THE USE OF ELECTRONYSTAGMOGRAPHY

IN CLINICAL PRACTICE

by

Arnold G. D. Maran

M.B., Ch.B., F.R.C.S.E.

**Thesis submitted to the Faculty of Medicine
of the University of Edinburgh for the Degree
of Doctor of Medicine.**

October 1965.

CONTENTS

	<u>Page</u>
<u>Introduction</u>	
<u>Acknowledgements</u>	
<u>Section 1</u>	
<u>The Maintenance of Posture</u>	
The Stretch Reflex	1
Motor Unit Activity	4
The Spinal Animal	4
The Decerebrate Animal	5
The Vestibular Reflexes	6
The Vestibular Nuclei	7
The Vestibulo-ocular Reflex	9
Tonic Neck Reflexes	11
The Body Reflexes	13
Vision	14
Standing	15
<u>Section 2</u>	
<u>Review of the Literature on applied vestibular physiology and vestibular function tests.</u>	
Historical	17
Rotation Tests	20
The Coriolis Reaction	27
The Caloric Test	30

	<u>Page</u>
Position Tests	43
Parallel Swings	51
Torsion Swings	53
Tests of the Vestibulo-spinal Reflex	55
The Galvanic Test	59
Optokinetic Tests	61

Section 3

Review of the literature on nystagmography.

Mechanical Nystagmography	65
Optical Nystagmography	66
Electronystagmography	68
Methods of Electronystagmography	69
Factors Measured in Electronystagmography	72

Section 4

Authors Methods

Apparatus	80
Electrodes	83
Calibration	86
Calculation of Slow Phase Speed	88
The Position Test	89
The Caloric Test	93
The Coriolis Reaction	96

Section 5

Results

Normal Series

Experiment 1	97
Experiment 2	98
Experiment 3	99
Experiment 4	100
Experiment 5	100
Experiment 6	101
Experiment 7	101
Experiment 8	102
Experiment 9	103
Experiment 10	104

Pathological Series

Group 1	106
Group 2A	109
Group 2B	111
Group 2C	112
Group 3	115
Group 4	116
Group 5	118
Group 6	119
Group 7	124
Group 8	126

	<u>Page</u>
 <u>Section 6</u>	
<u>Discussion</u>	
Effect of Fixation on Nystagmus	128
What Happens When the Eyes Close	131
Calibration	132
Horizontal Nystagmus in Normals	135
Vertical Nystagmus in Normals	136
Positional Nystagmus	138
Vertigo of Cervical Origin	144
Nystagmus and the Eustachian Tube	147
Ocular Nystagmus	152
Temperature, Duration and Eye Speed	157
End Point of Caloric Nystagmus	165
Directional Preponderance	166
Canal Paresis	170
Different Wave Forms	173
Nystagmus and Memory	180
Nystagmus and Alertness	182
Frequency	184
Errors in the Method	185
Disadvantages	186
D.C. and A.C. Amplifiers	188
Conclusions	191

APPENDIX

BIBLIOGRAPHY

INTRODUCTION

Electronystagmography is a method of measuring the results of vestibular function tests. For many years it has been used as a research tool especially in Europe, but only in a few centres is it in routine clinical use. It was hoped in the early days, that electronystagmography would solve many questions outstanding in vestibular function testing; while it has been of great use, it has, in turn, created many new problems and posed many new questions.

The aim of this thesis is to show how electronystagmography can be of use in the routine examination of patients complaining of vertigo. The work consists of an eighteen months study of 130 normal subjects and 200 patients with disorders of equilibrium.

The thesis is presented in six sections.

In section one the maintenance of posture is briefly outlined in order to show what a relatively small part the inner ear plays in this. In section two there is a review of the literature on applied vestibular physiology and tests of vestibular function. Section three consists of a review of the more important parts of the literature on nystagmography. The development of the technique and the different methods employed are discussed. Sections four and five describe the methods used in this work and the results in normal and pathological subjects. Finally, in section six, the points raised in this work are discussed.

ACKNOWLEDGEMENTS

My interest in nystagmography was first aroused during a visit to Professor Arslan's clinic in Padua in 1962. Later in that year, during visits to Amsterdam and Utrecht, I saw what could be accomplished with the method. My thanks are due to the doctors of these clinics for their time and help, and especially to Dr Adrian Philipszoon of the Wilhelmina Gasthuis in Amsterdam.

I am deeply indebted to Dr Kenneth McLay, Senior Lecturer in the Department of Otolaryngology of Edinburgh University for starting me off on electronystagmography and also for letting me draw on his large experience of the subject; acknowledgement is also due to Dr McLay for the use of figure 4.

To Dr A. Brownlie Smith, Surgeon in Charge of the Ear, Nose, and Throat Department of the Royal Infirmary, I must express my gratitude for allowing me to do electronystagmography on patients under his care; I must also thank him for his constant encouragement.

My thanks are due also to the surgeons and physicians in the Departments of Otolaryngology and Medicine who have referred patients for investigation.

To Dr Nils Henriksson of the University of Lund, Sweden, are due my thanks for giving up so much of his time earlier this year to discuss nystagmography with me. I have quoted from these discussions frequently during this thesis in the

form of personal communications.

I must also thank Dr Hugh Begbie of the Department of Physiology of Edinburgh University for his help and guidance on the vestibulo-ocular reflex and the rotation tests.

Mr Paul Geissler, F.D.S., kindly helped me with the translation of the German references quoted.

I am grateful to Miss Dorothy Wardle, Librarian to the Royal College of Surgeons, for her help with the bibliography.

Finally my thanks are due to Mr Eric Leask of the Department of Medical Physics for his technical help, Mr John Paul for doing the photographs of the nystagmus records, and to Mrs E. McDonald for typing the manuscript.

SECTION 1

THE MAINTENANCE OF POSTURE

Patients with lesions causing vertigo complain primarily of disturbance of posture, and for this reason the mechanisms involved in the maintenance of posture will be briefly reviewed.

To achieve balance we use rather a wide variety of input information - not only from the vestibules, but also from the eyes, the muscle and joint proprioceptors, from skin receptors, and often we deduce the correct response indirectly from other sensory information such as hearing. With the galaxy of test procedures at his disposal, it is only too easy for the otologist to think of the inner ear as the only structure involved in posture.

Posture is the ability to maintain the upright position under all physiological conditions. It should be remembered, however, that one does not need to be upright to have posture.

Tone is a state of reflex contraction of a muscle, and the proper distribution of tone in the various muscles, which is in turn controlled by higher centres, maintains posture. Tone depends on the myotatic or stretch reflex.

The Stretch Reflex.

Limitation to movement is ultimately anatomical. Normally, physiological mechanisms come into play to prevent structural damage but in tabes dorsalis, for

example, where afferent fibres are damaged, joints may be forced unknowingly beyond their limits with the resultant Charcot joint.

If the tendon of a healthy muscle, in full connection with the nervous system, is pulled on by any type of force, the resistance from that muscle will be a reflex contraction, the stretch reflex. It is a feed back or 'servo' system, the contraction of the muscle being regulated according to the extent needed to prevent the fibres being substantially elongated. The fact that the stretch reflex is a monosynaptic reflex, means that there is no after discharge, a short delay and no recruitment.

A muscle spindle consists of intrafusal fibres each attached at one end; their free ends are attached to the annulospiral ending. When the intrafusal fibres contract, they stretch the annulospiral ending.

In the anterior horn there exist α cells and γ cells. The α efferent fibres supply the ordinary muscle fibres but not the spindle; the γ efferent supplies the intrafusal fibres of the spindle. If we want to contract a muscle, we do it by means of the cortico-spinal tract firing on to the γ efferent cell which causes a contraction of the intrafusal fibres. This stretches the annulospiral ending and the afferent impulses fire on to the

Figure 1: A schematic representation of the stretch reflex. A muscle spindle is represented between two muscle fibres. A.S. = annulospiral afferents, F.S. = flower spray afferents, G.T. = Golgi tendon organ, R.C. = Renshaw cell.

α cell and this leads to a muscle contraction (Figure I). Flower spray afferents also come from the muscle spindle to fire on to the α cell but their function is unknown.

If the cortico-spinal tract fired directly on to the α cell (as it does for a sudden or unexpected movement), the response would be much quicker; the γ efferent system is a self regulating feed back mechanism and so a much smoother movement results. While the cortex knows what any muscle is doing at any time, it does not know the exact state of contraction, so by means of the γ efferent system the muscle has some control over what it is going to do and can grade its own contraction.

When a muscle contracts its antagonist relaxes because the annulospiral afferent fibre sends an inhibitory fibre to the antagonist. The antagonist also sends an inhibitory fibre to the protagonist. If a muscle is stretched more than usual, inhibition to stop injury occurs from the Golgi tendon organ (Figure I). In normal activity it may be stimulated to inhibit the reflex, but it may be that the organ is only protective at the extremes of movement to prevent injury. A third inhibitory factor is the Renshaw cell which is situated near the α cell. If impulses are going too fast from the α cell, the Renshaw cell will fire back on it to damp it down.

Muscles always have tone, i.e., are always contracted, and to discover why they do not fatigue it is necessary to examine motor unit activity.

Motor Unit Activity.

Skeletal muscle if stimulated once will respond with a single contraction which, unlike cardiac muscle or nerve fibres, has a very short refractory period. If the muscle is stimulated at higher rates, it shows, firstly a partial tetanus and then a complete tetanus.

Postural movements are smooth and sustained. A partial tetanus is sustained but not smooth and a complete tetanus is smooth but not sustained. What is used, therefore, are two motor units in asynchronous partial tetanus and the result is both smooth and sustained.

When more work needs to be done, more and more motor units are brought into play (recruited).

Muscles are mixtures of pale and red fibres. In postural movements mainly red fibres are used as they are slowly contracting and have slow metabolic rates.

The Spinal Animal.

The spinal animal shows the myotatic reflexes by themselves, completely, and also certain intersegmental tracts that connect all the myotatic reflexes together.

When spinal shock passes off, flexor reflexes recover first but there is only a slow recovery of extensor reflexes. This means that flexor muscles recover tone but extensor muscles do not, because while both flexor and extensor muscles are affected by higher centres, the extensors are more influenced.

The Decerebrate Animal.

The decerebrate animal is one in which the forebrain has been removed by a section of the midbrain. There still exists the vestibulo-spinal tract, the cerebellum, the neck reflexes and part of the facilitatory lateral reticular formation. The main differences between this animal and the spinal animal are due to the vestibulo-spinal tract.

The decerebrate preparation shows greatly increased tone in all extensor myotatic units which, in the normal, are damped by higher centres. If the vestibulo-spinal tract is then divided, the extensor rigidity vanishes and it becomes very like a spinal animal.

Consider now, two decerebrate animals A and B. If the cerebellum is removed from B, there is not much change apart from a little more rigidity. If all the dorsal nerve roots are now divided in both animals, B will remain in the same state, but A loses all its extensor activity and becomes a spinal animal. Thus, the cerebellum directs

the vestibulo-spinal tract to fire on to the γ efferents. When the afferent fibres are divided, the γ efferent is still being bombarded in animal A, but since the afferents are cut, nothing returns to the α cell. In animal B, the vestibulo-spinal tract fires directly on to the α cell and so no effect ensues on cutting the afferent fibres. If the cerebellum is now removed from animal A, it will regain its tone and not be flaccid because the vestibulo-spinal tract fires directly on to the α cells, (POLLOCK & DAVIS, 1927).

The vestibulo-spinal tract stimulates the extensor myotatic reflex but has not much effect on the flexor reflexes.

The Vestibular Reflexes.

The vestibular apparatus supplies information to the brain about the position and movements of the head. This information is used as a basis for determining the postural adjustments which are necessary in the musculature of the limbs, trunk and eyes. It is not the only source of information about the position of the head in space; both proprioceptors and visual sources of information provide important clues about posture, and these alternative sources become particularly important when the function of the labyrinth is disturbed.

The decerebrate animal is able to orientate its head in space and shows the vestibular reflexes better than the normal.

If a person leans forward and the head tilts forward, the vestibular reflexes bring the head up to the right place again. Fibres go from the vestibule to the vestibular nuclei and from there, via the vestibulo-spinal tract, the neck muscles contract to re-orientate the head in space.

The function and physical properties of the otoliths and semicircular canals will be examined more fully in the next section.

The Vestibular Nuclei.

There are four vestibular nuclei on each side - medial, lateral, superior and inferior - of which the medial nucleus is the largest. It lies on the floor of the fourth ventricle and is situated both in the pons and medulla.

The fibres of the vestibular nerve bifurcate to form ascending and descending branches. The inferior nucleus lies at the lower end of the descending tract. Near the termination of the ascending tract are a number of multipolar nerve cells designated as the lateral nucleus (Deiters); the upper part of this, at the level of the facial nerve, is called the superior nucleus.

Other ascending fibres pass into the anterior and posterior lobes of the cerebellum. From the lateral nucleus, fibres pass medially to the medial longitudinal bundle; they pass upwards to the nuclei of the oculomotor, trochlear and abducent nerves and form the basis of nystagmus movements. Other fibres pass downwards to the nucleus of the accessory nerve to concern the regulation of head movement. Fibres arise from the lateral nucleus to pass directly to the spinal cord to form the vestibulo-spinal tract. The superior nucleus sends fibres directly to the midbrain, chiefly to the oculomotor and trochlear nuclei.

Various groups of afferents end in specific regions in the lateral nucleus, (BRODAL, 1964). Afferents from the cerebellar cortex and fastigial nucleus have specific areas of termination within the nucleus. These pathways, like the vestibulo-spinal tract, are both somatotopically organised. There is thus a route for impulses from the 'spinal areas' of the cerebellum to the cord which explains that somatotopically localised responses can be obtained on stimulation of these cerebellar areas.

The fibres from the utricle and saccule have destinations that are distinct from those of the semicircular canals (LORENTE de NO, 1933). Stimulation of the

vestibular nuclei show predominantly ipsilateral effects on somatic musculature (THULIN, 1953); contralateral effects were restricted to the medial nuclei.

Damage of the vestibular nuclei gives rise to signs which are similar to, but more severe than, labyrinthectomy on the same side. Even after labyrinthectomy on both sides, damage to the vestibular nuclei gives rise to disturbance of posture.

The Vestibulo-Ocular Reflex.

When the head moves, the eyes move; since the eyes are attached to the head they must share in its movements. If the head makes small movements, however, there may be movement of the eyes relative to the head. These movements concern the vestibule and constitute the vestibulo-ocular reflex.

The vestibule, through its system of linear and angular accelerometers, can give information about movements of the head and this forms the afferent side of the reflex arc; the efferent side involves the passage of impulses along the motor nerve to the extra-ocular muscles.

If the head moves one degree to the right, the eyes move one degree to the left with respect to the head, and the result is that the image of any object will continue to occupy the same point on the retina. Thus, the reflex

is compensatory to prevent the blurring of the retinal image during head movements and ensures that visual information is always available. If the head moves through a larger radius, however, only partial compensation occurs and the retinal image speed is only diminished. Also, if the head moves very slowly, no compensation occurs. If compensation does occur it is only momentary because the eyes soon drift back to the more useful central position, (BEGBIE, 1965).

If the centering device were peripheral it would necessitate the cupula being locked to the central position of the eyes. More likely, however, is the explanation of BENDER (1955) who showed that if areas in the paramedian zone of the diencephalon and brain stem were stimulated, the eyes snap back to centre from an initially deviated position. The fact that the fast phase of nystagmus is part of the vestibulo ocular subcortical reflex was shown by HENRIKSSON (1955) with decorticated animals, where it was possible to elicit the vestibular nystagmus with both components.

MONTANDON and MONNIER (1964) showed that in animals, stimulation of a diencephalic dorso-lateral region, medial to the lateral geniculate body, always elicited a horizontal nystagmus with the quick component to the

opposite side. Direct stimulation of the vestibular nuclei (superior and medial) with the same stimulus resulted in a horizontal nystagmus to the same side and, in some cases, a vertical nystagmus. If both the diencephalic and vestibular nystagmogenic areas were stimulated at the same time, the effects cancelled each other out; if the stimulations were crossed, the effects were additive. They concluded that the diencephalic nystagmogenic area moderated the nystagmogenic action of the ipsilateral vestibular nuclei and increased the action of the contralateral vestibular nuclei.

Tonic Neck Reflexes.

In addition to the position of the head affecting posture through the otolith organs, it also affects posture through the receptors in the neck.

The afferent part of the neck reflex is from C1, 2 and 3. When the vestibule brings the head up in the leaning forward position, the neck reflexes bring the rest of the body up in line with the head. C1, 2 and 3 afferent fibres are connected with all other myotatic units in the body by intersegmental tracts.

If the decerebrate animal is labyrinthectomised to see the reflex free from the complications of the otolith effects, it is found that turning the neck towards one side

of the body gives rise to an extension of the limbs on the same side and a flexion of those on the opposite side.

Some of the receptors at least concerned with these reflexes lie in the joints of the neck, especially the atlanto-axial and atlanto-occipital joints (McCOUCH, DEERING & LING, 1951). The effects of these endings are ipsilateral, for if the joints on one side are denervated, the neck reflexes operate only on the contralateral limbs.

BIEMOND (1939) examined the relationship between the cervical nerves and dysequilibrium by section of the upper posterior cervical nerve roots in a rabbit. A positional nystagmus appeared a few minutes after section and only appeared when the head was moved on the trunk. Tracing the degeneration of C2, 3 and 4 posterior roots by the Marchi method of staining, he followed it to the vestibular nucleus. Posture experiments in the lower orders (fish, birds and quadrupeds) are not often applicable to man, who has a totally different posture, but BIEMOND (1940) also described a case of posterior cervical root section in a man for torticollis done under local anaesthesia; the patient showed similar positional nystagmus for about an hour after the operation.

COHEN (1961) showed that when C1, 2 and 3 were bilaterally anaesthetised in baboons, all showed severe

disorientation, imbalance and motor inco-ordination like labyrinthectomised animals.

It is as important for an organism to know the position of its head in relation to its body as it is to know the position of its head in space. Orientation of the head in space is due to the vestibular apparatus, but this cannot inform the brain of the angle formed by the head and body. This can only be done by neck proprioception, and without this information it is inadequate merely to have information indicating the position of the head in space as, in COHEN's experiments, severe defects in equilibrium and orientation occurred despite the fact that the vestibular structures were completely intact. It has been shown that proprioceptive information derived from the muscles and joints of the neck have a considerable importance in visual tracking carried out while the head was moved with respect to the body, if the latter remains stationary (BARLOW, 1962).

The Body Reflexes.

Other parts of the righting reflex depend upon information derived from nerves supplying the skin. MAXWELL (1923) pointed out that a labyrinthectomised dogfish does not right itself when placed supine in the water, but will do so as soon as it comes in contact with

the bottom of the tank.

If a decerebrate animal is placed on its left side, afferents come from the left side to the spinal cord and activate the intersegmental tracts to myotatic reflexes in the neck; the neck then moves to bring the body back into line. This is the body to neck reflex.

In a similar fashion, a labyrinthectomised decerebrate animal with its head in a plaster collar will still make attempts to get up, but unsuccessfully. This is the body to body reflex.

Similar cutaneous reactions may be important in man.

Vision.

The previously mentioned reflexes are all components of the righting reflex. The last but by far the most important in man is the visual component.

In animals, the lower visual centres are good enough, but in man, the higher visual centres are needed for the righting reflex. Man uses many visual cues for orientation and the features of the environment considered significant will depend largely, if not entirely, on learning.

Some idea of the effect of vision and the vestibulo-ocular reflexes can be had from referring to the excellent descriptions of post-labyrinthectomy sensations in man by

FORD and WALSH (1936) and CRAWFORD (1952).

Standing.

In man, the centre of gravity is elevated high over a small base of support, and so, when a person stands, he is rarely, if ever, perfectly still; the body moves back and forward and from side to side irregularly, and these movements are a little increased if the eyes are closed.

