MINERS

NYSTAGMUS.

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MINERS' NYSTAGMUS.

SCHEME OF THESIS.

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MINERS' NYSTAGMUS.

INTRODUCTION.

For the last few years I have been in practice in West Cumberland and closely associated with Miners working in a small seamed coal pit belonging to the Allerdale Coal Company, at Buckhill, Broughton Moor, by Maryport. During this period I have had occasion to examine and treat patients suffering from Miners Nystagmus, as a practitioner, and quite apart from any examination in regard to workmens' compensation.

My interest was first aroused by the complexity of the disease, and the different reactions of the condition on the patients' nervous system. Furthermore, it appealed to me, as it has done to others, as a condition of great national importance; and, as the etiology is still obscure, and the treatment of great importance, the subject presented itself to me as one worthy of more consideration.

As has been inferred, this is an occupational disease, and one of considerable frequency, almost entirely confined to the coal

mining industry, although cases are not unknown in the Ironstone Mines, where safety lamps are used. Cases occuring in other occupations have been described, the most outstanding being that of Nystagmus in a compositor, described by Snell (1) in 1890. Two years ago (1925) I had a case occuring in a fireman employed locally, and the history of and the conclusions derived from this case will be discussed later.

The disease when manifest produces

definite physical signs, the most characteristic

of which are oscillation of the eyeballs, and

involuntary twitching of the eyelids, (that is,

blepharospasm). In addition to these signs,

nodding of the head may be present, and the

patient may suffer from extreme nervous symptoms.

The disease may be latent, only to appear with

all its characteristics after an accident or

severe strain.

The ultimate effect of the disease is to prevent the miner from fixing the eyes accurately on a given object, thus materially reducing his usefulness as a workman.

That this disease, so marked in a proportion of our working classes, should come to be placed on the list of industrial diseases.

was not a surprise, and May 22nd 1907 saw the condition defined as "Nystagmus - process mining" added to the Schedule of diseases of the workmens' compensation act of 1906.

Prior to this date no accurate information on the condition was available, although many articles had been written on the subject. Since this date the number of cases receiving compensation has been on the increase, with a somewhat alarming rise during the period 1913 - 14, when the Schedule was altered from the previous definition to "The Disease known as Miners' Nystagmus, whether occurring in miners or others, and whether the symptoms of oscillation of the eyeball be present or not".

In the present thesis I intend to discuss the condition from the standpoint of recent developments, basing the work on my own observations, and on the study of the works of various authors on the subject.

Many theories have been postulated as being the sole etiological factors in this condition, but I feel convinced that we are dealing with a disease as complex in its origin as it is in its development; and I hope to correlate those factors which to my mind have a very definite part in producing this distressing malady.

My

observations have not been purely surface examinations, as I have descended the Buckhill Colliery and others, and made myself thoroughly conversant with the conditions under which the Miner works, so that I might be able to judge the more carefully and thus form my own conclusions.

5.

HISTORICAL OUTLINE.

Prior to the introduction of the
Davey Safety lamp into the coal mining industry
generally, in the year 1850, there were no
recorded cases of Miners' Nystagmus; but in the
year 1861 Decondé (2) in a paper, dealing with
nystagmus as a whole, and published in the Archives
of the Belgian Academy of Medicine, described
cases occuring in coal miners. Snell (1)
however, in his book claimed that a Dr. Gillett
of Sheffield was quite conversant with the
condition as far back as 1854, and Nieden (3)
claimed that Peppnuller described cases during
the period 1860 - 63.

In 1875 C. Bell Taylor (4) published a paper in the "Lancet" entitled "Miners' Nystagmus - a new disease" considering it to be analagous to writer's cramp, and he thought that overburdening of the eye muscles and sustained efforts to see in a deficient light were the fundamental causes.

Later, in the "British Medical Journal" (5) he says, "There is no central lesion, the affection is a pure myopathy."

In this country our first and foremost investigator was Dr. Simeon Snell of Sheffield,

and he treated the subject in a very thorough and systematic manner, and called attention in 1875 (6) to the position assumed by the "holer" at work - namely a strained posture with the eyes directed obliquely upwards; and he laid great stress on its importance as an etiological factor. This view was generally accepted and considered an adequate explanation of the condition. is, we have fatigue as the etiological factor irrespective of the mode of illumination. this period onwards Snell's various publications (7) led to much greater knowledge of the disease and no doubt stimulated other investigators at home and on the continent. Amongst the most notable of the latter may be quoted Dransart (France), Nieden (Germany), and Romiée (Belgium).

Dransart (8) published his first paper in 1877, and stated that nystagmus was due to a myopathy of the elevators of the eye: and, after calling attention to the works of Nieden, Gräfe and Nuel (1874), he quotes Arlt (1867), who presumed that the movement of the eyes in nystagmus was an attempt to bring a fresh part of the retina to bear on the same point as an aid to vision. Later Dransart (9) states that a large percentage of cases are anaemic

and that refractive errors have nothing to do with nystagmus, and still later in 1891 (10) he agreed with Snell in regard to posture being the prime cause; and, in a communication with Famechon, he lays the greatest stress on fatigue as the essential cause.

Nieden (11), whose book published in 1894 is the standard in Germany, found the disease in those workers who were compelled to work in a strained position with the eyes directed upwards, but he held the cause to be, in the first place, lack of light, and likened the condition to that of tremors, so often seen in the old as a result of fatigue. He summarises the causes as follows:- lack of light; defective vision; insufficiency of the interni with a corresponding weakness of the associated movements of the eyes; the general ill effects of the work performed.

