the first attempt to explain the nature of nystagmus is that of Wilbrand, who regarded it as "due to derangement of the volitional impulse or of the general reflex centre for the eyeballs, so that there is a defect of harmony between the two."

Gowers does not agree with this view because nystagmus is unknown with disease of the cerebral hemispheres above the cerebral ganglia, unless pressure is exerted on lower structures. He points out (Proc: Roy; Soc: Med. 1908.1, p. 71. Neurological Section) that in all movements steadiness is based on reciprocal action, the opponents yielding and yet giving due support, and he advances a theory based on Sherrington's experiments with "spinal" animals. Sherrington
divided the cervical cord in animals, and found that stimulation of the extensors of a joint for example, caused them to act up to a point; then they stopped suddenly and the flexors contracted, and this alternating extension and flexion went on for some time.

By dividing the nerve to the antagonists and stimulating the central end, he showed that the relaxation of the muscles first stimulated was due to inhibition of the spinal centre by afferent impulses from the antagonists. Such stimulation immediately inhibited contraction of the acting muscles. As the nerve was solely muscular the centripetal impulses must have arisen in the antagonists by tension acting on the muscle spindles.

Gowers in his theory of the mechanism of nystagmus assumes there is a centre which combines and coordinates the action of the two eyes, and also subserves the muscle reflex action. He calls it "the Mid-brain ocular centre," the mid-brain situation being presumed, because all lesions causing nystagmus are situated in or near the mid-brain and pons, or in the cerebellum. This centre controls the equilibrium of the antagonistic ocular muscles, and a deficiency or excess of the many influences acting on it may disturb its stability and allow the muscle reflex alternation to assert itself. The light reflex
is also governed through the same centre.

Before considering the validity of this theory when applied to various forms of nystagmus, it might be as well to mention the connections of the ocular mechanism with the labyrinth and certain other parts of the nervous system.

The vestibular nerve from the semicircular canals carries fibres to the nucleus of Deiters, which is connected by the vestibulo-spinal tract with the anterior horn cells of the spinal cord, and with the nuclei of the eye muscles through the posterior longitudinal bundle, which lies ventral to these nuclei beneath the floor of the fourth ventricle.

The cerebellum also is intimately connected with Deiters' Nucleus and the ocular nuclei.

Stimuli from the optic nerve are also capable of an effect on the body via the corpora quadrigemina and the tecto-spinal tracts. The nuclei of the eye muscles are closely connected with one another on the same and the opposite sides, and they receive impulses from the cerebral cortex; though disease of the cerebral hemispheres does not, as already mentioned, produce nystagmus.

It has been suggested that the nucleus of Deiters is the centre whose disturbance is responsible for nystagmus.
Accepting Gowers' view as a working hypothesis I shall now apply it to see if it can explain the various clinical forms of nystagmus.

In the congenital and infantile groups it is not inconceivable that the disordered afferent impulses could so disturb the centre as to manifest the muscle reflex action of Gowers, resulting in Nystagmus. The influence of light on the centre is seen in infantile nystagmus associated with head nodding. Similarly the gross pathological causes such as syringomyelia, disseminated sclerosis, Friedreich's ataxia, and cerebellar disease could produce nystagmus by destruction of the centre or of the fibres connecting it with the ocular nuclei. Friedreich's ataxia, for instance, is a disease of the spinocerebellar system, and this might easily upset the stability of the centre through the influence of the cerebellum over it. Again in Syringomyelia there are often signs of glial tissue in the fourth ventricle, and around the aqueduct of Sylvius.

Those cases of nystagmus which occur in chronic poisoning by such poisons as lead, alcohol, and arsenic are explicable by the assumption that they produce degeneration in or near the centre.

Labyrinthine nystagmus again, as produced for instance by syringing the ear, is easily explicable
as due to deranged impressions from the labyrinth to
the centre. The acute disturbance of the ocular
centre seems to allow the muscle reflex action to
become insubordinate for a short time.

Peters (Arch. Ophth. 36, 667) has even suggested
that Miners' Nystagmus is of purely labyrinthine
origin—a redistribution of the endolymph occurring,
owing to the head being titled backwards at work.
He calls attention to the backward carriage of the
head and asks, "Is it primary or secondary?" It has
been pointed out however that other abnormal positions
of the head such as torticollis do not cause nystagmus.
The backward carriage of the head seen in some cases
of Miners' Nystagmus serves, as Llewellyn points out,
the simple purpose of getting the eyes into a position
of stability while the gaze is still directed hori-
zontally forward. Besides Miners' Nystagmus is not
like a purely labyrinthine nystagmus, which is rhythm-
mical and not pendular.

With regard to Miners' Nystagmus, Gowers seems
to have considered it possible for the poor light to
produce nystagmus in a manner analogous to the infan-
tile cases; but he does not stress the point, and in
another place (Albutt & Rolleston's System of Medicine)
he gives as causes deficient illumination and upward
oblique gaze. He also attributed some possible
importance to bad air.

The only criticism that can be offered against his view as applied to Miners' Nystagmus is that a rate of 350–500 oscillations per minute as are reported by some writers hardly allows time for complicated nerve reactions.

If we consider the above theories of Miners' Nystagmus, we will see that, with the exception of Snell's and Bell Taylor's, they nearly all contain the idea of incoordination of some centre. The reasons for rejecting Snell's and Bell Taylor's are mentioned above. Jeaffreson's is somewhat on the usual plan too, for his dissociation of movements normally associated" would appear to involve the production of incoordination somewhere, though his cerebral anaemia theory cannot be accepted.

Apart from these and the three recent views mentioned above all the authorities are agreed on the importance of insufficient illumination. Though the various writers include other subsidiary factors and explain the mechanism of production in different ways, they are nearly all agreed that deficiency of light is the essential factor in the aetiology of Miner's Nystagmus.

The multiplicity of views expressed recently is itself an indication that all is not well with
our knowledge of the condition. It must however, be admitted that the evidence is strongly in favour of the advocates of the "Light" theory. If we consider some of their strongest arguments their conclusion seems inevitable.

Until Llewellyn made photometric measurements at the coal face it was not realised how very feeble was the light with which the miner worked. Llewellyn showed that the average amount of light falling on the working area varied between 0.02 to 0.2 of a foot-candle. Owing to the fact that coal absorbs 90-97% of the incident light the amount of light reflected into the eye is 0.002 to 0.02 of a foot-candle. He proved that the illumination in a candle pit was on an average five times greater than that in a safety lamp pit. The candle has the great advantages that it can be placed nearer the working place, which from the law of inverse squares means a great deal; and that there are no shadows cast as in the case of the safety lamp, where the bonnet, standards and reservoir considerably reduce the amount of light. The light of a safety lamp, already bad, is made worse by the distance it has to be placed from the working place so as to avoid the swing of the pick, the absorption of the light by the coal and coal-dust covered surfaces, and often
the angle of incidence; the illumination with the rays at right angles being, in accordance with the cosine law, double that with the rays at an angle of 60°.

The coal dust fouls the lamp glasses, and this may reduce the illumination by as much as one third. The workman is apt to leave his lamp too long in one place - the lamp may be as much as nine or ten feet away, and with his body perhaps obstructing the light, the light falling on the coal face may be reduced to less than one hundredth of a foot-candle. (Llewellyn)

Many of the electric lamps in common use are less efficient than some oil lamps, for they are often very badly looked after and they too are at a distance from the coal face.

I shall now give some of the chief points in favour of the theory of deficient light.

In the first place very strong support is given by the history of the condition. The first case was reported in 1861 though it seems to have been recognised about 1855. Safety lamps were introduced in 1851, and it was not until after the extensive introduction of safety lamps that we find the literature growing rapidly. It is difficult
to believe that our fathers and grandfathers who were such excellent observers could have missed the condition if it had existed for any length of time before.

In naked light districts the condition is rare or almost unknown. In America, again, where the only change in illumination was from naked lights to the electric cap lamp there is no problem of nystagmus. Any reference to it in the literature deals with European cases.

In Scotland where safety lamps are much less widely used the condition is much less common than in England. Court's figures seem to bear no other interpretation than the one he put upon them. He examined a total of over 1000 men, and found 34% affected in safety lamp pits and 5.6% in the candle pits. In over 200 cases who worked with torch lights he found no nystagmus.