Afferents from the feet and ankles appear to be unimportant as shown by ORMA (1957) who abolished sensibility by cooling. He made the interesting observation that females swayed more than males when standing in ice cold water. In females, sight and cutaneous sensibility of the soles are, by the side of other postural reflexes, of importance, but not in men in whom other postural reflexes dominate over those stimulated by the soles and sight. Males are, on average, taller than females, but there is no correlation between height and amplitude of sway. ORMA came to the conclusion that if muscular strength is great, muscular myotatic reflexes are used almost exclusively in the maintenance of standing equilibrium and in this case the amplitude of swaying is small; if muscular strength is not great, other afferent parts, the soles and sight, play a more important role in postural mechanisms, and in this case the amplitude of

swaying is wide.

During standing, KELTON and WRIGHT (1949) found that soleus was inactive for periods from 1 - 5 seconds, whilst tibialis anterior was inactive from 1 - 3 minutes. All other muscles they investigated were inactive for long periods of time. JOSEPH and NIGHTINGALE (1952) found constant activity in soleus and a variable amount of activity in gastrocnemius.

It is obvious, however, that since the weight line falls slightly in front of the ankle joint, the calf musculature is called on to counteract a gravitational pull.

SECTION 2

REVIEW OF THE LITERATURE ON
APPLIED VESTIBULAR PHYSIOLOGY
AND VESTIBULAR FUNCTION TESTS

In this section an attempt will be made to review the more important parts of the literature concerning applied vestibular physiology, and in so doing, the development of the various tests of vestibular function will be described.

Historical.

BROWN-SEQUARD (1860) pointed out that thermic stimulation of the vestibular apparatus caused vertigo. Professor CRUM BROWN (1874) of Edinburgh University was the first to point out that the end organ in the ampulla of a semicircular canal is stimulated by a flow of endolymph in the canal. About the same time, BREUER (1874) and MACH (1875) came to the same conclusion. BREUER (1889) showed that vestibular stimulation caused nystagmus.

In 1892, EWALD published his famous series of experiments on the semicircular canals of pigeons. He sealed off a semicircular canal with dental cement and then inserted a pneumatic hammer between the blocked area of the canal and the ampulla, thus enabling him to exert a positive or negative pressure on the endolymph.

His findings have had the force of laws. A flow of endolymph towards the ampulla (ampullopetal) in a lateral canal is a maximal stimulus, whereas a flow away from the ampulla (ampullofugal) is a minimal stimulus. In the case of a vertical canal, a flow of endolymph away from the

ampulla (ampullofugal) is a maximal stimulus, whereas a flow towards the ampulla (ampullopetal) is a minimal stimulus.

In lower animals (fish, frog etc.) Ewald's law must be modified, as the canal response is unidirectional, in the direction of the so called maximal stimulus of Ewald only (LAUDENBACH, 1899; MAXWELL, 1920; STEINHAUSEN, 1931; ROSS, 1936).

According to BARANY (1906), cold water causes ampullofugal movement, and hot water, ampullopetal.

LORENTE de NO (1931) studied eye muscle reactions to labyrinthine stimuli in rabbits. He concluded that Ewald's laws hold if the stimulation of the labyrinth was within physiological limits, but if the stimulation was excessive or carried out in an unusual plane, then the reactions may not follow Ewald's laws.

GRAHE (1921) reported that clinical reactions to hot (47°C) caloric stimulation was not so definite as reactions to cold (27°C) stimulation.

FITZGERALD and HALLPIKE (1942) found that in 80% of their patients, the reactions to cold caloric stimulation were slightly in excess of the reactions to hot caloric stimulation. Since they were using equal and opposite stimuli (7°C above and below body temperature), they concluded that Ewald's laws do not apply to man.

The present concept of vestibular activity is as follows. At rest, the whole vestibular system is in a state of tonic activity, manifested by a steady discharge of action potentials in its peripheral neurones. The vestibular tonus of the two sides has equal and opposite actions on the eyes and skeletal muscles. If the action of one side is reduced, then the normal side will predominate, leading to nystagmus with its quick element to the normal side.

All vestibular neurones have a resting discharge; alterations in the movement of the head, be they by angular or linear acceleration forces, bring about fluctuations of this discharge. For the lateral semicircular canal, for example, ampullopetal displacement leads to an increase in the resting discharge level of action potentials, and ampullofugal displacement leads to a decrease.

Vestibular dysfunction is shown by spontaneous manifestations (e.g. nystagmus and abnormalities of muscle control) and abnormal induced manifestations, which are abnormalities of basic reflex responses.

Rotation Tests.

Post-rotatory reactions, both nystagmus and after sensation, have been used in vestibular function testing since BARANY (1907) introduced his rotation test.

When a person is rotated with the head inclined 30° forward, at constant velocity, the deviation of the cupula provoked by the initial acceleration will disappear completely. In this state of equilibrium, the subject has no turning sensations. When the movement is stopped, the deceleration causes the cupula to be deviated in the opposite direction, and both the sensations and reactions have a direction opposite to that during the acceleration phase.

In the original Barany test, the subject was rotated ten times in twenty seconds, and then the movement was abruptly stopped. On the average, 22 seconds are needed after stopping, for the eyes to be at rest again.

There are many criticisms of this test. Since the period of constant velocity lasts less than 20 seconds, the cupula, which has been deviated by the great initial acceleration, does not return to its original position. The sudden cessation of rotation stimulates the cupula while it is still in a deviated position, and so it is impossible to give an accurately dosed stimulus to the cupula.

VEITS (1931) evolved a slightly better method whereby the subject was rotated subliminally , so that the cupulae were not stimulated until a speed of $180^{\circ}/\text{sec.}$ was reached. This velocity is maintained for a few minutes until there is no nystagmus or sensation, and only then is the turning stopped. The stimulus is, however, very severe, unphysiological, uncomfortable and liable to damage a sick labyrinth (VAN EGMOND & JONGKEES, 1948).

STEINHAUSEN (1931) discovered that the cupula in the living animal (the pike) reaches to the roof of the ampulla, acting like a hermetically sealed swing door with spring like properties. It had been thought by previous investigators that the cupula did not reach further than half way to the roof of the ampulla. Steinhausen's finding produced a totally different picture, and he was able to demonstrate that during angular acceleration, the inertia of the endolymph caused the cupula to be deflected. After the initial acceleration, the deflected cupula returned slowly to its rest position under the action of its own restoring force, and it is to this slow return that the long after sensations of turning could be attributed.

On these observations, Steinhausen based his well known torsion pendulum concept of the cupular mechanism which was taken up by Van Egmond and his colleagues, who formulated a precise mathematical treatment of the cupulo-

endolymphatic system in the human (VAN EGMOND, GROEN & JONGKEES, 1949) and in the ray (GROEN, LOWENSTEIN & VENDRIK, 1952).

A semicircular canal is a physical system that is, in principle, amenable to exact analysis - a body of fluid in an annular structure, a folding flap which tends to keep in one position and returns to it when bent, and a viscosity between the fluid and the walls. Unfortunately, two difficulties arise. The first is that not all the physical constants are known. The second is that there is some uncertainty whether the usual laws of hydrodynamics may be applied to such a small structure and what should be the valid approximations (BEGBIE, 1965).

The Utrecht workers took as their basis, the differential equation of the torsion pendulum where there are no external forces. $\ddot{\xi} + \frac{\pi}{\Theta} \dot{\xi} + \frac{\Delta}{\Theta} \xi = 0$.

where, Θ = moment of inertia of the endolymph

π = moment of friction of the endolymph

Δ = restoring force for unit angular deviation of the cupula

ξ = angular deviation of endolymph in relation to the skull

$\dot{\xi}$ = angular velocity of endolymph in relation to the skull

$\ddot{\xi}$ = angular acceleration of endolymph in relation to the skull

The values of the constants were established as follows:-

$$\pi/\Delta = 10 ; \quad \theta/\Delta = 1.$$

Thus the behaviour of the cupula in response to any given rotational stimulus could be theoretically predicted. The approximate solution of the above equation relating to a sudden stopping of the chair from a constant velocity γ is:- $\xi = \gamma \frac{\theta}{\pi} \left[e^{-\frac{\Delta t}{\pi}} - e^{-\frac{\pi t}{\theta}} \right]$ with the limiting condition, $t = 0, \xi = 0$ and $\xi = \gamma$.

The slow return of the deflected cupula is determined by the term, $e^{-\frac{\Delta t}{\pi}}$. If the after sensation expires when the cupula has reached a minimal perceptible deflection $\xi_{min.}$, then the duration of the after sensation t_u is given by $t_u = \frac{\pi}{\Delta} \log \left[\frac{\theta \gamma}{\pi \xi_{min.}} \right]$. If a number of determinations are made of the after sensation durations using different stopping velocities, then a logarithmic plot of the impulse against time should yield a straight line curve, the slope of which is the constant $\frac{\pi}{\Delta}$.

This graph is called a cupulogram and the tests required for its determination are known as cupulometry. The after nystagmus is plotted in the same way.

The nystagmus cupulogram does not have the same slope as the sensation cupulogram and Van Egmond et al (1949) suggest that the two curves are independent expressions of

the activity of the vestibular organ.

It is as well at this point to note that the term, torsion pendulum, is semantically incorrect. The cupula is not a true torsion pendulum (cf. a quartz rod) but the formulae of a torsion pendulum apply to it.

The above approaches assume that the endolymph moves as a solid block, i.e. they leave aside the more hydrodynamical aspects. DE VRIES (1956) takes a more rigid view about the physics and relates cupular displacement to subjective sensation of rotation only. Some of the constants he assumes are taken from a wide variety of organisms and are unlikely all to apply to any one creature.

GROEN (1956) makes the further assumption that cupular displacement is exactly paralleled by eye displacement and, therefore, he feels able to use his equation to describe eye movements and not just cupular movements. If cupular movements were exactly paralleled by eye displacement, one might expect that the eyes would be maintained for some time in a deviated position and only very slowly regain their central position. What happens, however, is that the central centering device (BENDER, 1955) brings the eyes back to the central position and nystagmus beats result which become increasingly less in velocity and amplitude. The slow phase is abolished by bilateral labyrinthectomy and the fast phase by barbiturates.

BYFORD, HALLPIKE and HOOD (1952) were able to confirm the validity of the results of Van Egmond et al in similar tests also using the duration of the subjective turning sensation as an indicator.

HALLPIKE and HOOD (1953) attempted to measure the speed of the slow component of the nystagmus induced indirectly, using GRAYBIEL'S (1946) hypothesis that the oculo-gyral illusion is dependent on nystagmus and its slow component. Their investigation of the oculo-gyral illusion consisted of tests where the subjects had their eyes open and fixed on a small dimly lighted point. In these tests, the not unimportant ocular influence on nystagmus had to be taken into account.

ASCHAN (1955) considers that two movements occur in cupular deviation - torsion of the cupula itself and also sliding of the cupula as a whole over the surface of the crista. He quotes DOHLMAN'S film (1938), in which the cupula of a cod was made visible by contrast injections. From this, the conclusion is that when acceleration ceases, the torsion of the cupula itself disappears very quickly, probably within a second; the phenomena which occur later are due to the fact of the cupula sliding slowly back over the crista to its rest position. The abrupt change of speed of the slow component of the nystagmus noted in his human rotation experiments is ascribed to the torsion of

the cupula itself. Any further reactions are due to the sliding movement of the cupula.

Although the stimuli used in cupulometry are rather weak, habituation, or as Hallpike and Hood (1953) called it, 'response decline', will occur.

In conclusion, it can be said that since in rotation tests, both labyrinths are stimulated simultaneously, deviations from the normal response are difficult to interpret in terms of disordered function of a particular labyrinth; to this extent the diagnostic value of these tests is limited.

The physical forces engendered within the canals by rotational stimuli, however, can be calculated with considerable precision. This has been the reason for the great advances in knowledge over the last 20 years of the physical characteristics of the canal mechanism in the normal human subject.

The Coriolis Reaction.

One important set of phenomena connected with rotatory systems, is known as the Coriolis effect or Coriolis force. While its practical application is mainly in the realms of space flight and space stations, it is occasionally a useful tool in the investigation of vestibular function.

It is produced if a subject, who is rotating with the head upright, is asked, when at constant velocity, to bend the head forward on to the chest and then bring it up again. There is a most unpleasant sensation, often amounting to nausea, and the subject tends to fall sideways.

The following theoretical points may make the reaction clearer.

The cupula has approximately the same density as the endolymph (GROEN, 1956), and the closed canal system is an efficient detector of angular acceleration because of unequal tangential acceleration vectors on opposite sides of the canal. The horizontal canal is a circular canal, off centre. During rotation there are two diametrically opposed points, r_1 and r_2 , which are the nearest and furthest points respectively on the canal from the centre of rotation. During angular acceleration α , αr_2 is greater than αr_1 , and this difference amounts to an inertial couple which tends to rotate fluid in the canal. Hypo-

thetically, if the canal were centred on the axis of rotation, r_1 and r_2 would be equal radii and $\propto r_1$ and $\propto r_2$ would be equal magnitudes, but their directions would complement one another, resulting in the same inertial couple as before.

If a chair with a person sitting upright on it, is propelled along a straight track with an instantaneous acceleration (a_{rel}) and velocity (v) relative to the track, and if the track is on a turntable which has an instantaneous angular velocity (w) and angular acceleration (α), the vestibular system has an acceleration (a_T) at the instant relative to the earth; these factors can be analysed into several components - linear acceleration (a_{rel}) relative to the turntable, centripetal acceleration ($w^2 r$) and tangential acceleration (αr) which are linear accelerations relative to the earth, (where r represents the distance of the vestibular system from the centre of rotation), and the Coriolis acceleration, $2vw$, the added linear acceleration.

Assuming the otolith system to be a perfect transducer of inertial force, it would respond with a magnitude and direction proportional to the vectorial sum of these four components and gravity.

If the lateral canal is in the plane of rotation (and no other canal) then the canal system is little affected by

Figure 2: P_1 and P_2 are points on a vertical semicircular canal. The canal is at right angles to the plane of rotation. The dotted lines show the head bending forwards. The Coriolis acceleration vectors are in the plane of the canal and are not opposed by the tube walls.

$2vw$.

The situation is very different if the head is tilted during rotation. Mechanical couples develop as P_2 in a vertical canal translates, relative to the plane of rotation, faster than its antipode, P_1 (Figure 2). Thus P_2 has greater v and hence greater $2vw$ than P_1 . The torque exerted by Coriolis forces around the entire circumference of the circular canal is a gyroscopic torque.

$2wv_2$ and $2wv_1$ can be resolved into $2w(v_2 - v_1)$ which is the Coriolis acceleration couple dependent on the relative velocity of v_2 and v_1 .

In the situation of a vertical canal being brought into a field of constant velocity previously occupied by the lateral canal, the Coriolis acceleration vector will be parallel to the canal walls, and the Coriolis couple will be maximally effective.

According to GUEDRY and MONTAGUE (1961), the greatest velocity and discomfort during clockwise turntable rotation, results from head return to upright from right tilt, and the greatest apparent displacement results from head return to upright from left tilt. While the intensity of the nystagmus can be equal in two subjects, the apparent velocity and discomfort experienced may differ markedly. Their experiments showed that with the Coriolis vestibular reaction, habituation occurred very quickly.

The Caloric Test.

The caloric test is one of the most generally used methods of examining the labyrinth and was first described by BARANY (1906). He suggested that the change in temperature at the drum membrane caused by syringing is conducted towards the semicircular canals by the structures in the temporal bone. The specific gravity of the endolymph in the lateral part of the horizontal canal is lowered by heating and raised by cooling, and so a flow of endolymph is produced which is dependent on the position of the canal. The movement of the endolymph is the cause of the vestibular reaction.

Older theories of the reaction: BARTELS (1911) suggested that heat had a stimulating effect, and cold a depressing effect, directly on the nerves.

KOBRAK (1918) put forward the opinion that the caloric response was caused by vascular reactions; the vessels on the periphery of the labyrinth being constricted by cold, and the central vessels reacting with dilatation resulting in a flow of endolymph and cupular deviation.

BORRIES (1922) showed that a normal caloric reaction could be caused even when the semicircular canals were extirpated. In his opinion the caloric reaction was a reaction of the whole labyrinth especially of the otolith.

BRUNNER (1921) thought that caloric nystagmus was central in origin and not due to cupular deviation.

VAN CANEGHEM (1946) explained the caloric reaction as an alteration in intra-labyrinthine pressures. Heat causes an increase in pressure and cold, a decrease. In his opinion, the increase of the intra-labyrinthine pressure had its origin in the utricle.

Of these, the theory of Barany is the only plausible one. It explains clearly the nystagmus, the turning sensation, and the deviations in walking and pointing. It also gives a good explanation of the reversal of the nystagmus when the position of the body is reversed (prone), for in this case the stream of endolymph will move in the opposite direction in the horizontal canal.

DE KLEIJN and STORM VAN LEEUWEN (1921) found that there was total ablation of the caloric reaction when the horizontal canal was exactly horizontal on a binaural axis and also when the vertical canals were placed in such a position that the thermal stimulus could produce no effect.

Evidence for extra vestibular influence on caloric nystagmus has been put forward by several authors. DE KLEIJN and VERSTEEGH (1924) showed the influence of irritation of the nasal mucosa on the caloric nystagmus.

While FUJIMORI (1924) showed that removal of the cervical sympathetic nerve had no effect on the caloric reaction, the experiments of TERRACOL (1927) showed that manipulation of the sympathetic ganglia in the neck could produce a nystagmus. THIELEMAN (1924) stopped the caloric reaction by cocainisation of the middle ear mucosa. HENNEBERT (1946) showed that brushing the temple and similar manipulations caused a diminution of nystagmus and a disappearance of the sensation.

Methods: The test may be quantitative or qualitative. The Dundas-Grant test in which air, cooled by ethyl chloride, is blown into the meatus, relies on an all or none indication of vestibular function.

KOBRAK (1918) used a quantitative evaluation by irrigating the ear with short thermally graded stimuli to estimate the caloric threshold. He used 5 ccs. of water of increasing coldness, till he arrived at a threshold value for the nystagmus provoking stimulus. This method was also used by DE KLEIJN and VERSTEEGH (1927). The test is tedious to carry out, the threshold value rather diffuse, and it is not particularly delicate (FITZGERALD & HALLPIKE, 1942). The work of DE BARRENE and DE KLEIJN (1923) and BARRE (1937-38) showed that the responses also depended on directional preponderance, i.e. that nystagmus to one side was increased and that in the opposite direction was

decreased.

In 1942, FITZGERALD and HALLPIKE described a new caloric test. It followed the lines recommended by GRAHE (1920 and 1921) and VOGEL (1929), who used water at 17° or 27°C and 47°C in small quantities. Since the latter temperature is very uncomfortable, FITZGERALD and HALLPIKE used water at 30°C and 44°C , i.e. equidistant from body temperature. The lateral canal is brought into the vertical plane by the patient lying supine with the head flexed 30° . The irrigation is for 40 seconds and not less than 250 ccs. of water are used. They found that this gave the most constant reactions and tended to overcome slight temperature differences which resulted from loss of heat in the apparatus. The test was designed to fulfil the following requirements (CAWTHORNE, DIX, HALLPIKE & HOOD, 1956).

1. That it should be easy to apply and comfortable for the subject.
2. That the results should provide an accurate and repeatable measure of the sensitivity of the lateral canal.
3. That it should be capable of revealing a directional preponderance.

The induced nystagmus lasts for about $1\frac{1}{2}$ - $2\frac{1}{2}$ minutes and is observed from a distance of about 12 inches with

reflected light. The gaze is fixed straight ahead on a mark, and so the nystagmus is of the second degree. The time interval selected for measurement extends from the application of the stimulus to the end of the nystagmic response, and a stop watch is used. The interval between the tests should be at least 5 minutes.

The results are recorded on the figure shown below, (Figure 3).

FIGURE 3.

The reactions are numbered 1 to 4 from above downwards. Reactions 1 and 4 consist of nystagmus to the right, and reactions 2 and 3 consist of nystagmus to the left.

A canal paresis can be simply represented by the formula $(1 + 3) - (2 + 4)$, and a directional preponderance by $(1 + 4) - (2 + 3)$. Combinations can occur (mixed lesions) and may be crossed or uncrossed.