The question of poverty of illumination being the prime factor in causing Miners'

Nystagmus, soon began to be further advanced, and in 1878 Romiée of Belgium and Court of this country in 1891 made some publications dealing with this point of view. Romiée (12) 1892 advanced a theory that excessive accommodation for a long time in a bad light was the cause, and he again stated that the safety lamp was

recommended for use in the Belgian mines in 1851, while in 1876 the Mueseler lamp was insisted upon, and in the two years following this he noticed an increase in the number of cases. He also states that our forefathers were every bit as good observers as we are, and that had the disease existed prior to the use of the safety lamp, they would have noticed it, and he quotes figures to prove that as illumination improved, the percentage of cases fell. He quotes Lewuillon of Mons (13):- Percentage of cases with Mueseler lamp 38,

Percentage of cases with Electric lamp 19, and concludes his communication in the following words (14): - "L'éclairage insuffisant est la cause unique de la production du nystagmus des houilleurs"

This then gave rise to the question of the comparative influence between naked and safety lamps, (the naked lamp being the strongest luminant); and Court (15), Britain, in a report to the Derbyshire Miners' Association 1891 and in the "British Medical Journal" (16) 1892 lays the greatest stress on the illumination, and says that when open torches of two and a half candle power are used there is no nystagmus, and that in a systematic examination of one thousand one hundred and sixty nine colliers he found that

of five hundred and ninety seven men with safety lamps, two hundred and seven had nystagmus, and of five hundred and seventy two with naked lamps only thirty two had nystagmus, (of these thirty two, twenty nine had previously worked with safety lamps).

This view being in direct opposition to the strained posture theory of Snell led to much discussion, but the trend of opinion favoured that of Snell.

Jeaffreson (17) 1887 propounded his view, which is really only an elaboration of Snell's posture theory. He believed that the disease was a general one with nystagmus as one of a group of symptoms, and suggested the name of "Miners' Neurosis". He thought that it was caused by fatigue, which follows the disassociation of movements normally combined, for example flexion of the head with elevation of the eyes, that is, walking in the pit, the head bent and the eyes up to avoid hitting the roof. Normally the head and eyes should go up together.

This view was upheld by Rutten (18) and he stated that the movements of the eyes are involuntary, inimitable, and rhythmic, not choreic. He says "Il doit exécuter constamment des mouvements de la tête, tantôt l'inclinant sur le

côté, tantôt la rejetant en arrière, ou bien il doit combiner ensemble ces deua mouvements".

This view is termed "Gegenrollung" by the Germans.

Mlle. Toteyko (19) thinks that the study of the tics and occupational cramps throws a clear light on the pathology of Miners' Nystagmus.

She agrees with Dr. Rutten, and thinks the mental attitude should also be taken into consideration. She regards the disease as a neurosis and summarises her views as follows:- (a) attitude at work, (b) unbreathable air, (l'air irrespirable des mines), (c) absence of sunlight, (d) absorption of gases from the coal.

J. Tatham Thompson (20) says the disease is rare in naked light pits.

In the year 1906 the equilibration disturbance hypothesis made its appearance. A. Christie Reid (21) attributes the condition to imperfect fixation due to a dim light, and frequent disturbance of vision. All the muscles are involved, as there ought to be hyperphoria present if the elevators alone are affected. Fatigue alone is not sufficient.

Harrison Butler (22) points out that a fatigued muscle gives a tetanic curve and not a clonic one and lays stress on the imperfect fixation, on the crystalline surface of fresh coal impairing binocular vision.

May 22nd 1907 was a memorable day in the history of the coal mining industry, as the disease was then included among the scheduled industrial diseases of the Workmens' Compensation Act 1906. Further prominence was given to the subject when in 1913 the definition of the disease under the 1906 Act was altered by the Secretary of State from the original description. (vide introduction).

In 1908 Nuel (23) maintained that the chief factors in the production of the disease are the darkness of the mine and the crystalline fracture of the coal, with prolonged elevation of the eyes upwards; and that one factor alone is not sufficient; and he quotes cases of nystagmus in polishers of black marble, who always worked looking upwards.

Another view put forward in regard to the pathology of the condition was that of Peters (24), who wrote a paper entitled "Is Nystagmus of Labyrinthine origin", and lays stress on the backward tilt of the head, and queries whether this is a primary or an acquired position. Ewald and Ach oppose this view, and point out that in torticollis, nystagmus does not follow, although the labyrinth has an influence on muscular tone.

Labyrinthine irritation was assumed by Trombetta (25) in the year 1907 to be the cause, and he attributed this to be brought about by frequent and rapid changes in the atmospheric pressure and to the incessant blows of the pick and to shot-firing.

The one object of Snell's work was to discount the influence of feeble illumination in the causation of the affection, and his theory held its ground up to about 1900, when a fresh generation of investigators and workers seemed to have reversed the current opinion; and the view that the chief factor in the etiology of the disease was a deficient illumination present in the mine, so stoutly defended by Romiee and Court, gained prominence and acceptance; so much so that the Opthalmological Congress in 1912 passed a resolution endorsing this view During this period the unanimously. investigators were numerous; the most outstanding being Llewellyn, Stassen, and Ohm.

Elewellyn (26) was the first Tyndall Research Student appointed in 1910, and he proved (by actual photometric measurements) the amount of light falling on the coal face, and found that the miners work in very deficient illumination.

In 1912 Coppez (27) made a study of

the tracing of nystagmic movements, and came to the conclusion that nystagmus was due to an incomplete tetanus, the result of fatigue, and that all the muscles were affected.

Dransart (28) returned to the subject in 1913, and while still holding that the prime cause lay in a direction of the gaze, he thought that deficient light was a secondary cause, and that, with improved illumination, the primary cause would disappear.

Wilson (1913 - 15) (29) held that the feature common both to miners' nystagmus and nystagmus from other causes was imperfection of the retinal images. Shufflebottom (30), in his Milroy Lectures 1914, considered the disease to be due to deficient light, cramped position, refractive errors and ocular injuries while at work.

"That fatigue in the execution of ocular movements is only the clinical manifestation of miners' nystagmus" has been put forward by Stassen (31). He lays great stress on the physical conditions of the work producing a nervous syndrome characterised by inco-ordination and exaggeration of the visual reflexes. After treating the subject from every point of view he gave his final opinion in these words (32):- "La cause nécessaire

et suffisante des troubles visuels des ouvriers mineurs est le travail prolongé dans de mauvaises conditions d'éclairage! Again (33) "Le fait est donc certain: les troubles visuels des mineurs relèvent de la physiologie de la fatigue, dans de mauvaises conditions d'éclairage".