Stassen in over 11,000 men found 5% to 6% of serious cases in safety lamp pits and 1.3% in candle pits. In the candle pits there were no grave cases.

Stassen estimates the gravity of a case by the amount of nystagmus, and by the ease with which it is brought out.

In the Nystagmus Committee's first report (p.32) it is shown that in Bristol and the Forest of
Dean, and in the South Wales candle pits where the percentage of safety lamps was nil, the rate per 100 underground workmen was .03 and .023 respectively, while in the South Wales safety lamp pits with 100% safety lamps the rate was .38 per 100 underground workers.

Further there are many instances of an improvement in illumination reducing the amount of nystagmus. Probably the best of these is the case given by Romiéé, for this is the only case where the true incidence of nystagmus before and after improvement of illumination has been contrasted.

Romiéé made a complete examination of the coal-getters in four Belgian pits in 1892 and again in 1908 after improved illumination had had time to do its work in three of the pits. In these three pits there was a reduction of fifty per cent in the number of coal-getters showing nystagmus. In the other pit where the illumination had remained unchanged the incidence had shown a slight increase.

In the discussion of a paper by Llewellyn (Trans. Inst. Min. Eng. Vol. LVIII part 3) Mr Caleb Johnson gives the following experience. Electric lamps were introduced in one of two pits working the same seams under identical conditions. About 70-75% of the men at the coal face were supplied
with the new lamp, which was a distinct improvement on the oil lamp it displaced. In that pit the cost of nystagmus six years after was one thousandth part of a penny per ton of output. In the oil lamp pit it was three fifths of a penny per ton. Many other instances could be quoted (see Mitton (discussion) Trans. Inst. Min. Eng. Vol. LXVIII, Part 1. p. 70 & 71, and Nystagmus Committee, First Report pp. 44 et seq).

Ohm has produced nystagmus experimentally in puppies and kittens by shutting them up in a dark cellar.

They also exhibited head tremor.

It is found that one method of eliciting nystagmus is to shut the patient in a dark room for a short time.

I myself have known cases who could not keep working with an oil lamp but continued with an electric lamp, though some cases find the electric lamp worse. This does nothing to overthrow the light theory, for many cases of Miners' Nystagmus are sensitive to bright light, and would find the hard light and glare of the electric lamp uncomfortable.

I went down the pit and saw the men at work to obtain some idea of working conditions. I must confess I was unprepared to find such appalling conditions of illumination. It must be a great strain to work for eight hours in such conditions. I can testify to the discomfort caused by the light shining directly into
one's eyes. I saw men at work with oil lamps giving just over 1/2 c.p. average for a shift of eight hours, and compared their lamps with an electric lamp giving an average of 0.75 c.p. for the shift. The difference was not easily appreciable, and I am quite sure those electric lamps would not go very far in the reduction of nystagmus.

I obtained figures from one colliery in which the electric lamps mentioned above were introduced in 1923. I append the figures.

<table>
<thead>
<tr>
<th>Year</th>
<th>Cases of Nystagmus</th>
<th>Number of men underground.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1922</td>
<td>7</td>
<td>720</td>
</tr>
<tr>
<td>1923</td>
<td>8</td>
<td>735</td>
</tr>
<tr>
<td>1924</td>
<td>10</td>
<td>755</td>
</tr>
<tr>
<td>1925</td>
<td>3</td>
<td>786</td>
</tr>
<tr>
<td>1926 (7 months strike) nil.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1927</td>
<td>2</td>
<td>764</td>
</tr>
</tbody>
</table>

It appears tempting to attribute the decline to the introduction of the electric lamps, but having seen the lamp in use I can hardly credit it with such powers. I am more inclined to attribute this decline to the alteration in the compensation rate in 1924, when it was altered from 35 shillings per week to half the weekly wage being obtained previous to the onset of
disability. This was in force in 1924 but the clerk who supplied me with the figures volunteered the information that the men did not realise this and nearly all the cases in 1924 came to enquire why they were not receiving 35 shillings per week. He himself suggested that the decline in 1925 and 1927 was due to the realisation of this fact.

I obtained from another small colliery using the oil lamp referred to above, and this supports my explanation of the above decline. The figures for this colliery were:

<table>
<thead>
<tr>
<th>Year</th>
<th>Cases of Nystagmus</th>
<th>Number of men underground</th>
</tr>
</thead>
<tbody>
<tr>
<td>1922</td>
<td>3</td>
<td>298</td>
</tr>
<tr>
<td>1923</td>
<td>6</td>
<td>237</td>
</tr>
<tr>
<td>1924</td>
<td>4</td>
<td>207</td>
</tr>
<tr>
<td>1925</td>
<td>nil</td>
<td>211</td>
</tr>
<tr>
<td>1926</td>
<td>nil</td>
<td>207</td>
</tr>
<tr>
<td>1927</td>
<td>nil</td>
<td>216</td>
</tr>
</tbody>
</table>

It will be seen that since 1924 there have been no cases, and there is no improvement in illumination to which we may attribute it.

I made a special point of observing the men at work while underground and with a seam about three feet thick, from the top of which the miner had to clean a few inches of clod. Their eyes were in a position...
very closely resembling that which Snell thought so important, and after a trial of hewing I was convinced that it would cause fatigue of the eye muscles concerned with elevation. While admitting this I cannot agree with Snell's theory that this strained position is the cause of the nystagmus. I do not consider that recent criticism has done much to disprove the view that deficient illumination is an essential factor in the production of the actual eye movements in Miners' Nystagmus. At the same time a careful examination of the literature and my own clinical experience has convinced me that there is more in the problem than meets the eye.

A few facts are worthy of consideration. The examination of large numbers of underground workers has established the fact that a considerable proportion of these men exhibit actual nystagmus. The figures of Romiéc, Stassen and Coppez in Belgium, of Court and Llewellyn in this country, and of Dransart in France agree fairly closely. The figure is 20 to 25 per cent of all underground workers. This proportion would fit in with an explanation on a physical basis. But the majority of these cases have no immediate interest to those concerned with Miners' Nystagmus as an economic problem. A great many of them are bliss-
fully unconscious of their condition. Some have marked nystagmus without being incapacitated. Compare case 12 of my series.

The percentage of men claiming compensation for nystagmus represents only a small fraction of the above figure. Granted that some environmental condition or combination of conditions is an essential factor in the production of nystagmus, there must be some personal factor that is important in determining disability. Clearly the presence of nystagmus in the majority of cases does not produce incapacity. In fact the relation of nystagmus to incapacity, as I remarked above, is extremely variable. Many cases are seen who show total incapacity with a minimum of oscillation, and some cases with no oscillation at all. In cases Nos: 3 and 20 I could not elicit a trace of nystagmus. The medical officer of a large group of collieries in this district assures me that he sees many such cases. Again subjective symptoms and incapacity may persist for a long time - months or even years - after the disappearance of nystagmus and all other signs.

Consider cases 8 & 9 of my own series. I can see no reason why these men should not be at work. Yet two are idle and one is employed on light work on the surface. To what is their capacity due? Is it due to nystagmus, to laziness and malingering, or has it a
more subtle psychoneurotic origin? Nystagmus has disappeared, so it cannot be due to the eye condition. We can dismiss the second alternative with confidence in most cases if not quite in all. The solution seems to lie in the third question. The consideration of a few cases is most instructive. I think the most convincing case I have seen in life or in print is one recorded by Llewellyn (Miners' Nystagmus, Colliery Guardian Coy. p.4) and I quote it at length. The case was one of "a collier with 20 years of underground life, all spent with safety lamps. On Aug. 26th he received a slight blow on the head from a stone. He resumed work on Aug. 31st, and was suddenly seized with blindness and had to be led home. He had never experienced any symptoms before. Examination showed violent rotatory nystagmus even at the horizontal level. He had severe headache, ataxy and photophobia. He became very depressed and drank heavily. He showed no improvement for months and expressed suicidal intentions. After 18 months he was little if any better and then accepted a lump sum in settlement of his claim. Within a month he was working underground without ill results. His explanation of his cure was: - 'I have been taking Dr. Williams' Pink Pills for Pale People for three weeks, two after each meal, with threepenny worth of gin each time. I came well at once.'"
The man was obviously either malingering or psychoneurotic. No malingerer has been known to produce nystagmus so we must exclude that possibility. It would be interesting to know if this man had nystagmus before the accident. Personally I consider he must have had it without perhaps knowing it. Anyway I consider there can be no doubt that his incapacity was psychoneurotic, or the suggestive power of a few dozen Pink Pills could hardly have charmed it away. Was ever a case of Railway Spine more promptly cured by the settlement of a claim? In this case and others like it one cannot avoid the conclusion that the patient's disability rests on a psychoneurotic basis. It is admitted by all writers that it is easy to produce a claim for compensation by telling a nystagmic who is unconscious of his condition that he has the disease. The onset of disability in several cases after an accident (as in cases 13 and 15) and in economic crises, is suggestive. The cases 11, 19 and 20 of my own series illustrate how an economic disaster can produce claims.