HALLPIKE, HARRISON and SLATER (1951) examined 93 normal subjects in respect of canal paresis and directional

preponderance. In both, the mean value is close to zero with the standard deviations being 15.5 and 15.7 seconds respectively.

JONGKEES (1948), THOMSEN (1953), HAMERSMA (1957), STAHL (1958) and PREBER (1958) found a great variability for the normal response to the caloric test.

It is false to use the traditional absolute values for the difference in excitability of both labyrinths and for the presence of a directional preponderance. A difference of 40 seconds between left and right at an excitability level of 50 seconds per irrigation may be pathological, whereas the same difference of 40 seconds at a level of 300 seconds will fall completely within normal limits. For this reason JONGKEES and PHILIPSZOON (1964) decided to express the difference in excitability between two sides and a directional preponderance with a relative figure, i.e. the percentage of the total excitability. A canal paresis would be represented as:-

$$\frac{(1 + 3) - (2 + 4)}{(1 + 2 + 3 + 4)} \times 100\%$$

When there is a right canal paresis the result is a positive value, and when it is a left canal paresis, it is a negative value. A directional preponderance is represented as:-

$$\frac{(1 + 4) - (2 + 3)}{(1 + 2 + 3 + 4)} \times 100\%$$

A directional preponderance to the right gives a positive value, and a directional preponderance to the left gives a negative value.

In their normal series, JONGKEES and PHILIPSZOON (1964) found that the standard deviation for a canal paresis was 6.7% as regards duration, and 7.5% as regards maximum eye speed. For a directional preponderance, the standard deviation was 15% for duration and 8.7% for eye speed.

To confirm their opinion that relative and not absolute values should be used, they analysed the normal material of HAMERSMA (1957) and PREBER (1958).

Temperature Effects: The pathway of flow of temperature has been described by SCHMALTZ (1932) who was able to calculate a possible speed at which the thermally induced current travelled.

A cold irrigation cools down the lateral part of the horizontal canal and the rest of the labyrinth retains the same temperature. The difference of temperature between the different parts of the labyrinth produces a variation in density of fluid in the horizontal canal at different places and a stream of endolymph. If the stimulus acts for too long or is too cold, the cooling of the canal is not limited to its most lateral part, but reaches other parts of the labyrinth as well. Even though the loss of temperature at one point of the wall of the canal is much

larger, the difference in temperature between two different parts in the canal is less than in the first instance.

That the flow of temperature in an ample and strong irrigation reaches deeper, is shown by the fact that minimal irrigation causes a pure horizontal nystagmus, and strong irrigation, a nystagmus with a rotatory component.

VOOGD (1946) irrigated some subjects with ice water for about 20 minutes continuously. He noted that in about 5 minutes, there was no longer any sensation of turning and the nystagmus was very slight. In his experiments it was proved that the caloric stimulus even influenced the function of the cochlea, causing an experimental diplacusis.

JONGKEES (1948) points out the following facts which cannot be explained by Barany's theory.

1. That occasionally, the caloric reaction may still be elicited after the semicircular canals, or even the entire labyrinth, have been destroyed. If the cochlea is reached by the thermal stimulus (VOOGD, 1946), then the otoliths will be within reach also.
2. The above fact could also explain why the sensation of turning is usually accompanied by a sensation of changed position. Stimulation with cold water gives the same sensation as being in a position in which the otoliths of the stimulated side are lower than the other otoliths, i.e. the same stimulation as a decrease

of the pressure of the stones on their maculae. With hot water it is the reverse.

3. The predominance of the cold over the hot caloric nystagmus. Ewald's law would suggest a different result.
4. The predominance of the nystagmus in the normal position over the thermal reaction in the reversed position.
5. That the reversal of nystagmus does not take place in two planes which are separated by 180° .
6. Directional preponderance.
7. In the caloric test, nystagmus has a lower threshold than sensation, whereas in the rotation test, the opposite is the case (HULK & JONGKEES, 1948).
8. That the caloric test may be positive and the rotation test negative.

He concludes that these disharmonies may be explained by vascular influences, influences from the internal auditory meatus and also from the central nervous system.

PINNAROLI (1911) and DOHLMAN (1925) using the cadaver, showed that most of the thermal stimulus exerted its effect on the lateral canal. SCHMALTZ and VOLGER (1924) measured calorically induced temperature changes on the outer surface of the labyrinth in the human temporal bone and in radical mastoid cavities. CAWTHORNE and COBB (1954) measured the

temperatures inside the lateral canal, at the drum, and in the cannula carrying the water to the meatus in the living human subject. They found that the temperature at the drum changed to that of the water, and after the flow had ceased, it returned to its former level following a roughly exponential curve. Recovery of temperature to normal levels had not always occurred after 10 minutes. The temperature inside the lateral canal showed little change during the first 10 - 15 seconds. The greatest rate of change usually took place between 20 and 70 seconds. The peak value with water at 30°C was reached in 105 seconds and with water at 44°C it was reached in 101 seconds. The peak change with cold water was a mean of 0.67°C and with hot water the mean of the peak change was 0.8°C . The temperature in the lateral canal had returned to within 0.1°C of normal in 10 minutes.

The intensity of the reaction varies with the length of time the stimulus is applied to the labyrinth (DOHLMAN, 1925). The lateral canal is most affected, the superior canal next and the posterior vertical canal is least affected by the temperature change.

Habituation: This is the phenomenon of progressive reduction of the nystagmic response to either repetitive rotatory or caloric tests.

Habituation to rotatory tests is well documented.

In man it has been reported by GRIFFITH (1920a, 1924), HOLSOPPLE (1923), DODGE (1923), DUNLAP and DORCUS (1926), WENDT (1951) and ASCHAN (1954). In animals it has been noted by GRIFFITH (1920b), DETLEFSEN (1923), MAXWELL, BURKE and RESTON (1922), GOULD (1926), FEARING (1926, 1941), MOWRER (1934), HALSTEAD (1937) and HOOD and PFALTZ (1954).

Habituation has three characteristics - acquisition, retention and transfer (HALSTEAD, YACORZYNSKI & FEARING, 1937). Acquisition is the progressive decline of the nystagmic response and this may vary from a moderate response decline to absence of response. Retention is the persistence of the neural process underlying the response decline; it is transitory both in man (McCABE, 1960) and animals (FEARING, 1941). Transfer means that habituation to rotatory stimuli in one direction produces almost equal habituation to rotation towards the opposite side. The work of HALSTEAD, YACORZYNSKI and FEARING (1937) strongly suggests that habituation is partially associated with a functional change in the cerebellum.

Whether habituation occurs to repetitive caloric tests is a moot point. In man, HAMERSMA (1957) showed that response decline occurred in the maximum eye speed, but HOOD and PFALTZ (1954) failed to show any response decline in the duration or frequency of the nystagmus. The latter authors produced no habituation in rabbits, but DUNLAP

(1925) showed that it did occur. This was confirmed in cats by HENRIKSSON, KOHUT and FERNANDEZ (1961); they also showed that habituation was abolished by barbiturate anaesthesia. According to HENRIKSSON (1965), habituation to the caloric stimulus also occurs in man.

With every movement of our head, we are using the vestibular reflexes and there is no sign of habituation. It seems as if suppression of these reflexes occurs when the magnitude of the stimulus is above normal physiological values as in the rotation or caloric test. McCABE (1960) observed that in figure skaters, not only the vestibulo-ocular reflex arc is suppressed, but also the postural reflexes. The experiments of HENRIKSSON et al (1961) support previous opinions that neither fatigue nor adaptation of vestibular receptors cause habituation. The cerebellum may participate in the process (HALSTEAD et al, 1937) but the medial reticular formation could also play a part.

Efferent Activity: GRANIT (1955) demonstrated a nervous centrifugal control of many peripheral receptor organs. The olivo-cochlear bundle of RASMUSSEN (1946) is well known. Efferent fibres to the vestibule were first found by RASMUSSEN and GACEK (1958). Electron microscopy of the crista has indicated a double innervation (WERSALL, 1956; ENGSTROM, 1958).

LEDOUX (1958) recorded a change in the resting activity of the vestibular nerve in the frog during stimulation of the other side.

HENRIKSSON et al (1961) reported that repeated monaural caloric irrigations in cats, caused a response decline in the non irrigated ear, provided that the test stimulus provoked nystagmus in the same direction as the preceding repetitive tests. The existence of nervous connections between the labyrinths might be the mechanism of transfer of habituation to equivalent stimuli.

Conclusions: The caloric test is the most widely used test of vestibular function. The stimulus is applied in a controlled manner to a single known end organ. Higher centres, however, exert an unknown and variable action on this end organ. As distinct from the rotation tests, the method has yielded comparatively little information about the exact physical characteristics of the canal mechanism, however, as the physical forces caused by the stimuli within the canals cannot be readily computed. There are many difficulties in the accurate performance of the test and its interpretation. These will be discussed in Section 6.

Position Tests.

The otoliths act as linear accelerometers and are always subject to the force of gravity. Their responses vary with their inclination, and thus they signal the orientation of the head.

Terminology: ALEXANDER classified nystagmus into 1st, 2nd and 3rd degree according to the direction of the visual axis. This is applicable to the clinical observation of vestibular nystagmus whether spontaneous or induced.

Since Barany's early writings it has been known that nystagmus may depend on the position of the head and may change in character on changing posture. Positional nystagmus was defined by NYLEN (1931) in contrast to spontaneous nystagmus; he suggested that the term spontaneous nystagmus be restricted to nystagmus behaving in the same manner no matter what the position of the head. JONGKEES (1953) states that positional nystagmus is spontaneous nystagmus which changes in direction or intensity with changes in posture. Probably the best classification of positional nystagmus is NYLEN's (1950), which is as follows:-

1. Type 1 is direction changing nystagmus. This is nystagmus beating in one direction in a certain head position and beating in the opposite direction when the head position is changed to another.

2. Type 2 is direction fixed nystagmus. This is nystagmus which always beats in the same direction and is present in all or only some head positions, and which may change in intensity on change of head position.
3. Type 3 is irregular positional nystagmus and consists of nystagmus which may change in direction in any given position. It also embraces other forms of nystagmus influenced by posture and not referable to one of the other groups.

The terms direction changing and direction fixed were first introduced by SEIFERTH (1937). FRENZEL (1938) combines types 1 and 3 under the heading true positional nystagmus and terms type 2 as spontaneous nystagmus released by position stimulus.

Provocation nystagmus (FRENZEL, 1955) is the nystagmus present after repeated posture testing, or nystagmus present after stimulation of vestibular function tests, which is present in a position where none could previously be recorded.

Persistent nystagmus is nystagmus of unlimited duration in a given position. Transitory nystagmus is nystagmus that in a given position can only be noted for a short period. FRENZEL (1955) states that 30 seconds is the limit between these two types of nystagmus, while LINDSAY (1951) puts the limit at 1 minute. Transitory

nystagmus is generally classified under Nylen type 3.

The benign positional nystagmus described by BARANY (1921), NYLEN (1927) and DIX and HALLPIKE (1952) is characterised by a latent period of 5 - 6 seconds, a nystagmus to the affected ear when this is underneath, with distress, a quick decline and disappearance of the nystagmus, and a reversal of the nystagmus for a short time on sitting up. Repetition of the test ultimately produces extinction. Caloric and audiometric tests show no abnormality.

Methods: The standard method to test for positional nystagmus (usually employed with electro-nystagmography) is to test in the following positions - upright, prone, supine, head right and head left (usually in the supine position but occasionally in the upright position), and head hanging.

To test for benign positional nystagmus the following procedure is adopted. The subject, from the sitting position, is quickly laid supine with the head lowered 30° and turned $30 - 45^{\circ}$ to one side. The movements are made with the examiner holding the patient's head.

ASCHAN et al (1957) examined the two questions,

1. Is the releasing factor, the actual position of the head?
2. Is change of position of the head significant?

In the answer to these questions lies the best method of testing - whether it is better to use a very slow turning posture table, or whether it is better to examine the patient on a couch with fairly rapid movements of the patient's head.

They used a special electrically driven posture table and found that when dealing with persistent forms of positional nystagmus, the position of the head was the determining factor for releasing the nystagmus. For transitory and transitional forms of positional nystagmus, the movement of the head into the new position also played an important part, often the dominating one, in the releasing mechanism.

They concluded that for clinical purposes, the best way to examine for positional nystagmus is to turn the patient fairly rapidly into the different positions. When turning slowly, transitory forms of positional nystagmus might be overlooked.

Theories of cause of positional nystagmus: According to NYLEN (1929), the origin of positional nystagmus is to be sought in changes of intravestibular pressure produced by changes in position.

Centrifuging the otoliths from their maculae gave rise to positional nystagmus in several experiments, (HASEGAWA, 1925; NYLEN, 1926).

Removal of the macula sacculi did not influence positional nystagmus caused by alcohol intoxication (DE KLEIJN & VERSTEEGH, 1930). Introduction of amalgam into the inferior part of the labyrinth caused positional nystagmus if the amalgam touched the utricular membrane.

JONGKEES (1949) showed from his experiments in rabbits that positional nystagmus can be caused by damage to the peripheral labyrinth even when the function of the semi-circular canals seems to remain normal. Positional nystagmus with changing direction may be caused by lesions of the peripheral labyrinth.

ASCHAN, BERGSTEDT and STAHL (1956) showed that alcohol given in even a small single dose produces two phases of positional alcoholic nystagmus (P.A.N.). In phase 1, the nystagmus beats to the right in the right lateral position, and to the left in the left lateral position; the directions are reversed in phase 2.

LINDSAY and HEMENWAY (1956) argued that "the continuation of active impulses from part of the labyrinth receptors, with sudden loss of other receptors, appears to be the situation which is characterised by postural vertigo and a positional nystagmus."

CITRON and HALLPIKE (1956) suggested that benign positional nystagmus was due to a qualitative disorder of the otolith apparatus; this was based on the histological

findings in a human temporal bone. This showed an absence of the otolith membrane, disorganisation of the sensory epithelium, and certain gross tissue changes in the connective meshwork underlying the epithelium of the utricle of one ear.

The postulate that nystagmus was a qualitative response of the affected otolith apparatus was based on the fact that the positional nystagmus was eliminated by surgical destruction of the affected labyrinth in two cases.

RIESCO-MAC-CLURE (1957) postulated that the incoming normal signals, which arise in the vestibular receptors through change in the position of the head, produced a qualitatively abnormal response of the nerve cells in the vestibular nuclei. The abnormal response determines the transient and paroxysmal characteristics of the postural nystagmus. He supports this hypothesis with the surgical finding in a human case, of an astrocytoma of the inferior vermis of the cerebellum. The patient had benign positional nystagmus and postural vertigo with no signs of peripheral lesions.

FERNANDEZ, ALZATE and LINDSAY (1959) carried out a large series of experiments in cats to try to elucidate the physiological basis underlying postural nystagmus.

Section of one utricular nerve produced benign positional nystagmus showing that a lesion in the otolith

apparatus may be accompanied by positional nystagmus.

The fact that sudden unilateral loss of vestibular function was not followed by positional nystagmus, was shown by doing a unilateral labyrinthectomy.

Unilateral section of the utricular nerve and nerves to the lateral and superior canals produced positional nystagmus, showing that sudden unilateral partial loss of vestibular function may be followed by positional nystagmus.

Stimulation of the utricular nerve did not produce nystagmus, while stimulation of the nerves to the canals provoked horizontal and vertical nystagmus, supporting the observation that positional nystagmus is not likely to be of otolithic origin.

Lesions of the flocculonodular lobe of the cerebellum produced a benign positional nystagmus, suggesting that positional nystagmus may be due to the release of vestibular centres from cerebellar inhibition.

Positional nystagmus produced by ablation of the nodulus was temporarily eliminated by cocainisation of both labyrinths, and bilateral labyrinthectomy produced a permanent disappearance. Bilateral labyrinthectomy followed by ablation of the nodulus failed to produce postural nystagmus. This indicated that the presence of signals with information as to the position of the head in space is necessary for producing the postural nystagmus of

cerebellar origin. Disappearance of positional nystagmus by disease or unilateral labyrinthectomy does not necessarily mean that positional nystagmus originated in the vestibular sense organ.

A further series of experiments in 1960 showed that the nodulus acts as an inhibitor of the vestibular centres.

The flocculonodular lobe is phylogenetically the oldest division of the cerebellum and it develops from the vestibular nuclei. Its afferent and efferent connections are mainly with the vestibular system.

It is as well, at this point, to recall (see page 12) that BIEMOND (1939) produced positional nystagmus in rabbits by cutting the posterior cervical nerve roots.

Conclusion: Testing for positional nystagmus is an important, informative and widely used test of vestibular function. The genesis is by no means clear as yet. There is evidence for a peripheral, a cerebellar and a cervical cause.

Parallel Swings.

The origin of the sensations of the normal human subject to linear motion has been investigated since the late 19th century. Neither DELAGE (1888) nor TRAVIS and DODGE (1928) were convinced that vestibular receptors were important.

Methods: MACH (1875) placed a person on a large balance and noted the conditions under which, with the eyes covered, movement could be perceived as the balance swung up and down. The sense of rising came at, or just before, the bottom of the excursion, the sense of falling, at or just before, the top. The sensation was thus most striking when the acceleration was greatest. For rhythmic horizontal movements, the sense of motion is again ahead of the motion.

DELAGE (1888) used a parallel swing. BOURDON (1914) used a trolley pulled by a falling weight, and TRAVIS and DODGE (1928) used an oscillating platform. JONGKEES and GROEN (1946) used a parallel swing and GRAYBIEL and PATTERSON (1955) used a human centrifuge. With all the above methods, the accelerations at threshold varied from 2 - 26 cm./sec.².

WALSH (1961) described a parallel swing which was a stretcher suspended with wires 1.6 metres long, giving a period to the motion of 2.5 seconds. Both longitudinal

and transverse movements were used, and he suggested that with care, yawing motion could be avoided.

JONGKEES and PHILIPSZOON (1964) did not agree with this and used a square frame of steel tubes 2 metres wide, and in the middle of this, two other tubes were welded, between which, canvas was fixed as in a stretcher. The length of the cables was 3.38 metres and the maximum acceleration was 167 cm./sec.^2 . The torsion was very slight.

Compensatory eye movements occur, consisting of rotation about an axis of the eye, perpendicular to the direction of the force of gravitation.

Mechanisms: LOWENSTEIN and ROBERTS (1949) showed that the sense endings in the maculae of the thornback ray normally show a resting discharge, the frequency of which is increased or decreased by positional changes.

When the head is tilted 30° forward, the saccules lie vertically with the otoliths falling outwards, and the utricles lie horizontally with the otoliths uppermost. Present evidence suggests that the otolith organs are stimulated by movement in their own plane, i.e. the saccules are stimulated by vertical motion and the utricles by horizontal motion. When the head is in an altered position these relationships may be reversed.

WALSH (1960) emphasised the importance of the shearing force required for maximal stimulation. He also showed

that after unilateral labyrinthectomy there is a variation in sensitivity according to the orientation of the head, and that a person is relatively insensitive when lying on the side of the lesion.

Animal work has long pointed to the fact that the utricles play a more important role than the saccules. The change in the shearing force on the membrane will be greater for a given angular tilt for an otolith organ that is horizontal rather than vertical. This backs up the fact that normal posture can still exist following destruction of the saccules or their nerves but not of the utricles.

The Torsion Swing.

The torsion swing enables a rotatory test to be done in a few minutes (DE BOER, CARELS & PHILIPSZOON, 1963). Its use was originally described by MACH (1875) and later by VAN EGMOND, GROEN & JONGKEES (1949). With its use, the lateral canals are stimulated with angular accelerations. When it is swinging freely, damped sinusoidal movements are obtained.

It consists of a seat hung by two cables from the ceiling. The seat of the swing is coupled by means of a flexible shaft to a linear potentiometer on the floor and so the movements are recordable.

When there is clockwise acceleration, the nystagmus is to the right, and it is to the left as the direction changes. The number of beats to the left and right during the first ten swings is counted. The threshold of the slow phase of the nystagmus is much lower than that of the rapid phase which only occurs when the slow phase has reached a critical value (JONGKEES & PHILIPSZON, 1964).

