MINERS' NYSTAGMUS.

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In presenting this Thesis on Miners' Nystagmus, my object is to review the literature on the subject, with emphasis on factors accepted now, or in the past, to be of aetiological importance; to give my personal views of the significance of these factors; and to include my original contributions to the recorded knowledge of the disease.

During twelve years in practice in one of the largest coal mining areas in this country, I have had the opportunity of examining nystagmus cases from 16 collieries, referred by the Yorkshire Coal Owners Mutual Indemnity Society for assessment of incapacity, as well as in the course of my present duties as Examining Factory Surgeon.

INDUSTRIAL IMPORTANCE.

It is estimated that Miners' Nystagmus costs the community annually about £1,000,000 in loss of production and compensation, to say nothing of the suffering and discontent provoked. It accounts for 90% of the cost of all industrial diseases. The number of persons in receipt of compensation for this disease averages nearly 10,000 a year, of which about 2,000 are new cases.

The industrial importance of Nystagmus is officially recognised by the appointment, at the request of the Home Office, of a Miners' Nystagmus Committee which has issued Reports in 1922, 1923, and 1932.

DEFINITION.

Physicians in the past, observing the coexistence in the disorder of childhood known as 'Spasmus nutans' of nodding of the head and oscillations of the eyeballs, termed the oscillations "nystagmus" from the Greek "nustagmos" meaning drowsiness or nodding.

So far as I know, no comprehensive definition of Miners' Nystagmus has been recorded. Court (1891) suggested "A definition of the disease should include night-blindness, nystagmus, photophobia and head symptoms....." For want of a better, I suggest the following. Miners' Nystagmus is an occupational disorder of the nervous system, characterised at the onset by oscillations of the eyeballs and later having a tendency to develop incapacitating psychoneurotic symptoms.

SYMPTOMS.

The term "symptoms" as used in this Thesis also denotes "signs", following the method adopted in the official Committee's Reports.

The symptoms of miners' nystagmus may be classified as local and remote, subjective and objective.

LOCAL OBJECTIVE SYMPTOMS.

These include oscillations of the eyeballs; blepharospasm---by which is meant blinking of the eyelids, in contradistinction to narrowing of the interpalpebral fissure, which may also be present---; forced ocular movements, such as spasm of convergence, spasm of the superior rectus causing the pupil to be hidden under the upper eyelid, and semi-voluntary irregular movements of the eyes; undue slowness in reading Test-Type---e.g., hesitancy over 6/24 line yet eventually correctly reading 6/6 line; frequent lowering of visual acuity below 6/12; difficulty in maintaining a fixed gaze e.g., on examiner's fingers; bulging of the eyeballs and visible hyperaemia of the sclerotics on bending forwards.

LOCAL SUBJECTIVE SYMPTOMS.

Symptoms referable to the eyes are included under this heading though, of course, vision is a function also of the brain.

These symptoms include night-blindness and photophobia---evidenced underground by difficulty in seeing going to and coming from the coal face, and on the surface by bad vision at dusk, early morning, and in bright artificial light or sunlight; dazzling by lights; spinning of lights; double vision; transient loss of vision after stooping, severe exercise or continuously gazing upward; difficulty in focussing the coal face or small objects on the ground; apparent sudden movement of the coal face.

REMOTE OBJECTIVE SYMPTOMS.

These include a "vibratile" tremor of the head, felt by the examiner's hand firmly placed on the top of the patient's head and preventing its extension when his gaze is directed upwards; a vibratile tremor felt on placing the hand on the patient's shoulder---especially after bending exercises; tremors of the extremities varying in degree to chorea like movements; staggering ---especially after bending exercises;---tachycardia; visible sweating on slight exertion; increased reflexes; the nystagmic "facies". (vide p. 5.)

REMOTE SUBJECTIVE SYMPTOMS.

These include giddiness; headache--frontal or occipital;---sleeplessness; bad dreams; nausea and even vomiting, especially in the morning; diarrhoea and precipitate micturition on

excitement; various phobias, including fear of being underground, or of blindness; mental depression and even suicidal tendencies.

It should be noted that though all the above symptoms may be present in any one case, this would be extremely unusual; in most cases only some of the symptoms described are present. Further, though at some stage of the disease, oscillations may be absent, still, as the Departmental Committee Report of 1938 puts it, "Oscillation of the eyes is of the essence of the disorder."

MOVEMENTS OF THE EYES IN NYSTAGMUS.

Oscillation of the eyeballs is not confined to miners' nystagmus. A brief survey of the conditions in which nystagmus is found, and how the oscillations may be described, is given as follows.

Many authorities have shown that the movements of true nystagmus are invariably rotatory, and that the oscillations may be of two kinds. (a) "pendular", when the movement in each direction is equal in speed, and (b) "jerky", when a slow outward movement is succeded by a quick inward one. The movements may vary in amplitude and are commonly described by the terms, "coarse", "medium" and"fine". The movements in both eyes may be symmetrical, (conjugate nystagmus) directly opposite, (disjunctive nystagmus), entirely unrelated, (dissociated nystagmus), or present only in one eye, (unilateral nystagmus). The direction of the movements may be vertical, oblique, horizontal or rotatory.

MOVEMENTS OF THE EYES IN MINERS' NYSTAGMUS.

The movements of the eyes in Miners' Nystagmus have their own characteristics. As Brock (1938) and many others point out they are more persistent in duration, are severer in degree, and more varied in type. They may be vertical, horizontal, oblique or circumductary; but typically are more truly rotatory in direction. Movements in both eyes are almost invariably symmetrical, though the amplitude may vary in each eye. The typical oscillations may be present in a man with well marked squint, or having only one eye. The oscillations may be accentuated, or latent ones made apparent, by elevation obliquely upwards of the visual regard, by confinement in a dark room, or by shovelling exercises, preferably in the dark. In my cases the following method of examination and classification was used; it is an elaboration of the method used by Pooley of Sheffield. The miner was first examined in an erect posture in daylight, the visual regard being directed horizontally forwards, when if no oscillations were found, the visual regard was directed obliquely upwards. Oscillations present under these conditions constitute "Nystagmus No. 1." If the oscillations were not elicited by this manoeuvre,

he was put through shovelling exercises in the daylight for about two minutes. The eyes were then examined, the body being held in the bent forward position, whilst the visual regard was directed obliquely upwards. Oscillations neccessitating this manoeuvre for their production constitute "Nystagmus No. 2." If the oscillations were still absent, the miner was kept seated in a dark room, the body bent well forwards, whilst the visual regard was directed upwards. Oscillations now elicited constitute "Nystagmus No. 3." If after half an hour in a dark room no oscillations were found, shovelling exercises in the dark were done, and kept up for a period of ten minutes. If the oscillations were then provoked, this constituted "Nystagmus No. 4." When dealing with a large number of cases each of which was examined at one or two monthly intervals, this method of classification will be found most helpful, and when combined with the following abbreviations, has the effect of conveying "much in little." E., & N.E., signify that the oscillations were easily elicited
P N.P.
and persistent, or, not easily elicited and not persistent, respectively. H.T., signifies the presence of head tremors; B., the presence of blepharospasm or blinking of the eyelids. Ph.,existence of photophobia. S., staggering or swaying movements; and the pulse rate was given in beats per minute. Thus the following entry B. Sykes 30/9/37 NYS. No.4. N.E., H.T.,
N.P.
B++., Ph., 120, signifies that the person on the date mentioned had oscillations of the eyeballs only after exercises in the dark; that they were elicited only with difficulty and were not persistent; that head tremors and blinking of the eyelids were marked; photophobia was present; he staggered when being put through his exercises; and that his pulse rate, at rest, was 120 per minute.

Cases in which no nystagmus was found after exercises in the dark were noted as nystagmus free, and examined again in a month's time. In many cases "Nystagmus No.4" would then be present. If oscillations were again absent, a third examination was made after a lapse of another month, and if no oscillations were then found, they were certified as being free from nystagmus, a note being made, however, as to the presence or absence of head tremors, blepharospasm or blinking of the eyelids, staggering, and the pulse rate was recorded. In my opinion, it is unwise to certify a man as being free from oscillations of the eyeballs unless three consecutive examinations are made over a total period of two months. In my experience it not infrequently happened that cases in which I could detect no oscillations and had certified as nystagmus free, were examined by an ophthalmic consultant before contesting the case in Court, the miner having appealed against my decision. The consultant has then found,

on ophthalmoscopic examination, very fine oscillations, and consequently was unable to certify that the man was free from nystagmus. The experience I had on taking a case in which I could not detect oscillations to Dr. Eminson, ophthalmic consultant in Doncaster, taught me always to ask the patient before certifying him free from nystagmus, if he himself could bring on the oscillations. Dr. Eminson, having put the workman through prolonged exercises in the dark, also failed to discover any oscillations. The room curtains were then drawn apart, it being daylight, and we retired to another room leaving the workman seated in the light. After about ten minutes we returned to the room, and the Consultant informed the man that he was unable to find, after an exhaustive search, any trace of nystagmus. The miner then said, "Shall I bring them on for you Doctor?", and on being somewhat sceptically encouraged to do so, he vigorously moved his head and neck in a peculiar, almost "concertina-like" fashion for about ten seconds, whilst he remained seated, then fixing his head in an horizontal position and gazing obliquely upwards at the ceiling declared, "They're going now, Doctor! and they were!

When examining a crowd of about a hundred men suffering from all types of injuries, it is remarkable how one can detect the nystagmic cases from the others, even where oscillations are not present until provoked. There is a definite nystagmic "facies" which is easier appreciated than described. The head is held well back, there is a narrowing of the interpalpebral fissure, the visual regard tends to be inclined slightly downwards, and there may be an apparent inability steadily to gaze in one direction giving a "shifty" look to the afflicted person. When the hand is held on the top of the workman's head or shoulder, and he is asked to look at the examiner's finger, so held that the visual regard must be directed obliquely upwards, tremors, varying in degree, of the head and neck can often be felt. After several seconds in this position, the oscillations may become apparent. When the workman is then informed, "That will do", he vigorously closes his eyes, shakes his head, and frequently the provoked oscillations will abruptly cease. Though I have only on one occasion seen a case (above described) where the miner could himself bring on oscillations when thorough attempts by myself to do so had failed, observations made from many hundreds of examinations of nystagmus cases leave me in no doubt that nystagmic miners can consciously or subconsciously adjust the position of the head, with regard to the body, in space, and, his visual regard, so that he can, with least effort, provoke the oscillations. Further, the oscillations having been provoked by this head and eye manoeuvre, he can, by some other head and eye movement, arrest them.

AETIOLOGICAL CATEGORIES OF NYSTAGMUS.

A concise classification of the conditions in which nystagmus is found is given by Duke-Elder as follows:-

1. OCULAR NYSTAGMUS.
   - (a) Pseudo-nystagmus.
   - (b) Nystagmus of eccentric fixation.
   - (c) Optico-kinetic nystagmus ("train nystagmus")
   - (d) Nystagmus due to deficiency of light.
   - (e) Amaurotic nystagmus.
   - (f) Amblyopic nystagmus.
   - (g) Latent nystagmus.
2. VESTIBULAR NYSTAGMUS.
3. OCCUPATIONAL NYSTAGMUS.
4. NYSTAGMUS from sensory stimulation of the skin about the ear.
5. NYSTAGMUS from auditory stimulation.
6. CENTRAL NYSTAGMUS. (a) Vestibular and cerebellar.
   (b) Cortical.
7. HYSTERICAL and VOLITIONAL NYSTAGMUS.
8. IDIOPATHIC and HEREDITARY NYSTAGMUS.

To this list might be added the Normal Physiological Ocular Nystagmus which, though invisible to the naked eye, has been shown by Dodge, (1903) Marx, (1913) Borries, (1926) and others, to exist. It is due to the constant muscular activity resulting from the Optical and Psycho-optical Reflexes.

1. OCULAR NYSTAGMUS. The constant muscular activity required to produce fixation, results in fine rotatory movements of the eyes which, although they may seem at rest, are never completely immobile. Thus we have normally a physiological nystagmus always present but invisible to the naked eye. When the eyes change from one fixation point to another, these oscillatory movements are increased, but still unnoticed.

1(a). A PSEUDO-NYSTAGMUS is merely the exaggeration of these normal oscillations. It usually indicates fatigue of a weak muscle, or is associated with some central nervous disease.

1(b). NYSTAGMUS OF ECCENTRIC FIXATION. This is observed in 50% of normal subjects when the fixation axes are deviated to either side beyond the normal limits of binocular vision. It is a horizontal, jerky nystagmus and is induced most readily in states of fatigue.

1(c). OPTICO-KINETIC NYSTAGMUS occurs when succesive moving objects traverse the field of vision. It is known also as "train nystagmus". It is produced by the eyes following one moving

object until it is in the peripheral field when the attention is attracted by another object. The first impulse causes a slow movement of the eyes outwards, the second, a quick jerk back.

1(d). NYSTAGMUS OWING TO DEFICIENCY OF LIGHT. Children born and reared in dark surroundings may develop a pendular nystagmus which is frequently associated with a nodding movement of the head (spasmus nutans). It seems to be due to a failure in the development of fixation. (Raudnitz. 1897). The condition is normally seen in the cat in dim illuminations. (Ohm 1918). Similarly, if dogs are kept in dim illuminations for six or seven weeks, a nystagmus develops, which is not seen when the stimulus for fixation is lost, as would obtain if the animal were kept in complete darkness, or if the optic nerve were cut. (de Kleijn and Versteegh, 1920., Bartels, 1924., and others). In my opinion, the interpretation of these facts is that in conditions of incomplete illumination, the fixation and optical reflexes are inadequately stimulated, resulting in failure of equilibration of the extra-ocular muscles, which is rendered visible as "nystagmus". This is almost of a compensatory nature and may be interpreted as an attempt by nature to focus objects on that part of the retina which is best suited to bad light, also to gain the greatest visual field.

1(e). AMAUROTIC NYSTAGMUS. A pendular or jerky nystagmus may occur in those who are congenitally blind, or have lost their eyesight for some time. This is accepted by many authorities but is in part denied by Ridley and Sorsby who state, "Indistinctness of retinal images due to disease acquired after normal development of fixation, (i.e., a few weeks after birth) never results in nystagmus."

1(f). AMBLYOPIC NYSTAGMUS. When fixation is impossible due to some grave defect in vision, e.g., an opacity in the media, or a central scotoma, nystagmus may occur. Common causes, therefore, are central corneal opacities, as from ophthalmia neanatorum, congenital cataract, or other deformities especially those affecting the macula, total colour blindness, albinism, congenital syphilis &c. The nystagmus is pendular, usually horizontal, but sometimes rotatory.

1(g). LATENT NYSTAGMUS. A rare condition first described by Faucon (1872) and Von Graefe (1878). It is elicited on covering up one eye, develops in the covered eye, and the nystagmus is in the direction of the open eye. It is nearly always jerky. The cause is unknown.

2. VESTIBULAR NYSTAGMUS.

This results from either stimulation of the vestibules, e.g., by rotation, heat, cold, inflation of the middle ear, or,

from disease of the labyrinths or their associated paths. Vestibular nystagmus is usually "jerky", the slow movement outwards being the true feflex whilst the jerk back may be regarded as merely corrective. Experiments by Bauer and Leidler (1911) and Magnus and de Kleijn (1923) show that vestibular nystagmus may be entirely subcortical and involve only the afferent impulses from the labyrinth, the oculomotor grey column and the efferent oculomotor nerves. The existence of such a path is shown when considering the "Labyrinthine Reflex."