Cases like these have brought the psychoneurosis hypothesis into prominence. Those who regard the condition as purely psychoneurotic in
origin point out that "nystagmus per se does not dis-
able; that symptoms admittedly psychoneurotic are	en often the actual cause of disability.
The ocular symptoms associated with nystagmus are
typically psychoneurotic; photophobia, blepharospasm,
head tremor, extreme convergence on examination of
the eyes, and night blindness were familiar during
the war as mental results of such stimuli as gassing
and burial." (Culpin.) The non-ocular symptoms
are claimed to be identical with those following war
strain, and Herbert Page's book on Railway Injuries
describes nearly all the symptoms except oscillations
of the globes.
Many trivial injuries determine the onset of incapacity,
such as slight cuts on the head, a foreign body in
the eye, or even a sprained back. The nystagmus often
disappears as the more obviously mental symptoms in-
crease. Exactly similar phenomena were seen in the
war-strained.
Fear is a powerful emotion and, in spite of the
Freudians, a potent reason for repression and mental
conflict. There is no gainsaying the fact that
the collier's life is both arduous and dangerous.
He may see his friend killed or seriously injured a
few feet away from him while he himself has the
narrowest of escapes. He can hardly help wondering if it will be his turn next. I have seen a well educated young man - an electrician - who literally looked into the grave but escaped with nothing but a shaking. Now, several weeks after, he exhibits tachycardia and tremor of the hands made worse by the least excitement. He was not an underground worker and of course had no nystagmus, but had an ordinary miner had his experience I should not have been in the least surprised to see him put in a claim for Nystagmus.

In addition to such incidents there is the miserable feeling of being penned in, and the realisation that it will be difficult to get away if danger does threaten. No doubt the miner gets accustomed in some degree at least to his environment, but the old emotional experiences have left their mark.

It is not then difficult to conceive that some men will develop neurotic symptoms when a sufficient stimulus is forthcoming.

Eddison in his paper in support of the psychoneurotic theory draws attention to three points:

(i) The psychoneurotic predisposition. He quotes a case who admitted to a phobia of being stalked in the dark before the onset of symptoms. I have not found such definite admissions but it is worth considering the type of person who goes on compensation for nystagmus. They are
all of the neurotic type, who are very much concerned with every little ailment they have.
Eddison found psychoneurotic symptoms in 199 out of 310 cases. These preceded the nystagmus in 26 cases and accompanied it in 51. In the remainder they developed later.
It is notorious that a latent case can be suggested into a manifest case with ease. And in this fact, I think, lies the explanation of the "epidemics" which make Fergus invoke the aid of some microbic agency. There were similar experiences with "shell-shock" cases in the war, especially among men with poor morale.

(ii) The neurotic colour of the clinical picture as seen in
(a) The prevalence of anxiety symptoms.
and (b) The fact that obviously neurotic symptoms and nystagmus may both be evoked by the same stimulus e.g. fright or excitement, and that one cannot be evoked without the other.

(iii) The reaction of the cases to compensation.
I have already dealt with this at some length and shall mention other points later.
Eddison suggests that the bad lighting constitutes a strong suggestion for the adoption of ocular symptoms as part of the developing psychoneurosis by producing eye-strain and fixing the patient's attention on his eyes. The nystagmus is, according to him, a tremor analogous to other tremors, while the danger element is the real origin of the condition.

There are two stumbling blocks in the way of those who would support this view that the condition is purely psychoneurotic in origin. The first is the difficulty in explaining the difference in incidence between naked light and safety lamp pits. It must be remembered that the investigations of Court and others have shown a greater incidence in safety lamp pits, as far as the true incidence is concerned, while the number of claims for compensation is also greater.

Those who support this view maintain that the primary difference between the two classes of pit is one of danger and not of illumination. I find myself unable to accept this statement.

It probably was so in the early days with frequent explosions, but the younger generation has forgotten nearly all about gas explosions, and probably many a young miner is not quite sure why he uses a safety lamp.
Are we to believe that in America where, owing to the occurrence of explosions the electric cap lamp was introduced, the men know no fear?

Secondly, how can their view explain those cases who have nystagmus without symptoms or disability? Why does anyone develop a psychoneurosis? The raison d'être of a psychoneurosis is the removal of the victim from his uncongenial environment, or the obtaining of gain in some form or other. There is not much gain in developing nystagmus and then continuing to work for years with it. There are a number of such cases who know they have nystagmus and continue to work. My case No. 12 has been working with such knowledge for five years, and Martin describes a case who had worked for seventeen years.

It is instructive to consider the influence of the factor of compensation. I have already made some remarks in connection with the explanation of the increased number of claims for compensation; and in the introductory section I have shown the effect of alteration of the definition in 1913, and of economic stress in 1922. Now I shall pay more attention to the prolongation of disability.

In 1908 the proportion of old cases (i.e. continued from the previous year) to new was 15:100. This has risen year by year and in 1922 was 124:100 and in 1925 was 228:100.
In other European countries where the compensation regulations are more stringent the protracted disability now seen in this country is unknown. Over 75% of Stassen's cases in Belgium return to work in three months, and only 2% are unable to resume their work at the end of six months. In this country too, before the inclusion of Nystagmus in the list of conditions for which compensation should be paid, the cases were practically never off work for more than three to six months, according to the evidence given before the Departmental Committee on whose recommendation Nystagmus was scheduled for compensation in 1907. Nowadays one frequently sees cases whose incapacity has lasted for periods of two to more than ten years. A somewhat similar experience arose in the matter of pensions for psychoneuroses of war. I have no doubt that the prolongation of disability and a great deal of the increased number of claims are directly attributable to compensation.

The following instance cited by Haldane (Trans. Inst. Min. Eng. Vol: LXXXIII Part 2) is convincing. After the coal strike of 1921 a colliery discharged 250 men. In 1920 out of a total of 650 men 3 cases were certified. In the four months following resumption of work at the pit 23 out of the 250 men dismissed were certified, but not a single case was
reported among the 400 men retained. Obviously the
disability was either feigned or psychoneurotic in
origin.

At one colliery I know, where the average
number certified was one or two yearly, there were
eight certified within two months of the commence-
ment of a strike. Cases 11, 19 and 20 are also
interesting in this connection.

Further there is a tendency for workmen,
especially those of advanced age, who are really
incapacitated from other causes, arteriosclerosis,
bronchitis etc., to seek compensation for nystagmus.

It has been pointed out by indemnity companies that
the death rate of nystagmus receiving compensation is
high. Case 16 was for many months on compensation.
A few days after resumption on the surface he died
suddenly from cardiac failure.

Cases like Llewellyn's, quoted above, and a
number of my own (Nos. 8, 9, 11, 19 & 20) show for how
much compensation is responsible.

Many cases are nothing but compensation neurosis, and
this applies especially to these with protracted dis-
ability. I am extremely doubtful whether Miners'
Nystagmus qua nystagmus produces disability.

It certainly does not cause the prolonged invalidity
we see in many cases. The worst nystagmus I have
seen was in that case who has continued to work for years. He says he has very little trouble, and has no desire to go on compensation while he can work. If he were dismissed and could not find employment it might be otherwise.

What conclusions then can be drawn from the above?

For the reasons given I do not consider the whole matter can be explained on a purely psycho-neurotic basis. Nor can I agree that the factor of deficient illumination explains the whole case.