The Neck Torsion Swing.

This is the same as the torsion swing but the head is fixed by means of a head rest and a mouth clamp.

Nystagmus can be provoked in this swing in bilaterally labyrinthectomised patients (streptomycin) (JONGKEES & PHILIPSZON, 1964). The relationship between vertigo and cervical nerves has already been pointed out on page 12.

Tests of the Vestibulo-Spinal Reflex.

A commission appointed by the Collegium Oto-Rhino-Laryngologicum in 1950 recommended investigation into the study of vestibulo-spinal reflexes. Their importance has also been stressed by WODAK (1957).

As seen in the first section of this thesis, constant impulses from the labyrinth on the anterior horn cells affect the tone of the striated muscles. This is mediated by three descending tracts - mainly the vestibulo-spinal tract, but also the medial longitudinal bundle and the reticular formation.

The first attempts to study the reflex were the goniometer test of VON STEIN (1904), the past pointing test of BARANY (1910) and the arm deviation test of BARANY (1911), GUTTICH (1914) and WODAK and FISCHER (1922).

To increase the accuracy of his past pointing test, BARANY (1926) obtained a recording of the stretched index finger on a sheet of paper. FISCHER and WODAK (1924) modified this to obtain a direct graphical recording by letting the patient hold a pencil in each hand, pressed against a downward moving sheet of paper. TALPIS (1927) mounted electric lamps on the fingers and made a photographic recording of their movements. This method was further developed by NYMAN (1945). BEHRMAN (1936)

examined 634 normal subjects using different tests and found that past pointing practically always occurred in the predicted directions.

In 1938 UNTERBERGER described a test in which he measured rotation on their own axis, in persons doing the stepping test. HIRSCH (1940) described much the same phenomenon and called it the "waltzing test". He found that normal subjects could do the stepping test without rotating on their own axis and without moving forwards.

In 1959, FUKUDA, described his stepping test. He placed his subjects in the centre of two concentric circles which were $\frac{1}{2}$ and 1 metre in diameter and divided into angles of 30° . The subjects were blindfolded and stepped in the one position for 50 - 100 steps. The parameters measured were the rotation on the subjects' own axis, the angle which the subject formed on his original sagittal plane and the distance moved forwards, backwards or to the side. Hirsch and Unterberger were concerned mainly with alterations in the stepping test after vestibular stimulation, while Fukuda emphasised that spontaneous changes were most important.

ZILSTORFF-PEDERSEN and PEITERSEN (1963-4) carried out a thorough study of the stepping test with the subjects stepping for 1 minute blindfolded. The normal variation with no stimuli was so great that it was impossible to set

up normal values. Weak rotatory stimuli resulted in changes in the stepping test that were not consistent, while stronger rotatory action gave consistent changes. With galvanic stimulation, they concluded that it did not result in a definitely unilateral stimulation of the vestibular system. Their irrigation tests showed that caloric stimulation had preponderantly unilateral actions on the vestibulo-spinal reflex, and that the stepping test is more sensitive than nystagmus observations at weak caloric stimuli.

While, theoretically speaking, this test ought to be one of the most valuable in the evaluation of disorders of equilibrium, it is of no use on its own and only of limited use in conjunction with other tests (PEITERSEN, 1965).

TOROK (1960) noted that at faster speeds of rotation, the trunk deviated visibly in one direction at the start of rotation and in the other direction after the chair was suddenly stopped. He called this "lateropulsion" and described a method for recording this.

An electromyographic method applied in rotatory examination was described by HARA, TOTSUKA and SUZUKI (1960).

With the lateropulsion test, which is performed in the sitting position, any lateral sway in response to a rotatory stimulus is automatically counteracted by a

compensatory movement of the body to prevent falling. Theoretically, therefore, the deviation should be small. Tests involving the presence of complicated body movements necessitate intercurrent cerebral activity and this is apt to change nystagmus patterns.

For this reason, HENRIKSSON, DOLOWITZ and FORSSMAN (1962) devised a test where the nystagmus and vestibulo-spinal reflex were provoked by the same caloric stimulus. Caloric tests are performed with the patient reclining backwards in a dentist's chair with the head resting on two inflated cuffs. The changes in pressure in these cuffs, made by movement of the patient's head, are transmitted to a pressure transducer and thence to a recording device. The lateral twist of the head, neck and body which follows labyrinthine stimuli occurs in the direction of the slow phase of the nystagmus. Henriksson called this laterotorsion as the movements have the characteristics of twist rather than pull. He quite accurately calls this a test of cristospinal reflexes.

While the test certainly measures induced neck torsion it is not a measure of the vestibulo-spinal reflex, which is more complicated.

In a later paper, DOLOWITZ, FORSSMAN and HENRIKSSON (1962), indicated that the cristo-ocular and cristospinal reflexes had different receptor cells and neurones despite

the common origin of the stimulus. Their basis for this was the fact that cases of Meniere's disease had decreased caloric nystagmus and vertigo but normal laterotorsion; cases of meningoencephalitis showed a normal response of nystagmus but a decrease in laterotorsion.

The Galvanic Test.

PURKINJE (1853) was the first to show that a galvanic current flowing through the head effects equilibrium.

There are two methods.

Methods: In the unipolar method, both electrodes are wrapped in gauze soaked in saline and the large flat one is held by the patient while the ball electrode is placed on the tragus and pushed into the meatus. The normal reactions are a cathode nystagmus to the same side and an anode nystagmus to the opposite side. The milli-amperage is read.

In the bipolar method, the two poles of a continuous current are applied, one in front of each ear. In the normal, there is an inclination of the trunk and head to

the positive pole when the current reaches about 5 milliamps, and a nystagmus towards the negative pole.

Mechanisms: After a bilateral labyrinthectomy galvanic nystagmus can still be produced. This shows that endolymphatic movement and cupular deviation are not the only mechanisms involved in the production of a galvanic nystagmus.

DOHLMAN (1929) found typical galvanic responses to stimulation of Scarpa's ganglion. If, in pigeons, both labyrinths and ganglia of Scarpa are removed, galvanic nystagmus can still be elicited by a strong current (HUIZINGA, 1931). Thus the ganglion cannot be the only site.

LOWENSTEIN and SAND (1940) and LEDOUX (1958) showed that a galvanic current affects the eighth nerve, increasing or decreasing its excitability. Since a galvanic current is D.C., augmenting it gives a stronger reaction.

The eighth nerve is not the only site where galvanic current acts, because a nerve-muscle preparation only reacts on opening or closing the circuit; augmenting a continuously flowing current has no influence on the state of the muscle (DOHLMAN, 1957).

Optokinetic Tests.

Although the test of optokinetic nystagmus is still used by neuro-otologists, it is not a true test of vestibular function.

Methods: The most usual way to do the test is with a Barany drum with three speeds and a variable axis of rotation. The subject views the drum from a distance of 50 cm. The nystagmus is noted in respect of its amplitude and direction, both horizontally and vertically. A normal person gets nystagmus whose direction is opposite to that of turning. Frequency increases with greater speed of revolutions and with closer spacing of the black stripes.

McLAY, MADIGAN and ORMEROD (1957) showed that with this method, precise control of the speed of the drum did not yield regular nystagmoid strokes. They evolved a hollow hexagonal drum which could be revolved round the subject at a velocity, acceleration and deceleration, all electronically controlled. There are six vertical white stripes inside the drum, of such a width that the image produced by a single stripe exactly covers the macula. The nystagmoid movements of the eye with this method are very regular.

Mechanisms: According to OHM (1936) the vestibular nuclei constitute a cell station common to the reflex pathways of

both vestibular and optokinetic nystagmus.

SMITH and BRIDGMAN (1943) showed that optokinetic nystagmus is essentially a brain stem reflex dependent upon direct nervous pathways from the optic tracts to the superior colliculi and eye muscle nuclei. If these are preserved, then optokinetic nystagmus may be induced following removal of the hemispheres, provided only that the visual stimulus involves a large part of the retina. If the stimulus is a small one, presented on a large stationary background, then in most animals the nystagmus can only be obtained if the cerebral cortex is present.

RADEMAKER and BRAAK (1948) described subcortical optic nystagmus which occurs when all parts of the visual environment are moving in the same direction, and cerebral optic nystagmus which occurs when successive moving objects, which arouse the instinctive interest, traverse the field of vision.

WINSTON et al (1948) showed that streptomycin affects the central vestibular neurones. DIX, HALLPIKE and HARRISON (1949) carried out tests on subjects with severe streptomycin intoxication and found that although the caloric responses were absent or reduced, the optokinetic responses were brisk and normal. This showed that the reflex pathways for optokinetic nystagmus were independent of the vestibular nuclei.

CARMICHAEL, DIX and HALLPIKE (1954) showed that areas in the supramarginal and angular gyri of each cerebral cortex were concerned with the directional control of optokinetic nystagmus. Each is thought to exert a controlling action on optokinetic nystagmus occurring in a particular direction by virtue of a modifying influence on lower reflex centres, located, probably, in the superior colliculus. A unilateral lesion of the supramarginal and angular gyri is characterised by a weakening or suppression of the nystagmus to the opposite side.

McLAY, MADIGAN and ORMEROD (1957) found that when an observer closed his eyes during an optokinetic test, there was a cessation of nystagmus for a short time, followed by a resumption of nystagmoid movements in the same direction as the primary nystagmus. This must be a form of 'memory' nystagmus (McLAY, 1965).

SECTION 3

REVIEW OF THE LITERATURE
ON NYSTAGMOGRAPHY

It will have been seen from the previous section that almost all assessments of vestibular function depend for their evaluation on the observation of nystagmus.

It had long been felt that many of the problems would be solved by some method of more accurate analysis of the various nystagmic parameters. In a review article by CAWTHORNE et al as recently as 1956, it was stated that "the practical advantages of the caloric test procedure carried out and interpreted in the manner described, do not preclude the need for further development, for which we must await improvements in the technique of nystagmus recording which should make possible a full and rapid analysis of the entire course of the eye movements that constitute a nystagmic reaction."

Although attempts at nystagmus recordings have gone on since the end of the last century, it is only in the last few years that nystagmography has been in routine clinical practice in a few centres.

Nystagmography, however, although it has been a great help in the analysis of vestibular function tests, has raised almost as many problems as it has solved. Many of these problems, but not all, are inherent in the methods used. This problem, although not referring to nystagmography, was well put by MAX PLANCK in 1936 - "it looks as though it must be impossible to distinguish between the

"event in itself", and the apparatus by which it is measured."

Nystagmography has been approached from several angles, and the methods used have been, broadly speaking, mechanical, optical and electrical.

Mechanical Systems of Nystagmography.

HOGYES (1881) recorded post rotatory nystagmus in rabbits by piercing the anaesthetised eye with a wire which had a Marey's capsule applied to the opposite end.

BERLIN (1891) transmitted the eye movements to a watch glass by means of a pin attached to an ivory shell fitted on to the cornea.

OHM (1914) made a device consisting of a lever attached to the upper eyelid and writing on a kymograph.

BUYS (1924) described a pneumographic method for recording nystagmus. The cornea of the moving eye acted on a membrane, and the pressure in a closed air-filled system increased and decreased with the movement of the eyes. The variations in pressure were recorded.

DOHLMAN (1925) used a combination of mechanical and optical systems. He placed over the cornea, a rubber teat to which was attached a small mirror. From the movements of this, he was able to produce photo-kymographic recordings of high quality.

GEMERT et al (1954) described a mechanical system whereby a contact glass was placed on the eye; a parallel plate air capacitor was connected to the glass. Movements of the eye changed this capacitance. The capacitor forms part of the capacitive voltage divider.

KUILMAN (1958), in an attempt to record rotatory nystagmus, used a combination of mechanical and optical systems. He placed a small cap of black silk on the anaesthetised cornea and fixed a tiny platinum pin on to the silk. The two platinum knobs reflected the light of a small electric bulb into the camera. A beam of light entering the objective is divided into two halves and by comparing the angles of the curves it is possible to analyse the rotatory movement of the eye.

Generally speaking, mechanical systems are no longer used, mainly because their application is painful and sometimes risky.

Optical Systems of recording nystagmus.

DODGE and CLINE (1901) photographed the light reflex from the eye and recorded nystagmus.

TSCHIRREN and VIESINGER (1947) were the first to describe and introduce into clinical practice, the photo-electric method.

TOROK, GUILLEMIN and BARNOTHY (1951) made further improvements to the photoelectric method. Photoelectric cells were utilised to record variations in the reflection of the infra-red light beam thrown on to the eye surface. The photocell-generated electrical signal is amplified and recorded by an oscillograph. The difference in the intensity of reflection from the sclera and iris was utilised. The advantages over electronystagmography according to the authors were that no electrodes were near the eyes, no electromyographic or electroencephalographic interference was picked up, the pick-up unit fixed on to lightweight goggles gave absolute freedom of movement, and the lesser amplification required did not require meticulous isolation and wire mesh insulation. They also claim that it is technically easier and consumes less time.

While TOROK et al used one photocell and eccentric illumination, PFALTZ and RICHTER (1956) used two photocells lighting the central part of the eye. Records were obtained in the vertical and horizontal plane by photoelectric registration, and information was provided about the direction, frequency, amplitude and duration of the nystagmus.

Electronystagmography.

It would be almost impossible to review the entire world literature dealing with electronystagmography. The development and major advances, however, will be reviewed.

Principles: Electronystagmography depends on the existence of the corneo-retinal potential difference first described by DU BOIS-REYMOND in 1849.

In the retina, electrical processes are always taking place causing the retina to be charged negatively as against the cornea. The eye can thus be regarded as a dipole, which during nystagmoid movements alternates around a vertical axis. When the eye moves from side to side, the field power in the region of the eye changes and with the nystagmogram, the eye movements are recorded via these changes in field power. (Figure 4).

Figure 4: Showing the way in which the potential difference between the cornea and retina is utilised to record eye movements.

The eye movements occur equally well behind closed eyelids and this is the condition under which recordings are usually made. The main reason for recording with the eyes closed is that fixation inhibits nystagmus, and by closing the eyes and abolishing this fixation, a great many more cases of 'latent' spontaneous nystagmus show up. Fixation can equally well be abolished by recording with the eyes opened in a dark room.

Methods: SCHOTT (1922) and MEYERS (1929), using string galvanometers intended for electrocardiography, were the first to make use of the above principle. Schott used copper electrodes in the conjunctival sac and he believed that the recorded currents were mechanical and due to eye movement. Meyers believed that the action potentials came from the eye muscles.

MOWRER, RUCH and MILLER (1936) were the first to suggest that recorded potentials were related to the corneo-retinal potential difference; they used a D.C. amplifier and a ballistic galvanometer and led off potentials with silver-silver chloride electrodes in isotonic saline.

FENN and HURSCH (1937) found the potentials to be proportional to the deviation of the eye.

HOFFMAN, WELLMAN and CARMICHAEL (1939) showed that bitemporally led potentials gave greater voltage as opposed to those in the conjunctival sac. They used a photographic

method and compared potentials arising on movement of the eye through an angle of, at most, 12° from the mid position, and found that the potentials were a linear function of the tangent of the angle of rotation of the eye.

JUNG (1939) pointed out the advantages of electrical registration of nystagmus with closed eyes thus avoiding fixation. He registered both vertical and horizontal nystagmus in each eye separately, and from his normal and clinical subjects, he analysed the direction, frequency, amplitude and duration of nystagmus.

LEKSELL (1939) found that potentials picked up from the temples were proportional to the sine of the angle of rotation of the eye; and as the sine of a small angle is roughly proportional to the angle itself, the potentials may be regarded as proportional to the angle. He replaced the string galvanometer used by previous workers by a recording oscillograph.

MILES (1939) measured potential differences with eye deviations of 30° to right and left and found variations of 0.3 - 2.5 millivolts for each eye. He also pointed out the rapid changes in potential (sometimes to several millivolts) that occur on induction of emotional states because of the psychogalvanic reflex.

GLORIG and MAURO (1950) recorded caloric nystagmus by means of an amplifier and a portable electrocardiograph.

HERTZ and RISKAER (1953) used an A.C. amplifier with three channels connected to three pens of an electroencephalograph. They revealed both vertical and horizontal nystagmus of both eyes simultaneously.

GROEN (1953) reported the use of electronystagmography in cupulometry.

DIETERLE and MONNIER (1955) placed two electrodes infraorbitally and one supraorbitally, and they suggested that the electrical potential of the periorbita is greatest in the infraorbital region nearest the eye bulbs. They also found that the voltage is directly proportional to the angle of displacement of the eyes, and that the electrical potential is equal in horizontal or vertical eye movement.

To ASCHAN, BERGSTEDT and STAHL (1956) must go the credit for producing the first monograph on electronystagmography from which they drew on experience gained from 1500 cases and experimental work on 500 normal subjects. They used a direct writing electrocardiograph (Elema's Mingograph) with a D.C. amplifier; there was a thin ink jet writer which created no friction on the paper. They used a D.C. pre-amplifier with a long time constant, as the longer the time constant, the greater the accuracy of the reproduced slow phase; when the nystagmus is of low frequency, the slow phase will not be represented linearly. With their method and a time constant between 1.5 and 3

seconds, the slow phase was recorded linearly. The error of the method of deducing the eye speed during the slow phase was estimated at $\pm 10\%$.

In the method described by McLAY, MADIGAN and ORMEROD (1958), an amplifier with a time constant of 10 seconds was used, and it was worked out that the error in amplitude for a nystagmus stroke lasting 2 seconds was under 10%.

JONGKEES and PHILIPSZOON (1964) produced another comprehensive monograph on the subject, dealing mainly with the clinical application and uses of electronystagmography. They used A.C. amplifiers and a direct writing electrocardiograph.

Factors measured in nystagmography.

Analysis of nystagmus records obtained by electronystagmography yield information on the direction of the nystagmus, the maximum eye speed, the frequency and the amplitude. Other qualities of the nystagmic response have also been studied.

OHM (1939) using mechanical nystagmography preferred to determine the nystagmus reaction by its "Energie", by which he meant the product of amplitude times frequency (A.F.). This was also the approach of MITTERMAIER (1954) and MITTERMAIER and CHRISTIAN (1954).

While Ohm stated that the AF implies the angular deviation of the eye during one second in one nystagmus stroke, the eye speed and AF are not identical properties. The AF is the angular deviation of the eye per time unit, but the eye speed is the angular deviation of the eye per time unit if it had not been interrupted by the fast phases (HENRIKSSON, 1956). The difference between eye speed and AF is small in reactions with a low eye speed (i.e. not so many fast phases), but when there are many fast phases the eye speed may approach twice the value of AF as shown in Figure 5.

Figure 5: See text. V_e = eye speed.

POWSNER and LION (1950) designed a special arrangement in which the original potential variations at the temples of the patient were transformed electrically so that they gave a direct measurement of the angular speed of the eye on a

cathode ray oscilloscope; the angular acceleration of the eye was recorded by a second oscilloscope.

HENRIKSSON (1955) designed a method of estimating the eye speed during the slow phase based on the method of Powsner and Lion for electronic derivation of the slow phase. He completely disregards the fast phase, because the difference in speed during the slow and fast phases is sometimes so great that they cannot be satisfactorily registered together (DOHLMAN, 1954), and because the fast phase speed is always the same and is of no interest regarding vestibular reaction. He assumes that the slow phase of nystagmus does not last less than 0.2 seconds and uses an 0.05 time constant.

The method is to convert ordinary nystagmograms, so that the rate of angular movement of the eye in one direction is recorded as a line above a base line, providing that the eyes move for a sufficiently long time in the direction studied. The main advantage of the method is that the nystagmus record gives an amplitude corresponding to the speed of the slow phase of nystagmus, and the record is compressed and easy to handle. The disadvantages are that all derived curves are in the same direction, and the direction of the nystagmus must be known in advance; if it changes direction, the polarity of the diode clipper has to be changed. This is offset by the fact that underneath

the derived curve, Henriksson now also records nystagmus in the conventional manner.

BUYS (1924) showed that the slow phase of vestibular nystagmus increased in proportion to the rate and duration of the acceleration, and could be regarded as a manifestation of the vestibular stimulation.