Ohm of Westphalia (34) thought that miners' nystagmus was a disorder of tone produced by the labyrinth in different muscle groups, and stated that lack of light and unfavourable conditions at work were the occasioning factors of the disorder and that these gave rise to too strong and too infrequent innervation of the labyrinth (35), thus giving rise to a disorder of muscle tone. Contending that the disease varies directly with the amount of labyrinthine disturbance, and inversely with the illumination present. He believed that a pit lamp giving two to three candle power under favourable conditions would diminish the number of cases (36).

In 1920 Martin (37) thinking that there was a similarity between cases of nystagmus and anxiety neuroses of the warstrained, put forward the theory that the disease was essentially one of exhaustion.

Anderson also 1920 (38) held that

astigmatism was the most frequent cause of the condition, and he suggested examination of all men before employment.

Upon the recommendation of the Miners' Light Committee, the Medical Research Council appointed a Miners' Nystagmus Committee in 1920 (39). The result of their deliberations was the cry for more light!

Dr. F. Robson of Penarth (40) in

South Wales contended that the absorption of mine gases, carbon monoxide, or some symbiotic combination of gases was the cause of miners' nystagmus. He also sought to prove that the incidence of the condition bears some ratio to the nature of coal worked. Various kinds of coal show differences in the percentage of volatile contents; therefore, according to Dr. Robson, the higher the volatile content of the coal worked in an area, the greater will be the incidence of nystagmus in that area.

This gas poisoning theory was enunciated by Pechdo (41) in 1893 and supported by Harrison Butler at the Oxford Opthalmological Congress in 1912, by Coulter (42) in 1914, and by Leighton Davies in 1920 (43).

It is interesting to note that the strained position theory of Snell has given way

to the deficient light theory; and Drs. J. S. Haldane, and T. L. Llewellyn, at a meeting of the Institution of Mining Engineers (55), combated certain criticisms made against this most widely accepted conclusion (44), "That defective illumination is the essential cause of miners' nystagmus". This has however been called in question by Dr. Freeland Fergus of Glasgow and by Dr. Robson of Penarth. The latter (45) still holds the opinion that volatile or gaseous substances are the cause, and states that oneeyed men get nystagmus! Dr. Fergus (46) on the other hand suspects infection, from a toxin or a microorganism, and states that the very fact that there are so many new recorded cases in one year differing from another in so marked a degree, indicates that the disease is not due to lack of illumination; for, had this been so, the number of cases each year would be more or less constant. He also states that the disease seems to appear in epidemic form. He also draws attention to the fact that there is general disturbance of the innervation of the heart, that invariably hemeralopia, and nyotalopia are present in association with miners' nystagmus. He concludes "I am agnostic as regards the origin of miners' nystagmus, except that I feel confident that it

has not been proved to be due to defective illumination".

Edgar L. Collins (47), a Member of the Miners' Nystagmus Committee, gives his observations obtained in the United States of America, and says "There was no need for waiting on leaving the cage to 'get your eyes'; the walls of the working were white, and reflected the light from the lamps, stone dusting being always done with limestone or gypsum. The electric safety lamps used gave more light than ours, and were different in shape, being worn in the cap with the battery attached to the waist. No further reason need be sought for the entire absence of nystagmus amongst American coal miners!

This brings us to the conclusion of the historical outline, in which I have mentioned each author's views, as we advance from the previously considered myopathic theory, to the present day theories of gas poisoning, general neurosis, or general fatigue of the whole oculomotor system; or, in other words, the views on the nature of the disease in a state amounting to chaos. We will now turn our attention to the causation and pathology, in which section I will discuss some of the theories propounded and attempt to correlate my findings.

ETIOLOGY.

Miners' nystagmus, (although I see no reason why it should not be classified with the ordinary varieties of nystagmus), is quite different from other varieties of nystagmus in that it occurs among men employed in an occupation involving ocular strain in a dim light. Not only do they work in a dim light under strenuous conditions, but they are a class of the community prone to inbreeding, over-crowding in dwellings, and, in West Cumberland, to bad wages; factors all of which lead to lowered vitality and poor bodily resistance.

Fuchs (48) in his text book on pphthalmology classifies the etiological factors of nystagmus as follows:-

(a) Conditions producing bi-lateral amblyopia in early infancy. (i.e. visual or so called congenital nystagmus). Such conditions are opacities of the cornea, especially from ophthalmia neonatorum; congenital cataract haemorrhages; disease of, or abnormalities of the retina and chorioid; total congenital colour blindness, and albinism. Refractive errors, even when great, he says do not

usually cause nystagmus, although they may do so.

- visual difficulties, such as are usually incidental to the patient's occupation (occupational nystagmus), the commonest form of which is miners' nystagmus, which seems to be mainly due to insufficient illumination causing reduction in light sense and foveal insensitivity (49). Nystagmus due to insufficiency of illumination occurs in other occupations (Frost); and a twitching (probably not a true nystagmus), which Snell ascribed to strained position of the eyes, occurs in compositors.
- (c) Irritation arising from the labyrinth,

 (vestibular nystagmus). This may be

 produced by disease of the labyrinth and its

 connections; or artificially by changes in

 the labyrinthine circulation and pressure due

 to rapid rotation of the body; syringing the

 ear with hot or cold water or galvanization

 of the ear rotation, caloric (or thermic),

 and galvanic nystagmus.
- (d) Nervous Diseases :-

Cerebellar abscess.
Tumour at Cerebello - pontine angle.
Tumour of Cerebellum.
Multiple sclerosis (12% Uhthoff).

Hereditary ataxia (usually denoting affection of fibres going to Deiter's nucleus).
(Wilbrand and Saenger).
Encephalitis (10% Uhthoff), especially Encephalitis Lethargica.
Syringomyelia.
Little's Disease (16%).
Idiocy (28%).
Other nervous diseases rare.

(e) Poisons:-

Ether; Chloroform; Alcohol; Morphine; Cocaine; Sulphonal; Veronal; Arsenic; Lead; Quinine; Ergot; Sewer Gas; Mustard Gas; etc.
Botulism.