I think it can be concluded with a feeling almost of certainty that the defective light is the causa causans of the actual nystagmus, but the nystagmus, at least in the vast majority of cases, is not the cause of the disability. If deficient illumination and the ensuing nystagmus were everything one would expect a patient on being removed from the cause to show a steady improvement in his symptoms. Many have good and bad spells. Fergus likens the attacks to ague, but similar phenomena were seen in war cases and in all kinds of psychoneuroses. In other cases the symptoms complained of get worse with the lapse of time until the
patient is what the layman calls "a case of nerves."

If everything were due to the physical cause, deficient illumination, I should expect a much greater percentage of those exposed to this condition to be disabled.

In my opinion the cases fall into groups which may be described as follows:

1. Those cases with dramatic onset after some injury or fright, (e.g. cases 13 and 15) and a host of subjective symptoms but without nystagmus. These are psychoneurotic from beginning to end. Cases like No. 19 and 20, who develop disability promptly on dismissal but show no nystagmus, are of the same nature, if malingering can be excluded.

2. Cases who have nystagmus without symptoms (Case No. 12) and without disability. These are the so-called latent cases. It is perhaps possible for nystagmus itself if very severe to cause so much discomfort as to produce disability, but I have never seen a case without other symptoms of a neurotic type who was disabled.

It must be assumed that the cases in this group are the fortunate majority of those who exhibit
the sign nystagmus but are endowed with a stable nervous constitution. Those who succumb are men less well fitted by heredity, environment, and training for life's battles, and have a consequent tendency to psychopathic symptoms.

3. Other cases again, forming the majority of those certified, show gradual onset of symptoms, though disability may appear suddenly after an accident, and exhibit nystagmus along with neurotic symptoms such as spasm of eyelids, giddiness, tachycardia and tremor of the hands. The late W.H.R. Rivers pointed out that the head tremor is probably related to the ocular part of the disease for it is rare in other psychoneuroses. Further it occurs as a part of Ohm's "dark tremor."

These seem to be cases of nystagmus combined with psychoneurosis. The victim of the condition first notices some slight difficulty in executing the finer part of his work, and feels a "strain" of his eyes. He finds he has some difficulty in fixing points accurately. For instance he has trouble in notching timber or in striking a particular spot with his pick. The strain he complains of is no doubt a sense of fatigue of his centres at some level, probably
a high one, due to a difficulty in interpreting the images as in reading in a dim light.

His visual trouble causes him sooner or later to develop three well defined fears although he will probably not admit the force of them.

Firstly, he fears he may lose his sight, and in this connection it must be remembered what an important place the eye takes in a list of our organs. The depressing effect of this fear must be very great.

Secondly, he develops an economic fear that he may not be able to continue to earn his living, and it is from its bearing on this that compensation is so important.

His third fear is that his difficulty in seeing may render him more liable to accidents. I do not consider the element of physical danger so important as the advocates of the purely psychoneurotic theory would have us believe. If it were so, we should find that the danger of the occupation would result in a more even distribution in safety lamp and naked light pits.

Any slight eye trouble resulting from nystagmus acts as a nucleus round which to build up a psychoneurotic picture, just as in the war cases an old wound localised a hysterical paralysis. It serves as a locus minoris resistentiae.

With a man in this condition a mere trivial injury is
often the last straw and disability sets in.
The introduction of compensation for nystagmus has
added another factor. It has in the first place
familiarised the miner with the condition. At present any and every uncomfortable sensation about
the eyes, which in the old days would have gone unnoticed, is apt to be attributed to nystagmus, and to arouse all sorts of thoughts and fears of illness and incapacity.
Secondly, it has made the financial aspect loom large in his mind. I consider compensation directly responsible for those cases who put in a claim after dismissal. But for the presence of compensation there would be no disability. That is not to say that these cases are malingerers. There are very few who wittingly mali
The familiarity with the condition and the importance of the financial aspect so act on the whole personnel of the mine as to prepare a fertile soil for future cases.
Compensation further acts by prolonging the disability once it is established. If the man is lazy — and all of us are not gifted with a love for work — having once had a taste of idleness he wants a feast of it.
In the olden days after perhaps a few weeks’ rest the victim would have attempted to resume work, and under the stimulus of necessity would have won through.
Now, especially if there is little pecuniary advantage
to be gained by resumption of work, the effort is not made.

In those cases who are afraid of losing their sight or that their condition will reduce their value in the labour market, the instinct of self-preservation points to compensation rather than resumption of work, just as those cases who are dismissed seek compensation from resentment, or from the fear of not finding other employment.

Further, if the man is kept idle too long he loses his physical and moral tone. He thinks he must be a hopeless case and this, together with feelings of annoyance with his employers as the party responsible for his sad plight, is not conducive to a speedy recovery.

Such is my opinion of the problem of nystagmus. To explain the mechanism of production of the nystagmus itself it seems plausible to accept Gower's suggestion of an ocular coordinating centre in the mid-brain.

It needs no great exercise of the imagination to picture this centre as receiving stimuli from various sources, in particular the eye, and to a less degree the labyrinth. Disordered stimuli might well be able to induce incoordination and nystagmus. Personally I think the view of Ohm which emphasises the importance of deficiency of the light sense and
regards the muscular contractions which we recognise as nystagmus to be as it were an exaggeration of a normal process seems the most attractive explanation. Or the "nystagmus" might be a tremor resulting from the removal of control or inhibition by a higher centre, just as the tremor of the Parkinsonian is explained by the removal of striate control over lower centres in the mid-brain. Exactly where this controlling centre is situated would be a matter of very difficult speculation. Whatever the exact explanation of the mechanism of production may be I think that the evidence to hand is very strongly in favour of the view that deficient illumination is an essential factor in the production of the actual nystagmus, and if we can reduce or prevent the incidence of the condition it is not of paramount importance from a practical standpoint to know the exact mechanism.

It would be well perhaps to consider briefly some subsidiary factors which are usually regarded as having an aetiological bearing.

Age.
The liability to nystagmus increases with advancing age and consequent longer exposure to the cause. It has been found as early as 13½ years (Stassen) after
only five months of underground life. The average age is given by Llewellyn as 42 years, and the average duration of underground life as 26 years. My own figures are 41.6 yrs and 23.6 years.

There is a tendency for older men to blame nystagmus for any ocular disability of which they are conscious.

Hereditary Predisposition.

Several instances are recorded of nystagmus among two or more members of the same family. This factor might act in two ways. Some personal factor such as deficiency of the light sense may be inherited. Or a psychopathic taint may be handed on from father to son. As sons follow their father's occupation the mere working of chance would ensure some cases occurring in families.

Error of Refraction.

Anderson (B.M.J. 1920, ii, 313) has suggested that this is the most important etiological factor and that presbyopic changes explain the maximum incidence at the age period of 40-45 years.

Great difference of opinion prevails on the importance of refractive error in Miners' Nystagmus. An examination of 1200 cases (Miner's Nystagmus Committee, First Report p. 53) discovered presence of error in 71.5%. Parsons gives the incidence among
the general population as 70%. The influence of this factor can hardly be great.

Pooley, after a comparison of nystagmus miners with unaffected miners and with workmen other than miners, concludes (Miners' Nystagmus Committee, Second Report p. 33) :-

"Errors of refraction in themselves have no effect whatever either on the incidence of Miners' Nystagmus or on the age at which incapacity from Miners' Nystagmus commences."

Among 15 of my cases whose refractive condition I could ascertain 9 had error of refraction. The number is however too small to render the observation of any value. I have no doubt that errors of refraction may have some influence in cases with marked error, for in some such cases no other explanation can be found for the nystagmus. These cases occur in men who are not miners. Correction of any error nearly always gives increased comfort to a nystagmic.

**Accident.**

The influence of this factor has already been mentioned.

It does not produce nystagmus, but may make manifest a latent case and may be the origin of neurosis.

**Illness.**

Frequently the onset of disability occurs after influenza and other debilitating conditions.
Alcohol.

The chief influence of alcohol is in retarding recovery. Idleness leads to alcoholic indulgence and this completes a vicious circle by prolonging disability.

Nature of Work.

The actual coal-getters provide 80% of the cases. Timbermen and firemen provide a comparatively large share of the remainder. Those men are affected who use their eyes most but no underground worker is immune.

Position at Work.

This may have some influence in producing fatigue of the ocular muscles and so fixing the patient's attention on his eyes, but I do not think it has any essential role in the production of the eye movements.