KOIKE (1959) and FERNANDEZ (1960), in cat experiments, concluded that the eye speed in the fast phase is not constant and is intimately correlated to the slow phase of vestibular nystagmus.

DOHLMAN (1925) showed that during the slow phase, the rate of increment of the eye is directly proportional to the vestibular stimulation. While irrigating the ear of each normal subject with water of different temperatures, he found that the speed of the slow phase was a linear function of the difference in temperature between the water used for irrigation and the body temperature of the patient. With thermo-elements, he measured the differences in temperature occurring in prepared temporal bones when irrigating with water of varying temperature. The shape of the curves he obtained, closely resembled the variations of the speed of the eye in the slow phase of nystagmus in humans when the ear was irrigated with water of corresponding temperature. On this basis he claimed a

correlation between vestibular stimulation and the rate of travel of the eye during the slow phase of the nystagmus.

JONGKEES (1949) and MITTERMAIER (1954) showed a poor correlation between the physical stimulus and the duration of nystagmus.

VAN EGMOND and TOLK (1954) concluded from their work that the sensitivity of the labyrinth was not determined by the duration of the nystagmus but by the speed of the slow phase.

HENRIKSSON (1956) compared the eye speeds and durations after caloric stimulation with different temperatures, and found that while the eye speeds varied linearly with the temperature, the durations remained about the same.

HENRIKSSON (1955) also showed that during constant acceleration, the speed of the eye in the slow phase of vestibular nystagmus varied linearly with the acceleration of rotation and with the duration of the stimulus; in the same series of experiments he showed that when using cupulometry, the speed of the eyes is not correlated to the duration of the nystagmus.

STAHL (1958) measures the mean speed of the slow phase over a ten second period.

MITTERMAIER (1955) measures the mean speed of the slow phase over the entire duration of the nystagmus. He showed that the maximum amplitude and total amplitude were very different, as the greatest amplitude is usually between 30 and 60 seconds of nystagmus duration.

According to STAHLE (1958) the best criterion for judging the excitability of induced nystagmus would be the estimation of the amplitude in the first 30 - 90 seconds.

JUNG and TONNIES (1948) measure the total amplitude (i.e. the sum of the amplitudes in the nystagmus strokes). This sum has to be computed from the conventional curves, which takes a considerable time.

TOROK, GUILLEMIN and BARNOTHY (1951) measure the top frequency of nystagmus, while MITTERMAIER and CHRISTIAN (1954) measure the average frequency.

SECTION 4

AUTHOR'S METHODS

In the series of 200 cases of dysequilibrium about to be presented, the following procedure was adopted in almost every case, the exceptions being cases of ocular nystagmus and some cases of chronic middle ear disease.

The history was taken on a preprinted questionnaire. Information was obtained on the date of the first attack of vertigo and of the latest attack, the durations and number of attacks, the longest free interval, the warning of onset, any precipitating factors, the time and place of onset, and whether there was nausea and vomiting. The direction of falling or rotation was noted as was the effect of change of head position, both during and in between attacks. The subjective state of the hearing was inquired into and details were noted regarding the presence of tinnitus, otorrhoea, otalgia, and whether there was a feeling of fullness in one or both ears. Questions were asked regarding the state of consciousness during attacks, the number of syncope if any, diplopia, headaches, and neck or root pain. Previous treatment was noted as were the drugs the patient was taking at the time. A full medical past history was taken and at the end, the patient was invited to indicate any points he felt had been missed.

In the examination, the first thing that was done was to examine the ears and upper respiratory tract. The Rinne, Weber and absolute bone conduction tests were done

with tuning forks at 256 and 512 c.p.s.

The presence of clinically visible spontaneous nystagmus was noted, taking care not to make the patient gaze too far to each side. This was done only in the upright position and any other head position in which the patient complained of vertigo.

Motor and sensory power was tested and the motor and sensory reflexes elicited. The cranial nerves were examined in the usual manner. Vibration sense was tested in all extremities and diadokinesia was examined.

Rombergism was done with the patient blindfolded, and he was asked to walk forwards and also backwards along a straight line while blindfolded. Past pointing was looked for by the finger to nose test. The patient was asked to stand upright, feet apart, with the arms held out and the index fingers touching two fixed points. He was then asked to close the eyes and the points were withdrawn a few inches. Deviation of the arms was noted.

The fundi were examined, the blood pressure noted and peripheral pulses felt in upper and lower extremities, and also with the shoulder depressed and the arm pronated.

Radiographs were taken of the mastoids and petrous bones with lateral oblique, sub-mento-vertical, Towne's and Stenver's views. Tomograms were done if indicated.

The cervical spine was X-rayed with antero-posterior, lateral and lateral oblique views.

If indicated, blood was taken for haematological study, serology, virus studies and agglutination tests. At a later date, again if indicated, lumbar puncture was done.

Audiometry was always done before vestibular function tests in order not to disturb the endolymph. Pure tone audiometry was done with an Amplivox 82 audiometer and Fowler recruitment tests were done if a non-conductive gap existed. Occasionally the audiogram was repeated, first with the head extended and then with the head flexed.

Bekesy audiometry was done with continuous tones and pulsed tones. For this, the patient sat in a sound damped booth, and the audiometer used was a Grason-Stadler type E-800. Recruitment was accepted as being present if the pen amplitude was four squares or less.

After all this had been done, the patient went for vestibular function tests with electronystagmography.

Apparatus.

The apparatus used has been a Devices High Speed Recorder Type R.1, which is a single channel direct writing instrument; while it is suitable for mains or battery

operation, it has consistently been used off the mains supply (Figure 6.).

Figure 6: The Devices electronystagmogram.

It is portable, weighing $22\frac{1}{2}$ pounds and is 6 x 14 x 12 inches in dimension. The amplifier has a sensitivity of 0.15 - 2 cms./mV. A.C., an input impedance of 1.5 mega-ohms and a time constant of 4.5 seconds. The time constant is regulated by means of two condensers placed in the small box situated to the right of the main unit in Figure 6.

The pen recorder has a flat frequency response, and records by means of an 8 cm. heated stylus on heat sensitive paper in roll form, 6.2 cm. wide and ruled in millimetre squares overprinted every 5 millimetres. The paper drive is powered by a D.C. motor servo controlled by a transistor amplifier to maintain constant speed against

variation of supply voltage or paper tension.

There are six speeds - 1, 2.5, 5, 10, 25 and 50 mm./sec. Calibration is normally done at 2.5 mm./sec., and recording at 5 or 10 mm./sec.

The controls on the pre-amplifier of the unit from right to left are:-

- (a) CAL button:- this is the very small button on the right, and by depressing it, calibration signals can be injected. The amplitude of the CAL signal is determined by the setting of the Range switch, e.g. on the 0.1 mV. range, the CAL signal is 0.1 mV. As the CAL signal is injected in series with the red input lead, it is possible to calibrate either through the signal source or with the input short circuited (Zero position).
- (b) Range:- this is a 9 position knob calibrated 0.1 to 100 mV. and is a sensitivity selector. It is usually set at 0.5 or 0.25 mV. for recording purposes.
- (c) Offset:- this is variable and is to adjust the position of the pen on the paper and to counteract mains interference.
- (d) Operate:- this has four positions.

S (standby), pre-amplifier only on; O (observe), pen on and stylus pre-heated; R (record), pen on and paper drive on; RF (record with filter), high frequency

filter brought in which gives a 6dB cut in signal amplitude at 35 c./s.

- (e) Gain:- this is a variable gain potentiometer, ratio 2:1, which adjusts the standardisation of the amplifier.
- (f) Input:- this switch has three positions. At Zero, it short circuits the signal input leads to the chassis earth. At 3K, the amplifier is at normal gain and the input impedance is 1.5 K.ohms each input to the chassis. At 30K, the amplifier is at one tenth of normal gain and the input impedance is 15 K.ohms each input to the chassis.

The average eye signal is 40 microvolts for 1° of eye movement, and as the apparatus is calibrated for 1 millimetre to equal 1° of eye movement, then 40 microvolts are equal to 1 millimetre of deviation on the paper.

Electrodes.

The patient's skin in the centre of the forehead and at the outer canthi is prepared by rubbing with anaesthetic ether until a good erythema is produced. Ordinary methylated ether was discarded in favour of anaesthetic ether as the former tended to leave a white residue on the skin.

The electrodes are slightly domed silver plates, 8 mm. in diameter and joined to silver plated wires by a silver

solder. While this silver solder is necessary for animal work, it has been replaced for routine purposes with ordinary solder as this is stronger; this has not affected the recordings at all.

The electrodes are filled with Cambridge electrode jelly so that it is flush with the surface; the electrode jelly acts, like many other substances, as a conductor but it has been found that using tomato ketchup in its place gives recordings of comparable quality.

The electrode leads are coloured black, red and orange. The black one is an earth and is attached to the centre of the forehead; in recording horizontal nystagmus, the red electrode is placed on the left side of the patient's eye, and the orange one on the right (Figure 7.).

Figure 7: Showing the placement of the electrodes.

The electrodes are fixed by means of sellotape; collodion was found not to be stable enough and its use is too time consuming for routine use.

For vertical nystagmus recording, one electrode is placed above the orbit (above the eyebrow) and the other below the orbit in line with the pupil, the patient being earthed as before.

The greatest care must be practised in applying the electrodes, as almost every technical difficulty we and other writers have encountered, has been due to poor preparation of the skin, faulty placement and fixing of electrodes and damage to the leads (usually done by ripping them off a patient too roughly).

If electrodes become displaced by the patient screwing up his eyes, or by sweat, or by tears loosening the sellotape, they must not be replaced in a different position as this upsets the calibration. If it is not possible to keep them in the same position then calibration must be repeated.

In horizontal nystagmus, an upward deviation of the pen represents eye movement to the right and a downward displacement means the eyes have moved to the left. The slow element of a nystagmus is shown as a gentle slope up or down and the quick movement is represented as a

vertical line (Figure 8.).

Figure 8: The upper record shows nystagmus to the right and the lower record, nystagmus to the left.

In vertical nystagmus, the direction of the beats is shown usually by arrows, 'up' meaning towards the forehead, and 'down' meaning towards the chin.

All records are read from left to right.

Calibration.

Once the electrodes are in place, it is necessary to calibrate in order that deviations on the records measured in millimetres can be translated to degrees of rotation of the eye.

The patient sits upright, six feet from a board on which there are lights which can be flashed on and off by a hand switch at the apparatus. There are three sets of lights on the board subtending angles of 20, 30 and 40 degrees at six feet (Figure 9.).

Figure 9: Showing the board used for calibration.

The first pair of lights which are 26.2 inches apart (20° at six feet) are the only pair used routinely.

With the paper running at 2.5 mm./sec. the amplitude of the eye movements from right to left are adjusted until the 20° movement results in a 20 mm. deviation on the paper. In this way 1° equals 1 mm. for future calculations. Quite often 1 mm. equals $10/11^{\circ}$, $10/9^{\circ}$ or some such fraction and adjustments in calculations have to be made.

The resistance over the electrodes is usually about

2.5 - 3 K.ohms, slightly less in women and slightly more in men. Provided that this resistance does not undergo any major change, the calibration will also remain unchanged.

Calculation of the speed of the slow phase.

It is important to know the speed of the slow phase as this is the expression of the vestibular element. Since the apparatus has been calibrated, then millimetres of deviation on the paper can be translated into degrees of rotation. The speed of the paper during recording is either 5 mm./sec. or 1 cm./sec. If it is 5 mm./sec., then one large square on the paper represents one second; if it is 1 cm./sec., then one second is represented by two large squares (Figure 10.).

Figure 10: Showing how to measure the speed of the slow phase. At 5 mm./sec. the speed is the distance marked 'a', and at 10 mm.(1 cm.)/sec. the speed is the distance marked 'b'.

The speed of the slow phase is the number of millimetres of deviation which occur in one second and can be worked out by applying a ruler along the slow phase line and marking off any one second period on the paper (even deviating the line backwards or forwards) and measuring the height in millimetres which the slope traverses in one second.

The Position Test.

With the patient's eyes closed, recordings were made in the following positions - upright (sitting), prone (head down sitting), supine, head to the right supine, head left supine and head hanging (Figures 11 - 16.).

Figure 11: The upright position.

Figure 12: The prone position.

Figure 13: The supine position.

Figure 14: The head right position.

Figure 15: The head left position.

Figure 16: The head hanging position.

Recordings were done for at least 40 seconds.

Occasionally, posture tests were carried out in the upright head right and upright head left positions. When indicated, the tests for benign positional nystagmus were carried out, i.e. upright with the head turned to one side 45° , then lowering the patient so that the head is lowered 30° over the end of the table still turned to the side, and then after one minute, resuming the upright position again.

From the records, the speeds of a few of the typical beats in each position in which nystagmus was shown were measured and the mean taken.

A positional nystagmus was regarded as pathological if it was present in more than two positions or if it had a speed of greater than 4° /sec. in any one position.

The Caloric Test.

The caloric tests were performed with a modification of the method of FITZGERALD and HALLPIKE (1942). The ears were irrigated with about 250 ccs. of water at 30°C and 44°C delivered from thermostatically controlled tanks for 30 seconds (Figure 17.).

Figure 17: Showing the thermostatically controlled tanks used for the caloric test.

The duration and maximum eye speed of the slow phase of the nystagmus were recorded. The end point was usually indicated by square waves (vide infra) and the maximum eye speed was taken as the mean of the eye speeds between 60 and 70 seconds. The irrigating time of 30 seconds rather than the usual 40 seconds was chosen as it is enough to produce about a 3 minute recording of nystagmus from normal ears.

To evaluate the result it was thought important not to use the traditional absolute values for the presence of a canal paresis or a directional preponderance. These were expressed as a relative figure, i.e. the percentage of the total excitability.

The numbers, 1, 2, 3 and 4 were allotted according to the traditional standards to the irrigations, i.e. 1 = left 30°C, 2 = right 30°C, 3 = left 44°C, and 4 = right 44°C.

A canal paresis was expressed in the percentage of the total excitability as follows:-

$$\frac{(1 + 3) - (2 + 4)}{(1 + 2 + 3 + 4)} \times 100\%$$

A right canal paresis gave a positive value and a left canal paresis, a negative value.

A directional preponderance was expressed as:-

$$\frac{(1 + 4) - (2 + 3)}{(1 + 2 + 3 + 4)} \times 100\%$$

A directional preponderance to the right gave a positive value and a directional preponderance to the left, a negative value.

The results, using the above techniques, of HAMERSMA (1957) and JONGKEES and PHILIPSZOON (1964) show that a canal paresis was pathological as regards duration when it exceeded the values $+14\%$ or -14% , and regards the maximum eye speed, $+15\%$ or -15% .

A directional preponderance was regarded as pathological if the duration exceeded the value $+30\%$ or -30% , or if the maximum eye speed exceeded $+18\%$ or -18% .

For this series these values have been applied as the technique used does not differ much from that of the above authors. There has also been difficulty in collecting sufficient numbers of normal subjects willing to have caloric tests performed to make a series statistically significant.

When a response was not obtained from one ear in the caloric test, the Dundas-Grant method was used. While blowing the cold air into the ear, the nystagmogram was run continuously until a nystagmus appeared or for three minutes, whichever came first.

The Coriolis reaction.

Occasionally, use was made of the Coriolis reaction in the following way.

The subject was seated in the upright position in a Barany chair and rotated. If, during clockwise rotation, the head was tilted forward, the subject felt as if he were rolling sideways. If the right vestibule was not functioning, the patient did not experience this sensation, but a response would occur if the direction of rotation was reversed.

SECTION 5

RESULTS

NORMAL SERIESExperiment 1.

Aim: To see if horizontal nystagmus in the standard six position tests was normal.

Method: Thirty five normal subjects, 18 males and 17 females, whose ages varied from 19 to 55 years, were tested with electronystagmography in the standard six positions for horizontal nystagmus. None of them had any previous ear disease or episodes of dysequilibrium. All drums were intact, normal and mobile.

Results: (Appendix 1). Twenty two out of the 35 (63%) had a positional nystagmus in at least one of the positions. The average eye speed was 2° /second, the slowest speed being 1° /second and the fastest, 5° /second. In only 2 instances, however, was the speed 5° /second. In no case was there a regular frequency. Of the 22 cases with spontaneous nystagmus, 8 had it in only one position, 11 in two positions and 3 in three positions; none had nystagmus in more than three positions. Ten cases showed nystagmus to the right and 11 to the left. Case 10 showed position changing nystagmus, but on repeating the test later, no nystagmus was present. It transpired that this had been a post alcoholic nystagmus as described by ASCHAN et al (1956).

Conclusions: Nystagmus is to be regarded as pathological in the position tests if the eye speed in any one position exceeded 4° /second, or if nystagmus was present in more than two positions. A pathological nystagmus must also have a regular frequency.

Experiment 2.

Aim: To see if vertical nystagmus in the standard six test positions was normal.

Method: Twenty five normal subjects with no previous ear or balance complaints and with intact, normal drums, were tested in the standard six positions for vertical nystagmus. One electrode was placed under the eye on the infraorbital rim in the midline, and the other above the eyebrow in the midline. There were 11 males and 14 females and the ages varied from 19 to 55 years.

Results: (Appendix 2). Seventeen cases had vertical nystagmus (68%): the direction of the slow component was downwards in 15 cases and upwards in 2 cases. The average eye speed was 6° /second. Twelve out of the 17 cases showed a regular frequency. Of the 17 cases showing vertical nystagmus, 1 showed it in one position, 3 in two positions, 5 in three positions, 3 in four positions, 2 in five positions and 3 in all six positions. The distribution of positions in which vertical nystagmus was

shown was almost exactly equal.

Conclusion: That vertical nystagmus in the standard six positions is a normal finding and with a single channel recorder it is not worth the extra time involved to test for vertical as well as horizontal nystagmus.

Experiment 3.

Aim: To test the linearity of calibration.

Method: The calibration board shown in Figure 9 has lights subtending angles of 20° , 30° and 40° . Six normal subjects, with the electrodes placed right and left, looked alternately at the lights, first at 20° , then at 30° and finally at 40° .

Results: (Appendix 3). The apparatus was calibrated initially for the 20° lights so that 1° represented about 1 mm. deviation on the paper. The mean of the six readings for 20° gaze was 18.9 mm. deviation; for the 30° gaze it was 29.1 mm. deviation and for the 40° gaze, 40 mm. deviation.

Conclusion: That calibration is linear. This means that the fast speeds of induced nystagmus can be worked out accurately from the initial 20° calibration.

Experiment 4.

Aim: To investigate the effects of different lighting conditions on calibration.

Method: Six normal subjects were asked to stretch their hands out in front of them and to look at each hand alternately. They were then ordered to shut their eyes and to carry on looking at where the hands still are (but this time memorising the position).

Results: (Appendix 4). The mean eye deviation with the eyes open was 1.9 mm. and with the eyes closed it was 2.1 mm.

Conclusion: That although calibration is carried out in a lighted room with the subject's eyes open and the recordings of nystagmus are taken with the eyes closed, the calibration is still applicable.

Experiment 5.

Aim: To examine whether upward deviation of the eyes on closing affects calibration.

Method: Six normal subjects performed the calibration manoeuvre sitting with the head extended 10° but with the axis of fixation horizontal. The movements were then repeated with the head flexed 20° .

Results: (Appendix 5). The mean of the six readings in the 10° extended position was 17.9 mm. deviation and in the 20° head flexed position, 19.3 mm.

Conclusion: That upward deviation of the eyes did not appreciably alter the signal of response.

Experiment 6.

Aim: To study the effect of memory on induced eye movements.

Method: Six normal subjects were asked to follow the movements of a pendulum for three minutes. At the end of this time they were asked to close their eyes and remain relaxed.

Results: Three of the six subjects showed a pendular movement of the eyes after closing them. One subject showed a brisk left beating nystagmus.

Conclusion: That eye movements may still occur after an induced nystagmus. This may be ascribed to a memory of the induced eye movements.

Experiment 7.

Aim: To investigate the effects on the maximum eye speed and duration of nystagmus of irrigation of normal ears with water of different temperatures.