3. OCCUPATIONAL NYSTAGMUS.

This is typically seen in Miners.

4. REFLEX SENSORY NYSTAGMUS.

This may occur on pinching or pulling the skin of or about the ear. It is horizontal and towards the side stimulated. The cause is unknown. (Borries 1923).

5. REFLEX ACOUSTIC NYSTAGMUS.

Occurs from stimulation by a loud sound. (Urbantschitsch 1910). It may be due to a direct vibratory stimulation of the labyrinths. (Tullio 1924).

6. CENTRAL NYSTAGMUS.

This results from some lesion of the vestibular nerve or its secondary connections after it has entered the brain. It occurs, therefore, in diseases of the medulla, pons, mid-brain, and cerebellum. It is nearly always jerky, and may be horizontal, vertical or rotatory. It is not usually spontaneous and usually develops only on eccentric fixation.

7. VOLUNTARY AND HYSTERICAL NYSTAGMUS.

This can be diminished by distracting the patient's attention. It is increased on fixation and is pendular in character.

8. IDIOPATHIC AND HEREDITARY NYSTAGMUS.

This is usually horizontal and pendular, and occurs in otherwise normal persons perhaps inclined to be neurotic. It is transmitted hereditarily.

AETIOLOGY AND PATHOGENESIS OF MINERS' NYSTAGMUS.

That the cause of nystagmus is not yet finally settled is the view still held by some authorities, although the general consensus of opinion is in agreement with the views expressed thus in the First Report of the Miners' Nystagmus Committee (1922), "The essential factor in the production of Miners' Nystagmus is deficient illumination;" and, "Miners' Nystagmus is a disease of extraordinary complexity, and it is probable that the conditions which have at one time or another appeared to observers as the sole aetiological factor nearly all have an influence in the production of the disease."

Not only has there been an almost complete repudiation of the earlier accepted cause (i.e., "constrained position") of the disease, but the disease itself has, in Law, completely changed its character. When first added to the Third Schedule of the Workmen's Compensation Act 1906 on May 22nd., 1907 it was described as "Nystagmus". This description was altered on July 30th 1913 to, "The disease known as Miners' Nystagmus, whether occurring in miners or others, and whether the symptom of oscillation of the eyeballs be present or not." (underlined I.C.) A further alteration of the description to "Miners' Nystagmus", is suggested in the Departmental Committee's Report of 1938.

Before considering the causal factors, all of which have, or have had, their supporters, a review of the literature is necessary. This must, to be accurate, be given in some detail as what appeared to one observer to be the sole cause, may be accepted by another authority as being only, though perhaps an important predisposing cause.

Though Snell stated that a Dr. Gillett of Sheffield recognised the disease as early as 1854, and Neiden stated that Peppmuller described cases in the period 1860 to 1863, and C. Turner Thackrach writing of colliers in 1832 says, "Their eyes from swelling of the lids appear small, are affected with chronic inflammation and are intolerant of full light, and many, after a few years trial, are obliged by the injury which their health has sustained, and especially by the weakening of their eyes, to leave the mine," the distinction of being the first to record the disease is held by Deconde (1861).

Deconde considered that anaemia and alcoholic excess were the chief aetiological factors.

Arlt (1867) contended that the movement of the eyes in miners' nystagmus was an attempt to bring a fresh part of the retina to bear on the same point in the interest of vision. Later (1891) he agreed with Snell that position was the prime cause.

Snell (1875 onwards) was emphatic and stated "The essential and prime cause is to be found in the kind of work performed (i.e., "holing") in which the gaze is directed upwards in a more or less oblique manner, whereby fatigue is induced in the elevator muscles of the eyeballs and the internal and external recti. It occurs irrespective of the mode of illumination." He considered it solely a local myopathy.

Bell Taylor (1875) decided, "Miners' Nystagmus appears to be a pure myopathy and strictly analogous to Writers' Cramp. There is no central lesion." He considered, however, that deficient light increased the strain on the eye muscles.

Romiee (1878 onwards) after most comprehensive investigations concluded that deficient illumination was the chief factor in producing the oscillations. He showed that the incidence of nystagmus varied inversely with the illumination.

Oglesby (1880) considered the disease to be of central origin. He states, "As wet and cold are the immediate cause of so many serious diseases of the spinal cord, we may, I think, say that the frequent immersion of the lower half of the body in water is the primary cause of the disease." He further points out that working "when the head and neck are much bent" causes venous engorgement of the medulla, consequent on the pressure of the large blood vessels of theneck." He concludes..... "venous engorgement is the chief factor in this curious phenomenon."

Wilbrand (1884) was the first to assume an equilibration centre of the eyeball in the mid-brain. He associated nystagmus with the want of harmony between the centre for the common reflex action of the eyeballs and the volitional impulse.

Jeaffreson (1887) advanced a theory somewhat resembling the views of Oglesby and Wilbrand. He considered the constrained position of the miner was the chief aetiological factor by producing

a. "Cerebral anaemia" (flexion and extension of the head interfering with the circulation of the blood through the vertebral arteries; extension of the head causing the anterior free margin of the tentorium cerebri to press on the upper part of the pons) and

b. A dissociation of movements normally combined ("The miner is obliged to direct his gaze above the horizontal median plane and at the same time to flex his head upon his chest")

He considered the oscillations "only one out of a number of symptoms which point to some central change in the nervous apparatus" and was the first to suggest that thedesignation "Miners' Neurosis" would be more applicable.. He did not consider that defective illumination was a factor.

Dransart (1887) stated that the prime cause was the elevated visual regard, whilst the chief secondary cause was defective illumination. He believed (1913) that with improved light the primary cause would cease to be an aetiological factor.

Tathan Thompson (1891) pointed out that "the question of insufficient light stimulation of the retina bears at least an equal if not a greater claim" [i.e., than strain of the ocular muscles). He considered nystagmus the result of imperfect stimulation of the co-ordinating centre, and that "strain, either mental or muscular, anything that brings about either general bodily or nervous exhaustion, must necessarily increase the tendency to the disease."

Court (1892) expressed views directly opposed to Snell, whose "constrained position" theory held sway until 1912 when the Home Office officially endorsed Court's views. Court believed, "It is the want of light that is the only cause of the mischief." Court has the distinction of being the first English observer to declare this view.

Pechdo (1893) suggested that "gas" poisoning, from some unknown hydrocarbon might be the cause. He advanced this theory because of the much greater prevalence of nystagmus in safety-lamp pits than in naked light pits.

Neiden(1894) although finding the incidence of nystagmus nearly a hundred times greater in safety-lamp pits than in candle pits following an examination of over 11,000 men, stated, "The first prime cause of this affection is the particular kind of work which the hewer has to do in holing, in a stretched position of the body, head and eyes." He considered, however, that lack of light was a necessary factor.

Peters (1907) introduced a labyrinthine disturbance theory. He stated, "Nystagmus is caused by afferent impulses from the semi-circular canals whose function has been modified by the position of the miner at his work. It is due to irritation in the vestibule associated with the redistribution of the endo-lymph in the vestibule, when the head is bent backwards." He, therefore, from a different aetiological view point, gave support to Snell's "constrained position" theory.

"Departmental Committee's Report on Compensation for Industrial Diseases" (1907). This Committee apparently convinced by the evidence of Dr. Snell, reported "This disease..... is due primarily to fatigue of the elevator muscles of the eyes from the constrained position, in an obliquely upward direction, in which the eyes have to be kept."

Trombetta (1907) also considered labyrinthine disturbance the main factor. He suggested that this was caused by the alteration of pressure, and the incessant blows of pick and shot-firing.

Nuel (1908) considered that bad light was as important a factor as the constrained position. Later he supported Christie Reid's theory. (q.v.)

Rutten (1908) supported Jeaffreson's theory.

Elworthy (1910) considered that insufficient lighting and the entire absence of colours were the main causes, producing "fatigue of the eyes". This fatigue, resulting in a breakdown of the co-ordinating mechanism of the eyeballs, was further aggravated by glare from other miners' lamps. He suggested also that the presence of ultra-violet light rays (twice as numerous in electric lamps as in oil lamps and candle lights) accounted for the incurable cases of nystagmus, due to their exhausting and irritating effect on the retina, with resultant "burns".

Weekers (1910) suggested failure of dark-adaptation as the cause.

Harrison Butler (1912) supported both the deficient light theory and the absorption of poisonous gases given off from the coal.

Coppez (1912) made tracings of the nystagmic oscillations and concluded that they were due to fatigue, producing a form of clonus in the voluntary muscles of the eyeballs. He held that the oscillations were not influenced by the labyrinths.

Brown and Ross Mackenzie (1912) considered that "the factors contributing to the production of nystagmus in miners, arranged in order of relative frequency and importance were,

1. "Inadequate light.
2. Errors of refraction.
3. Straining of the ocular muscles.
4. Neurotic temperament."

Norman (1912) declared, "Miners' Nystagmus is a perversion of the function of central fixation due to the conditions under which the coal miner works, but determined by a personal factor which, in the majority of instances, is an error of refraction." The pathogenic working conditions he considers are,

1. "Defective illumination in the pits" and
2. "Cramped or stooping position at work" producing fatigue of the elevators.

Llewellyn (1912) believed, "It is the long continued inability to make use of foveal vision that causes mine workers to develop nystagmus after years of work underground."

Coulter (1914) considered "gas-poisoning" a factor.

Shufflebotham, (1914) like Jeaffreson, considered nystagmus "a general disease, one symptom of which is oscillation of the eyeballs." He considered that the chief aetiological factors were bad light, position, refractive errors, and injuries, especially to the eye itself. He considered the good type of workman more prone to develop the disease.

Stassen (1914) stated, "The real cause of nystagmus must be sought, not in the upward look, but solely in the faulty lighting conditions" (1922). He emphasised the importance of both local and general strain e.g., the shock of increased atmospheric pressure induced by the rapid lowering of the cage, the swift plunge into darkness before the eyes have got a chance to become accommodated, the ocular strain of working in deficient light, and the constant struggle between two visual fields, as each eye fixes a different facet of the crystalline fracture of the breaking coal. He believed, "all these factors produce a nervous syndrome characterised by inco-ordination and exaggeration of the visual reflexes." Like Coppez, after a study of nystagmographs, he concluded that the oscillations were not the result of labyrinthine disturbance.

Ohm (1916) stated (1918) "This bewildering array of phenomena (i.e., the oscillations) is the result of a disturbance of the vestibular (underlined I.C.) control of the eye muscles," and other groups of muscles, due to lack of light and other unfavourable conditions of work which bring about a too strong and too infrequent innervation of the labyrinths. It was a disorder of muscular tone. He found the disease varied inversely with the illumination and directly with the amount of labyrinthine disturbance. After shutting up puppies and kittens in a dark cellar for 10-48 days he found oscillations in all of them, the tracings of which resembled those of miners' nystagmus. He considered that disturbances of dark-adaptation, alcoholism, and stature above the average were important predisposing factors, whilst errors of refraction and injuries to the eye had no causal bearing.

Oliver (1916) in part returned to Snell's idea. He stated, "It is due primarily to fatigue of the elevator muscles of the eyeballs when the men are working in a constrained position in a dim light and are looking upwards in an oblique direction."

Anderson (1920) made the remarkable statement, "Improvement of the illumination will unquestionably reduce the strain, and at the same time the incidence, of nystagmus, but will never stamp out the disease from our mines whilst refractive errors exist amongst our colliers."

Leighton Davies (1920) declared that the oscillations were "part and parcel of a general toxaemia" and suggested this might be caused by either "gas" poisoning or bacterial infection.

Martin (1920) considered "the disease is essentially one of exhaustion." He accepts that "deficiency of light is the exciting cause" whilst the main predisposing causes are,

1. "Nature of work done".
2. "Excess of alcohol and tobacco."
3. "Illness."
4. "Accidents, especially to the eyes."
5. "Heredity."
6. "Exhaustion due to want of food, and prolonged strain without rest is the most important, most effective predisposing cause of the disease."

Christie Reid (1920) declares, "There is no evidence of strain or fatigue. What has taken place is that the afferent impulses that control the normal equilibrium of the eyeballs have been put in abeyance in the dark, and replaced by other disordered impulses, as from the rhythmic swinging of the pick, acting through the semi-circular canals."

Kestenbaum (1921) suggested, as did Ohm, that "the normal minute ocular excursions present in the active fixation reflex become less minute and thus visible as nystagmus when fixation is insufficiently developed."

Priestley (1922) accepts deficient illumination as the exciting cause but considers "refractive errors of eyesight are the great predisposing cause."

"The First Report of the Miners' Nystagmus Committee" (1922). This is largely the work of Haldane and Llewellyn. The Committee unanimously concluded, "The essential factor in the production of Miners' Nystagmus is deficient illumination."

"The Second Report of the Miners' Nystagmus Committee" (1923). This is written by Pooley and amplifies the First Report by stating, "Errors of refraction in themselves have no effect whatever on the incidence of Miners' Nystagmus....."

Healey (1923) considers the oscillations of the eyeballs the result of "a physiological effort to see" in a degree of illumination which allows only of rod vision.

Pooley (1926) states, "Its causes I still regard as unsettled, the existing evidence is strongly in favour of its being a compensatory effort on the part of the central nervous system to overcome the effect of working in deficient light."

Robson (1923) after an exhaustive study of the incidence of nystagmus in coal mines concludes, "The incidence of nystagmus varies directly with the percentage of volatile matter in the coal." "It is conceivable that toxaemia from CO or some symbiotic combination of gases may be the chief cause or genesis of nystagmus...

Gallant (1924) and Cridland (1925) supported the "gas poisoning" theory.

Freeland Fergus (1925) stated that the disease might be due to a specific infection. It did not seem to be due to posture or defective illumination.

J. Prosser Davies (1926) suggests that nystagmus may be due to "the lowering to great depths at very high speed of those engaged in the mining industry." He considers that, "a rapid vertical descent of the body as a whole through space may set up a functional disturbance of the labyrinths and cerebellum and so produce nystagmus."

Cooke (1931) made the original observation that in fifty seven nystagmic miners examined, the Arneth Count deviated to the left. From this he concluded that, accepting the essential cause of nystagmus to be defective illumination, some "activating" or "precipitating" factor was necessary for it to become manifest and this factor was some "endogenous source of toxaemia", e.g., pyorrhoea alveolaris, septic tonsils &c.

Roche (1931) considers there are three causal factors in the production of nystagmus.

1. Defective illumination, causing "the reserve nervous energy of the retina to be used up". "This manifests itself in inco-ordination of the eye movements."
2. "Malposition at work" causing irritation to the labyrinthine system with alteration of reflex efferent impulses to the ocular and other muscles.
3. Heredity.

Lane (1931) states, "While there is evidence to show that light determines the incidence of the disease, I have found more nystagmus where the temperature goes above 72°F, the wet and dry bulb readings approach each other, and where there is a poor circulation of air."