Ventilation.

The conditions of ventilation can, I think, be disregarded as a factor nowadays, but it is interesting to note that Haldane found that reduction of the oxygen in the air supply by 1% caused a reduction of 34% in the light given by an oil safety lamp.
Treatment.
This must be considered under the heads:—

A. Prevention
B. Treatment of the developed disease.

Prevention.
As the condition in many cases proves so intractable to treatment, it is prevention rather than cure that calls most for our attention.
It really seems as if the problem is twofold. We shall have to consider how we can prevent not only nystagmus, the physical objective manifestation but also the disability which often accompanies it and may even occur without it, and above all the protracted idleness which will ultimately convert an apparent flower of manhood into a weed.
The evidence to hand indicates almost beyond doubt that the essential factor in the production of the ocular movements is deficient illumination.
So our first effort must be to improve this. The two possible methods of doing so are to increase the reflecting power of the coal, and to increase the amount of light that reaches the coal face.
It is not economically possible to increase the reflecting power of the coal throughout the mine, so we must depend on the second method.
If the experience in American Mines is any criterion, it is obvious that the best method of increasing the
amount of light is the adoption of electric cap lamps. In this way the light is brought nearer the coal face and is at right angles - the cosine law and the law of inverse squares being ever present in our minds. -

Further the lamp moves with the workman giving light just where it is wanted, and there are no shadows.

Cap lamps have been introduced on an experimental scale in a few places but did not find favour. This must be due to a conservative trait in the miner, for I should think that if the miner can accustom himself to his work, he should, with a little encouragement and persuasion, have no difficulty in overcoming his dislike for a heavier and unusual lamp. After a very little experience with it the improved illumination and greater comfort would heavily outweigh any disadvantage such as weight or awkwardness.

Failing wide introduction of the cap lamp the only course left is to increase up to three or more the candle power of the hand safety lamps - oil and electric. This is being steadily achieved, and a candle power of three has been obtained with combustion tube oil lamps, but when colliery managers have at great expense introduced any particular lamp they are loth to scrap their outfit when an improvement is made. The number of views being circulated on the subject of the causation of nystagmus, which tend to discredit the light
theory, hardly afford them any inducement to do so. However, it would be a thoroughly sound investment, especially for those collieries with a high incidence of the disease.

They must not be too sanguine that the effects will be discernible immediately, for the condition of nystagmus seems to be of slow development, and the benefits from improvement in the illumination will not be fully felt for some years. There have been cited many cases of remarkable reduction in the incidence of Nystagmus following improvement in illumination. Following introduction of electric lamps in one pit the nystagmus cases receiving compensation were reduced from seventeen to nil in four years. In another instance quoted by Mr Eustace Mitton the percentage in one colliery was reduced from 2.19% to .72%.

Other cases are quoted above in appraising the claims of the deficient illumination theory.

Frosting or tinting of the glasses might also be of appreciable value in reducing glare, if it does not involve an excessive absorption of light.

The miner will hardly need to be told to keep the direct light from the lamp out of his eyes as much as possible for the frequent alternation of light and dark adaptation is extremely trying, even to a normal person. The nystagmic finds it one of his greatest difficulties. The tinted glasses have produced good
results at Ebbw Vale, but if it involves too great a reduction of light it must not be over-done, for our ultimate aim must be to abolish nystagmus, not to make matters tolerable for nystagmics.

Another important point, as Llewellyn has insisted, is the care and upkeep of the lamps. All care given to lamps will be well repaid, and lampmen should be appointed because they are specially fitted for the task; not because they are not fitted for any other occupation. It is neither business nor commonsense to introduce expensive machinery, and then not make every effort to get the best out of it. A simple photometer should be fitted up in the lamp room and every lamp tested at regular intervals.

A great deal can be done for the comfort of the miner and the benefit of the owner by whitewashing the pit-bottom and the roads as far in-by as possible. In this way illumination may easily be increased three or fourfold. Light stone dusting may also be useful in this direction. The victims of Nystagmus most commonly find travelling to and from the working place the most trying part of their work, especially if the lamps are unshaded, and every miner should have a tin or cardboard shade to place behind his lamp, so that it may cause no inconvenience to those behind. I can testify from personal experience to the discomfort an unshaded light
can cause to a normal person. The nystagmic's power of adaptation to altered conditions of illumination is reduced, and he finds great difficulty in "getting his sight" when he goes down the mine or leaves it. We all know the temporary blindness that follows the approach of a car with bright headlights on a dark night. By improved illumination of the roadways the workman on a long journey may save five to ten minutes, and this multiplied by the large number of men will work out to several shifts in the course of a week. Any improvement in illumination of the roadways or the coal face will greatly improve the amenities of the mine, and the miner's environment is not so attractive that we need hesitate to improve it.

Rivers (Nystagmus Committee, First Report, 1922) says:-
"It is highly probable that the defect of illumination acts as a condition which enhances the psychoneurotic aspect of Nystagmus by increasing the element of danger, real or imaginary, so that in improving the illumination we shall be attacking the troubles which are now disabling so many miners, from two different sides." If low illumination enhances the tendency to apprehension and consequent repression, the improvement of the illumination should, in addition to reducing nystagmus in the strict sense, lead to a diminution in the number of those cases of psychoneurosis in which nystagmus is absent."
It might be mentioned in passing that improvement of illumination will result — and has indeed been proved to result — in greater output and fewer accidents. In the meantime the improvement seems to be going on but slowly and something more must be done.

If the conclusion reached above that compensation is often the determining factor in producing disability and a claim, and especially in prolonging already existing disability, is correct, some alteration should be made in the Workmen's Compensation Act as regards Nystagmus. Inasmuch as the neurotic cases themselves might be regarded as due to the nature of their employment it might seem hard not to allow them compensation, but it is an unfortunate fact that compensation, far from doing these cases good, does them harm both morally and materially. Without the complicating factor of compensation the sufferer would make an effort to work, and with the help of confidence acquired by increasing success would recover rapidly. Allow compensation and it is the factor that determines his "set" away from work.

In my view the widening of the definition in 1913 was a mistake, as it allowed compensation to purely psycho-neurotic cases. Percival has brought forward a suggestion, based on his view of hereditary predisposition, that compensation...
should be allowed for a definite period - he proposes one year - during which time the afflicted miner could learn a new trade. Those receiving compensation would be called upon to sign an undertaking never to work in a colliery again. In this way he thinks we should get rid of the susceptible material and prevent it from begetting more susceptible material into the colliery villages. I do not think this would be very successful for the neurotic type is, like the poor, always with us, and not a peculiarity to colliery areas. It has been shown that twenty five per cent of underground workers exhibit nystagmus on examination. These are all cases of potential disability, given conditions of enough stress. Without improvement of the illumination there will continue to be this quota, and a greater or smaller proportion of them will be disabled according to condition of employment, wages and the like.

Besides, the collier is a skilled worker, the product of many years' experience. Most of the men are getting on in years, and will not easily adapt themselves to other walks of life.

To judge from conditions on the continent it would seem that some definite nystagmus should be present before compensation is allowed. This would be tantamount to reversion to the old definition, and such an action would hardly find favour in political
circles. It would provide a good opportunity for ranting and raving in some quarters.

Probably the best plan would be to fix a period for the duration of compensation, as in Belgium. There a maximum of six months is allowed but 78% of cases are back at work in three months. This would at any rate prevent the protracted disability seen especially in some of the older cases. I am not lacking in sympathy for these men, but it seems unfair that the industry should have to bear the cost of compensation in such cases. The Act makes it very easy to establish a claim to compensation, but does not provide any convenient method of terminating a claim. Unless the miner voluntarily returns to work it involves arbitration before a county court judge.

This is slow and expensive, and the suggestion (Nystag. Committee, 2nd Report) of a medical administrative deserves support. Failing a time limit to compensation means should be established for reassessing periodically the capacity or incapacity of a case, on which assessment compensation should be based.

If some lump sum method of compensation could be used it would be a great advantage for it would remove the weekly or fortnightly stimulus to nervous disturbance and be for the good of the victim. Unfortunately it might provide too tempting a method of acquiring lump
sums, and repetition of a claim would have to be barred, but it would prevent the prolonged idleness we often see, and it has been seen that the economic problem of nystagmus lies as much in the protraction of disability as in the number of new cases certified. Meanwhile we have compensation with us and we must try to surmount the difficulties it creates as best we can by means of peaceful persuasion and suggestion.