Method: Fifteen normal subjects (with the same provisos of normality as before) were subjected to irrigation of one ear with water at 37° , 35° , 33° , 31° and 29°C . for 30 seconds. At least 10 minutes were allowed to elapse between each

irrigation. There were 5 females and 10 males and ages varied from 19 to 55 years. Each subject was tested for spontaneous nystagmus in the supine position prior to testing them; none of the subjects in this or ensuing caloric experiments had spontaneous nystagmus in the supine position. Eye speeds were measured every 10 seconds from the moment of onset until the end point and the maximum eye speed was taken. The temperatures of the water were taken at the outlet by means of a thermometer.

Results: (Appendix 6). It will be seen from the Appendix that there is a wider variation in the eye speed than in duration. The mean eye speeds were 5.1° /second (37°C), 12.2° /second (35°C), 22.1° /second (33°C), 27.4° /second (31°C) and 34.3° /second (29°C). The durations (mean) at the same temperatures were 106.9, 141.5, 155.3, 162.6 and 178.4 seconds.

Conclusion: That eye speed rises linearly as temperature falls, while the duration increases only slowly. A small difference in temperature makes a large difference to the eye speed, but not much difference to the duration.

Experiment 8:

Aim: To study the relationship between eye speed and frequency.

Method: The same group as in Experiment 7 were studied in

the same manner. The maximum eye speed was found to occur between 50 and 70 seconds after the commencement of irrigation. Due to the difficulties in accurate measurement of frequency, the total beats between 40 and 80 seconds were counted and a mean taken.

Results: (Appendix 7). The maximum eye speeds were the same as in the previous experiment at the different temperatures. The mean of the frequencies (in beats per second) was 0.86 (37°C), 1.4 (35°C), 1.9 (33°C), 2.1 (31°C) and 2.3 (29°C).

Conclusion: That frequency rises parallel to eye speed as temperature falls.

Experiment 9.

Aim: To study the effect of different temperatures on the onset of nystagmus.

Method: In 10 normal subjects, a continuous recording was taken before, during and after irrigation of one ear with water at 37° , 35° , 33° , 31° and 29°C . At least 10 minutes were allowed between irrigations.

Results: (Appendix 8). The mean of the onset of nystagmus times was 35.1 seconds (37°C), 30.9 seconds (35°C), 27.1 seconds (33°C), 24.0 seconds (31°C) and 21.1 seconds (29°C). The comparable eye speeds in degrees per second were 5.8, 12.3, 19.2, 21.9 and 27.9.

Conclusion: That nystagmus starts sooner as the temperature falls. In almost every case, nystagmus is present before the end of the irrigation.

Experiment 10:

Aim: To examine the effect of irrigation for different durations with the same temperature.

Method: Six normal subjects had one ear irrigated with water at 30°C for 10, 20 and 30 seconds; at least 10 minutes were allowed to elapse between each irrigation. The recordings were continuous, before, during and after irrigation. The following parameters were noted - time of onset of nystagmus, duration of nystagmus, maximum eye speed and frequency.

Results: (Appendix 9). The onset of nystagmus was about the same at 10 and 20 second irrigations (23.2 and 23.3 seconds), but much sooner at the 30 second irrigation (18.3 seconds).

The duration rose progressively as the time of irrigation increased.

The eye speed was $23.3^{\circ}/\text{second}$ at the 10 second irrigation, $26.2^{\circ}/\text{second}$ at the 20 second irrigation, but $34.2^{\circ}/\text{second}$ at the 30 second irrigation. The point at which the eye speed became maximum rose as the length of irrigation increased.

The frequency rose very little as the length of irrigation increased.

Conclusion: That to irrigate for less than 30 seconds gives a very much smaller response in all parameters, but the results would be sufficient for a differential caloric examination (a 3 minute duration and over a 20° /second eye speed). The subjective sensation of vertigo induced, however, did not vary much; the 30 second irrigation has been used in this series, as opposed to the shorter times, to make sure of getting results in the pathological series.

PATHOLOGICAL SERIES.

This consists of 200 patients who were either seen first in the Department of Otolaryngology in Edinburgh Royal Infirmary, or were referred from medical wards within the hospital.

For ease of presentation they have been divided up into 8 groups, the full results of which are shown in Appendices 10 to 17 inclusive.

Group 1. (Appendix 10).

This consisted of 44 patients suffering from Meniere's disease and 2 patients suffering from the Lermoyez syndrome.

The criteria of diagnosis were a typical history of the attacks and progress of the disease, the presence of tinnitus, a recruiting neurosensory deafness either on Bekesy audiometry or the Fowler test. Eighty per cent of the patients complained of a feeling of fullness in the ear during or just before an attack. The two cases of Lermoyez syndrome both had a marked improvement in hearing during the attacks.

Eight of the cases were bilateral. Nine cases showed no evidence of recruitment (8 Meniere's and 1 Lermoyez). Two out of the 9 were totally deaf in the affected ear, 2/9 were bilateral and found Bekesy audiometry difficult, and 5/9 had a variable slight low tone loss on the affected

side which was not sufficient to allow for recruitment.

Of the 46 in this group, there were 18 females and 28 males. Only 4 showed visible spontaneous nystagmus in the upright position, but with electronystagmography, 43 cases showed spontaneous nystagmus.

Seventeen were direction fixed towards the affected side, 10 were direction fixed towards the healthy ear, 7 were position changing and 1 was direction changing; all 8 cases of bilateral disease showed spontaneous nystagmus.

Spontaneous nystagmus in the different positions was as follows:-

<u>Number of positions in which nystagmus was shown</u>	<u>Number of cases.</u>
0	3
1	3
2	9
3	10
4	12
5	5
6	4

In the Hallpike position test, 8 cases showed a nystagmus with subjective symptoms and of these, 3 had a position changing nystagmus.

With the head right and head left in the upright position, spontaneous nystagmus was present in 4 cases when not present in the equivalent supine positions. In 6 cases where nystagmus existed in the head right and head left supine positions, none was present in the corresponding upright positions.

All 46 cases had caloric tests performed. Under the older method of estimating the results there were 24 canal pareses, 18 directional preponderances, 3 mixed lesions and 1 normal. Using the values described in Section 4 of estimating the results there were 23 canal pareses, 14 directional preponderances, 3 mixed lesions and 6 normals.

As regards the canal pareses, both the maximum eye speed and duration agreed in 15/23 cases and disagreed in 8/23; of these 8, the eye speed gave the pathological result in 7 and the duration in one.

Nine cases had a spontaneous nystagmus to the same side as the paresis, 7 had a spontaneous nystagmus to the opposite side, 2 had no spontaneous nystagmus and 5 had a position changing nystagmus.

The canal paresis was of the affected side on 18 occasions and was present in 5 of the 8 bilateral cases.

When directional preponderance is considered, the maximum eye speed and duration agreed only 4 times and disagreed 10 times, the eye speed always giving the pathological result.

All 14 cases of directional preponderance showed spontaneous nystagmus; 12 were to the side of the directional preponderance, 1 had a position changing nystagmus and 1 had a direction changing nystagmus.

Ten out of the 14 cases had a spontaneous nystagmus in the position in which the caloric test was performed.

Nine had a directional preponderance towards the affected side and 5 had it towards the healthy side.

Group 2. (Appendices 11A, 11B and 11C).

This consisted of 66 cases and cervico-vascular has been chosen as the group generic term. It has been split into three sub-groups.

Group 2A.

This consisted of 30 cases of true vertebro-basilar insufficiency. The main symptomatology consisted of transient bouts of vertigo especially on change of head position, occasional syncope, and other signs of vascular involvement such as a history of coronary artery disease, intermittent claudication, peripheral embolic episodes, cardiac arrhythmia, or hypertension.

There were 18 females and 12 males, and the average age was 59.9 years (range 37 - 73 years). Audiometric examination revealed no group pattern and the results were very variable.

Two cases had what was originally thought to be visible spontaneous nystagmus in the upright position, but this was refuted on both occasions by electronystagmography;

they were shown to be irregular senile eye movements and not true nystagmus. Twenty three out of the 30 cases showed spontaneous nystagmus with electronystagmography. Of these, 22 were direction fixed and 1 was direction changing. The following shows the number of cases having spontaneous nystagmus in each position:-

<u>Number of positions in which nystagmus was shown</u>	<u>Number of cases.</u>
0	7
1	9
2	5
3	6
4	1
5	1
6	1

When the Hallpike position test was done, 5 showed position changing nystagmus in at least one position. Although nystagmus was often recorded in the head to one side and down position, the worst subjective sensations were always on resuming the upright position.

Twenty nine out of the 30 had caloric tests performed. Under the older method of working out the results there were 7 canal pareses, 10 directional preponderances, 2 mixed lesions and 10 normals. Applying the values used in this series, there were 7 canal pareses, 5 directional preponderances, 2 mixed lesions and 15 normals.

Of the 7 canal pareses, the maximum eye speeds and durations agreed 5 times; the maximum eye speed gave the

pathological result on the 2 occasions of disagreement. Three had no spontaneous nystagmus, 3 had spontaneous nystagmus to the opposite side to the paresis, and 1 had a spontaneous nystagmus to the same side.

Of the 5 directional preponderances, only one had agreement between the maximum eye speed and duration; of the 4 that disagreed, the maximum eye speed gave the pathological result each time. Three had a spontaneous nystagmus to the same side as the directional preponderance, 1 had a spontaneous nystagmus to the opposite side and 1 had no spontaneous nystagmus. Only 2 cases showed spontaneous nystagmus to the side of the directional preponderance in the supine position.

Group 2B.

This is really intimately connected to Group 2A, as it consisted of 5 cases (3 female and 2 male) who had sudden unilateral deafness and vertigo, which could presumably be regarded as a labyrinthine vascular accident.

Three had total deafness on one side and 2 had a 60dB neurosensory loss.

Two showed visible spontaneous nystagmus in the upright position but all 5 showed spontaneous nystagmus with electronystagmography. Four cases showed it in all six positions, and 1 showed it in four positions. All 5 had

a direction fixed spontaneous nystagmus beating towards the healthy side.

All 5 had caloric tests performed. Two had a canal paresis, 2 had a directional preponderance and 1 had a mixed lesion.

Both canal pareses agreed as regards the maximum eye speed and duration, and both had a spontaneous nystagmus beating towards the opposite side from the paresis.

In one directional preponderance, the maximum eye speed and duration agreed, and in the other, the eye speed gave the pathological result. Both directional preponderances were directed towards the healthy side, and both had a spontaneous nystagmus in the supine position in the direction of the directional preponderance.

Group 2C.

This group consisted of 31 cases (13 females and 18 males) who complained of transient bouts of vertigo precipitated mainly by change of head position, but with no syncope and no evidence of other peripheral vascular disorders, hypertension or cardiac arrhythmias. They all, however, complained of neck pain or root pain. These were considered to be cases of cervical radiculitis (MARAN, 1963). The average age was 60 years (40 - 79 years).

Only 4 had a visible spontaneous nystagmus in the upright position, but all 31 cases had a spontaneous nystagmus on electronystagmography. Fourteen cases had a direction fixed spontaneous nystagmus but on Hallpike position testing, 5 of these showed some change of direction on change of position. The most revealing feature of this group was that 16 had a position changing spontaneous nystagmus; 1 case had a direction changing spontaneous nystagmus.

The following shows the relationship between the number of positions in which spontaneous nystagmus existed and the number of cases:-

<u>Number of positions in which nystagmus was shown</u>	<u>Number of cases.</u>
0	0
1	0
2	5
3	4
4	13
5	7
6	2

In the head right position, 13 showed spontaneous nystagmus to the right, 12 to the left, and 5 had no spontaneous nystagmus. In the head left position, 17 had spontaneous nystagmus to the right, 11 to the left and 2 had no spontaneous nystagmus. One case showed a direction changing spontaneous nystagmus in the head right and head left positions. Thirteen had a position changing

spontaneous nystagmus in the head right and head left positions.

If the head right and left in the upright position were also considered (and this was only done in 13/30 cases), another 6 had position changing spontaneous nystagmus.

Of the 16 cases of position changing nystagmus, 12 were examined in the right and left lateral positions (i.e. with no head rotation); none of these cases showed nystagmus in the lateral positions.

Twenty eight caloric tests were performed. Using the older values, there were 9 canal pareses, 8 directional preponderances, 4 mixed lesions, 6 normals and 1 was impossible to interpret due to blinking. Applying the values stated previously there were 11 canal pareses, 8 directional preponderances, 2 mixed lesions, 6 normals and 1 impossible to interpret.

Of the 11 canal pareses, 5 agreed with regard to maximum eye speed and duration, and in the 6 that disagreed, the eye speed always gave the pathological result. Five had spontaneous nystagmus to the opposite side from the paresis, 1 was to the same side and 5 had a position changing nystagmus.

Of the 8 directional preponderances, only 1 agreed with regard to maximum eye speed and duration and 7

disagreed; of these, in 5 the eye speed gave the pathological result, and in 2, the duration. Three had a spontaneous nystagmus to the same side as the directional preponderance, 1 to the opposite side, and 4 had position changing nystagmus. Four out of the 8 directional preponderances had spontaneous nystagmus in the supine position in the direction of the preponderance.

Group 3. (Appendix 12).

This consisted of 16 patients (10 females and 6 males) who had what was considered to be vestibular neuronitis. The criteria for diagnosis were a normal audiogram, a severe bout of vertigo with the patient being rotated or pushed in one direction, and with this attack settling and perhaps being followed over the next few weeks by more minor attacks. All cases resolved fully.

One case had a visible spontaneous nystagmus in the upright position, but 15 showed a spontaneous nystagmus with electronystagmography. Fourteen had a direction fixed spontaneous nystagmus and 1 had a position changing nystagmus. The number of cases having spontaneous nystagmus in the six positions was as follows:-

Number of positions in
which nystagmus was shownNumber of cases.

0	1
1	3
2	5
3	2
4	2
5	0
6	3

All 16 cases had caloric tests. With the older standards, there were 3 canal pareses, 12 directional preponderances and 1 mixed lesion. Applying the aforementioned values, there were 3 canal pareses, 7 directional preponderances, 1 mixed lesion and 5 normals.

All 3 canal pareses agreed with respect to duration and maximum eye speed; 2 had spontaneous nystagmus to the opposite side from the paresis, and 1 had no spontaneous nystagmus.

Five out of the 7 directional preponderances agreed in both maximum eye speed and duration, and the 2 that disagreed were both rendered pathological by the eye speed. All the directional preponderances had spontaneous nystagmus to the side of the directional preponderance, and 6 out of the 7 had spontaneous nystagmus in the supine position.

Group 4. (Appendix 13).

This consisted of 10 cases (7 males and 3 females) who

had post traumatic positional vertigo. These patients complained of vertigo in certain positions, or on head movement after a head injury. Deafness, if it existed, was usually caused by the head injury.

In the upright position, 3 showed visible spontaneous nystagmus, but with electronystagmography this was shown in 9/10 cases. In 5 cases, the spontaneous nystagmus was direction fixed, in 2 position changing and in 2 it was direction changing; in 1 case the spontaneous nystagmus was both position changing and direction changing.

Using the Hallpike position tests, 4 patients had a rotatory nystagmus in the critical position but this was not recordable. In 1 case, who was quite clear about the symptomatology, nystagmus could not be produced at all at the time of testing. Of those with a recordable positional nystagmus (5), none reversed on resuming the upright position, but one reversed in the head down and to the right position. Of the 4 showing clinical rotatory nystagmus, 2 had a direction fixed nystagmus, 1 had a position changing and 1 had no nystagmus in the six test positions.

Eight cases were tested with the caloric test according to Hallpike, and one had a Dundas Grant test. Of the former group, using the older standards, there were 2 canal pareses, 4 directional preponderances, 1 mixed lesion and

1 normal. Using the values for this series, there were 2 canal pareses, 3 directional preponderances, 1 mixed lesion and 2 normals.

Both canal pareses agreed as regards maximum eye speed and duration; 1 had a spontaneous nystagmus to the same side as the paresis, and 1 had a position changing nystagmus.

Two of the directional preponderances agreed as regards the maximum eye speed and duration, and in 1, the maximum eye speed gave the pathological result. All 3 had nystagmus to the same side and 2 had spontaneous nystagmus in the supine position. Two directional preponderances were to the side of the lesion and 1 was to the opposite side.

Group 5. (Appendix 14).

This consisted of 12 cases who had an anatomical abnormality of one middle ear and were complaining of vertigo.

Two had visible spontaneous nystagmus in the upright position, and 1 had what was originally thought to be spontaneous nystagmus but which was later proved not to be a true nystagmus by electronystagmography. Eleven out of the 12 cases showed spontaneous nystagmus with electro-nystagmography. Ten were direction fixed and 1 was position

changing. The number of cases having spontaneous nystagmus in each position is as follows:-

<u>Number of positions in which nystagmus was shown</u>	<u>Number of cases.</u>
0	1
1	1
2	4
3	0
4	3
5	2
6	1

The side to which the spontaneous nystagmus beat depended on whether the lesion was irritative or paralytic.

When caloric examination was done, it was usually by means of the Dundas Grant method or by cold stimulation to see whether or not function existed.

In one case a modified Hallpike caloric was done, and the result was a directional preponderance to the affected side, in which the maximum eye speed and duration agreed, and which had a spontaneous nystagmus to the side of the preponderance in two positions including the supine position.

Group 6. (Appendix 15).

This consisted of 17 cases who complained of disorders of equilibrium with the causal lesion either within the central nervous system, or at least, central to the vestibule. There were 3 cases of acoustic neuroma, 7

cases of disseminated sclerosis, and 1 case each of a parietal lobe glioblastoma, a posterior fossa meningioma, trigeminal neuralgia, Ramsay Hunt syndrome, Paget's disease, cerebellar ataxia and Bickerstaff's encephalitis. Also included in this group, for ease of presentation, are 2 cases of syphilitic ganglion neuritis and 2 cases of mumps labyrinthitis.

There were two females and one male with acoustic neuromas. One case had total unilateral deafness and two had a non-recruiting neurosensory loss.

Only one had a visible spontaneous nystagmus in the upright position, but all 3 showed spontaneous nystagmus on electronystagmography, 1 in one position and 2 in four positions. In 2 cases the spontaneous nystagmus was position changing and in 1 it was direction fixed.

There were 2 canal pareses and 1 directional preponderance on caloric testing.

Both canal pareses agreed as regards maximum eye speed and duration, and in one, the spontaneous nystagmus was position changing, and in the other, the spontaneous nystagmus was to the opposite side. Both cases had a neurosensory deafness and the canal pareses were of the affected side.

The maximum eye speed gave the pathological result in the directional preponderance. The patient was totally

deaf in the ear opposite to the side of the directional preponderance. The spontaneous nystagmus was position changing, and in the supine position, was opposite to the side of the directional preponderance.

There were 4 females and 3 males with disseminated sclerosis. Audiometry was variable - 2 had unilateral neurosensory deafness (1 of which recruited on one occasion), 3 cases had bilateral neurosensory deafness, 1 had unilateral conductive deafness, and 1 had normal hearing.

None of the cases had visible spontaneous nystagmus and 6/7 had spontaneous nystagmus with electronystagmography. Two were direction fixed, 2 were direction changing and 2 were position changing.

On the 3 occasions the patients were tested in the head right and head left upright positions, there was a difference in speed but not in direction from the corresponding supine positions.

In the 7 caloric tests, applying the older standards, there were 2 canal pareses, 4 directional preponderances and 1 normal caloric. Applying the values for this series there were 2 canal pareses, 2 directional preponderances and 3 normals.

Both canal pareses agreed with regard to the maximum eye speed and duration. One had spontaneous nystagmus to the same side and one had no spontaneous nystagmus.

The maximum eye speed gave the pathological result in both directional preponderances. One had a position changing nystagmus and one had a direction changing nystagmus. One had a spontaneous nystagmus in the supine position.

The Ramsay Hunt syndrome had a left sided neurosensory deafness and a left sided facial palsy; there was no visible or electronystagmographic evidence of spontaneous nystagmus. The caloric test showed a left canal paresis in which the eye speed and duration agreed.

The trigeminal neuralgia had no deafness, no visible spontaneous nystagmus, a position changing nystagmus in three positions with electronystagmography, and a normal caloric.