Stephens (1932) considered that the large majority of miners have evidence of a disturbed metabolism with raised basic blood pressure which "is followed by a disturbed exudation so that the nerve tissues are not fed satisfactorily and so do not exercise their usual control over the body movements." Replacement of fresh foods by tinned foods was largely responsible for the disturbed metabolism.

"The Third Report of the Miners' Nystagmus Committee" (1932). This agrees with the First Report and considers that, "the fundamental cause of miners' nystagmus in the narrower sense of the term (i.e., oscillations of the eyes) is the low illumination under which the miners in general work." Millais Culpin emphasises the part played by the accompanying psycho-neurosis in causing the other symptoms of the disease, e.g., defect of visual acuity, night-blindness, photophobia, forced ocular movements, tremors of the head and limbs, and disordered action of the heart.

Zeiss (1932) postulates two cerebral centres,

1. "Position keeping," and upset by dim light, and
2. "Co-ordinating," and upset by chronic gas poisoning.

His gas poisoning theory suggested itself because of his finding a direct correlation between the incidence of nystagmus and the concentration of carbon particles in the air.

Alabaster (1933) advanced a theory that in the journeying up and down in the cage, infection was sucked up the Eustachian tubes resulting in labyrinthine upset.

Ferguson (1933) declared, "I am convinced that the sole cause of nystagmus lies in defective light interfering with macular vision." "The lesion of miners' nystagmus is to be found in the eyes themselves and nowhere else."

Wiedershein (1934) supported Zeiss' theory.

O'Sullivan (1934) stated, "Illumination, however, cannot be accepted as the sole cause." "The essential cause of the disease has not yet been definitely established." From observations made, he concludes that, "repeated rises in blood pressure caused by descent into mines, play a part in the aetiology of Miners' Nystagmus", presumably by causing cerebral hyperaemia.

Boyd (1936) suggests that the disease has a definite pathological background rather than being a functional disease of the nervous system.

Brock (1938) considers that the low illumination and the constant sense of danger bring about physiological and psychological breakdown resulting in the complete picture of miners' nystagmus. "It is a breakdown of the man's ability to withstand the stress of the mine."

"Report of the Departmental Committee on certain questions arising under the Workmen's Compensation Act." (1938). This Committee, considering miners' nystagmus, reported, "There

is no longer any room for doubt that this (low illumination) is in fact the main determining cause." They consider the oscillations the result of a physiological effort to see, "which become perpetuated by habit spasm."

Caiger (1939) considers that defective illumination produces the oscillations, but "the breakdown of a normal inhibitory mechanism" produces the incapacity. In 75% of cases this breakdown is the result of "industrial fear" ("including fear of long continued unemployment, and chronic financial stress for himself and his family and finally the prospect of losing his job permanently"); "the other potent fear is fear of blindness."

Ridley and Sorsby (1940) consider the oscillations to be an exaggeration of the normal minute ocular movements, due to insufficient stimulation of the fixation reflex because of insufficient illumination and absence of surfaces differing in illumination or colour.

The review of the literature on Miners' Nystagmus shows that whereas some consider it a local disturbance confined to the eye muscles themselves, others consider it a general nervous disease, functional or organic. Let us consider the various views in detail.

Snell's "constrained position" theory can, I believe, be confidently ruled out as the essential cause of nystagmus. This position is adopted by the miner when "holing", and is described by Snell as "a recumbent one with the head upon the right shoulder" and the eyes directed obliquely upwards. He considered this resulted in strain, then atony, of the elevator and internal and external recti muscles of the eyeballs, which manifested itself in the oscillations. That such a position would result in strain and fatigue of the elevator muscles no one will deny but nystagmus is found frequently in men who have never adopted this position. This has been my personal experience. In most of the seams in this area "holing" is never done yet I have seen well marked nystagmus producing incapacity among men who have always worked in such seams. Of 2000 cases reviewed in the First Report of the Miners' Nystagmus Committee, over 50% had done little or no "holing". Tathan Thompson (1891) observed that in thin seams in naked light pits, holing is commonly done but nystagmus is rare.

Snell's "holing" position theory is, therefore, not supported by facts. It must be recognised, however, that even

in thick seams, the miner, both at work and in going to and from work, may be in a cramped position. In such cases, however, except when looking at the roof, I have found the gaze directed more generally forwards, or downwards and forwards, though the constraint on the skeletal muscles generally and the neck muscles especially may be obvious. Such a position, however, is assumed by workers in occupations other than coal mining where nystagmus is unknown. Elaboration later of the neuro-muscular mechanism involved in the production of nystagmus confirms my belief that a constrained position, whether that described by Snell or otherwise, though not the essential cause of nystagmus, should be included among the predisposing causes.

Inadequate illumination is now accepted by most, though not all, authorities as the essential cause of nystagmus. Romiee (1878), Court (1892), Llewellyn (1912), Ohm (1916), and Ferguson (1929) are among the observers who have made personal investigations into this factor, and the evidence produced is convincing. Miners' Nystagmus is confined to coal mines, or to metalliferous mines in which a coal seam is present, but is mainly found only in such mines where safety lamps are used. Though nystagmus does occur in candle pits, its incidence is low and resultant incapacity still lower (Stassen). Where really adequate methods of lighting are possible, as in America, (Owings 1934) nystagmus is unknown. Also, the incidence of nystagmus among miners not incapacitated by it, which is, as will be shown later, the only accurate guide as to the true incidence of the condition, varies directly with the illumination (Romiee (1878), Court (1892), Neiden (1894) and others).

Further, Llewellyn (1932) and Ferguson (1929) have shown that after improving the illumination under which a given group of miners, affected with oscillations, worked, the incidence of nystagmus was reduced, in this group, whilst many of those who still had oscillations were affected to a lessor extent. In these experiments the only change made in the working conditions lay in increased illumination. Such observations appear to me to leave no reasonable doubt that the essential cause of nystagmus is inadequate illumination.

Reference to the various reflexes later described explains how the faulty lighting may result in the oscillations. As to why it may do so is explained by a brief consideration of the physiology of vision.

The human eye is adapted for clear form vision only when the degree of illumination is sufficiently strong to allow the specialised cone elements in the fovea centralis and perifoveal area to function. When the illumination of the retina

falls below a certain level, vision is mediated exclusively by the rods in that part of the retina without the macular area. Whereas stimulation of one cone, in virtue of it having its own nerve connection, results in the projection of a clear image, stimulation of several rods is required, in virtue of there being only one nerve connection to several rods, before a reasonably intense image is projected. In night-prowling animals this is afforded not only by the hunter and the hunted moving, but is farther facilitated by each rod having a separate nerve filament. In man perception of a stationary object, when vision is mediated exclusively by the rods, will be facilitated by projecting the image slightly to one side of the fovea, either by moving the head, the eyeballs remaining stationary, or by moving the eyes, the head remaining stationary. This latter action is accomplished by the oscillations of Miners' Nystagmus. This "physiological" or "compensatory" nystagmus theory is, in my opinion, the significance of the views of Arlt (1867), Ohm (1916) Healey (1923) and others.

Other observers (Ohm (1916), Christie Reid (1920), Roche (1931) and others) hold that the bad lighting of the pits results in inadequate stimulation of the fixation reflex, (q.v.) allowing the labyrinthine reflex (q.v.) to become dominant. The unopposed labyrinthine reflex is stimulated by constrained position, jarring effects of picking coal, &c., resulting in inco-ordinate movements of the eyeballs.

Another explanation concerns the optical reflex (q.v.) When foveal vision is abolished, as demonstrated by Ohm in keeping puppies in the dark, the normal optical reflex is insufficiently stimulated and the tone of the ocular muscles is lessoned. This results in an exaggeration of the normal minute oscillations which then become visible as nystagmus.

The fixation reflex is further strained when the miner, whilst concentrating on one point of the dark coal face, has brighter illuminated point-images, owing to the crystalline fracture of the coal, projected into consciousness, thus tending to alter the direction of gaze. This tendency is readily appreciated if one endeavour to concentrate one's gaze on a star-free patch of a starry sky.

The essential factor in the "deficient light" theory is that the illumination under which the miner is compelled to work is such that foveal vision is rendered impossible.

The questions now arise:-

(1) What is this threshold of foveal vision, below which vision is mediated by the retina periphery?, and

(2) What intensity of illumination does the miner have to work under? If it can be shown that the miner generally works with a degree of illumination rendering foveal vision impossible, then this explanation for the almost undoubted fact that bad light is the essential cause of nystagmus may reasonably be accepted.

(1) What is the threshold of vision?

The Miners' Nystagmus Committee 3rd Report (1932) accept the evidence of J. H. Shaxby to the effect that the "critical illumination" of the retina, i.e., the intensity of illumination above which we have foveal vision, below which, peripheral vision, is represented by 0.0068 ft. candle. As it is not known just how much above this figure the illumination must be to ensure that foveal vision is maintained, they double this critical figure; hence they contend that if the retinal illumination is 0.0136 ft. candle, foveal or macular vision will be complete. They further contend that if the surface brightness of the coal face is 0.0136 ft. candle, then, the miner looking at this, foveal vision will be maintained.

Forbes Sharpley (1936) points out, however, that retinal illumination is a function of the pupil as well as of the objective brightness. This was brought to my notice in a striking manner in the course of the experiment made on J.O. ae. 35 described in detail later. When the man's pupils were fully dilated and fixed with homatropine he stated he could see the coal face better than he had been able to do for some years. Also, a Home Guard who had occasion to have one pupil only dilated found such an appreciable difference in dark vision in his eyes that he suggested all Home Guards should have their left pupil dilated when going on night duty, the left eye to be used when observing, the right, when shooting!

The only unit which allows for this is the photon, introduced by Troland (1916) and defined by him as "the illumination of the retina corresponding with a stimulus--- surface brightness of one candle per square metre, and an effective pupillary area of one square millimetre." Whereas the Committee Report above mentioned, gives the surface brightness illumination in foot candles, by which, I interpret, they mean the total illumination from a point on a surface, Sharpley's surface brightness units are, more accurately, (since "foot candle" is a measurement of intensity of illumination falling on a point) "candles/sq. foot", which, I interpret, is the total illumination from, or objective brightness of, 1 sq. foot of a surface, one point of which has been subjected to a given intensity of illumination as measured in foot candles.

Sharpley, whose observations are, I think, of considerable practical importance, reviewing the original work of Konig (1897) Hecht (1924), Porter (1902), Ives (1912) and Lythgoe and Tansley (1929) found that the average threshold figure given by these observers for foveal vision is represented by an objective brightness of 0.0026 c/sq. ft., and that the point where the change over from rod to cone vision is complete is 0.0035 c/sq.ft. He suggests, accepting these figures, that an objective brightness of 0.004 c/sq.ft. would ensure the maintenance of foveal vision in the subjects examined. He observes, however, that these investigators did not take into account the normal variations in size of the pupil. Accepting that their subjects had normal sized pupils, 0.004 c/sq. ft. would correspond to a retinal illumination of 0.89 photon. To give this illumination in a subject with the smallest normal size pupil as given by Lythgoe (1932) an objective brightness of 0.01 c/sq. ft. would be required.

In brief, therefore, Sharpley's work shows that to ensure the maintenance of foveal vision in miners with average sized pupils, the coal face must be illuminated so as to give a surface brightness of 0.004 c/sq. ft. In miners with smallest normal-size pupils, the surface brightness would require to be 0.01 c/sq.ft.

The results of these two observers can be easily translated into terms of intensity of illumination required at the coal face by the formula $E = \frac{\pi B}{Y}$ where E is the intensity of illumination required in foot candles, B = the surface brightness in c/sq. ft., and Y = the reflecting factor.

With regard to the reflecting factor, coal absorbs 88-97% of all incident light (Llewellyn 1912). Calculations I have made show that the Miners' Nystagmus Committee 3rd Report estimate the average reflecting factor as 5.4%. Though this may appear low, it allows in our final calculations (to estimate the intensity of illumination necessary at the coal face) for absorption of light by the coal-dust laden atmosphere, dirty lamp glasses, &c. Accepting a reflecting factor of 5.4% the Miners' Nystagmus Committee consider that an intensity of illumination of 0.25 ft.c. is required to maintain foveal vision. Calculations I have made accepting Sharpley's surface brightness figures and a coal face reflecting 5.4% show that an intensity of illumination at the coal face of 0.23 ft.c. and 0.58 ft.c. is necessary for miners with average and smallest normal-size pupils respectively. As the pits employ underground about 600,000 men in this country it is reasonable to assume that an appreciable number have the smallest normal-size pupil, so

to ensure foveal vision in all miners the illumination necessary for those with smallest size normal pupils should be provided i.e., 0.58 ft.c. at the coal face. This now brings us to our second query.

(2) What intensity of illumination does the miner have to work under?

Llewellyn published photometric measurements taken up to 1912 and found that the average illumination at the coal face in safety lamp pits was 0.018 ft.c., and in candle pits 0.09 ft.c. The Coal Mines General Regulations (Lighting) 1934 ensured that by the end of 1936 a complete change over to new type lamps giving greatly improved illumination must have been effected. The best type of safety hand-lamp in use is the 4.5 Candle Power Alkali hand lamp. In this area these are represented by the Ceag and Concordia makes. Photometric tests at the coal face show, with these lamps, an intensity of illumination of 0.091 ft.c. (Jones 1931). With the 2-volt cap-lamps in use in this country (in some pits only), photometric tests at the coal face show an intensity of illumination of 0.30 ft.c. after allowing for the angle of incidence, and the lamp 2' 6" from the coal face. (Barber, Emnett and Jones 1933). These cap-lamps therefore, though they provide foveal vision for the miner with average size pupil do not necessarily do so for the miner with smallest normal-size pupil. Further, in the South Yorkshire Coalfield, the use of the cap-lamp is confined to haulage hands and is not used by the collier. According to Owings (1934), the cap lamps in general use in American mines, where, it may be noted, cap lamps have always been used and nystagmus is unknown, give an intensity of illumination at the coal face of 6.55 ft.c. and 1.85 ft.c. for the new and old type lamps respectively. It is shown therefore that apart from the use of the cap lamp, the miner, even with the new type hand-lamp works with a degree of illumination which renders foveal vision impossible. This, it will be remembered, was the condition requiring to be shown obtained, before accepting deficient light as the essential cause of miners' nystagmus. I believe this theory to be correct, and that Miners' Nystagmus as represented by oscillations of the eyeballs will continue to manifest itself until the illumination provided is such that the miner can continuously throughout his shift use his foveal vision. Elworthy's opinion that the absence of colours in the pit is an aetiological factor is, in my opinion, dependent chiefly on the fact that colours other than black would reflect more light.

That nystagmus was due primarily to labyrinthine disturbance has been suggested by Peters (1907) and Trombetta (1907), the former considers this the result of position at work, the latter, of the incessant blows of pick and shot-firing, to which might now be added coal-cutting machines. That these factors are the essential cause of nystagmus can, I think, be confidently ruled out in that workmen in occupations other than coal miners who are subjected to the conditions these observers have described, do not develop nystagmus.

That such conditions may be contributory factors, I accept. The answer as to how they may influence the movements is found by a study of the Pathology of nystagmus. This labyrinthine influence, becoming dominant because of deficient light, is held by Ohm (1916) Christie Reid (1920) Roche (1931) and others.