Treatment.

A great deal in the treatment of Miners' Nystagmus depends on the medical adviser and on the treatment meted out to these patients by the colliery managers.

The doctor's first step must be to disabuse the man's mind of the idea that he may lose his sight or be unable to go down the pit again. He must himself show some degree of enthusiasm and confidence, and assure the nystagmic that he will certainly get better, and that nearly all cases make a rapid recovery. The patient may know of cases who have been idle for several years and this will not make for his ready acceptance of that assurance, but until something is done in the matter of compensation, we must try to get over this difficulty by assuring him that his case is "different." However if he has confidence in his
medical adviser and is assured of rapid recovery, a much more helpful frame of mind is created. He acquires some confidence himself.

There is some controversy as to how soon a man should be put on surface work. If the man complains of severe symptoms it is undoubtedly best to give him some weeks' rest, for he is quite convinced of the gravity of his condition and anything that might have the appearance of unsympathetic treatment will only annoy and anger him, which will tend to create a resistance on his part. If the case does not present such severe symptoms a short rest of two or three weeks only need be allowed.

After this rest of three to eight weeks the men should be encouraged and persuaded to start work on the surface - the sooner the better. Surface work is as important in the treatment as underground work in the production of the trouble. Almost any workman left idle too long sooner or later reaches a stage when he is unfit for employment of any kind. This is even more true of these cases. If they are left idle they have nothing to do but think of their symptoms, and when they adopt an attitude of self pity they are indeed in a bad way. Some of them attempt to drown their sorrows in alcohol with disastrous results.

It is in the provision of graduated surface work that the managers can do their share in the re-
habilitation of these fallen soldiers of industry. Some managers are apt to form, and even to express, the opinion that certain cases are malingering. This is doubly unfortunate. For in the first place it arouses annoyance and anger in the patient towards those whom he often holds responsible for his condition, and this can only have the effect of delaying his recovery. Secondly the man feels this is unjust and clings to his symptom to save himself from being despised both by himself and others.

If the work is graduated the patient will gradually acquire more and more confidence, and comparatively rapid recovery will ensue. In the same way graduated exercise was found the best treatment for D.A.H. cases in the war. The difficulty is that many managers are averse to providing work for these cases, many of whom, he will tell you, are more trouble than profit. Where however a conscientious effort has been made to provide appropriate surface work, it has been well repaid by the reduction in compensation costs. It would be an advantage if they could be allowed to work for part of a shift at first, and then this and the number of shifts could be gradually increased.

When cases have sufficiently recovered I see no reason why they should not be allowed to return to work under-
ground. It is true they may develop it again, but many cases continue underground work for years after an attack. When a case has been free from signs and symptoms for two or three months he should certainly be allowed to return to underground work.

With regard to actual drug treatment we cannot do much to attack the condition. The general health should be attended to, and the tonics such as nux vomica, quinine, hypophosphites and iron are often helpful, especially in those cases which have followed illness. Again, if a troublesome symptom such as sleeplessness can be relieved the patient gets encouragement and confidence in his physician, both of which are essential to recovery.

Errors of refraction should be corrected for this often relieves headache, and the patient feels more comfortable generally.

Alcohol should be prohibited, unless such prohibition will do more harm than good. At any rate temperance must be strictly observed. Smoking should also be restricted in heavy smokers.

When the patient returns underground he must be provided with a good light. Many cases from safety lamp pits do well by going to naked light pits.

A few cases will not respond to any treatment, and the best way to deal with these is by settlement of
the claim by a lump sum.

Cases of very old men, or of repeated failure are apparently not suited to their employment and should not be allowed to work underground. In the same way very young patients showing a relapse should be drafted out of the industry.

Let us hope that satisfactory illumination will soon be achieved and so do away with the necessity for compensation or treatment.

LIST OF CASES.

Case 1. E. T.  Age 35.

He has spent 15 years underground, some years being missed in the war. About 4 years ago he had nystagmus, the dancing of lights being the chief trouble. He left underground, and went straightway to surface work. After 9 months of this he resumed underground work until 6 months ago, with an electric lamp. He preferred this lamp to the oil lamp, especially one he obtained with frosted glass. Six months ago he was on the point of giving up when he cut two fingers, and after compensation for this ceased he resumed work on the surface.

At this time he complained of a great deal of giddiness and headache - "terrible" on some days -
across the brow. He could not endure walking behind a lamp, and had great difficulty in seeing "between the lights" (i.e. in the dull light of twilight and before dawn) and at night. Otherwise his sight was "excellent." His sleep was "not very good," and he had dreams of an unpleasant character though he remembered none of the details. His pulse was rapid, 96 per min. Rotary nystagmus on elevation of eyes was definite but not marked.

Now after 6 months on surface work there is no nystagmus even after exertion and confinement in the dark. His chief complaint is "specks before the eyes when walking and reading." Excessive stooping, ascent or descent of a number of steps, excitement or cross words with an official will still bring on headache, giddiness and dancing of lights. If he avoids these he is quite well. His vision is 6/6. His pulse is 80. All his coalgetting has been in a thin seam, two feet eight inches. He had to clear the clod at the top and as far I could gather from his description and poses his eyes or head would be in the position Snell thought so important.
Case 2. T.R. Age 62.

He has worked underground for 40 years. He was a coalcutter up to four years ago, and since then a repairer.

In 1923 his doctor noticed he had nystagmus and told him so. Since then he has suffered from giddiness on exertion, frontal headache and occasional jumping of lights. There was a tendency to fall in dull morning light.

He gave up coalgetting but continued underground with an electric lamp which he found much better than oil lamps.

He worked for two months after the great strike of 1926 and then in February 1927 developed influenza. He was "on the Health Insurance" for five weeks, since when he has been on compensation for nystagmus.

In February, with the symptoms mentioned above, he had nystagmus - rotatory oscillations of 180 per min. and a pulse rate of 100. There was no lid spasm and no head tremor.

In July 1927 he was recommended for surface work but none was provided.

Now in December 1927 he is still idle, although willing to work. No nystagmus can now be elicited but he still complains of giddiness and dancing of the lights on strenuous exertion. His pulse rate is 74.
He says he feels fairly well and would like to try work. His systolic blood pressure is 185 mm: and he shows definite arteriosclerosis.

Refraction:--

R and L +2 D Sphs.


He has been underground for 38 years, for 22 years as a fireman. He first noticed symptoms in the form of giddiness and dancing of lights about eight months before. Matters got worse and he gave up work in January 1928. He has always used oil lamps.

He found great difficulty in seeing in dim light, and could not read because the print was jumping. He also suffered from severe spasmodic headache just behind the eyes. He has recently suffered also from sleeplessness, and dreams almost every night. Details of these are not forthcoming but he remembers once dreaming of an explosion - it was at the time of the disaster in Cwm when a large number of men were killed - when "he woke up in a cold sweat."

His eyes showed no definite nystagmus, after repeated examinations, but his eyelids are constantly blinking.
There was no head tremor, but a very fine tremor of the hands.
His pulse rate was 120.
One month afterwards his sleep was much better and all his symptoms were better except that he still complained of giddiness on slight exertion. He resumed work on the surface in March 1928.

Refraction: \( R = \text{normal} \quad L = +0.50 \text{D Sph.}\)

He worked underground for 28 years.
In December 1925 he gave up work after noticing symptoms of headache, giddiness and dancing of lights for 10 months previously.
He showed definite rotatory nystagmus with a rate of 230 per min. There was no lid spasm, but a fine tremor of the head could be felt. His pulse rate was 110.
He was idle for 4 months, since when he has been on surface work. He said that the lights of an oncoming car "seemed to be drawing him so that he was afraid he would be run over."
Now he shows no signs whatever and complains of no
symptoms.

His vision is myopic.

Refraction :- 3.75 D, both eyes.

This case is, in my opinion, fit for underground work again.


He was employed as a rider underground, and had been underground for 12 years at the time of onset. He had noticed symptoms for 3 months. I did not see him at the time, but I have seen the notes of the Compensation doctor of the Company, and he classed him as a moderate case with nystagmus and typical symptoms. He had a slight cut head a day or two before giving up.