Nystagmus is a condition in which there is on conjugate movement of the eyes, poorly sustained ocular deviation, and is affected by a succession of slow or quick, course or fine, rotary or oscillatory movements of varying range. It may be divided into three varieties. (1) Searching movements. (2) Pseudo-nystagmus. (3) Nystagmus proper.

- 1. Wide, purposeful, and slow movements of the eye in all directions usually seen in people who are born blind or have lost the power of fixation. The eyes never rest on any definite object.
- 2. Rapid, jerky movements of the eyes when they are carried to an extremity of an excursion in any direction. The eyes do not remain fixed but recede and return to their position at the rate of four or five oscillations per second.
- 3. Nystagmus proper, in which the eyes make

regular rapid oscillations, about a fixed point, vertical, horizontal, or rotary, not only when the eyes are at the extremity of an excursion but when otherwise at rest. This is usually bilateral, but can occur in one-eyed men.

Nystagmus whether visual or due to nervous disease is sometimes hereditary.

Some persons can produce it voluntarily.

(Case) No. T.8.

Snell, Nieden, and Dransart all bring evidence forward pointing out the strained position maintained by the holer at work, with the eyes directed obliquely upwards, and agree that the disease is a local myopathy affecting the elevator muscles of the eye. strained position is the case in nearly all the West Cumberland pits, and especially so at Buckhill Colliery where most of my observations were made. There the average seam is about two feet thick, but I contend that nystagmus is not confined to the coal hewer alone as is evidenced from the Statistics appended hereto, (Vide p. 52) where it will be noticed however that out of four hundred and sixty three cases 81.4% were hewers, 7.775% shift men, 1.725% deputies, .866% pony drivers, and that nearly every worker

in the pit is liable to the disease. The hewer is undoubtedly the man chiefly affected, but then he is no doubt the hardest worked and more liable to be continually in small areas where the ventilation is bad compared with his fellow workmen. I do not think the strained position theory is sufficient to explain the condition, because my experience is that the man at his work does not look upwards, except occasionally, and the nystagmus, which is manifest in the eye, is accompanied by other symptoms such as rapid pulse and tremors in the muscles in other parts of the body; and, as I have already said, all workers in the pit are liable to the disease. Furthermore, in testing cases for the presence of nystagmus, it was not always possible to elicit it by means of asking the man to look at an object with the eyes in an oblique and elevated position. Several of my cases with well marked nystagmus could be best demonstrated by asking them to stoop suddenly and touch their toes. This was usually sufficient to elicit the nystagmus and some cases actually fell to the floor on stooping. Metalliferous mines (50) have no nystagmus although they work in a strained position.

Romiée, Court and Thompson agree as to the condition being due to strain as a result of

working in a deficient light, and Romiée and Court give figures of the comparitive influence of naked and safety lamps. The naked lamps are the stronger, while the safety lamp pits have the greater number of cases of nystagmus, leading these authorities to discard the posture theory in favour of defective illumination. The lamps now used in my district are electric and even yet the miner works in a defective light. However, most of my cases have in their time used the oil safety lamp and some have used candles, but, if they are questioned, the usual reply is "It's them oil lamps as does it"! (That is the disability, in their opinion, was not due to defective light, but to the fumes which were given off from their lamps). No one can dispute the statement that the miner does work in a defective light, from the statistics given by Romiée and Court (12,13,14,15), and from the excellent book published by T. L. Llewellyn on Miners' Nystagmus (51), (in which he gives actual photometric measurements of the amount of light falling on the coal face); and furthermore, he points out that, owing to coal dust, the lamp becomes deficient as the man's shift nears an end; and his experiments (52) also proved that the illuminating power of the lamp fell off

rapidly as the oxygen percentage in the atmosphere diminished. An important point, as, if the diminished oxygen is sufficient to effect a lamp. it must have some effect upon the human system either directly or indirectly, immediate or cumulative. I thoroughly agree that illumination is a prime and important factor, and that strain either general or of the ocular muscles also plays a part, but I am not yet satisfied that these conditions alone are sufficient to produce all the symptoms met with. If it was simply a case of long standing muscular strain in an unnatural position. how is it we do not find nystagmus amongst decorators and painters and allied occupations? They work in a good light one would at once say, and they certainly do work in a better light than the miner; but what about the metalliferous miner? - he uses a safety lamp and is not affected, or to a lesser extent . Case No. T 9 who worked in an iron ore mine complained of the ill effects of bad ventilation and this at once suggests to me that nystagmus is not due to lack of illumination alone but to the combined effects of several agencies of which strain and defective illumination are a part.

Gowers (53) thinks that miners' nystagmus is due to an alternating contraction of opposing

muscles. He thinks there is a central lesion in or near the mid-brain and pons and in the cerebellum. A "Mid-brain Optical Centre". There is in my opinion good grounds for believing that there is such a centre, and also I agree with Gowers in believing nystagmus to be in the nature of a reflex action. Numerous reflex actions are known to occur in the brain. winking of an eye; the light reflex on the sphincter muscle of the iris; are examples. The conscious reactions of the brain are not included among the reflexes, because reflexes are involuntary, but it should be remembered that so far as nervous mechanism is concerned the conscious reactions do not differ from the true reflexes. The activities of the brain are carried on by a mechanism of one neuron acting upon another, just as in the case of the reflex arc. believe there is a mid-brain optical centre. Well it must be activated in the same way by one neuron acting on another, and its normal way of acting is by co-ordinated movements of the eye, and as we grow older this becomes more or less reflex. For instance, when one learns to ride a bicycle, it is done by voluntary co-ordination; nevertheless, in proportion to the skill obtained, this becomes more or less