That disturbance of cerebral circulation might be the essential aetiological factor was suggested by Oglesby (1880). His theory is dependent chiefly on the position of the miner at work and for this reason, as already discussed, can be discarded. That vascular disturbances are important contributory causes is upheld by Jeaffreson (1887) and O'Sullivan (1934).

Failure of dark-adaptation as the cause of nystagmus is suggested by Weekers (1910). This factor with its resultant hemeralopia, I have investigated somewhat fully.

The association between failure of dark-adaptation, or night-blindness, and Miners' Nystagmus has long been recognised, and commented upon by many observers. C. Turner Thackrach (1932) writing of colliers remarks, "their eyes.....are intolerant of full light." Court (1892) states "a definition of the disease (i.e., Miners' Nystagmus) should include night blindness, nystagmus.....because these symptoms all occur together in miners working with the inferior light." The First Report of the Miners' Nystagmus Committee (1922) states, "This failure of dark adaptation, or night-blindness, is undoubtedly one of the most characteristic symptoms of the disease", whilst Roche (1931) states, "One of the first symptoms of nystagmus is deficient vision at twilight. All miners suffering from nystagmus complain of twilight blindness."

That hemeralopia is found in the "pre-nystagmus" stage is observed in the Departmental Committee's Report on the Workmens' Compensation Act (1938). ".....there are some men who ultimately develop oscillations of such an extent as to become cases of miners' nystagmus who have complained of earlier subjective symptoms such as mal-projection, intolerance of light from lamps of other workers, difficulty of seeing on entering the mine workings or leaving them....."

Failure of dark-adaptation, it has already been noted, has even been suggested (Weekers 1910) as the cause of miners' nystagmus (1st Report Miners' Nystagmus Committee 1922). Oglesby (1880) states, "I was at first impressed with the idea that pigmentation of the retina lay at the root of the mischief" but discarded his theory on negative ophthalmoscopic findings.

Ohm (1916) considers that "Disturbances of dark-adaptation have proved factors which predispose to nystagmus."

Though most observers have noted the occurrence of night-blindness in miners' nystagmus, few have suggested an explanation of it. Jeaffreson (1887) states, "The interference (i.e., obstruction of blood flow through the vertebral arteries) with the nutrition of the visual centres may be one of the chief features in the production of hemeralopia which is almost always present."

Roche (1931) suggests that it is probably due to two causes:-

(1) "The stratum pigmentum produces visual purple. As a result of continuous over production these cells become defective and nearly cease to secrete altogether, or they secrete a visual purple which is too diluted and ceases to stimulate the rods on exposure to light," or

(2) "Normally, several rods have only one central nerve connection, and continued use provokes fatigue, and consequently poor transmission of impulses."

The Third Report of the Miners' Nystagmus Committee (1932), on the other hand, considers the night-blindness of miners' nystagmus among the psycho-neurotic manifestations of this disease.

Recent work done on the association between deficiency of Vitamin A and night-blindness lead me to consider the association of diet and miners' nystagmus. On this subject Martin (1930) believes that "exhaustion, due to want of food and prolonged physical strain is the most important, most effective predisposing cause of the disease," whilst Stephens (1932) considers the nature of the miners' food all important. His views he expresses thus, "Since the introduction of tinned foods and preserved meats, the feeding of the collier is much below what it used to be. Formerly the Welsh collier used to live on good home-grown produce, with milk, eggs and cheese.....(underlined I.C.). To-day ill-feeding tinned foods are the chief features of the household board in a large number of homes, and it is from these homes that the army of nystagmus patients is recruited."

Investigations of the diet of miners in this area show that with the improvement in housing conditions giving most miners a garden of their own in which they are keenly interested and proficient, the supply of Vitamin A (or its precursor) from green vegetables, carrots &c. is, in the summer months, plentiful, but in the winter months, when this home-grown source is at a minimum, the intake of Vitamin A is greatly reduced. It is just at this time also that the seasonal variation in the Vitamin A value of dairy produce manifests itself. "The Vitamin A value of milk, butter and eggs tend to be at their lowest in the early part of the year....."("The Vitamins" Crookes Laboratories.)

Further, when the intake of Vitamin A is at its lowest, the demand for it, in the interest of vision, is at its highest, for the miner, in the dark winter months, has a greater need for dark-adaptation <u>apart</u> from the time actually spent below ground.

It is interesting to note that many observers have found miners' nystagmus to be more prevalent in the dark winter months. The following Table, from the First Report of the Miners' Nystagmus Committee, supports this view. For the last entry I am indebted to Mr. W. E. Gillhespy of the Yorkshire Coal-Owners Mutual Indemnity Society. The entries I have converted into percentages.

<u>Cases arranged according to the Quarter of the year in which failure took place.</u>

	No. of cases.	1.	2.	3.	4.
Dransart & Famechon	201	33.33%	17.91%	17.41%	31.34%
Ohm	521	30.51%	26.29%	20.34%	22.64%
Llewellyn	1649	34.26%	18.79%	22.61%	24.36%
Yorkshire Coal-Owners Indemnity Society. (1930-1939 inclusive).	1847	30.44%	26.06%	22.19%	21.24%

It is now generally recognised that the essential factor in Miners' Nystagmus is deficient illumination and that "during all the years when the miner is working at the coal face his macular vision is in more or less complete abeyance." (Departmental Committee 1938). This means that vision is mediated by the rod elements of the retinal periphery which calls for an adequate secretion of visual purple which, in turn, demands an adequate supply of Vitamin A.

The following experiment was carried out with the use of Crookes Dark Adaptation Tester. The Crookes Dark Adaptation Tester is an instrument designed for testing the rate at which the eye recovers its power to distinguish a faintly lighted object following a period in which it has been subjected to strong illumination, and also to discover the threshold of visibility after complete adaptation.

Light control is by means of two polarising discs, one fixed and one moveable, the angle between which determines the amount of light transmitted to the test object. (vide. Trans. Ophthalmol. Soc. Vol. LVIII, Part 1, 1938.) Three people living on an ordinary mixed diet were tested and an average curve was taken. This was accepted as the "normal" for the instrument used. Ten miners, all working, were selected at random apart from being of a "nystagmus age". The average age for the group was 38, and the average number of years worked underground 23. An average curve was plotted for this group. Three were selected from this group, one of whom,it was noted, had oscillations in daylight, at the horizontal level, before exercises. These were put on intensive Vitamin A treatment until each was able to see the arrow at the lowest finger post position used. An average curve was then plotted for this group.

The same technique was carried out in each case. Half an hour seated in the dark, four minutes "bleaching", and up to forty minutes examination. All the tests were carried out in January or February of this year (1941) and all were taken between 8 p.m. and 10.30 p.m. The finger post positions used were:-
Light Neutral Glass, 75, 50, 30, 10, 5.
Dark Glass, 30, 10, 5, corresponding to an intensity of illumination of 0.7, 0.47, 0.28, 0.093, 0.047, 0.028, 0.0093, 0.0047 millifoot candles respectively. The results were:-

3 NORMALS.

FINGER POST POSITION.	NEUTRAL GLASS. 75	50	30	10	5	DARK GLASS. 30	10	5
	TIME IN MINUTES.							
L.C.ae.38. Housewife.	3.25	5.45	7.40	9.40	13.50	14.20	16.20	17.35
P.B.ae.16. Clerk.	3.35	6	6.15	9.25	11.20	13.20	14.55	18.20
C.V.G.ae.31. Schoolteacher	2.55	3.45	3.57	4.18	6.10	6.50	10.30	24.25
Average.	3.18	5.10	5.57	7.47	10.26	11.30	13.55	20.6

Note. L.C. and P.B. each had for 2 days before the test 4 capsules Vit. A (Oral) a day. C.V.G. neither had, nor was taking, any vitamin preparation.

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10 COLLIERS.

FINGER POST POSITION.	NEUTRAL GLASS. 75	50	30	10	5	DARK GLASS. 30	10	5
	TIME IN MINUTES.							
J.O.ae.35. Collier	9.15	9.30	12.10	13.55	24.30	27.20	____*	____*
P.E.ae.36. Collier	7.55	9.3	9.30	12.40	35.10	36.30	____*	____*
A.C.ae.36. Onsetter	8.35	8.55	10.10	12.10	14.59	15.55	23.15	____*
C.B.ae.43. Shot-firer	8.2	10.5	10.40	12.55	18.10	19.40	30.35	____*
W.S.ae.30. Loader	5.55	7.30	8.35	9.35	17.29	____*	____*	____*
H.O.ae.61. Packer	13.25	13.55	14.20	19.30	30.10	____*	____*	____*
G.S.ae.36. Trammer	4.28	5.10	6.5	8.43	11.55	12.20	21.10	____*
S.S.ae.42. Haulage	7.30	8.10	8.25	10.25	12.55	13.45	39.30	____*
L.J.ae.29. Collier	5.45	6.55	8.35	10.55	15.25	16.30	34.55	____*
A.M.ae.40. Collier	7.5	7.40	8.25	12.5	15.10	16.15	____*	____*
Average	7.47	8.41	9.41	12.17	19.35	23.49*	34.55*	____*

Note:- The times marked * signify that the arrow was not seen up to 40 minutes. To estimate an average time, the dark glass being used and the finger post position at 30 and 10, those failing to see the arrow after 40 minutes were counted as having seen it at 40 minutes. The last two average figures should therefore be greater than shown.

3 COLLIERS AFTER TREATMENT.

FINGER POST POSITION.	NEUTRAL GLASS. 75.	50	30	10	5	DARK GLASS. 30	10	5
	TIME IN MINUTES.							
J.O.ae.35. Collier	3.30	5.2	5.32	7.10	15.10	16.10	20.20	27.10
P.E.ae.36. Collier	5.30	7.10	7.45	11.10	13.5	17.20	20.50	38.50
L.J.ae.29. Collier	3.15	4.10	5.30	9.20	11.5	12.25	15.30	38.10
Average	4.5	5.27	6.15	9.13	13.6	15.18	18.53	34.43

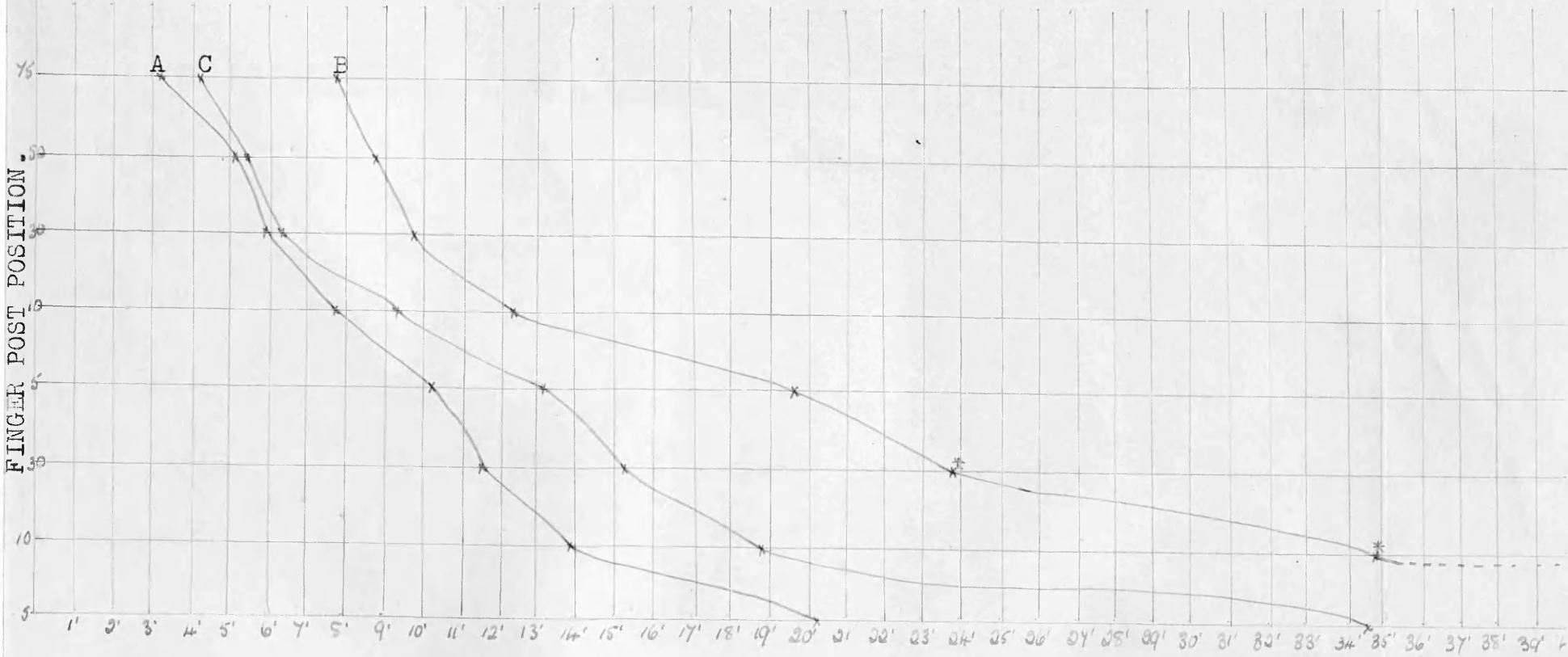
Note:- L.J's results were given after taking 6 capsules Vit. A (Oral) Crookes a day for 7 days; P.E's results after 3 capsules a day for 3 days followed by 6 capsules a day for a further 9 days; J.O's readings were given after 6 capsules a day for 27 days. He was examined weekly, and as already mentioned, had the oscillations of miners' nystagmus in daylight, at the horizontal level, before exercises.

Average 3 Normals.	3.18	5.10	5.57	7.47	10.26	11.30	13.55	20.6
Average 10 Colliers.	7.47	8.41	9.41	12.17	19.35	23.49*	34.55*	____*
Average 3 Treated.	4.5	5.27	6.15	9.13	13.6	15.18	18.53	34.43

These results are shown in the following graph.

Curve A = Average 3 Normals. Curve B = Average 10 Colliers.

Curve C = Average 3 Colliers after additional Vitamin A.



NOTE* At finger post position 30 and 10, in Curve B, two and five colliers respectively were unable to see the arrow after 40 minutes. These were included, for estimating an average time for the ten, as having seen the arrow at 40 minutes so that properly this curve should go further to the right.

Thesis Committee.

'PHONE
MEXBORO 3252.

FROM DR. IAN CAMPBELL.

CLIFF FIE[illegible] HO[illegible]
SWI[illegible] I, YORKS.

The Dean

Dear Sir,

About two months ago I lodged a Thesis o[illegible] Nystagmus. Since then I have been notified by British Medical Journal that they intend publishing observations I made. I would like, if possible, to make enclosed addendum, which could be pasted on after the [illegible] paragraph on page 30 ending "..... the peculiar tendency collier to develope Miners' Nystagmus"

Yours faithfully,
Ian Campbell

Hck. pc. 16.9.41

1941) for publication as an Original Journal.

[illegible] results were accepted (20th July, [illegible] These results were accepted by the British Medical [illegible] original article by the

These observations show that in the group of miners selected, a deficiency of Vitamin A, as demonstrable by the dark-adaptation test, was present, and by the administration of Vitamin A, over a limited period, the power of dark-adaptation was considerably improved.