The case of Paget's disease had a bilateral neurosensory deafness, no visible spontaneous nystagmus, a spontaneous nystagmus in one position with electronystagmography, and a normal caloric.

The case of cerebellar ataxia was a post encephalitic episode, and while the patient showed a very well marked visible rotatory nystagmus, no nystagmus was recorded on electronystagmography.

The case of Bickerstaff's encephalitis presented with a visible spontaneous nystagmus, a left facial and abducens palsy and a total deafness on the left; vestibular tests

showed a left canal paresis and a direction changing spontaneous nystagmus in all six positions. The patient gained full recovery of vestibular function, cranial nerve function, and hearing in a few weeks.

The right parietal lobe glioblastoma was bilaterally severely deaf, and had no function in either ear with caloric testing. A pendular nystagmus was seen on electronystagmography, but the consciousness level was depressed although the patient was fully co-operative.

The right posterior fossa meningioma had no visible spontaneous nystagmus, but with electronystagmography had a direction fixed spontaneous nystagmus in 5 positions. There was a difference in speed between the head right and head left upright positions as compared to the corresponding supine positions. Spontaneous nystagmus to the opposite side of the lesion was present in the head right and down, and head right and upright positions. The hearing and caloric tests were normal.

One case of syphilitic ganglion neuritis had no visible or electronystagmographic spontaneous nystagmus, but had a canal paresis to the opposite side of the deafness as shown by the eye speed. The other case had a visible spontaneous nystagmus in the upright position, and a spontaneous nystagmus with electronystagmography away from the deaf side in 5 positions; there was a directional

preponderance to the same side as the spontaneous nystagmus which agreed as regards maximum eye speed and duration.

One case of mumps labyrinthitis had a visible spontaneous nystagmus, but both had a spontaneous nystagmus with electronystagmography, one to the right in two positions and one to the left in all six positions. Both cases had total unilateral deafness, and both had a canal paresis of the deaf side; one of the canal pareses agreed as regards maximum eye speed and duration, and in the other, the maximum eye speed gave the pathological result.

Group 7. (Appendix 16).

This consisted of 4 cases of hysterical vertigo and 20 cases in which a diagnosis could not be made.

The 4 cases of hysterical vertigo had normal audiograms and had no visible spontaneous nystagmus. Two had a direction fixed spontaneous nystagmus in two positions, but the highest eye speed recorded was 2° /second. One had a directional preponderance as regards maximum eye speed, with a spontaneous nystagmus to the side of the preponderance but not in the supine position. Two calorics were normal.

Of the vertigo causa ignota group, one was a haemophilic who, ten days after a car accident in which he

suffered no injury, began to have vertigo with a slight low tone loss in the right ear. The next day the vertigo was more severe and he had a 50dB neurosensory non-recruiting deafness in the left ear. He had a position changing and direction changing spontaneous nystagmus, and no caloric reaction from either side. He showed the curious phenomenon in nystagmography of having nystagmus visible with the eyes open but none recordable with the eyes closed. Even in the absence of other signs, it was presumed that he may have had a small intracranial haemorrhage, and so he was transfused with fraction for five days, with partial recovery of the symptoms, but the dysequilibrium persisted with the eyes closed as did the deafness in the left ear.

Another of this group had a rare condition of a block in his Vitamin A metabolism and had a 40dB bilateral neurosensory deafness which recruited. There was no visible spontaneous nystagmus but he showed direction fixed spontaneous nystagmus in four positions (but not in the supine position) with electronystagmography. From the maximum eye speed, he showed a directional preponderance to the side of the spontaneous nystagmus.

Of the remaining 18 cases there were 11 normal audiograms, 4 cases of bilateral and 3 cases of unilateral neurosensory deafness.

None of the cases showed a visible spontaneous nystagmus in the upright position but 13 showed spontaneous nystagmus with electronystagmography. Eleven were direction fixed and 2 position changing.

There were 7 canal pareses, 4 directional preponderances, 4 mixed lesions and 3 normal calorics.

Of the 7 canal pareses, 5 agreed with respect to maximum eye speed and duration, and 2 were taken for the maximum eye speed. Four had spontaneous nystagmus to the opposite side, 1 to the same side and 2 had no spontaneous nystagmus.

The maximum eye speed and duration agreed in only one directional preponderance and disagreed in 3, with the maximum eye speed being pathological twice and the duration once. Three had spontaneous nystagmus to the same side and 2 had spontaneous nystagmus in the supine position.

Group 8. (Appendix 17).

This consisted of 5 cases of ocular nystagmus. All were tested because of their curious eye movements to see if it was vestibular nystagmus or not. None of the patients complained of vertigo. The opinions of ophthalmologists were that two had the lesions of toxoplasmosis in the retina, one had tuberculous chorioretinitis, one was an albino and one had no lesion seen in

the fundus.

No audiometric or caloric examinations were performed.

The subject of ocular nystagmus will be dealt with in the discussion and will include the findings and impressions in this group.

SECTION 6

DISCUSSION

The Effect of Fixation on Nystagmus.

Prior to the introduction of electronystagmography, nystagmus was best observed with Abel's spectacles, Bartel's spectacles and Frenzel's glasses with inwardly directed illumination.

OHM (1939), ASCHAN (1955) and HENRIKSSON (1955) have questioned whether Frenzel's glasses do indeed eliminate fixation and its effect on vestibular nystagmus. It is the generally held opinion now that visual influences must be eliminated when observing and recording true vestibular nystagmus; the elimination of fixation by Frenzel's glasses is not sufficient. This is based on the following facts.

The duration of post rotatory nystagmus with Frenzel's glasses is about 85% longer than without the spectacles (ASCHAN, 1948). When electronystagmography is used with the eyes closed, the nystagmus is prolonged about 30% over the value obtained with Frenzel's glasses (ASCHAN et al, 1952).

The duration of caloric nystagmus with fixation is constantly shorter than when measured behind Frenzel's glasses, and is shorter still than when measured with electronystagmography and the eyes closed (DOHLMAN, 1925).

The eye speed during the slow phase of post rotatory nystagmus with fixation using Graybiel's oculo-gyral illusion test is about one tenth of the corresponding value

as measured by electronystagmography without fixation (HENRIKSSON, 1955).

From the present series it has been obvious that the longer the history of vestibular vertigo, the more likely is electronystagmography to reveal nystagmus rather than clinical observation. It also shows that it is desirable to record nystagmus unaffected by outside stimuli (i.e. with the eyes closed).

If the 5 cases with ocular nystagmus are disregarded, 22 cases showed clinically obvious spontaneous nystagmus, but 170 showed nystagmus in at least one position with the use of electronystagmography. This is roughly an eight-fold increase. If the standards set for the pathological value of positional spontaneous nystagmus are applied, i.e. more than 4° /second and/or present in more than two positions, 122/170 showed positional spontaneous nystagmus which was regarded as pathological. In the series of JONGKEES and PHILIPSZON (1964), of the 230 patients who showed positional spontaneous nystagmus with electro-nystagmography, only 52 showed clinical nystagmus visible with the naked eye.

The threshold of the human eye for the perception of nystagmus is much higher by direct inspection than by means of electronystagmography. It appears that, as a rule, a nystagmus, the speed of the slow phase of which is less

than 6° /second cannot be observed even by an experienced observer, whereas by recording a nystagmus, a slow phase of a speed of $1 - 2^{\circ}$ /second can still be clearly recognised.

Figures 18 and 19 show examples of nystagmus in the same patient with the eyes open and the eyes closed.

Figure 18: Case 44 (F.55) who had a left labyrinthectomy for Meniere's disease. Three months post operatively no nystagmus was seen clinically, but it is well demonstrated with the eyes closed.

Figure 19: Case 39 (M.53) who had a left labyrinthine vascular accident. Nystagmus was present with the eyes open, but increased in speed when the eyes were closed.

HENRIKSSON (1955) records nystagmus with the eyes open in a dark room. He is of the opinion that closing the

eyes results in a decrease in the level of wakefulness in some patients. More will be said about this later.

In one case in this series (Case 199) a well marked nystagmus was present with the eyes open but it disappeared when the eyes were closed (Figure 20).

Figure 20: Case 199 (M.23), a bilateral destruction of labyrinthine function in a haemophilic and a left sided deafness. Right beating nystagmus present only if the eyes are open. Eyes were closed at the first arrow and opened at the second.

According to HENRIKSSON (1965) this is not an infrequent occurrence. He ascribes it to a decrease in the level of wakefulness, but this was certainly not the case in the patient in this series; the nystagmus disappeared on eye closure, even if this only lasted a few seconds, and it could not be restored even with arousal stimuli such as mental arithmetic, clapping and conversation.

What Happens When the Eyes Close.

When the eyes close in a patient with a facial palsy, one sees an upward rotation of the eye on the paresed side.

Figure 21: Showing a typical result from Experiment 5. The first half of the record is the head 10° extended, and the second half, 20° flexed.

BJORK (1956) reported that the phenomenon is not present in normal subjects. If it does happen in normals, however, it may well influence the readings and perhaps explain the differences previously mentioned between records made with the eyes closed and open.

Upward rotation of the eye might be suggested to displace the projections of the electrical fields in the skin where the electrodes are placed, thus producing a smaller signal than expected from the pre-recording calibration.

Experiment 5 was set up to examine this problem. The normal subjects carried out the calibration manoeuvre with the head extended 10° and flexed 20° thus representing a total upward rotation of the eye of 30° . The results showed that upward rotation of the eyes of this magnitude did not appreciably alter the signal of response (Figure 21). This finding is in agreement with ASCHAN et al (1956). Either Bell's phenomenon does not occur when the eyes are closed or, if it does occur, then it does not alter the projections of the electrical fields.

Calibration.

A description of the routine calibration method was given in Section 4.

It is more accurate, and also easier for patients, if a board with flashing lights is used for calibration. An alternative is a board with strips of coloured paper separated by a distance subtending 20° at six feet. This, however, is difficult to see if visual acuity is poor (as in Case 99 who could not even see the board), and it is difficult to make the eye movements accurately. Such a small board is useful when testing patients who cannot get out of bed and the recordings have to be done in the ward.

The resistance over the electrodes is usually about 2.5 to 3 K.ohms, being slightly less in women and children, and slightly more in men. Provided this resistance does not undergo any major change, the calibration will also remain unchanged. If a patient is upset by a caloric stimulation it may be that the calibration will be slightly altered by autonomic phenomena. In this series, the calibration was repeated at intervals during the tests and also at the end, in many cases, and was found not to alter appreciably.

Calibration is performed in a lighted room with the subject's eyes open, and the recordings of nystagmus are taken with the eyes closed. The question arises of whether such calibration is applicable under different lighting conditions and whether the corneo-retinal potential alters behind closed eyes with a consequent effect on the calibration.

Figure 23: Showing a typical result from Experiment 3, showing the calibration at 20°, 30° and 40°.

MILES (1940) found a decrease of the human eye potential of about 10% after dark adaptation for five minutes. HENRIKSSON (1955) found that there was no decrease in potential after dark adaptation.

Experiment 4 was designed to see if calibration was altered at all by closing the eyes. The subjects were asked to look at their outstretched hands alternately, with the eyes open and then with the eyes closed, the hands remaining in the same position. It was found that the eye deviations are practically the same in both recordings (Figure 22).

Figure 22: Showing a typical result from Experiment 4. In the first part of the record the eyes are open, and at the mark, the eyes are closed.

This agrees with the findings of ASCHAN et al (1956).

Experiment 3 showed that the calibration with this apparatus was linear. Subjects were asked to look at two points 20° , 30° and 40° apart. This means that the fast speeds of induced nystagmus can be worked out accurately from the initial 20° calibration (Figure 23).

Horizontal Spontaneous Nystagmus in Normals.

There has been some disagreement in the literature as to the occurrence of horizontal spontaneous and positional nystagmus in normal subjects. STAHL (1958) found no spontaneous or positional nystagmus in 50 normal subjects. BERGSTEDT (1961) found it in 9 out of 26 (34%) normals. JONGKEES, MAAS and PHILIPSZOOM (1962) found it in one case out of 85. LANSBERG (1962) found it in 18 out of 230 (8%) normals. JONGKEES and PHILIPSZOOM (1964) found spontaneous nystagmus in 36 out of 149 (24%) healthy subjects. Eight per cent had nystagmus in more than two positions, and 5% had a nystagmus in more than three positions. Seven per cent had a frequency of more than one beat per second. HENRIKSSON (1965) always regards spontaneous nystagmus as pathological.

VISSER (1962) studying spontaneous nystagmus in 30 normals and 66 patients proposed a pathological limit for spontaneous nystagmus as a frequency greater than 0.5 beat per second.

In Experiment 1, horizontal spontaneous nystagmus was found in 22 out of 35 (63%) normals. Only 14% had nystagmus in more than two positions and no case showed nystagmus in more than three positions. In no case was there a regular frequency. The average eye speed was 2° /second and in only two instances was the speed 5° /second.

It seems obvious from the above that there must be a difference in interpretation of what constitutes a nystagmus. If, in this series, Visser's limits about frequency were applied, then no cases would have been said to have had spontaneous nystagmus. Although this small series shows a much higher percentage than that of Jongkees and Philipszoon (1964), the average eye speed was much lower and no case showed a nystagmus in more than three positions.

From these statements it can be concluded that if nystagmus has a regular frequency of more than 0.5 - 1 beat per second then it is to be regarded as pathological no matter what its speed or in how many positions it shows.

If it has no regular frequency, then nystagmus is to be regarded as pathological if it appears in more than two positions or if the speed is greater than 4° /second in any one position.

Vertical Nystagmus in Normals.

In a large percentage of normals (80%), FLUUR and ERIKSSON (1961) found vertical spontaneous nystagmus. JONGKEES and PHILIPSZOOM (1964) found vertical spontaneous nystagmus in 83 out of 104 normals (80%); in 74 it was an upward nystagmus, in 6 a downward nystagmus, and in 3 it was a direction changing nystagmus.

In Experiment 2, vertical spontaneous nystagmus was found in 17 out of 25 normals (68%); in 15 cases it was an upwards nystagmus and in 2 it was downwards. The average eye speed was 6° /second and 12 showed a regular frequency. Thirteen (76%) had a vertical spontaneous nystagmus in more than two positions (Figure 24).

Figure 24: Showing vertical spontaneous nystagmus (upward beating) in a normal subject.

The conclusion from this is that vertical spontaneous nystagmus is a paranormal finding and is thus of little value in the investigation of vestibular function. The vast majority of cases have an upward beating nystagmus. It is tempting to postulate that the normal position of the eyes during sleep is 30° of upward rotation, and that in the alert state, with the eyes closed, the subject tries to keep the visual axis horizontal and the eyes centred. Higher centres may regard this position as abnormal for the eyes shut state, and the eyes are snapped back up to the 'normal' 30° of upward rotation.

According to FLUUR (1962) the slow phase of horizontal and vertical nystagmus is controlled from separate independent centres. The rapid phase, however, is elicited from a centre common to horizontal and vertical nystagmus.

Positional Nystagmus.

There is often confusion in the literature as to nomenclature when positional nystagmus is discussed.

Nylen type 1 positional nystagmus is called direction changing and Nylen type 3 is "irregular positional nystagmus which may change in direction in any given position". The use of the word 'type' is also often confusing. Direction fixed spontaneous nystagmus is by far the commonest type and yet it is relegated to type 2, while the much rarer direction changing spontaneous nystagmus is type 1.

Benign positional nystagmus has been shown to have a vestibular, cerebellar and/or a cervical origin, and is generally classified under type 3, which already contains an important and totally different type of nystagmus. Benign positional nystagmus is a well defined, rather uncommon condition which has the characteristics of latency, fatiguability and reversal. Many patients, however, show nystagmus in the Hallpike position tests which has none of these characteristics, but which is, nonetheless, a definite

nystagmus entity.

For these reasons a new classification of positional nystagmus is suggested and will be adopted in this thesis. There should be four varieties of nystagmus, viz:-

1. Direction fixed spontaneous nystagmus - this corresponds to Nylen type 2 and is nystagmus which is seen or provoked during any of the position tests and which always beats in the same direction no matter which position is adopted.
2. Position changing spontaneous nystagmus - this would correspond to Nylen type 1 and is nystagmus which alters direction on change of position. It is seen most commonly in the cases of cervical vertigo (group 2C) to be discussed later. Figure 25 shows an example of this.

Figure 25: Showing position changing nystagmus in Case 125 (M.64) with cervical vertigo. Nystagmus to the right in the head left position, and to the left in the head right position.

3. Direction changing spontaneous nystagmus - this would correspond roughly with Nylen type 3 and is a most important sign of a central lesion. It is nystagmus which changes in direction during recording in one position (Figure 26).

Figure 26: Showing direction changing nystagmus in Case 165 (F.42) with Bickerstaff's encephalitis. Right beating nystagmus in the first half of the record becoming left beating in the second half.

The confusion between this and position changing nystagmus is the main reason for changing the conventional classification. This type of nystagmus was often referred to as Nylen type 3, but this category is a depository of all forms of nystagmus which do not fit into the first two types. If the term direction changing nystagmus is used, then there is no ambiguity.

4. Fatiguable spontaneous nystagmus - this represents the so-called benign positional nystagmus, and it allows for its still unknown genesis. Nystagmus to fall into this group should have the characters of latency and fatigability

in the head down and to the side position, and reversal on sitting up.

This new classification makes no claim to be utterly comprehensive but it is hoped that it is less ambiguous than the previous one.

In this series 126 cases showed direction fixed nystagmus. Of the 35 cases in group 1 (Meniere's and Lermoyez syndromes) who had unilateral lesions, 27 showed direction fixed nystagmus; of the other unilateral lesions, 5/5 in group 2B (labyrinthine vascular accident), 14/15 in group 3 (vestibular neuronitis), 5/9 in group 4 (post traumatic vertigo) and 10/11 in group 5 (conductively deaf) had a direction fixed nystagmus. Thus a total of 61 cases out of 75 (81%) with a definite unilateral peripheral lesion showed a direction fixed nystagmus. However, 22/23 cases in group 2A (vertebro-basilar insufficiency) showed a direction fixed nystagmus.

In group 1, 17/27 had a direction fixed nystagmus towards the affected ear, and 10/27 had a direction fixed nystagmus towards the healthy ear. In group 2B, all 5 had a direction fixed nystagmus towards the affected side. Three out of 9 cases in group 4 had a direction fixed nystagmus towards the affected side and 2/9 had a direction fixed nystagmus towards the healthy ear. It is impossible to tell which side is affected in a vestibular neuronitis

in the presence of a directional preponderance; the relationship of spontaneous nystagmus to directional preponderance will be discussed later. Only 3 cases had a canal paresis and of the 2 that showed spontaneous nystagmus, both were beating towards the healthy side. The side to which the direction fixed nystagmus was beating in group 5 depended on whether the lesion was irritative or paralytic.

From this, all that can be said is, that if a peripheral lesion exists, then the nystagmus is likely to be direction fixed. It does not mean, however, that the finding of a direction fixed nystagmus means the presence of a peripheral lesion. Direction fixed nystagmus on its own is of no help in deciding the side of the lesion; the side to which the direction fixed nystagmus beats depends on the activity of the diseased labyrinth.

In the series, 35 cases showed position changing spontaneous nystagmus. The majority of these (16 cases) were from group 2C (cases of cervical vertigo). No other condition showed more than 2 cases of position changing nystagmus and these could possibly be explained on the basis of intercurrent cervical root irritation that was not bad enough to give symptoms. The significance of position changing spontaneous nystagmus will be discussed later. As is to be expected, it is of no value in siding a lesion.

There were 9 cases of direction changing nystagmus; 3 of these were both direction changing and position changing. They consisted of two cases each of disseminated sclerosis and post traumatic vertigo, and one case each of cervical vertigo, vertebro-basilar insufficiency, Meniere's disease, Bickerstaff's encephalitis and a vertigo causa ignota in a haemophilic. This is too small a number from which to draw any conclusions, but HENRIKSSON (1965) regards this sign as nearly always signifying a non peripheral lesion. The impression from this small series is in agreement with this.