reflex, that is, involuntary. In other words one establishes ones reflexes and one must suppose that the continual ajustment of certain sensory impulses to certain co-ordinated movements results in the formation of a more or less complex reflex arc - or, in plain words, a set of paths of least resistance. Have we any such conditions in the miner's life, whereby the continual ajustment of certain sensory impulses to certain co-ordinated movements would be necessary? surely we have! First and foremost we have central fixation done away with, owing to deficient illumination. We have abnormal postures (disturbing the body equilibrium), the rhythmic swinging of the arms, and necessarily the head, the blow of the pick jarring the whole system as it strikes the coal and the continual efforts to fix the eye on the point of aim. Are not each of these conditions in its own way causing sensory impulses and giving rise to certain co-ordinated movements? Are not the eyes receiving different impulses to what they are accustomed? Is not central fixation done away with and the whole body equilibrium disturbed? It is not to be wondered at, that, with the loss of central fixation, the eyes are receiving stimuli to which they are unaccustomed, or are working in a new way compared

with their regular co-ordinated movements. rod is believed to end in a single knob-like swelling and a number of them make connection with the same nerve cell. Each cone connects with a single nerve cell and eventually perhaps with a single optic nerve fibre, and therefore the path for the cones seems to be the more direct and this is the normal reflex path. In other words the fovea, with which we see most clearly, contains the cones which by light are stimulated to give a regular coordinated stability to the eye, but in the dark, the rods are stimulated and in the absence of the normal reflex through constant working in the dark, a fresh path is established, but one which sets up rhythmic movements of the eyes. Would such conditions account for latent nystagmus? Yes, this path is developing but the miner is still able to control his eyes by will power (accommodation) until eventually he breaks down with the strain, or some accident or other cause makes it fully apparent.

c. Bell Taylor (4,5) and Mlle. Ioteyko (19)
both think that miners' nystagmus is analagous to
writer's cramp. It is well known that persons
whose occupations necessitate complicated movements
over long periods of time may suffer from irregular
contractions of the muscles converned. For

example, writer's cramp, and a probable emplanation of this condition is that there is a defect in the centres associated in the act of writing by a morbid lowering of resistance in the commissural connections between the centres. so that there is a radiation of impulses and so an overaction of muscles not necessarily engaged in the act. I do not hold that fatigue is the essential etiological factor, but I think we have a clue to the pathology of miners' nystagmus namely an upsetting in the centres normally associated with the act of writing. If we have centres for writing, why not a mid-brain optical centre, and why not an upsetting of the centres normally associated with the regular equilibration of the eyeball, due to the lack of stimuli for fixation, and over stimulation from other sources causing instability of the eye? In support of this, I may mention that A. Christie Reid (21) put forward an equilibration disturbance hypothesis. He says that in the miner we have imperfect fixation due to a dim light and frequent disturbances of position. He thinks the more or less rhythmical series of movements performed in the swinging of the mandril, the eyes remaining fixed, is an important factor in the production of the disease. Fatigue is not sufficient as all the muscles of the eyeball are involved, and there is

no hyperphoria as one would expect if the elevators alone were affected.

Jeaffreson looks upon the disease as a general one and that position alone is not the sole cause, but that when helped by the disassociation of mevements normally combined. nystagmus occurs. There is after all not much difference between Reid's equilibration hypothesis and Jeaffreson's dissociation of centres normally associated, in fact I fail to see how one can conclude otherwise than that the pathological seat of miners'nystagmus lies somewhere in a mid-brain optical centre: and that for a disturbance of this centre. we must look to the factors already stated as being fundamental in its upset. I do not agree with Jeaffreson's theory of cerebral anaemia, nor with Mr. Oglesby's theory of "Venous engorgement of the Medulla".

Dr. F. Robson of Penarth contends that the absorption of mine gases is the cause, and he gives in the following table the case incidence in proportion to the volatile content of coal (54).

Monmouthshire. V.C. 29.5% C.I. 4.56% East Glamorganshire.V.C. 29.5% C.I. 2.22% West Glamorganshire.V.C. 25.5% C.I. 1.90% Carmarthen. V.C. 11.86% C.I. 1.15%

V.C. = Volatile content. C.I. = Case incidence.

This theory of gas poisoning was also advanced by

other writers mentioned in the historical outline

and was recently upheld by Dr. Robson (45)
when Drs. Haldane and Llewellyn still contended
that the defective lighting was the essential
cause. There can be no doubt that the daily
absorption of mine gases must have a very
deleterious effect upon the miner's constitution,
and when you consider the heavy work involved,
the gases produced during shot-firing, and the
speed at which the men work, it is not to be
wondered at that great strain is thrown upon the
whole system.

I have been told by men working at the coal face that on occasion a deputy (carrying his oil safety lamp) has had to return and get an electric lamp to come and find them. If this is a case, these men must have been working in an atmosphere laden with poisonous gases. Furthermore, I contend that the introduction of the electric lamp makes it possible for men to work in an atmosphere which will not support combustion and yet be unaware of it. In the days of candles a miner had to leave work at the face when his candles went out due to lack of air. No doubt the ventilation of our mines is much improved, but in the majority of cases it is still very deficient. I am not trying to disprove my theory of disturbance of the "Mid-brain optical

Centre", but rather to point out the deleterious effects that gas must have upon the muscular and nervous systems especially when those systems are already in a state of fatigue. The men themselves propound a gaseous theory, but they attribute it to the fumes from the oil lamps.

Let us for a few moments consider what happens in simple fatigue itself. The waste products added to the blood act upon the nerve endings in muscle and upon the grey matter in the brain and create a sense of fatigue. Although the sensation of tiredness is referred by us to the overworked muscles, the location of the cause is less in the peripheral than in the central nervous system. Would not those waste products be increased by breathing impure air, and have we not got an important factor which may help to act upon the "Mid-brain Optical Centre" and throw it out of gear? if so, is it a factor, common to all workers in the mine who become afflicted with nystagmus? This factor is certainly common to all such workers and more pronounced in small seamed pits, and I suggest that the fatigue products accumulate, and as a result the mid-brain ocular centre is less receptive to the sensory stimuli which are the controlling influences over ocular stability; and also, central fixation is done away with, with the