As the miner depends for his working vision, and safety, on his retina periphery, and must continually adapt to low illumination and sudden changes in light intensity, it seems desirable that he should have maintained an optimum supply of Vitamin A. This need is further enhanced, in my opinion, by the peculiar tendency of the collier to develop Miners' Nystagmus.

Other aetiological factors producing metabolic disturbances have been suggested. Deconde (1861) suggested anaemia and alcoholic excess. Lane (1931) considered a warm, humid atmosphere and poor circulation of air at the coal face. Whilst accepting these as contributory factors they can, for the reason given in considering the "position theory", be dismissed, in my opinion, from the position of <u>essential</u> cause.

Whereas Snell considered that the position of the miner at work produced nystagmus due to local muscular fatigue of the eye muscles, Wilbrand (1884) and Jeaffreson (1887) considered that position produced a central nervous fatigue due to "dissociation of movements normally combined" (Jeaffreson) e.g., the miner flexes his head but <u>raises</u> his eyeballs. This, in their opinion, was the essential factor in the production of the disease, and as such, in my opinion, it can be discarded, for the same"dissociation" is encountered in occupations other than colliers where nystagmus has never been known. That it exercises a predisposing cause, I believe, and this can be readily appreciated by anyone who attempts, in a "constrained position", some fine adjustments underneath a car with a nine inch ground clearance. The phrase "to sit up and take notice" will then be found to have a sound physiological basis.

Many observers have suggested a toxaemic origin of the disease. This toxaemia has been variously considered to be gaseous, bacterial or endogenous.

Gaseous Toxaemia.

That nystagmus was due to toxaemia from the inhalation of some gas present in mine air, was suggested by Pechdo (1893) and supported by Harrison Butler (1912), Coulter (1914), Leighton Davies (1920), Robson (1923), Gallant (1924), Zeiss (1932), Wiedershein (1934), and others.

Haldane (1922) observes that we may rule out excess of carbon dioxide and diminution of oxygen in mine air as the cause, since both these factors are increased in the open light pits yet there nystagmus is practically absent. This leaves only two known gases to consider, namely carbon monoxide and methane. No point is gained by a consideration of "unknown hydrocarbons" or "symbiotic combination of gases" mentioned by some of the authorities above noted, since these have never been demonstrated. Haldane excludes methane as a factor since, he states, it is present only in about 1% concentration and is a physiologically inert gas like nitrogen.

The only gas we have to consider, therefore, is carbon monoxide, which is the offender suggested by most observers. Robson (1923) is the chief advocate in this country of this theory and has written a book on this question alone. The following quotations demonstrate his views. "There appears to be one outstanding basic factor largely responsible for the genesis of nystagmus and that is toxaemia, due to mine gases acting through the circulation upon a central synapse which may be in the mid-brain or cerebellum, and followed by a syndrome of symptoms." "It is not inconceivable that toxaemia from CO.....may be the chief cause or genesis of nystagmus in all forms in underground workers in coal mines."

That inhalation of CO in large quantities <u>can</u> produce oscillations of the eyeballs has been observed (Glaister and Logan 1914). Commenting on this, Haldane (1922) remarks "these movements are not comparable with the oscillation of the eyes found in miners' nystagmus." However, as to how CO might cause the oscillations of miners' nystagmus an explanation might, I suggest, be advanced that it interferes with the proper functioning of the reflex arcs described under the heading Pathology of Nystagmus (q.v.) A similar idea may have been envisaged by Robson's "central synapse" poisoning already mentioned. To accept that CO does cause, or is a factor in the production of, the oscillations, a consideration of the physiology of CO absorption is necessary. Haldane (1922) observes that "apart from its one very dangerous property of combining with haemoglobin, CO is simply a physiologically indifferent gas like nitrogen or firedamp (methane)." Its

affinity for haemoglobin is 300 times greater than oxygen's (Wright 1940) and the haemoglobin thus converted into carboxy-haemoglobin is thrown out of use in so far as its oxygen carrying power to the tissues is concerned. We have therefore produced a condition of anoxia. According to Wright (1940), however, the presence of COHb alters the dissociation curve of the functioning haemoglobin, so that the oxyhaemoglobin present will not as readily give off its oxygen. Further, (Wright,1931) there has been described an iron containing pigment present in the tissue cells themselves which acts as a catalyst in the splitting up of oxyhaemoglobin into available oxygen (for the tissues) and haemoglobin. This so called "Atmungs-ferment of Warburg" is itself fixed to a non catalytic compound in the presence of CO, so that the tissue cells cannot make use of available oxygen inthe blood.

We have thus described three ways in which COHb might interfere with the oxygen available for the myoneural systems (q.v.) involved in oscillations of the eyeballs.

This brings us now to the questions (1) Is CO present in mine air? (2) Is COHb present in the blood?
(1) Is CO present in mine air?

Haldane (1922) and Graham (1934) have demonstrated that CO is normally present in minute quantities, due to the action of coal or decaying timber on mine air. According to Graham (1921) the proportion present is seldom over 0.005%. At a most modern colliery in Yorkshire mine air is examined monthly for CO, and the following figures, for a working seam, representing percentage CO present, were shown me. 0.0012, 0.0014, 0.0008, 0.0004, 0.0008, nil, nil, 0.0008, nil, 0.0010, 0.0004, 0.0004, 0.0008. These percentages, I gathered, may be in some seams increased.

It may be accepted, therefore, that CO is normally present in mine air. We now come to our second question.
(2) Is Carboxyhaemoglobin present in the blood?

Robson (1923) states, "If CO can be demonstrated in the blood of miners even in small amounts, then I think we have gone a long way towards the discovery of the chief causal agent," but I can find no reference, in his book, or elsewhere, to any tests having been made for it. After correspondence with Professor P. L. Sutherland, he (Professor Sutherland) decided that the most suitable test for me to make would be the Wacholz test. The technigue used was that described by Hawke (1938).
Tannin Test.---"Divide the blood to be tested into two portions and dilute each with 4 volumes of distilled water. Place the diluted blood mixtures in two small flasks or large test-tubes and add 20 drops of a 10 per cent solution of potassium ferricyanide. (This transforms the oxyhaemoglobin into methemoglobin). Allow

both solutions to stand for a few minutes, then stopper the vessels and shake one vigorously for 10-15 minutes, occasionally removing stopper to permit air to enter the vessel. (This is done to free the blood from carbon monoxide hemoglobin). Add 5-10 drops of ammonium sulphide (yellow) and 10 c.c. of a 10 per cent solution of tannin to each flask. The contents of the shaken flask will soon exhibit the formation of a dirty olive-green precipitate, whereas the flask which was not shaken and which, therefore, still contains carbon monoxide hemoglobin, will exhibit a bright red precipitate, characteristic of carbon monoxide hemoglobin. This test is more delicate than the spectroscopic test and serves to detect the presence of as low a content as 5 per cent carbon monoxide hemoglobin."

I made the following examinations at Manvers Main Colliery. (I am indebted to Mr. Bell, the Agent, who on this and all occasions afforded me every facility, to Mr. Hall, Safety Inspector, for the collection of the samples of mine air and showing me the actual working conditions at the coal-face, and to Dr. Woolhouse, Mines Chemist, for the analysis of the air samples).

Six colliers were taken on coming up from underground at the end of a shift. Their ages were 23, 21, 23, 31, 47, 43, and the number of years worked underground was 9, 5, 9, 17, 34, and 28 respectively. I collected the blood from the arm veins and examined it by the method above described. In no case was the presence of carboxyhaemoglobin revealed.

The analysis of the mine air collected during this shift showed the following CO content for the samples taken.

SAMPLE.	1.	2.	3.	4.
% CO.	0.0012	0.0008	nil.	0.0016

The recent number of cases of compensated nystagmus from the particular "working" concerned was:—

YEAR.	1936	1937	1938	1939	1940
CASES.	1.	1.	1.	1..	1.

These investigations show that, in the group of miners taken, carboxyhaemoglobin could not be detected by the method used. Whether lessor quantities of COHb, if present, could affect the oxygen metabolism of the involved myoneural reflex systems to such an extent as to produce oscillations, I am not competent to say,

but consideration of the known action of CO shows that, for it to do so, in such small concentrations as is normally present in mine air, it would require to have a selectively greater action on the "atmungsferment" of Warburg of the tissue cells in this myoneural system only, which, I should imagine,is extremely unlikely. Haldane (1922) is emphatic, and states that the amount of CO normally present in mine air "is far too low to cause any appreciable physiological effects."

Robson's theory is supported chiefly by his showing that, in his area, the incidence of nystagmus varied directly with the percentage of "volatile matter" in the coal. Haldane observes, however, that at ordinary temperatures coal is not volatile, so that this factor cannot be translated in terms of "volatile matter" given off from coal. Again, Collis pointed out that in Robson's area the bituminous (volatile matter 30%) coal was worked with safety lamps whilst in the anthracite (volatile matter 5%) coal districts naked lights were still in use. Hence, the factors Robson uses to support his CO poisoning theory in fact support the "deficient light" theory.

Bacterial Toxaemia.

That nystagmus might be the result of some bacterial infection has been suggested by Leighton Davies (1920), Freeland Fergus (1925) and others. No causal organism has ever been demonstrated nor, so far as I know, have any experiments been done which in any way support this theory. Whilst it remains in the realms---however attractive---of fancy, it need not be considered here.

Endogenous Toxaemia.

It has already been noted that Cooke (1931) considered that whilst deficient illumination was the essential cause of nystagmus, some "activating" or "precipitating" factor was necessary for the oscillations to develop. His examination of the blood of 57 nystagmic miners showed that in all of them the Arneth Count deviated to the left "suggesting the presence of a source of toxaemia". In each case he was able to demonstrate some possible endogenous source, e.g., bad teeth (59%) tonsils, antra &c.

Cooke's "toxic activator" theory is based solely on the laevo-deviation of the Arneth Count; the presence,in the nystagmic miners tested, of some possible source of toxic absorption; and the assumption that they had not "a personal refractoriness to the effect of toxins."

In my opinion, though his observations are of interest and provocative, his conclusions have not been decisively demonstrated. As he himself points out, "the Arneth Count is

altered by a variety of conditions and many different agents." On the other hand, I agree that a miner suffering from endogenous toxaemia or, for that matter, toxaemia from any source, including, for example, acute infections or absorption from injured tissues, will, everything else being equal, be more prone to develop miners' nystagmus.

Errors of Refraction.

Errors of refraction have been held by several investigators to be either the chief, or an important, predisposing cause. Pooley investigated this question with mathematical thoroughness and his observations form the Second Report of the Miners' Nystagmus Committee. (1923). He concludes, "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." Priestley (1922), however, believes that by refusing underground work to those whose vision is less that 6/18 in both eyes, "grave nystagmus would be practically eliminated from the coalfields."

Having frequently observed miners' nystagmus producing total incapacity where the vision was 6/6 in both eyes, I cannot accept the latter view of the importance of refractive errors. On the other hand, I believe it is only reasonable to accept that, other aetiological factors being equal, the man with refractive errors will be thereby more prone to develop nystagmus.

Psychological Factors.

The last important contributory causal factor which has been suggested is emotional stress. This implies a wider conception of the disease than oscillations of the eyeballs, and includes "Miners' Neurosis", introduced by Jeaffreson (1887), and the malady defined (since 1913) in the Third Schedule of the Workmens' Compensation Act as, "The disease known as Miners' Nystagmus, whether occurring in miners or others, and whether the symptom of oscillation of the eyeballs be present or not."

It has already been noted that the symptoms of miners' nystagmus include, other than oscillations of the eyeballs, photophobia, blepharospasm, forced ocular movements, tremor of the head and limbs, and disordered action of the heart. It is obvious that all these symptoms cannot be directly attributable to the oscillations. The compensated disorder, as we now know it, is regarded as a composite disease comprising nystagmus proper, as represented by the oscillations, and a psychoneurosis, as represented by blepharospasm, tremors, tachycardia &c. This psychoneurotic aspect of the disease has been considered by Jeaffreson (1887),

Rivers (1922), Millais Culpin (1936), Brock (1938), Caiger (1939) and others.

W. H. R. Rivers, in an Appendix to the First Report of the Miners' Nystagmus Committee (1922) declares, "The state known as Miners' Nystagmus may be a psychoneurosis arising independently of any disorder of the eye-movements or of vision, while in other cases the psychoneurotic aspect of the disease, though secondary to nystagmus proper, (underlined I.C.) has come to be the more important part of the disease.

With regard to the first part of this quotation, namely, that "the state known as Miners' Nystagmus may be a psychoneurosis arising independently of any disorder of the eye-movements------", here, I think, confusion will arise unless it is agreed that, before accepting a group of symptoms as indicative of Miners' Nystagmus, oscillations of the eyeballs must be, or have been, present. If this is not adhered to, then the disease "Miners Nystagmus" will lose its undoubted clinical entity, and will include cases which, in other occupations, would be labelled "neurasthenia".

Rivers stresses the importance of the eye-movements themselves in producing the psychoneurosis, firstly, by causing disturbances of vision (e.g., inability to fix objects, mal-projection of the coal face, &c.), which in turn causes fears and anxieties, and secondly, "by providing a nucleus for his psychoneurotic symptoms, as in the psychoneurosis of warfare some old wound or organic affection will determine the locality of a hysterical paralysis or anaesthesia."

Brock (1938) concludes, "It might be expected that the low illumination and the constant sense of danger should bring about cases of physiological and psychological breakdown."

Caiger (1939) considers that the deficient illumination causes the oscillations of the eyeballs but "these of themselves are not disabling" nor do they produce any disturbance of vision or other symptoms, because "a normal inhibitory mechanism is set up." He believes that "the crucial factor in Miners' Nystagmus associated with disablement is that there has been a breakdown of the normal inhibitory mechanism concerned." This breakdown, he believes, is due, in 75% of cases, to "industrial fear" (fear of long-continued unemployment, chronic financial stress for himself and his family, and finally the prospect of losing his job permanently); in the remaining cases the cause lies in fear of blindness, toxaemia from some illness, or some accident in the pit.

Whilst agreeing with Caiger that deficient illumination results in the oscillations, and that it is the development of

a psychoneurosis which produces the incapacity, I am not convinced of his theory that "industrial fear" plays the chief part in the production of the psychoneurosis, the attention of the miner having been drawn to the presence of his oscillations.

Again, Caiger emphasises that "the oscillations of themselves are not disabling because a "normal inhibitory mechanism exists" "for inhibiting these undesirable consequences (i.e., impairment of vision and dizziness) of oscillations of the eyes". That some such inhibitory mechanism exists is undoubted since over 30% of miners have oscillations, yet only 0.44% are incapacitated by them. Since oscillations of the eyeballs are an abnormal phenomenon, however, this so called "normal inhibitory mechanism" would, in my opinion, more accurately be termed a compensatory inhibitory mechanism; and the existence of which implies a further strain on an already heavily taxed neuro-muscular mechanism. This, I maintain, justifies the acceptance with reservation only of the present day opinion that "the oscillations of the eyeballs are not disabling of themselves".

Millais Culpin contributes an Appendix to the Third Report of the Miners' Nystagmus Committee (1936). This Report states that, apart from oscillations of the eyeballs and the subjective movements of objects, the symptoms of miners' nystagmus, e.g., defect of visual acuity, night-blindness, photophobia,forced ocular movements, tremors of the head and limbs, and disordered action of the heart, are equally found in cases of psychoneuroses other than in miners.