He gave up underground work in November 1923. He was idle only a few days, and then started work on the surface. He remained on surface work for about one year, after which he resumed his old work.

I examined him in January 1928, when he had been on his old work for over 3 years. He felt quite well and complained of no symptoms. There was no nystagmus. His vision is 6/6 with:

R£ 2.75 D.  Lt. 2.5 D.

He first had trouble with his eyes in 1920. The lights "danced and turned round," and he had frequent headaches. For many years he carried on, during which he had spells of insomnia lasting for a week or two.

In January 1927 he gave up work after 36 years of underground work. This was eight weeks following the resumption of work after the eight months strike of 1926.

He complained of headache, giddiness on exertion and movement of lights. He showed rotatory nystagmus, with a rate of 170 per minute. The knee jerks were increased.

His pulse rate was 100, and he had some tremor of his hands.

On attempting to close his eyes a marked intention tremor developed in the lids.

He was idle for 6 months, when his nystagmus was much less marked but his symptoms were if anything worse, probably resulting from alcoholic indulgence to which he was very prone. He was however persuaded to resume work on the surface. Four months later he was remarkably improved. His nystagmus had almost disappeared and he only complained of occasional headache and giddiness on much exertion. His pulse rate is 70 and there is no lid tremor. He always used an
oil lamp until 3 years ago. Since then he has used an electric lamp and found it much better. He has worked for the most part on thin seams. He found big veins more trying because of having to look up.


He has worked underground for 50 years. He first had trouble "with the lights dancing" three years ago - 1924. He suffered from headache - occipital in distribution, and felt as if his head were "blown up like a balloon." He found it very difficult to see in a dim light, and found bright sunlight very "hard on his eyes." In February 1927 about ten weeks following his resumption after the long strike in 1926 he found the dancing of objects so trying that he had to give up work. He then had marked nystagmus and head tremor. The nystagmus was coarse and the rate about 100 per minute. After a little exertion such as bending up and down ten times it persisted at the horizontal level. There was much blinking of eyelids but not a proper tremor. His pulse rate was 120. The knee-jerks were increased. This man had always worked with an oil lamp until about 15 months before giving up. He found
the electric lamp supplied in its stead much better to work with.
He was very distressed about his plight. He had what amounted to a definite phobia of losing his sight, and it would keep him awake at nights.
One year after he is still idle. His nystagmus has almost disappeared but he still has a mournful tale to tell about his symptoms. If he is not persuaded to do some work soon he will go beyond recall.
He has an error of refraction.

\[
\begin{align*}
\text{R.} &= -1 \text{ D Sph.} & \text{L} &= -1.50 \text{ D Sph.} \\
& \quad -0.50 \text{ D. Cyl. Horizon.} & \quad -0.50 \text{ D. Cyl. Horiz.}
\end{align*}
\]

It is interesting to note that the above two cases along with two others whom I could not see all gave up work within two weeks. One would certainly expect them to be better after the rest of the eight months strike. It is another support for the view that idleness is not good for these cases. It almost seems as if they had lost their taste for work, or had their distaste for it accentuated by the strike period.
One of them was notoriously a bad workman.

He has worked underground practically all his life since he was 16 years of age. He has worked a great deal in thin seams 2 ft. 6 inches -

He suffered from giddiness and dancing of lights for a year before he gave up. The dancing of the lights was his chief trouble. He tried an electric lamp but found it no better than the oil lamp he had always used.

He could not see in the twilight or the dark and alleged that he nearly went into a river once in the dark. On inquiry it turned out that he had stumbled and fallen into a gutter about 30 yards, from the stream. However he had a fright and gave up work one week afterwards in August 1925.

One month later he had clonic spasm of the eyelids, with tremor of the head and hands. His nystagmus was the typical rotatory form at a rate of 190 per minute. His pulse rate was 120. He has been idle ever since. "At first he could hardly sleep at all owing to headaches."

Now in January 1928 his headaches are much better but he has exchanged them for a feeling as if "a band were round his head." He is subject to fits of depression, during which he eats as heartily as ever. He says he feels giddy if he rises suddenly, though I
found him gardening when I called to see him. There is no nystagmus now, but some tremor of the hands and head is still seen after exertion and excitement. His pulse rate is 98. His vision is presbyopic.

\[ R \& L. = +0.50 \text{ D Sphs.} \]

I am quite sure this case is nothing more or less than a compensation neurosis at present. If there were no compensation he would have been supporting himself long ago.


He had worked underground for 13 years when he first had trouble. This was in September 1914. He was idle for 2 years. He went back to work for 6 weeks but gave up again for another year. He then worked on the surface till 1919 when he returned underground.

In November 1923 he complained once more of giddiness, headache and dancing of lights and returned to surface work. He remained at this until March 1927. Then he said his symptoms got worse. His sleep too was often "very little." "He did not dream but just could not sleep." When I saw him for the first time at the end of March he was complaining of all the above symptoms,
but I could elicit no trace of nystagmus. His pulse rate was 80.
In December 1927 he was still idle. His symptoms were somewhat better. Giddiness was still complained of, and to a less extent difficulty in seeing in twilight. His pulse rate was 80.
His sight has not been tested since 1914 but he assures me that he "has excellent sight for reading and for distance."
He always worked with an oil lamp.
This patient is one of whom I am somewhat suspicious. I cannot help wondering, if his war-time incapacity had any connection with a possible call for service.
He has had absolutely nothing to show for his symptoms since I have seen him. He strikes me as being the type for whom lump sum settlement would be a specific.
The next case is somewhat like him:

Case 10. W. S.    Age 38.    Labourer (Underground)
He had been a collier previously for 12 years.
Three years ago he had to give up his work as a collier and after several months idleness he resumed work as an underground labourer. I have no details of this attack but he received compensation for nystagmus.
This was in 1924. He returned underground in 1925.
In April 1927 he again complained that the lights
moved from place to place - he makes out that they moved up to a distance of one foot - and that there were "splashes of light" from the lamp.

He felt a pain behind his eyeballs "as if his eyeballs were being squeezed." He complained of frequent headaches and giddiness on exertion. Examination revealed twitching of the eyelids but no nystagmus. His pulse rate was 125. There was no tremor apart from that of the eyelids.

After about 6 weeks' idleness he was persuaded to resume work.

At present he is still doing very light work on the surface.

When I saw him last he still complained of his symptoms although they were much better. The lid movements have ceased. His pulse rate is 100. He is still very worried over his eyes.

He thinks it is due to the electric lamp he was given about 4 years ago. "It dazzles too much," he says. Previously he had used nothing but oil lamps. He has an error of - 2.25 D in both eyes:

This patient is very well-known to me. He literally haunts our surgery. If he has a slight cough he comes to see if he is developing tuberculosis. A transient dyspepsia engenders a fear of organic disease of the stomach. He is hypochondriac in his expectation of illness. He seems to be suffering not from nystagmus,
but from a fear of nystagmus or blindness.

Case 11. J. R. Age 42. A Fireman.

This patient had worked underground for 25 years, always with an oil lamp.

For some reason he was dismissed. He had never had any symptoms before but promptly put in a claim for compensation. Nystagmus was present, and he received compensation for 2 months. Then nothing further was heard of him until it was discovered that he had obtained employment at another colliery. When I saw him about four months after his dismissal - he still had a fine rotatory nystagmus with a rate of over 250 per minute. He had no blephasospasm and no other signs or symptoms.

His vision he said was good. I was unable to test this. This is the type of case that makes one realize that the incapacity in Miners' Nystagmus must in some cases at least have no direct connection whatever with the presence of nystagmus. The next case drives this realisation home.

If this case had been unable to find employment for any length of time he would no doubt have continued to receive compensation for a very long time, for twelve to eighteen months of idleness would soon have created a distaste for work, and he would no doubt have deve-
loped the swarm of symptoms we see in many long standing cases.


This man has worked underground since he was 15 years old. He is an interesting case. He was discovered accidentally by a colleague, and I saw him in December 1925. Then he had most marked nystagmus. It was fairly coarse with a rate of 150 per minute. It was so marked that a friend had said that his eyes were jumping out of his head." He had known of his condition for five years. He noticed his vision was somewhat "blurred" and the lights were "not quite steady." He did not know he had nystagmus until he was told so five years ago. He has been recommended by his medical attendant to give up work, but he prefers to carry on as he earns quite a good living.