result that this centre is thrown out of gear and the lower centres take upon themselves the paths of least resistance, resulting in nystagmus. In addition we have the dis-association of movements normally combined, and afferent impulses passing from the semi-circular canals to maintain the body equilibrium in the various altering positions. In a few words the impulses passing to the lower centres are inexact and the controlling influence of the mid-brain ocular centre is interfered with, and, as stated already, first we have the will-power brought into play - the latent stage; but soon a stage is reached when the centre is unable to stand the strain any longer and nystagmus is manifest. The severity of the attack depending upon the extent of the breakdown. This explanation also confirms my observation that cases of nystagmus do not readily recover until after a very long rest. (The average duration of incapacity, total and partial, in my cases was one year seven months two weeks and four days). (Vide p.51) It also proves why drugs have little or no effect in remedying the condition. Also I think the same theory may be applied to explain the tremors in the limbs; the centres controlling those muscles are also affected by the waste products, and, as a result of morbid lowering of resistance in the

commissural connections, we get a radiation of impulses. In fact the whole of the nervous system is affected as is evidenced by the mental state of the patients; some strong-built men have broken down and cried with relief when told they would not go blind. The circulatory system is in most cases affected also. Nearly all my cases had pulses over ninety beats per minute and some one hundred and twenty per minute. Oral sepsis I also found prevalent, and the ill effects of this, accompanied by excess of smoking, or chewing of tobacco, associated with alcoholism, on a nervous system (struggling to carry on under such strenuous conditions), cannot be over estimated.

If a miner suffering from nystagmus be asked to press his forefinger on the table firmly tremors will be felt in his extensor muscles; and, likewise, ask him to press his toe on the ground, and tremors will be felt in his quadriceps muscle.

These facts in my mind bring miners'

nystagmus into line with other varieties of

nystagmus, for example in nervous diseases,

poisoning, etc., as classified in the beginning of

this section. Nystagmus is brought about by an

accumulation of factors in the miner's life.

One may say all miners do not get nystagmus. That

is true, but what is still more surprising is that

who suffer from nystagmus, and as these twenty five thousand, generally speaking, do not themselves know that there is anything wrong with them, no further proof is necessary to show that the oscillations, when not accompanied by subjective symptoms, are of negligible consequence (56).

I have a case, quoted hereafter, who did not wish me to find out that he had it, because he said he could work with it. To these facts I simply state that our nervous systems are not all built alike, and that our powers of resistance vary.

PATHOLOGY.

Very little is known or written about the pathology of miners' nystagmus, and it is a great pity that post-mortem examinations have not as yet been carried out.

When commencing this thesis I wrote to my brother-in-law, an Inspector of Mines in India, and asked him for some statistics as to the incidence of nystagmus in India. I take the liberty of quoting the reply here. "I have made enquiries in the office from R. R. S. and find that we have no statistics in the office on the subject. I have made enquiries from T.C.Murray, Argyle

Mackie, and Bathgate (Inspectors) and they all say that no case has ever been brought to their notice.

Dr. Rosario says it may exist but he has not had any cases.

existent in India. When seventy five per cent of the labour spend a few weeks in the mines and then go off to their villages and return for another spasm a month or so later; and permanent labour only work four to five days a week, there is very little strain on the eyes. As you know most of the labour here only works six months in the year and the rest of the time they cultivate. Then again open lights are used and there is no stooping.

I think you can safely say it is practically unknown in India and that the general conditions are not conducive to the production of nystagmys".

My criticism of this letter is simple.

Open lights are used and ventilation cannot get
too bad or, if it does, out go the lights, & work ceases.

Secondly, the miner only works for a short time in
the mine, and in the interval he tills his fields,
thus obviating any accumulation of poisonous products
in his system.

TREATMENT.

This important section must necessarily be divided into two parts.

- 1. Preventive treatment.
- 2. Curative treatment.

PREVENTIVE TREATMENT.

The results of my investigations have led me to believe that any miner in our coal pits is liable to become affected with the malady, and the most important factors in the preventive treatment are (a) The maintenance of improved miners' lamps and general lighting of the pit where possible, (b) Efficient ventilation and care that all workings are supplied with fresh air, (c) Prompt attention to all foci of infection, for example oral sepsis, etc., by all medical men and company doctors, (d) overwork to be avoided at all times, and especially when signs of nystagmus are commencing. Nowadays with modern means of transport, coal productionsis hastened in every branch, with the result that the hewer also works much quicker than he used to. These factors need not be elaborated upon except to remark that each one entails strict supervision on

on the part of medical men, engineers, surveyors etc, connected with the pits and the help of our Medical Officers of Health in the mining villages to prevent overcrowding and living in unsuitable dwellings.

T. L. Llewellyn in his book emphasises the need for the proper care of the miners' lamps, the use of shields, and good quality glass. Poor ventilation has a deleterious effect upon the amount of light given off by the lamps as well as affecting the miner's constitution, thus affecting him in two ways.

The Sherwood Coal Company have taken steps to prevent disease amongst miners and compensate them for loss of natural sun by giving their men a shower bath on coming to the surface and then giving them a few minutes artificial sunlight from special lamps. If this experiment succeeds it may have far reaching results, both medical and industrial.

CURATIVE TREATMENT.

The cure of this disease is I am afraid very difficult, as can be surmised from the views we have put forward as to its etiology. Each case must be studied separately. The miner's mental attitude taken into consideration, and all his fears

as also errors of refraction. I do not believe, not does Fuchs, that errors of refraction cause nystagmus, but relief must be given if such are corrected when present. Severe cases must rest completely and the milder cases, should be given fresh work on the surface; or, better still, a complete change of occupation, in which they are less liable to become despondent.

In the table, compiled at the end of this thesis, the total and partial incapacity averages are shown for a large number of pits in this district and emphasises the long duration of forced idleness.

Good feeding is essential, and, if
necessary, extra nourishment supplied through
local clinics, supplimented by good general tonics,
such as iron, nux vomica, strychmine and the
hypophosphites. In some cases sedatives may be
required.

To put a man on full compensation, unless he is very bad is a mistake; and surface work keeps him occupied. It is not necessary for him to leave the pit altogether, but, after a good rest, any employment given should be of a different nature, and special care should be taken with regard to ventilation, lighting and general hygiene.