Millais Culpin puts forward an explanation for the sequence of events in cases of nystagmus progressing to the stage of total incapacity. Owing to the specialised nature of his observations, they are quoted in detail.

"It has often been noted that this sequence (i.e., as the nervous symptoms increase, the oscillations diminish) occurs, but its significance has not been considered. Why does this disorder begining as a physical disability, change its apparent nature and become a disorder identical with psychoneuroses that have arisen independently of any association with nystagmus? The existence of a high percentage of men with oscillations and no nervous disability, and the existence, in miners, of nervous disability with no history of oscillations should, on statistical grounds, rule out oscillations as a cause of the nervous symptoms." "An explanation of the undoubted connection (underlined I.C.) must be sought in similar phenomena than can be observed under other conditions. Both in civil practice and in the War cases, it has become a commonplace that a bodily manifestation of hysteria can protect the patient against other symptoms; a man would be

quite fit except for an hysterical symptom---a paralysis or tremor, for example---but would show severe anxiety or even obsessional symptoms when that disappeared either spontaneously or as a result of treatment. It is perhaps not so well known that the converse may occur, and anxiety symptoms may sink out of sight when the bodily disability appears."
"The ocular disability (not the oscillations) thus behaves as an hysterical symptom;...."
"We can put the cases in a general scheme;
"A. Oscillation with no disability.
"B. Oscillation with subjective movement of objects. This appears to be a recoverable stage and may be organically determined. On the other hand, the added symptom may behave as an hysteria and for a time protect against further developments.
"C. The unfolding of further psychologically determined symptoms---hysterical, anxiety, or obsessional, with the oscillations now behaving as an hysterical tremor and tending to disappear.
"D. The disappearance of oscillation and the complete development of a psychoneurosis."

Culpin's explanatory scheme, to one unfamiliar with medical psychology, is somewhat involved. For consideration, I put forward a scheme (vide. p. 41) which, I suggest, also explains the sequence of events above mentioned. My views of the psychoneurotic factor in Miners' Nystagmus is as follows.

It is my firm belief that Fear plays the chief part in the production of the compensated disorder as we now know it. Coal-mining is a dangerous occupation and this fact is well recognised by the miner and his family, the male members of which will also probably become miners. Almost a half and over a third of all the fatal and non-fatal accidents respectively, reported yearly under the Workmens' Compensation Act, for the whole of Great Britain and Northern Ireland, occur in the coal mining industry. The chief danger is from falls of coal. The miner, whilst working, must continually watch the roof and coal-face and be prepared, at the first sign of danger, to "get clear". This involves good vision and hearing, for it is by these senses that he may anticipate danger and safeguard himself from it.

What is the position of the miner affected with oscillations? If he have no other sign of the disease, and is not conscious that his eyes do move, he is no more likely to develop a psychoneurosis, by reason of his oscillations, than his non-affected colleagues, provided, of course, that all the other aetiological factors are equal. If, on the other hand, the oscillations produce apparent sudden shifting of the coal-face; inability to focus clearly any point on the coal-face; inability accurately to judge distances e.g., from his lamp, or the swing

of his own or others' picks; and dizziness; then, in my opinion, we have, in these symptoms, the chief causal foundation for the building up of psychoneurotic manifestations such as headache, photophobia, blepharospasm, nausea, head and limb tremors, and tachycardia. This causal foundation engenders in the miner a sense---conscious or subconscious---of fear both for, and of, himself.

This fear the miner will not readily admit, in fact, will stoutly deny, but continual questioning----avoiding leading questions---does, in my personal experience, frequently reveal its presence. As one miner, after unproductive questioning on several previous occasions as to why the disturbances of vision should have made him give up work, said, "To tell you the truth, it puts the fear of God in you".

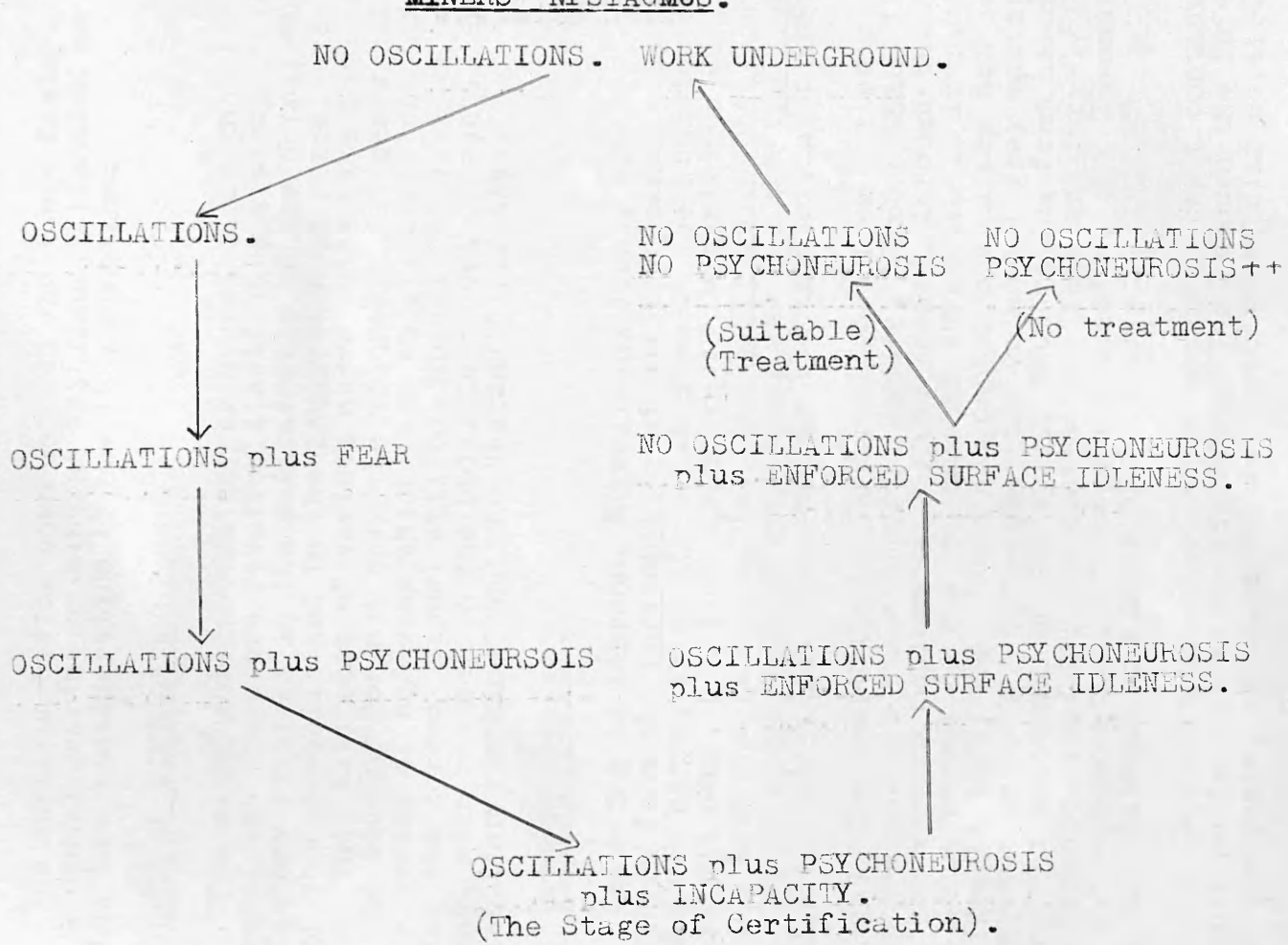
I believe this fear, for, and of, himself, plays a much greater part in the production of the incapacitating psychoneurosis than does the "industrial fear" described by Caiger; which latter fear, be it noted, is present at least in as great a degree in others entirely dependent on their work for their livelihood.

The sequence of events, as I see it, is briefly as follows. Work underground with deficient illumination produces the oscillations; these produce the disturbances of vision; these in turn make the miner afraid for, and of, himself; certifiable incapacity is then <u>unwittingly</u> achieved by the development of a psychoneurosis; with enforced surface idleness, the cause of the oscillations is removed, so that the nystagmus, per se, disappears; the psychoneurosis remains, but will tend to disappear if suitable treatment (e.g., surface work) is given, but will develop into a "compensation neurosis" if the miner is left to "retire on compensation".

This sequence is shown in the following diagram, (p.41) which is drawnin "circle" form as I believe that a miner who has once been totally incapacitated by miners' nystagmus is thereby rendered prone to develop a further attack if he return,after recovery, to his original work underground.

The personal views above expressed emphasise the importance of the presence of oscillations in first producing the psychoneurosis, and support my contention that the statement now generally made that "the oscillations of themselves are not disabling" should be accepted with reservation. The words of Llewellyn are apposite, "The eye has a greater influence on the mind than any other part of the body."

Though I believe fear for, and of, himself, to be the chief causal factor in the production of compensated Miners' Nystagmus, and that this fear is chiefly the consequence of visual disturbances themselves consequent on the oscillations, I accept that <u>any</u> emotional stress, other things being equal, should be accepted as a contributory causal factor.


MINERS' NYSTAGMUS.
NO OSCILLATIONS. WORK UNDERGROUND.
OSCILLATIONS.
NO OSCILLATIONS NO PSYCHONEUROSIS
NO OSCILLATIONS PSYCHONEUROSIS++
(Suitable) (Treatment)
(No treatment)
OSCILLATIONS plus FEAR
NO OSCILLATIONS plus PSYCHONEUROSIS plus ENFORCED SURFACE IDLENESS.
OSCILLATIONS plus PSYCHONEURSOIS
OSCILLATIONS plus PSYCHONEUROSIS plus ENFORCED SURFACE IDLENESS.
OSCILLATIONS plus PSYCHONEUROSIS plus INCAPACITY. (The Stage of Certification).

An investigation having been made of all the factors which have variously been described as the cause or chief contributory cause of Miners' Nystagmus, I am of opinion that the essential cause is deficient illumination, and that other aetiological factors described should be regarded, at most, as contributory or predisposing. The significance of Vitamin A intake is my original contribution to this aspect of the disease.

The question having been decided why these factors may have a causal bearing on Miners' Nystagmus, attention may be directed to a consideration of how they function.

THE PATHOLOGY OF MINERS' NYSTAGMUS.

This might more accurately be described as the Physiology of nystagmus, and therefore resolves itself into a study of the complex factors involved in the maintainance of the normal muscle-balance of the eyeballs and in the production of normal eye movements. The existence of various nerve paths have been demonstrated, and account for not only the voluntary movements but also for a series of reflexes which play an all important part in securing the correct visual attitude in all the various positions of the head, with respect to the body, in space. These I term broadly the "Labyrinthine" and the "Retinal" reflexes.

THE "LABYRINTHINE" REFLEX.

According to Langdon, Maxwell and Jones, (1918) 1. The fibres from the horizontal semi-circular canal pass through the 8th nerve, enter the brain stem at the junction of the medulla and pons, continue directly to Deiter's nucleus, and there divide into two pathways, (a) the VESTIBULO-OCULAR TRACT, concerned in the production of nystagmus, and (b) the VESTIBULO-CEREBELLAR-CEREBRAL TRACT, concerned in the production of vertigo.

(a) The vestibulo-ocular tract goes from Deiter's nucleus to the posterior longitudinal bundle, through which it passes to the various oculomotor nuclei, thence, through the 3rd., 4th., and 6th., nerves to the eye-muscles themselves. It is to be noted (Wright 1931) that these oculomotor nuclei are not separate structures, but actually form one long column of grey matter controlling the movements of the eyes. Enumerating from before-backwards (cranio-caudally) the control stations are for, 1/ Pupil reaction, 2/ Accomodation, 3/ Upward movements, 4/ Downward movements, 5/ Lateral movements of both eyes.

(b) The vestibulo-cerebellar-cerebral tract continues from Deiter's nucleus to enter the cerebellum through the inferior cerebellar peduncle, and runs to the three vestibular cerebellar

nuclei (N. Fastigii, Emboliformis and Globosus) of the same side, from which it proceeds upwards through the superior cerebellar peduncle, then continues to the cerebral cortex of both sides, but more particularly to the opposite side, through the crura cerebri. The cortical areas which receive these fibres are postulated by Mills to be the posterior portion of the second temporal convolution adjacent to the cortical area for hearing.

2. The fibres from the vertical semi-circular canal pass through the 8th nerve, immediately ascend in the pons, and at a point above its middle divide into two pathways similar to those of the horizontal canal from Deiter's nucleus, i.e., (a) the vestibulo-ocular tract, the fibres entering the posterior longitudinal bundle to be distributed to the oculomotor column of grey matter, and, (b) the vestibulo-cerebellar-cerebral tract, which reaches the cerebellum through the middle cerebellar peduncle, runs to the three vestibular cerebellar nuclei of the same side, thence to be continued upwards through the superior cerebellar peduncle to the cerebral cortex of the same and opposite side.

Further, according to Gray's "Anatomy", (1923) descending fibres of the vestibular nerve, including the vestibulo-spinal fasciculus arising from Deiter's nucleus, continue downwards in the anterior funiculus of the spinal cord to end round the spinal motor nuclei, thence to be distributed to the skeletal muscles. We have also a direct connection between the cerebellum and the retina, eye muscles, and skeletal muscles. Fibres arise from the dentate nucleus of the cerebellum, continue through the superior cerebellar peduncles, cross to the other side on the ventral surface of the cerebral aqueduct, then divide into ascending and descending branches. Most of the ascending fibres end in the Red Nucleus of the Tegmentum, thence to be continued downwards in the lateral funiculus of the opposite side of the spinal cord as the rubrospinal tract which synapses with the cells in the anterior horn. (skeletal muscles). Some of the ascending fibres synapse with the oculomotor nuclei (eye muscles), others, with the thalamus (retina).

It is seen, therefore, that this "Labyrinthine" reflex is a composite one. We may include in it the afferent nerve impulses received by the cerebellum from the muscles, skeletal and ocular, via the direct cerebellar and the anterior spinocerebellar tracts. Further, it has been shown, (Stein, 1894, Barany, 1906, Maxwell, 1920, and others) that the proprioceptive nerve endings of the neck muscles link up with the oculomotor muscles directly (apart from the neck muscles) via the oculomotor column of grey matter, to constitute the tonic neck reflex.

THE "RETINAL" REFLEX.