If he were one of those people on the lookout for symptoms he would no doubt have put in a claim for compensation long ago. He thinks compensation is too readily made use of, and seems to think it beneath him to claim it while he can work without any discomfort. Yet if circumstances arose which made the lure of compensation greater, he might succumb.

A careful examination has failed to reveal any other possible cause for his nystagmus.

He had been employed underground for 17 years. He always used an oil lamp.

This case supplies an answer to the case of Robson's mentioned in the text.

A fellow workman had been "gassed." He himself was not exposed to the gas, for he was at a safe distance.

The next day Nystagmus came on with acute onset.

He never had symptoms before but that morning on his way to work he could not see and returned home.

He complained of headache, giddiness on the slightest exertion and a "bursting sensation" in his head.

Everything, he said, was going round and round.

His pulse rate was 130. His knee jerks were very brisk. He had been unable to sleep the night before.

There was blepharospasm with tremor of the head and arms. There was a fine rapid rotatory nystagmus on looking up. I saw him four days after the accident for the first time, and his condition was exactly the same.

He was given three months complete rest and then started work on the surface. He was working on the surface four months after and felt much better.

I was unable to follow this case more closely owing to the distance at which he lived. If the patient had been gassed himself he would have provided a very good case for the advocates of the "gas theory."

This presents no unusual features. He had worked underground for 20 years, and had noticed symptoms for eight to ten months before he gave up work. His chief trouble was the dancing of the lamps as he made his way to work, and a liability to "dizzy attacks" when at work. Other symptoms were headache and a dislike for bright sunlight. He had a regular rotatory nystagmus with a rate of over 200 per minute, tremor of the eyelids and a fine head tremor. His pulse rate was 110.

He had always used an oil lamp.

He was given a complete rest for six weeks and in May 1927 resumed work on the surface. Nystagmus was still present but he said his symptoms were much better. In December 1927 his pulse rate was 78, there was no nystagmus, and except for occasional headache and giddiness his symptoms had disappeared.

He is still on surface work and in another four to six months he might be sent underground again.


This is an interesting case in that the onset immediately followed a head injury. He had been employed underground for 15 years, always using an oil lamp.

In November 1927 he received a scalp wound about two
inches long. It was more of a scratch than a cut, the depth being very shallow. He had never had any symptoms of nystagmus previously. The next day he went to work and noticed the lights dancing. He felt a feeling of intense giddiness and left the mine. He soon complained of all the usual symptoms. On examination he exhibited blephosospasm with head tremor, and a very fine and rapid movement of the eyeballs. The rate I could not count. His pulse rate was 126.

He was off work for two months at the end of which time his pulse had returned to a rate of 82, his nystagmus and head tremor had disappeared and the blephosospasm proper was no longer in evidence, though there was some blinking. He still said the lights danced at night and complained of some headache at times. He doubted if he could manage surface work but was persuaded to make an effort in the early part of January 1928. Two months later he said he felt much better.

His refractive condition is:

\[
\begin{align*}
\text{Rt.} & : + -0.0 & \text{Lt.} & : + -0.0 \\
-2.25 \text{D} & & -2.25 \text{D}
\end{align*}
\]
Case 16.  A. J.  Age 35.  A Labourer  
(Underground)

This man first noticed the dancing of the lights in November 1925.
He continued at work until March 1926. He had used oil lamps for fourteen years, and electric lamps for the last two years of his underground life. He had a rotatory nystagmus with a rate of 210 per minute. There was no head tremor and no true blephasospasm, but he frequently blinked his eyelids. His pulse rate was 94, and he complained of dancing lights, headache, giddiness and failing vision in dim light. Soon after he ceased work came the long strike of 1926. In December 1926 he resumed work on the surface and in January 1927 still complained of most of his symptoms, though they were not so troublesome. His nystagmus had disappeared. His pulse rate was 90 and he complained of extreme weakness. Examination revealed that he was suffering from mitral incompetence with some degree of dilatation. He was idle for another month during which he received compensation for nystagmus. Then he resumed work on the surface.
Five days later he developed oedema of the legs and was ordered to bed. He died suddenly while going upstairs.
Case 17.  R. P.  Age 42.  A Collier.

This case was discovered accidentally. He made no complaints about his eyes, and no exhaustive examination was made for fear of the effect it might have by attracting his notice to his eyes. The cause of his visit was a foreign body - a minute piece of coal - embedded in his cornea. During extraction nystagmus was observed, and confirmed by getting him to look up repeatedly while I was supposed to be in search of the foreign body.

The nystagmus was a fine rapid rotatory movement.

His eyes were not closely examined but a few guarded questions revealed the fact he was quite unconscious of any ocular or other discomfort apart from that caused by the foreign body.

This patient had worked underground for over 25 years, always using an oil lamp.

He was seen in February 1928 so there has been no time in which developments might occur.

This patient had worked underground for 16 years and had always used an oil lamp.

He first noticed the lamps dancing in May 1927.

The other symptoms giddiness, headache and tremor of the eyelids gradually developed, and in January 1928 he was compelled to give up work. He had a fine tremor of the head and his pulse rate was 92.

The only interesting feature of this case was that he is the only one I have seen who presented a difference between the two eyes. His right eye presented a fine rapid rotatory nystagmus; but the left eye exhibited slower and coarser movements, and there was more of a vertical excursion in it.

It is difficult to account for this difference unless it is connected with some defect in the musculature of one eye.

At the end of two months he was still idle. The difference was still observable though the nystagmus was much less marked.

His eyes had been tested over a year ago and there was a difference between the two eyes but hardly sufficient to account for the variation in the nystagmus.

The lenses he was then fitted with were:

Rt. -2 + -0.5
Lt. -2.75 + -0.5.
This and case 20 are recent and of interest.
This man was discharged with a number of other men five weeks ago, early in February. Within a week he made a claim for nystagmus. I could not convince myself that there was a definite nystagmus though there was an unsteadiness of the eyes. He complained of all the symptoms; headache, giddiness, dancing lights and bad vision in dim lights.
There is no lid spasm or head tremor. His pulse rate was 88.
He says his eyes have troubled him for five or six months.
He has been working underground for 26 years.

This case worked underground for five or six years in his youth. Since then he had not worked in a mine until four years ago. His history is exactly the same as that of case 19. He was dismissed about the same time and has recently made a claim for nystagmus.
He complains of all the usual symptoms but I cannot find a trace of nystagmus. There is no lid tremor but frequent blinking of the lids. His pulse rate is 94.
I am quite sure that had these two cases been retained they would have continued to work without any thought of nystagmus.
Summary.

The increase in the number of certified cases of Miners' Nystagmus and the increase in the duration of disability in a large number of cases have made the condition a question of economic importance. The essential factor in the production of the condition appears beyond doubt to be insufficient illumination. Some improvement has been made in the safety lamps both oil and electric in recent years, but the lighting of most mines is still far from satisfactory. The best method of effecting improvement is the introduction of the electric cap lamp as recommended by the Miners' Nystagmus committee. This it is hoped would soon result in the prevention of Miners' Nystagmus. Failing this the candle power of the hand safety lamps must be increased to at least 3 or 4 c.p.

Minor improvements can be effected by white-washing the roadways as far as possible and by providing lamp shields for the journey to the working place.

The factor of compensation is responsible for a great deal of the increase in the number of new claims and explains the increased duration of incapacity. Incapacity is, in most cases at least, not due to the actual nystagmus but to neurotic states developing
either alone or superimposed upon the nystagmus; and it is this aspect of the disease upon which compensation has had so great an influence.

Some alteration in compensation regulations is called for. The duration of compensation may be limited to a definite period, or the rate may be reduced after a certain period. Failing this there should be some machinery for periodic assessment of the capacity or incapacity of cases by medical men with special experience of the disease.

In the matter of treatment the important point is to impress the patient that his condition is a benign one, and to persuade him to resume work of some sort at the earliest possible date. Prolonged idleness is the worst possible treatment for these cases.

Sympathetic action on the part of the management in providing suitable surface work will be well repaid by results.

Errors of refraction should be corrected, and any distressing symptoms such as insomnia should receive suitable therapy.
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