LIST OF CASES.

It would be tedious reading and quite unnecessary to give full details of all the cases

I have seen, and a note of a few with some comments on these is preferable.

Case No. T 1. Age 42. First affected in 1911, when opening out a new area of coal (driving a winning). Worked with his father, who became a victim shortly afterwards. I quote a letter received from him. "Now it often happens, that ventilation is very poor. Baults or nip outs are come in contact with and these usually produce gas the oil lamps that we used also gave off a bad odour, very detectable in any place where the ventilation was bad. As a rule we were given a spare lamp as you never could tell when the light was going to be extinguished with the fumes. the commencement of the disease to the time I was compelled to give up work, occupied about two years. After being idle for twelve months, I resumed work as a surface hand..... in 1914 I returned underground using electric lamps and trying many different sorts of work".

He was very bad in 1925 - six months off work - now at work below, on the trolleys. He

agrees with me about bad ventilation and mentioned about the rapid work now needed. Also gave me the names of several other men affected who agreed This case is of interest as he with him. himself brings forward evidence of defective ventilation and the recurrence of his trouble whilst engaged in different labours under better lighting conditions, more or less, tells against any purely local myopathic or defective illumination theories. Case No. T 2. First suffered 12 to 13 years ago has worked all along and is still working. Was off sick some two months recently and eyes did not improve during this time. Surely a pure myopathic condition would improve with rest. This man's eyes were worse on looking directly upwards and he had to give up his position as a church organist on account of his eyes. He cannot give a cause. Admits ventilation is bad. Pulse one hundred and ten per minute. Tremors of head, arms and These facts point to nystagmus as being a legs. general condition rather than a purely local one affecting the eyes only. In my opinion they point to an affection of the whole central nervous system. This case also wrote to me about his condition and concluded his letter as follows: -"Miners nystagmus although not dangerous as the medical profession tells us, yet is a very

distressing and uncomfortable complaint".

Case No. T 3. Age 30. Worked at pit bottom 16

years - 7 to 8 years with oil lamps; rest with
electric. A hewer, moderate smoker, and total
abstainer. Cannot himself assign a cause.

Nystagmus most evident on being asked to stoop
suddenly, in fact he fell to the floor on being asked
to do so. This proves an upsetting of the body
equilibrium as a whole, and undoubtedly that of the
eyes as well. Tremors of eyelids, head, arms and
legs present. Pulse one hundred per minute. Oral
sepsis present. Backward tilt of head present and
cap worn over eyes.

Case No. T 4. Age 40. Stone worker. Used oil and electric lamp. Has done very little hewing, perhaps two years, (twelve years ago), but had no symptoms at that time. The stone work requires frequent up glancing at the roof, but he says it is not more trying to the eyes than ordinary work at the coal face. Some time ago he was bad and did surface work for about eighteen months and then tried down below again and in six months was as bad as ever. Is working below now at stone work. The movements of the man's eyes seem to be more or less circular, complete in the direction of the hands of a watch and were always worse at dusk.

Oral sepsis present. Pulse eighty four per

minute. Tremors in quadriceps only, and often complained of cramp in his feet.

This case is of interest because he had done so little hewing in his life, and, although stone work is somewhat constrained, it is not so bad as coal hewing, and yet he suffered very much. The cramp in the feet is noteworthy, proving again that it is not only the eyes that are affected but that other nervous signs are also present. The pulse in this case was normal and I could not elicit tremors in his hands or arms. Knee jerks slightly exaggerated.

Case No. T 5. Age 43. Heavy drinker. Smokes and chews tobacco heavily. Works in a two foot seam. Contributes cause to fumes from lamps, defective light, and states that he is no better even working with electric lamp. A very nervous man, with marked tremors of head, arms and legs. Nystagmus most marked on looking to the right and on toeing a line, which he did very badly. Marked rotary nystagmus was induced even on looking downwards.

This case also proves that wherever an equilibration test is employed nystagmus can as a rule be elicited without putting the eyes in a strained position.

Case No.T. 6. First noticed when attended for

influenza, but made no mention of his eyes but some few weeks after his return to work he complained of his eyes. Pulse one hundred and eighteen per minute. Pupils dilated. contended that the air was bad where he worked. This case proves the debilitating effects of other illnesses upon the condition. He is now at work upon the surface and is improving. Case No. T 7. A case occuring in a fireman, who was never near a pit in his lifetime. Gassed during the war and wounded in the face. Two years ago wound developed an abscess due to an old piece of shrapnel, and, whilst examining him one day. I detected a coarse lateral nystagmus. Not rotary. I asked him how he worked, and he told me he always kept his eye on his boiler fire when stoking and the coke was on his left. The heat and fumes often used to upset him and his record card shows many attendances for gastric catarrh and bronchitis. The shrapnel was removed and there is no septic infection now but he still has his nystagmus. interest because his position at work was in no way strained, and it was only at intervals that he had to stoke; but, when he did so, one can readily understand the dis-association of movements. The eyes fixed on the boiler

entrance and the swinging of the arms and body
to the left, combined with the effect of the heat
and the fumes inhaled, must have upset him. On
my advice he has now given up stoking.

Case No. T 8. "Yorkshire Post". 9/12/26.

Nystagmus and eye oscillation - simulation
suggested.

Although not my own case this is worthy of inclusion. Dr. Caiger, Sheffield in court said "Again and again I find they begin to strain in some peculiar way, and seem able to produce a certain amount of oscillation. I myself can produce a little movement that way."

Is it possible then that a man can inhibit his "Mid-brain ocular centre" and so throw his ocular stability out of gear. A few of my cases certainly seem to be able to set their eyes in motion, but then they are cases just recovering. However, if this be the case, it is a further proof and advance to the theory that the seat of nystagmus lies in the brain and the central nervous system.

Case No. T 9. Worked in Iron Ore mines with candles. Seams usually large. Giddiness often present on coming to the surface. No oscillations present at any time, but had tremors of hands and head. Is now working in the open

air and vouched this information on telling him

I was interested in mines. He concluded by
telling me that the ventillation often would not
allow the candles to burn.