The retinal fibres are continued as the optic nerve (Gray's Anatomy) to the optic chiasma where a partial decussation takes place. The fibres from the nasal half of each retina cross the middle line and enter the optic tract of the opposite side. The fibres from the temporal half of each retina do not cross but are continued backwards into the optic tract of the same side. The optic tract winds round the cerebral peduncle, then divides into a medial and lateral root. The medial root consists of fibres connecting the medial geniculate body of one side with the inferior colliculus of the opposite side via the optic chiasma, and constitute the Commissure of Gudden. The lateral and larger root ends in the lateral geniculate body, the pulvinar of the thalamus, and the superior colliculus, which together constitute the lower visual centre. From the cells of the lateral geniculate body and the pulvinar, fibres, the optic radiations, take their origin and pass to the cortical visual centre situated in the cuneus, and in the neighbourhood of the calcarine fissure. Many efferent fibres from the superior colliculus cross the middle line and descend in the medial longitudinal fasciculus to end by synapsing with the oculomotor grey column, thence to be distributed to the eye muscles; others, via the tectospinal fibres, with the motor nuclei in the anterior horn, thence to be distributed to the skeletal muscles. Further, we may include in this "retinal" reflex the afferent fibres running via the spinotectal fasciculus from the skeletal and ocular---(via the numerous afferent kinaesthetic fibres present in the oculomotor nerves and whose cell stations appear to lie on the trunks of these nerves (Wright, 1931)---muscles to the superior colliculus, thence, as the tect-cerebellar fibres to the cerebellum. Also may be included the "optical reflex"---stimulation of the retina by light brings about an increased tonus of all the extraocular muscles.

REFLEX MOVEMENTS OF THE EYES.

Duke-Elder (1932) divides reflex movements of the eyes into two classes; movements which are carried out involuntarily, but involve the mediation of consciousness, and true reflex movements which are subserved by a subcortical mechanism. Under the first heading he includes the reflex movements of fixation, and the corrective movements undertaken in the interests of fusion; these he terms the Psycho-optical reflexes. In the second class are the Postural reflexes associated with the labyrinths and the muscles of the neck. We may modify this classification by including under a third heading the Optical reflex. (See diagram p. 46.)

PSYCHO-OPTICAL REFLEXES.

A. The Fixation Reflex.
B. Corrective Fusion Movements.

A. The Fixation Reflex.

When a light falling on a peripheral part of the retina is appreciated by consciousness, the eyes are reflexly moved so that the image falls on the fovea.

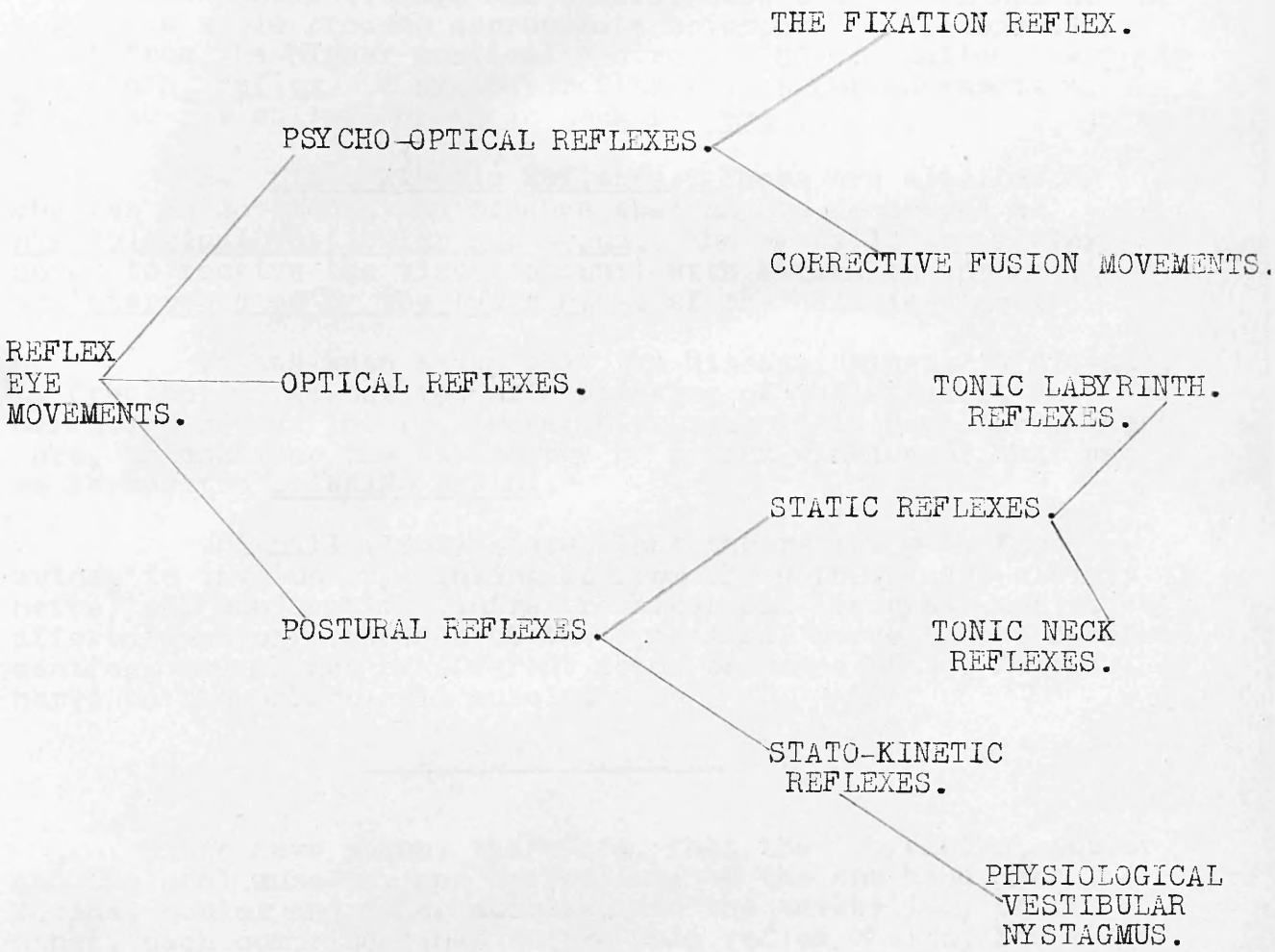
B. Corrective Fusion Movements.

To secure binocular vision, corresponding parts of each retina must be projected into space. This can be achieved only when the fixation axes of both eyes are parallel. It has been shown by many observers and by various methods that it is extremely rare for the muscles of both eyes to be so balanced that, in a state of physiological rest, the fixation axes lie parallel. In the great majority of individuals, therefore, there must always be, on stimulation of the retina, a reflex corrective movement to prevent double vision.

OPTICAL REFLEX.

It has been shown by Hering, (1878), Hoffman, (1913), and others, that when a light falls on the retina, the eye muscles are increased in tonus, apart from the mediation of consciousness.

REFLEX EYE MOVEMENTS. (Modified from Duke-Elder).



POSTURAL REFLEXES.

A. Static Reflexes.
B. Stato-kinetic Reflexes.

A. The Static Reflexes are elicited by changes in position. Experiments by Stein (1914), Parker (1918), Maxwell (1920), Benjamins (1928), and others, show that movements of the body as a whole produce appropriate movements of the eyeballs, apart from the higher cortical centres. This is called the tonic labyrinth. reflex. A similar reflex exists for movements of the neck and is called the tonic neck reflex.

B. Stato-kinetic Reflexes. These are elicited by changes in movement, and produce what may be described as physiological vestibular nystagmus. The eyeballs are reflexly moved to receive the visual stimuli with regard to the position, as interpretated by the labyrinths, of the head in space.

It has been shown that the disease, Miners' Nystagmus, is frequently associated with blinking of the eyelids, and narrowing of the interpalpebral fissure. It is necessary, therefore, to consider the physiology of reflex winking or what may be termed"the BLINKING REFLEX."

Whitnall (1921) states that the reflex path for automatic involuntary winking is from the cornea (naso-ciliary nerve) and conjunctiva (infra-trochlear and lachrymal nerves) by afferent sensory branches of the trigeminal nerve to the cerebral centres, and thence by efferent motor branches of the facial nerve to the orbicularis muscle.

We have shown, therefore, that the labyrinths, ocular and skeletal muscles, and cerebellum, on the one hand, and the retina, ocular and other muscles, and the cerebellum, on the other, each comprise links in two main reflex chains, and that these pathways have direct or indirect communication with the cortical areas of the brain. Their harmonious working must obviously depend on the functional and organic integrity of the complete system above described.

My view of Miners' Nystagmus is that it is a functional disturbance of the complex nervous and neuromuscular system above described. Whilst the essential factor in the production of this disturbance is deficient illumination, any factor, physical, physiological or psychological which may adversely affect the normal harmonious working of this system should be accepted as a contributory or predisposing cause.

THE COURSE OF MINERS' NYSTAGMUS.

Different observers have different methods of describing this disease. Roche (1932) recognises four stages:-

1. "Latent."
2. "Subacute".
3. "Acute".
4. "Neurasthenic".

The Miners' Nystagmus Committee (1922) gives a more clinical division which may be represented thus:-

1. The "Latent" stage.
2. The "Manifest" stage.
   - a/ "slight"
   - b/ "ordinary"
   - c/ "severe".

A more cumbersome but clinically more descriptive method I suggest as follows:-

1. The "affected with" stage.
2. The "suffering from" stage.
   - a/ The "pre-nystagmus" stage.
   - b/ The "nystagmus" stage.
   - c/ The stage of psychoneurosis.

It is essential, in my opinion, for the proper appreciation of this disease, to emphasise continually that it has one great inherent peculiarity, namely, that of those "affected with" it, less than 2% are "suffering from", or "incapacitated by" it.

I am convinced that the failure to appreciate this fact is responsible, more than any other factor, for the confusion, and lack of unanimity of opinion, which has existed and still exists concerning this disease.

1. <u>The "affected with" stage</u>.

In this stage the miner has oscillations of the eyeballs but no other symptom. I have seen cases where well marked oscillations were present, at rest, in daylight, at the horizontal level, where the miner could not tell <u>when</u> his eyes were oscillating. These men know that they have nystagmus only by being told.

Most cases in this stage are unaware that their eyes <u>do</u> oscillate, as the nystagmus is only present at work.

2. The "suffering from" stage.

(a) The "pre-nystagmus" stage. In this stage, the miner, usually about 35 years of age, is begining to develop incapacitating symptoms but is able to continue at work. He complains typically of difficulty in "getting his sight" on leaving the brighter-lit pit bottom to walk to the coal—face. After the shift, on reaching the surface, he is dazzled if it is daytime, by bright sunlight, or if it is night-time by car head-lights. He has some difficulty in seeing clearly the area of coal-face he is "working" and this may suddenly appear to be out of focus, or move. After working for some time, he may feel slightly dizzy and unsteady. When examined on the surface, oscillations will not usually be detected even after a quarter of an hour's bending exercises in the dark. The complaint of dizziness at work, however, suggests that oscillations are then present. The following case from my records is a typical example of this group.

"W. H.T. aged 43. Examined October 15th 1940. Worked as a collier since he was 17 years old. Was "buried" by a fall of coal in February 1940 and was off work two weeks. Six months ago asked for, and got, a light job instead of his coal-face work as he "could not stick it because of backaching and because of my head." He complained of headache and dizziness, had photophobia and blepharospasm. Pulse rate 102. No oscillations were detected after half an hour in the dark followed by bending exercises. I noted that when he had gazed at my finger, held so that his visual regard was directed obliquely upwards, just when I would have expected oscillations to develop in the typical "nystagmus" case, he closed his eyes quickly, shook his head, and when he opened his eyes again they were steady." I am convinced that by this manoeuvre he prevented his oscillations appearing, and that had he not "because of his head" changed his occupation of collier to general labourer underground, he would, by the time of examination, have reached the "nystagmus" stage.

There is, perhaps, even an earlier stage than represented by the case of W. H. T.,which, though not recognised, may, I suggest, be of industrial importance. The following case I would classify as a "pre-pre-nystagmus".

"T. R. ae. 31. Trammer. This man consulted me because of a troublesome cough. He had the depressed look sometimes associated with the nystagmic "facies" so I made enquiries as to whether he had any trouble with his vision down the pit. I could find not one symptom pointing to even the "pre-nystagmus" stage. Exercises in the dark on two separate examinations failed to reveal even a "flicker" of the eyeballs. Until the end of the second examination I gave him no explanation, nor had he sought one, as to what would normally appear an unusual way of investigating "a troublesome cough". To explain matters, therefore, I told him

that I had wondered, when he first came to see me, if he had nystagmus but after examination I was pleased to assure him that he had not the slightest trace of it. His melancholy reply was to the effect that if he had not got it now, he would sooner or later develop it, as all his family had it! On enquiry, I found that his father, his grandfather and his uncle all had to give up underground work because of it, whilst his brother, aged 24, was at present in receipt of total compensation for it.

Now this man, even in the present absence of any one symptom of miners' nystagmus, will, I believe, almost certainly develop the incapacitating disease later. This is the type, comparable, I suggest, with the Potential Effort Syndrome case of the Army, who, for the benefit of all parties concerned, should not be allowed ever to start work underground.

(b) The "nystagmus" stage. This emerges from the former stage. The visual disturbances e.g., inability to focus the coal-face, inability to judge distances, sudden shifting of the coal-face, spinning of lights, dazzle, night-blindness and photophobia, become much more troublesome. Headache, giddiness, staggering and loss of self-confidence are added to the picture and the man eventually is compelled to give up work at the coal-face. This is the stage of incapacity, partial or total. The oscillations will now be found to be present in daylight, at the horizontal level, before exercises.

(c) The stage of psychoneurosis. The predominant symptoms here are those of a psychoneurosis. The miner now complains of headache, giddiness, insomnia, nightmares, mental depression, and general weakness. Though the oscillations may be difficult or even impossible to elicit, photophobia, blepharospasm, and forced ocular movements may be much increased. Often there are present head and limb tremors, and irregular movements of the extremities, more marked after bending exercises. These exercises often cannot be long continued, the patient losing his balance and even falling to the floor. Tachycardia is a prominant and important feature. The chief phobia, in my opinion, now is, consciously or subconsciously, fear of having again to work underground.

(Note. A diagram showing the course of Miners' Nystagmus is given on page 41.)

INCIDENCE OF MINERS' NYSTAGMUS.

The incidence of Miners' Nystagmus is of peculiar importance as it is from variations in the incidence chiefly of the compensated disorder that observers draw deductions as to the significance of some suggested causal factor. Unless it

is clearly recognised that there are two separate incidences--- (1) Among those "affected with" miners' nystagmus, and (2) Among those "suffering from" or "incapacitated by" miners' nystagmus, then such "post hoc ergo propter hoc" deductions will result in inaccurate conclusions.

(1) Incidence among miners "affected with" Miners' Nystagmus.

This incidence, as recorded by the following investigators, may be given approximately as follows:-
Court (1891) 35% among coal cutters and 31% among all underground workers. The incidence among all underground workers is given by Neiden (1894) as 5%, Nuel (1907) 20%, Romiee (1908) 20%, Dransart and Famechon (1910) 10%, Libert (1910) 24%, Coppez (1912) 20%, Llewellyn (1912) 25%, Stassen (1914-1919) 24%, Roche (1931) 26 to 41%.

Factors influencing the incidence among those "affected with" nystagmus.

Whilst it is advisable to remember that the oscillations of miners' nystagmus may to some extent "lie in the eye of the beholder",---which would account for the variation in the incidence rate as given by different observers,---it has been shown that the "affected with" nystagmus incidence varies directly with the illumination.

Court (1891) found the incidence among all underground workers to be 31% where safety lamps alone were used, 0.87% among miners who had always used candles, and completely absent among miners who had always used torch lights.