This goes to prove that although other miners may not suffer from nystagmus proper, they do have some of the other associated symptoms, and that they too work in defective ventilation.

46.

SUMMARY AND CONCLUSION.

I am of opinion that -

- (1) Miners' nystagmus is an occupational disease, arising from a disturbance in the neuro-motor apparatus of the eye. That it can justly be classified with other forms of nystagmus, (as it frequently is in congenital and non-industrial cases, secondary to some defect in the sensory part of the visual mechanism).
- (2) That it is not a complete disease in itself connected with one particular cause. In mild cases the oscillations are limited to the eyes themselves, but in other cases there are associated tremors of the muscles of the eyelids, head, neck and even of the upper part of the trunk, shoulders, arms and legs.
- (3) That the oscillations and tremors are due to an over-action, or exhaustion of the higher motor nerve control centres in the brain, which may extend to any group of nerve centres.
- (4) That in my opinion the following conditions are all conducive to the onset of the disease.
- (a) defective ventilation and absorption of mine gases.
- (b) defective illumination, (which does away

with yellow spot fixation).

- (c) Conditions tending to disturb the body equilibrium, such as, rhythmic movements, the eyes remaining fixed, and the strenuous work involved in unnatural positions.
- (d) Contributary factors are, excess of tobacco and alcohol. Oral sepsis and other illnesses.
- of importance as to nystagmus as a disabling factor. In every country some men continue working although suffering with nystagmus, and there is no doubt that added importance has been given to the condition by the benevelent working of the Compensation Acts, especially by the admission of cases, when the wording of the Act was changed.
- (6) That it is a fact that the associated symptoms and the mental attitude are as disabling factors as the eye oscillations.
- (7) That errors of refraction have little or no influence on the production of nystagmus. Men with bad eyesight, corneal opacities, etc., can find employment in mines and earn full wages.

 Defractive errors where found should be corrected.
- (8) That miners' nystagmus is recoverable in a general way. Rest may be required for a year or

- more. (Vide table p. 49-51) The eyes resume their previous standard of vision, when steady, in uncomplicated cases.
- where possible, and if less remunerative partial compensation; or better still a lump sum should be offered in settlement. This has been the practice in West Cumberland lately and curiously enough often has a curative effect. That the economic problem is greatly complicated by the over staffing of our mines, and men, known to have been certified, cannot find employment; and, even if both parties are willing, work above ground cannot often be found.
- (10) That the ideal would be to form a colony for such men with outside work, which would be beneficial to themselves and the country.

TABLE 1.

WEST CUMBERLAND PITS.

COAL OWNERS' MUTUAL INDEMNITY SOCIETY COMPENSATION STATISTICS.

dolliery.	No.of	Aveg.	Aveg. Incapa	motal acity,	Aveg. Incap	Partial acity.	Aveg. T Durati	Total ion.
			Weeks.	Days.	Weeks.	Days.	Weeks.	Days.
Buckhill	26	8	44	2		4	53	1
Brayton Knowe	63	39.5	72	es		1	~	03
Clifton	52	9	36	1		23	63	CZ
Allhallows	13	43	72	4		23	0	Н
Brayton Domain	9	46	30	വ		03	~	Н
P	9	41	30	1		1	73	
Llimill	24	38.58	30	es	44	62	74	4
Oatlands	9	00	42	ເດ		7	10	
Oughterside	9	7 .	CS	7		വ	~	1
Seaton Moor	53	7	es	ıΩ		1	0	വ
ate	ω	35.8	100	4		7	123	വ
品	co	4	es	4			4	4
Outfields	23	0	181	٦		1	00	Н
seho	80	33.6	21	1	28	10	49	വ

TABLE 1. contd.

Colliery.	No.of Cases.	Aveg.	Aveg. To Incapac	otal city.	Aveg. Incap	Partial acity.	Aveg. To	otal
			Weeks.	Days.	Weeks.	Days.	Weeks.	Days.
t. He	25	10	55	03	46	ιΩ	102	٦
t.Helens No. 3	47	3.3	68	4	32	7	0	വ
t. Helens C.&B.	22	37,35	43	വ	11	4	22	63
t. Helens C.&B.W	25	4.8	20	7	27	1	47	es
arrington	39	0	46	വ	69	1	115	വ
cton No. 1	6/3	m	15	4	88	63	44	٦
arrington	14	34.5	23	4	23	Ω	47	4
arrington No. 1	٦	9	10	Н	1	ı	10	Н
illiam Pit	65	0	42	1	24		99	
ellington P	26	8.3	34	വ	13	જ	48	Н
dvsmith Pit	22	8	46	4	20	63	26	Н
ale Pit	03	0	23	ı	11	വ	34	വ
ythemo	Ω	00	37	1	46	Q	83	63

Period of Observations. 1911 = 1921.

N.B. Each workman's week = 6 days.

TABLE 2.

TOTAL CASES - No. 463.

YEARS 1911 - 1921.

TOTAL AVERAGES.

Aves.	Aveg. Total Incapacity	otal city.	Aveg. Partial Incapacity.	artial	Aveg. Total Duration.	otal
	Weeks.	Days.	Weeks.	Days.	Weeks.	Days.
38.26	46	ı	36	83	828	4

That is the Average Total duration was

1 year, 7 months, 2 weeks, 4 days.

N.B. Each workman's week = 6 days.



TABLE 3.

The total number of cases receiving

Compensation in the West Cumberland Pits was

463, from the years 1911 -1921 and the following
table shows the proportion in which they were

affected:-

Hewers	81.4%
Shift Men	7.775%
Deputies	1.725%
Putters	.216%
Firemen	.216%
Packers	.216%
Shift labourers	1.725%
Brushers	1.08%
Pump men	. 432%
Labourers	. 432%
Brakers	. 432%
Pony drivers	.866%
Coal cutters	.216%
Onsetters	. 432%
Haulage hands	. 432%
Rolleyway men	;216%
Bogey hands	.216%
Stone men	.216%
App. Hewers	.647%
Shift hands	.866%
Trailers	.216%

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