Romiee (1908) made an examination of coal getters in four pits, then examined them again after better lamps had been installed in three of them. His results were:-

PIT.	1877-1892	1908	LAMPS.
1	65%	17%	improved.
2	50%	22%	improved.
3	23%	10%	improved.
4	41%	50%	same.

Llewellyn (1932) examined the same miners before, and 6 months after, an issue of electric cap lamps and found that the "affected with" nystagmus incidence was 36% and 18% respectively.

Ferguson (1929) conducted similar experiments with similar results.

(2) Incidence among miners "suffering from" or "incapacitated by" Miners' Nystagmus.

This, obviously, is the incidence of the compensated disorder.

The official Blue Book figures for the years 1908 to 1936 (excepting the War years, for which records are not available) show the following percentage incidence of new cases among all underground workers to whomcompensation was paid for Miners' Nystagmus.

Pits in Great Britain.

Year.	% incidence of new cases.
1908	0.05
1909	0.08
1910	0.11
1911	0.16
1912	0.16
1913	0.26
1914	0.33
1919	0.29
1920	0.23
1921	0.22
1922	0.44
1923	0.40
1924	0.33
1925	0.39
1926	0.19
1927	0.22
1928	0.34
1929	0.33
1930	0.41
1931	0.39
1932	0.30
1933	0.24
1934	0.28
1935	0.30
1936	0.25

Factors influencing the annual incidence of the compensated disorder.

As already mentioned, when variations in the above figures are used to buttress, or discount, the aetiological importance of some one given factor, the logical fallacy, "post

hoc ergo propter hoc" must be borne in mind. There are so many factors involved, varying from year to year, any one of which may play a considerable part in causing a fluctuation of the annual compensated nystagmus incidence. The words of Collis (1923) are particularly apposite, "Examination of large numbers of underground workers have disclosed that well over 30% have oscillations of the eyeballs without being in any way aware of the fact; here is evidence of a deep pool from which cases could rise to the fly of compensation. The more tempting the fly, the more cases would rise; but the number that rise is no sure proof that the number of fish in the pool is increasing or diminishing."

Accurately to estimate the significance of some one factor in influencing the incidence of those "incapacitated by" nystagmus, two clear years should be given, during which this factor is operative, before the incidence is considered, as it takes this time for the operative factor to exert an appreciable influence; and during these two years every other possibly influencing factor should remain constant. A review of the years in question will show that this is well-nigh impossible. The disease itself has, in Law, altered from "Nystagmus" in 1907 to "The disease known as Miners' Nystagmus whether occurring in miners or others, and whether the symptom of oscillations be present or not" in 1913. Further, possibly influencing factors in the mining industry itself seem seldom constant for any length of time. During the War years, industrial conditions were abnormal. In 1919, total compensation was raised from 20/- to 35/- per week. In 1921 there was the Great Strike, in 1926, the General Strike, while the period between these Strikes was affected by long disputes, when the miner was either dismissed because there was no work for him, or was employed only part of the week, for the remainder of which he drew "dole". From 1909 to 1919, there was an eight hour shift, from 1919 to 1926, a seven hour shift, then from 1926 an eight hour shift. In 1920, wages were high, in 1928 and 1929 wages were low. There have been good working years, e.g., 1938-1940, and bad working years, e.g., 1931-1938. Improvements in lighting conditions have continuously been effected. The methods of coal getting have altered with the increasing use of coal-cutting machines and moving belts, resulting in less varied work, more output, and more noise.

In South Yorkshire there has been a marked decrease in the last three years i.e., 1938-1940, not only in the annual number of cases notified, but in the number of old cases, i.e., cases receiving compensation for over one year. Mr. W. E. Gillhespy, General Manager of the Yorkshire Coal-Owners Mutual Indemnity Society, gave me the following annual numbers of new cases of Miners' Nystagmus for which his Company had paid compensation.

YEAR.	1930,	1931,	1932,	1933,	1934,	1935,	1936,	1937,	1938,	1939,	1940.
NEW CASES.	238,	228,	213,	221,	236,	211,	161,	133,	122,	84,	82.

In an interview with the Manager of a large Yorkshire pit, I was told that, in his opinion, nystagmus was as prevalent now among the working colliers as it had been.

I believe the explanation of this apparent discrepancy of fact with opinion lies partly in the improved lighting conditions---(due to the installation generally of the new type Alkali hand-lamps, and, with the increasing introduction of coal-cutting machines and moving belts at the coal face, the fact that the collier, once he starts work, has no need to move away from his lamp, and is therefore not affected with the same variations in light intensity as he was under the old method of coal-getting)---which enables a miner "suffering from" nystagmus still to carry on his work, but chiefly in the belief the miner has, that if he once makes a claim for disablement he will find it difficult, in a bad working year, to get re-employed underground.

The decrease in the number of cases remaining on compensation for an extended period is due, in my opinion, to Medical Referees now taking a stricter view of the disablement caused by transient oscillations or occasional "flickers" of the eyeballs.

Among other influencing factors which have been suggested is the undue attention paid in some districts to the disease by enthusiastic Miners' Officials or medical men; the opening of new Miners' Convalescent Homes; and the payment of "lump sum" settlements. It will be readily understood that such factors, if operative, would tend to affect the incidence of new claims.

These observations show that many different factors may affect the incidence of miners certified, under the Workmens' Compensation Act, as suffering from nystagmus; that these factors may vary from year to year, and in different localities in the same year; and that the official Blue Book annual incidence figures are of doubtful value when used to demonstrate the importance of some <u>single</u> suggested factor in the disease itself.

<u>DIAGNOSIS OF MINERS' NYSTAGMUS</u>.

In the diagnosis of Miners' Nystagmus, a clear distinction must again be made between those "affected with" it, and those "suffering from" or "incapacitated by" it. The accurate diagnosis of the latter condition is of particular importance as this is properly the compensated disease known as "Miners' Nystagmus".

The diagnosis in the case of those "affected with" the disorder presents no difficulties and depends on finding the oscillations. (vide p. 3.)

The diagnosis in the case of the incapacitating disorder is, however, fraught with very great difficulties. The presence here of oscillations is not enough---some incapacitating symptom should be present. Unfortunately, the Law makes no such stipulation. The latest authoritative decision (Departmental Committee Report 1938) states, "A case may properly be certified if, when tested in the dark after stooping exercises, oscillations are found to persist for at least 15 seconds. We recommend this standard for adoption in all cases."

The First Report of the Miners' Nystagmus Committee (1922) states, "It is probable that 25% of all men over 21 working underground could obtain a Factory Surgeon's Certificate and be legally (underlined I.C.) disabled." That position obtains today. In an interview with Mr. Gillhespy, he told me of one case, in his experience, where a miner had made thirteen successful claims over a period of some years. He had this case investigated and found that the man was also a "bookmaker". The association between his claiming compensation and declaring off compensation with the Lincoln and November Handicap Meetings was then discovered! Now so long as this man was re-employed each time he declared off the Funds, he could legally carry this on indefinitely.

It is obvious that this is an unsatisfactory position.

Unfortunately, the diagnosis of the incapacitating disorder depends chiefly on the claimants own statements since it is the psychoneurotic symptoms and not the oscillations, which are the main indications of disability. In my opinion, giddiness, especially if accompanied by staggering after exercises, fine head tremors, and tachycardia, are the most important of these disabling psychoneurotic manifestations, and on the degree of their presence should be assessed the extent of incapacity. These symptoms may be most marked where the oscillations are fine or even absent, or absent where the oscillations are visible in daylight, at the horizontal level, before exercises. In all cases, however, "the condition cannot properly be described as one of miners' nystagmus unless oscillations either are or have been (underlined I.C.) present." (Departmental Committee Report 1938).

Under the present procedure, the claimant applies to the Factory Surgeon in the area for a Certificate under the Workmens' Compensation Act 1925. The Factory Surgeon, as a rule, is a general practitioner and has therefore a general knowledge of both the eye and psychoneurotic symptoms. Appeal against this certificate is made to the Medical Referee, who in most instances is an ophthalmic surgeon and will therefore have a special knowledge of the ocular condition but still only a general knowledge of the psychoneurotic symptoms.

The Departmental Committee (1936) suggests that miners' nystagmus cases should be dealt with, like Silicosis, by a specially constituted Board. This Board would consist of two members, an ophthalmologist and a physician.

I believe that the adoption of this method would be fairer to all parties concerned, and result in a great advance being made in the proper diagnosis of the compensated disorder.

PROGNOSIS IN MINERS' NYSTAGMUS.

The First Report of the Miners' Nystagmus Committee (1922) states, "Many men can work on the surface without any rest, and in these cases the prognosis is good. In more marked cases, a complete rest for six or more months is required, but even these men should be able to work on the surface within twelve months of failure. Speaking generally, all physical signs and symptoms are lost after the man has left the pit for two years."

"Cases in which the psychological factor is marked run a very different course. Although all physical signs may be lost, the symptoms may remain, and the man alleges total incapacity even after he has left the pit for years."

Pooley (Committee Report 1923) states, "Whereas in Belgium, France and Germany miners only stop work for severe attacks, the great majority of men appear to commence work on the surface either immediately or at least within a few weeks (underlined I.C.) and, with a few exceptions, to resume their original (underlined I.C.) work within two or three months. Anything like a year's absence is quite abnormal."

Stassen (Second Report 1933) who is personally responsible for the examination and certification of the miners in the Province of Liege---employing 30,000 miners---gives, from his wide experience, the following figures.

Period off work.	Percentage.
0 to 1 month.	28.
1 to 3 months.	50.
3 to 6 months.	20.
6 months and over.	2.

Mr. W. E. Gillhespy has given me the following data from his records. "Three-quarters of the nystagmus claims remain on compensation for over two years, and one third remain on compensation for over five years."

The above estimations show an apparent irreconcilable difference. The explanation, I suggest, lies in the appreciation

that the prognosis in Miners' Nystagmus depends entirely on the treatment. In Liege, the figures for which have been given, the miner off work with nystagmus receives, from his employers, a weekly subsidy equivalent to 25% of his previous earnings, in addition to which he may apply for (underlined I.C.) State Relief. In no case is this subsidy continued beyond six months. (underlined I.C.). If he remains on surface work---to which they are nearly all put---the compensation paid is proportionately less than with us. There is an urgent financial inducement, therefore, for the nystagmic miner to start surface work, and as speedily as possible return to his old work. This urgency is not present to the same extent with our nystagmic miner nor is that great curative factor---suitable work---so readily provided for him.

The First Report of the Miners' Nystagmus Committee states, "Complete recovery with return to work at the coal face is frequent, but relapse is common, and with each failure the outlook for complete recovery is less likely." This view supports my personal belief, already expressed, that a miner who has once been totally incapacitated by miners' nystagmus is thereby rendered prone to develop a further attack if he return, after recovery, to his original work underground. (See diagram p. 41.)

TREATMENT OF MINERS' NYSTAGMUS.

The treatment of miners' nystagmus may be active or prophylactic.

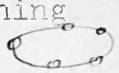
ACTIVE TREATMENT.

In my opinion, the factor of paramount importance in the treatment of a miner incapacitated by miners' nystagmus is the early and guaranteed provision of suitable work, surface or underground. Under the present system, the incapacitated miner, on full compensation, has the fear, at each examination by the Compensation doctor, that his compensation will be reduced to a partial basis, when no work will be available for him to supplement his weekly income. This medico-legal system results in an aggravation of the incapacitating psychoneurotic symptoms and is the chief cause of the abnormally prolonged period of total and partial incapacity found in this country. The difference in demeanour in the man, on a partial rate of compensation and working, and the man on a similar rate and idle, is manifestly apparent to anyone who has made Compensation examinations.

Of secondary importance are reassurance as to fear of blindness or indefinitely prolonged disability, correction of refractive errors, and removal of endogenous foci of toxaemia. Attention should be paid to personal hygiene, including provision of adequate rest and optimum diet.

Various treatments have been tried in an attempt to abolish the oscillations. Shufflebotham (1914) mentions that Romiee advised local applications of 3% eserine; and that atropine had been used without success. Cooke (1931) observed transient cessation of oscillations in "the few cases tried" after intramuscular injections of magnesium sulphate. Roche (1931) used injections of bulbocarpine, with no effect in some cases and transient cessation of oscillations in others.

With regard to the subjective movement of objects,--- the symptom which marks the commencement of the incapacitating disorder--Professor Millais Culpin, in the course of a correspondance I had with him, suggested his tentative theory that these subjective movements were due to a spasm of accomodation and most kindly gave me the opportunity of testing it. Unfortunately, I was able to do so only in one case, as the nystagmic miner, still working, is understandably afraid to have anything in the nature of an experiment done to his eyes. The results were as follows:-

J.O. ae. 35. Collier, worked underground since 17 years old. Had oscillations in daylight, at the horizontal level, before exercises. He complained of night-blindness, difficulty in seeing on coming from the dark into a bright light, and spinning of lights. A spinning light he represented so  rather than 

At times the coal face would apparently move. There was no headache, dizziness, head tremors or blepharospasm. Pulse 78.

The eyes were rendered cycloplegic(by three instillations at five minute intervals of 3% homatropine hydrobromide and 3% cocaine hydrochloride), three quarters of an hour before going to work on three succesive days and he was on each occasion examined the following day. The visible oscillations had not been affected, but he was emphatic that though lights still moved, the movement was not as rapid or as much as it had been before. He explained this diagrammatically thus:-  now  . During the

three days of the test, the coal face had not appeared to move. He could see much better than he had done for three years, i.e., since he had first noticed his eye symptoms.

In this case, therefore, cycloplegia, as induced by the method described, resulted in a diminution of the subjective movement of objects.

PROPHYLACTIC TREATMENT.

Recognition of the factors involved in the production of miners' nystagmus suggests the following prophylactic measures.

Adequate lighting at the coal face, and avoidance of glare, is required by encouraging the use of cap lamps, flood lighting, pneumatic lamps &c. The lighting of the underground roadways should also be adequate and may be improved by whitewashing and stone dusting. If the illumination at the coal face were maintained throughout the working shift at a uniform intensity of 0.58 foot candle, miners' nystagmus would, I believe, be completely abolished; if it were uniformly maintained at 0.33 foot candle, it would be appreciably reduced. In my opinion, this is the essential prophylactic factor and others should be considered as of secondary importance only.

Examination of the workman before employment underground is advisable. Attention should be paid to evidence of familial or inherent functional nervous instability; errors of refraction should be sought for and some standard, say a minimum of 6/18 for each eye, fixed; marked heterophoria or evidence of impaired labyrinthine or muscle-joint mechanism should be considered undesirable.

Ventilation should be carefully attended to, and warm humid atmospheres avoided. A definite time, at least 30 minutes, should be allowed for the meal eaten underground. A proper balance between the advantages gained in the mechanisation of coal mines---as represented by increased output, working of thin seams---and the disadvantages---as represented by increased noise and lack of variety in working, should be observed. The personal hygiene of the miner should be encouraged, attention being paid to diet including an optimum intake of vitamin A, early removal of endogenous foci of toxaemia, and a sufficiently prolonged convalescence after illness or injury. Lastly, the knowledge should be disseminated amongst the miners, that, owing to the nature of their work, they are liable to develop movements of the eyeballs---which fact they <u>already</u> know!---and that these movements are not of themselves disabling---which fact they do <u>not</u> know!

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