Case 165 (F.42) who was diagnosed as Bickerstaff's encephalitis had a direction changing nystagmus in all six positions. The pathology of this is a swelling of the myelin sheaths.

Although all 10 patients with post traumatic positional vertigo gave the classical history of extreme vertigo and nausea on putting the head into the critical position (usually lying down in bed at night), only 4 patients showed all the features of benign positional nystagmus in the Hallpike position tests. Since the nystagmus was rotatory it was not recordable. Of the 5 cases with a recordable positional nystagmus, none reversed on sitting up, but one reversed while in the head right and down position.

Many patients in the other group had a provocation

nystagmus in the Hallpike position tests, but none of these showed the characteristics of benign positional nystagmus. The character of the nystagmus provoked in these patients was of variable character.

Vertigo of Cervical Origin.

Since the beginning of this decade, a large number of cases of vertigo, especially in the older age group, have been designated as vertebro-basilar insufficiency. It is suggestive from this series that this group consists of two separate disease entities, namely, true vertebro-basilar insufficiency and a group in whom the pathology is primarily in the cervical nerve roots, cervical radiculitis.

The work of BIEMOND (1939, 1940), RYAN and COPE (1955), COHEN (1961) and BARLOW (1962) regarding the relationship of the posterior cervical nerve roots and vertigo was outlined in Section 1.

It is of interest in this series to compare groups 2A and 2C. The average age of patients in both groups was almost exactly the same (60 years). Group 2A consisted of 30 patients and 2C of 31 patients. There was no characteristic group differentiating feature with regard to audiometry or results of the caloric test.

All the cases in both groups had X-rays of their cervical spines, but some degree of osteo-arthritis or subluxation was

present in every case. SAMUEL (1965) has found that in the over 60 age group, osteo-arthritis in the cervical spine is present in 100%, and in the 50 to 60 age group, it is present in over 75%. None of the cases in this series had vertebral angiography performed as it was felt that this investigation, which is not without risk, would have no bearing on the future treatment of any case in the group. Atherosclerosis is so common in this age group that it would have been difficult to match angiographic findings to the symptoms. The possible vascular causes of vertebro-basilar insufficiency are laid out in a previous publication (MARAN, 1963).

The main symptomatology in group 2A consisted of transient bouts of vertigo especially on change of head position, occasional syncope, and other signs of vascular involvement such as a history of coronary artery disease, intermittent claudication, peripheral embolic episodes, cardiac arrhythmias or hypertension. Group 2C is clinically different, in that although the vertigo had the same quality, these patients had no attacks of syncope and no evidence of peripheral vascular disorders, hypertension or cardiac arrhythmias; the common feature in this group was, however, the presence of neck pain or cervical root pain.

Twenty three out of 30 patients in group 2A showed spontaneous nystagmus with electronystagmography, and all 31

Figure 27

patients in group 2C showed spontaneous nystagmus.

Figure 27 shows graphically the number of positions in which spontaneous nystagmus was recorded in each group. It will be seen that 90% of patients in group 2A showed nystagmus in three or less positions, while almost 70% of patients in group 2C showed nystagmus in more than three positions.

In group 2A, 22/23 patients showed direction fixed nystagmus, but in group 2C only 14/31 had a direction fixed nystagmus. The great differentiating feature was that 16/31 patients in group 2C showed a nystagmus in different directions in different head positions (position changing nystagmus). One case in each group showed a direction changing nystagmus in one position.

The vast majority of cases (13/16) in group 2C showing position changing nystagmus showed the difference in direction in the head right and head left positions. In group 2A only 7 cases showed spontaneous nystagmus in the head right position and 12 cases showed spontaneous nystagmus in the head left position. In group 2C, 25 patients showed spontaneous nystagmus in the head right position and 28 showed spontaneous nystagmus in the head left position; one case showed spontaneous nystagmus which changed direction in both the head right and head left position. There was no constancy as to the direction of

the nystagmus in the head lateral positions in group 2C.

In the head right position, 13 showed spontaneous nystagmus to the right, and 12 to the left. In the head left position, 17 had spontaneous nystagmus to the right, and 11 to the left.

That the nystagmus in the head lateral position was due to neck torsion and not to peripheral labyrinthine impulses, was shown by the fact that 12 of the 16 cases tested in the lateral position without turning the head (patient lying supine and on one side) showed no nystagmus.

In a previous publication (MARAN, 1963) it was postulated that this was due to a cervical radiculitis due to irritation of the upper cervical nerve roots on osteophytes. One case was tested on a swing to measure neck proprioception and this was found to be grossly abnormal; unfortunately, none of the cases in this series has been so tested. Ten have been treated with either neck traction or a plastic collar, and in eight cases, the position changing nystagmus has either disappeared or lessened greatly in speed, with disappearance of symptoms in all eight.

Nystagmus and the Eustachian Tube.

Can transient vertigo be caused by eustachian tube obstruction? It is well recognised that high speed jet pilots with disordered tubal function may become a little

disorientated during a quick descent.

If the eustachian tube becomes blocked and a negative pressure is caused in the middle ear, the drum will be drawn in and the round window membrane out. Cerebrospinal fluid would be drawn up the aqueduct of the cochlea when the round window membrane is drawn out. Since fluid is incompressible, no endolymph movement would take place and so no cupular deviation. It could happen, however, that a pulse wave travels through the inner ear fluids, similar to a left ventricular contraction and a peripheral pulse. According to Pascall's law, a pulse wave would pass through the endolymph and would probably be sufficient to cause cupular deflection and resultant dysequilibrium. The force of the pulse wave would be dependent on the diameter of the aqueduct of the cochlea (Poiseuille's law).

To a lesser extent this may be seen clinically, and in Case 67 (F.47) there is nothing else to explain the transient bouts of vertigo apart from blockage of the left eustachian tube. She had direction fixed positional nystagmus to the left in the supine and head right positions of 2° /second and 3° /second respectively. There was a directional preponderance to the left on caloric testing. The main complaint was of tinnitus, deafness and a feeling of fullness, all in the left ear, and transient bouts of dysequilibrium.

Case 8 (M.34) is interesting in that he was a case of mumps labyrinthitis resulting in a dead left ear and a left canal paresis. His mumps antibody titre was more than 520 units for both antigens. Four months after this had settled, he had two typical Meniere's attacks which were accompanied by tinnitus on the left side. Nystagmography at this time showed no spontaneous nystagmus, but this could be induced easily with a Valsalva manoeuvre (Figure 28).

Figure 28: Case 8 (M.34), see text. Patient started to do the Valsalva manoeuvre at the point marked, resulting in a right beating nystagmus.

If one accepts that the endolymphatic compartment is dilated in Meniere's disease, then it can be postulated that when Valsalva manoeuvre causes an increased venous pressure due to the reduction in cardiac output and increased mediastinal pressure, the reflected increase in pressure in the cerebrospinal fluid would cause it to escape up the aqueduct of the cochlea and cause a pressure wave in the dilated compartment and a spontaneous nystagmus.

Figures 29 and 30 show a similar finding in Case 188 (M.55) who had a left sided Meniere's disease and in Case 194 (M.61) who also had a left sided Meniere's disease. In this latter case there was no spontaneous or positional nystagmus in the standard six positions.

Figure 29: Case 188 (M.55) who had left Meniere's disease. Valsalva manoeuvre results in a right beating nystagmus.

Figure 30: Case 194 (M.61) who had left Meniere's disease. Valsalva manoeuvre results in a fast right beating nystagmus. Note the latent period.

When doing a Valsalva, there was a latent period of 7 to 14 seconds before nystagmus begins. This would support the aforementioned theory of venous back pressure causing the cerebrospinal fluid to go up the aqueduct of the cochlea.

The same explanation could be given for a curious finding in Case 88 (F.57) who had a Lermoyez syndrome in the right ear with a congenital deafness on the left side. She had a nystagmus of $2^{\circ} - 3^{\circ}$ /second in four positions to the left, and a left canal paresis (Figure 31).

Figure 31: Showing the audiogram of Case 88. There was a 20 - 25dB difference between the head extended and head down positions.

The audiogram showed a difference in thresholds in the right ear between the head down and head extended positions. This was reliable and repeatable. In the head down position, the intracranial venous pressure is raised due to constriction of the neck veins with resultant back pressure on the cerebrospinal fluid. This pressure would be passed back to the dilated scala media of the cochlea.

No previous report of this finding could be found in

the literature and it has not been found in any other patient in this series.

Ocular Nystagmus.

The healthy human eye is never completely at rest, but performs small movements estimated by MARX and TRENDELENBURG (1911) to amount to $4' - 5\frac{1}{2}'$ of a degree, the extent diminishing on fixation. In addition there is a coarser swinging movement. Both movements are present when the eyes are shut.

Ocular nystagmus consists of congenital nystagmus and gaze nystagmus.

Congenital nystagmus: This is defined as a nystagmus of a pendular type existing from birth or early infancy. In some cases there is an ocular cause (e.g. aniridia, opacities of the media, albinism) and some are idiopathic. There is also an acquired group which can occur after damage to the macula from a tuberculous or toxoplasmotic chorio-retinitis.

Congenital nystagmus is rare, and commoner in males than in females (HEMMES, 1924). There is frequently a hereditary basis for it. The eye movements are almost always horizontal - patients have no sensation of oscillopsia unless tired or emotionally upset, when there is an augmentation of the eye movements. The movements of the eyes are pendular in the centre position. On lateral gaze

they often change to a jerky type with the fast component in the direction of the gaze. When the neutral zone has an eccentric position in relation to the median plane of the head, the patient on fixation, usually turns his head to the side of the fast component of the nystagmus, and the eyes in the opposite direction to compensate; this brings them into the neutral zone in front of the body (FORSSMAN, 1963).

Congenital nystagmus in light, is nearly always suppressed, often completely, when the eyes are closed (ASCHAN & BERGSTEDT, 1955; ASCHAN et al, 1956; CATALANO & MADONIA, 1957; MOTTA, VON BERGER & BARAVELLI, 1957; BLOMBERG, 1959; and SUZUKI, 1961). ASCHAN and BERGSTEDT (1955), however, noted exceptions to this general rule in three cases which were not affected by eye closure.

Eye movements often decrease when a patient with congenital nystagmus fixes on a nearby object (NETTLESHIP, 1911; KASER, 1942).

Most authors consider ocular nystagmus to be released from the eyes (KESTENBAUM, 1948; ASCHAN & BERGSTEDT, 1955; MOTTA et al, 1957), because of the fact that nystagmus is more prominent in light than in darkness, and that patients nearly always have ophthalmological disorders.

Objections to this have been raised because often nystagmus does not disappear completely in darkness, and often there are no ophthalmological disorders found. Cases

Figure 32: Case 181 (M.14). The top record shows the pendular nystagmus and the lower record shows the slight increase in frequency on fixing.

Figure 33: Case 99 (F.36). The first part of the record shows the small fast movements on fixing. Fixing ceased at the point marked 'away'.

are also known of pendular eye movements which exist from the day of birth when the macula is undeveloped.

In this series only two cases showed true pendular nystagmus - one albino (Case 181) and one toxoplasmotic chorio-retinitis (Case 99). When Case 181 attempted to fix on a near object the frequency rose from 1.3 beats/second to 1.7 beats/second and the amplitude diminished a little (Figure 32); on the other hand, when Case 99 fixed on a near object, the frequency rose from 3 beats/second to over 4 beats/second and the amplitude diminished markedly (Figure 33).

When Case 181 closed his eyes, a nystagmus (as opposed to a pendular movement) to the right appeared which was sustained (Figure 34).

Figure 34: Case 181 (M.14). The top record shows the right beating nystagmus on closing the eyes. The lower record shows the pendular movements with the eyes open.

Case 91 (F.42) with tuberculous chorio-retinitis had a nystagmus to the left with the eyes open, and on closing the eyes, this disappeared (Figure 35).

Figure 35: Case 91 (F.42). Showing the difference between eyes open and eyes closed.
E.C. = Eyes closed.

A similar finding, only this time the nystagmus was to the right, existed in Case 111 (congenital nystagmus due to maternal toxoplasmosis), as seen in Figure 36.

Figure 36: Case 111 (F.49) who had toxoplasmotic signs in the retina, probably due to maternal toxoplasmosis. Showing nystagmus to the right with the eyes open and no nystagmus with the eyes closed.

Case 16, in whom no ocular abnormality existed, showed a right beating nystagmus which disappeared on closing the eyes.

Figure 37: Showing an example of deliberately contrived gaze nystagmus. In the upper tracing the eyes were deviated to the left (nystagmus to the left) and in the lower, to the right (nystagmus to the right).

These last three cases had a slow pendular movement on closing the eyes which may have been a 'memory' nystagmus.

Since none of these five cases complained of vertigo at any time, it cannot be postulated that those cases with a true nystagmus rather than a pendular movement, had a vestibular spontaneous nystagmus.

A pendular nystagmus can be explained as a necessary compensatory mechanism which has to be employed with a defective macula for maximum perception on the more sensitive parts of the retina around the damaged areas. The true nystagmus is a little more difficult to explain, however. While the rapid phase in these cases is a vertical line, the slow phase flattens out at the top of the stroke. This could be due to the fact that the time constant on the apparatus is not long enough, but the flattening off has very rarely been seen in cases showing true vestibular nystagmus.

Gaze nystagmus: That a jerky nystagmus can be observed in 50 - 60% of all normal subjects when the fixation axis is extremely deviated was shown by BARANY (1906) and NYLEN (1922).

Figure 37 shows an example of gaze nystagmus which was present on extreme deviation of the eyes to the left and right.

Gaze nystagmus which is present when the visual axis is deviated $20 - 30^{\circ}$ or less from the sagittal plane should probably be regarded as pathological (ASCHAN et al, 1956).

The clinical importance of gaze nystagmus is that if the eyes deviate from the central position during a vestibular nystagmus, the gaze nystagmus, if it exists, will add to or subtract from the vestibular nystagmus, thus modifying it.

Temperature, Duration and Eye Speed.

The water for the caloric test is delivered from the thermostatically controlled tanks at 30° and 44°C . With the apparatus used in this series, the temperature in the tanks was accurate $\pm 0.3^{\circ}\text{C}$. There was a loss of 0.5°C from the tank at 44°C by the time the water reached the ear, but the 30°C tank was accurate provided the water was allowed to circulate through the tubing for at least three minutes prior to usage.

The ideal method is to deliver 250 ccs. of water to the deep meatus at the exact temperature. To enable the water to reach the deep meatus, thin rubber tubes were attached to the nozzle of the main tubing. This is not possible, however, if there is a narrow meatus, as the deeply placed thin tubing blocks the outflow of water, and so less than 250 ccs. will be delivered.

The heating or cooling of the lateral canal will depend on the temperature of the water in the deep meatus, the pneumatisation of the mastoid, the patency of the eustachian tube, the thickness of the bony wall of the labyrinth, and the thickness of the drum. These factors will vary from person to person. From the work of CAWTHORNE and COBB (1954) it is known that irrigation of the external meatus with the above temperatures results in very little change of temperature within the lateral canal. These very small changes in temperature generally result, however, in large vestibular reactions. The decay of temperature in the meatus will depend on the above listed factors together with the amount of water left in the deep meatus at the end of douching, and the temperature and humidity of the room being used for the test, thus giving a different temperature decay pattern in almost every case.

JONGKEES (1949) found a certain increase of the duration when the difference in temperature between the water used for the irrigation and the temperature of the test person increased within a difference of less than 7°C . When larger differences were used, there was no corresponding lengthening of the duration.

Many of the investigations of earlier authors imply that the vestibular eye speed is a direct expression of the thermal stimulus applied to the labyrinth (see Section 2). No such

Figure 38.

close correlation between stimulus and duration has yet been proved, however, although the duration has generally been used as a measure of the excitatory effect provoked by the stimulus. To test the comparison between eye speed values and the duration of nystagmus in the same procedure, electronystagmography must be used to decide which of these effects of the stimulation is the most adequate and suitable for establishing the relationship between stimulation and effect.

It is important to know to what extent the qualities of eye speed and duration reflect stimuli of different physical values, i.e. to what extent an increase of stimulus conveys an increase of the eye speed on the one hand, and of duration, on the other. What is looked for is a quality producing not only evidence of a reaction of the labyrinth, but also a quantitative expression of the extent of the excitation of the vestibular apparatus.

To investigate this, Experiment 7 was designed. Fifteen normal ears were irrigated for 30 seconds with water at 37° , 35° , 33° , 31° and 29°C . Figure 38 is a graphic representation of the mean eye speed and mean durations provoked by each temperature. Both the eye speed and duration increase linearly as the temperature falls, but the eye speed curve is much steeper. When the temperature variation from body temperature increases from 2°C to 8°C ,

the increase in eye speed is from $12.2^{\circ}/\text{second}$ to $34.3^{\circ}/\text{second}$ (i.e. almost trebles), whereas the duration only increases from 141 to 178 seconds. An extrapolation of the eye speed curve almost hits the zero origo, which the duration curve does not. Thus the eye speeds at the different temperatures can be regarded as the true physiological answer to a physically determined stimulus. The eye speeds also reflect the course of excitation which the durations do not.

According to HENRIKSSON (1956), the duration of a caloric nystagmus agrees better with the times required for the restoration of normal ear temperatures, than with the excitatory effect on the sensory mechanism of the vestibular apparatus brought about by different stimuli.

Post rotatory nystagmus is due to the elastic forces within the cupula returning it. Post rotatory nystagmus seldom exceeds one minute no matter how strong a stimulus is used. In a caloric nystagmus, a duration of three minutes is quite commonplace, although the eye speeds indicate just moderate cupular deviation. The prolonged caloric nystagmus must thus be due to convection currents within the labyrinth delaying the regaining of normal position of the cupula.

Nystagmus decays exponentially. The above considerations make it possible that duration is a measure of the exponential decay of temperature, while the eye speed is a

measure of the reaction from the initial cupular deviation.

The accurate control of the temperature of the delivered water is essential. The methods employed for routine clinical caloric testing leave a lot to be desired from this point of view. What is needed is the thermostat at the nozzle, so that the water actually in the meatus is at 30°C and 44°C . If for example, the temperature at a cold irrigation was 31°C instead of 30°C , it would (according to Figure 38), influence the eye speed about four times more than it influences the duration.

As seen from Experiment 7, irrigation with water at 37°C produced a nystagmus with a mean eye speed of 5.1° per second. The temperature of the water at the nozzle end was measured each time and was exactly 37°C . The explanation must be that the body temperature of the subjects was above 37°C . Normal body temperature varies from 36° to 37.5°C ; the weather during the time of the experiments was rather warm and so the body temperature of the subjects was presumably at the upper level of normal. In the experiment, the finding is of little importance, but it becomes important when routine caloric testing is considered. Body temperatures will vary, and also slight differences in blood supply to the ear will bring about a variation in the thermic wave, affecting the eye speed more than the duration.

Figure 39.

In Experiment 9 it was shown that as the irrigating temperature falls, so the onset of nystagmus becomes sooner. The effect is shown graphically in Figure 39. It is seen that the eye speed and onset are linear functions of the temperature. According to McLAY, MADIGAN and ORMEROD (1957), a normal nystagmoid response to caloric stimulation begins within 5 or 10 seconds from the beginning of douching. In the experiment in this series, the mean onset at 29°C was 21.1 seconds with the quickest onset at 5 seconds and the slowest at 33 seconds. There was a similar wide variation at all irrigating temperatures. In almost every case (with a 30 second stimulus), nystagmus was present before the end of douching. In each case the eye speed was measured at onset, and thereafter at ten second intervals. The onset was quite sudden, being preceded by a few irregularly shaped waves, and rapidly built up to a maximum at between 50 and 60 seconds. This was verified in Experiment 10, where it was also seen that as the duration of the thermic stimulus was increased, the time at which eye speed was at its maximum approached 60 seconds. The eye speed continues at or around its maximum for a further 20 seconds and then begins to decay smoothly. At about 110 seconds, there was usually a sharper increase in the decay rate, and there was another sharp increase of decay rate at about 140 seconds. The normal duration was about 3 minutes with a 30 second

stimulus. There is no value in using the onset of nystagmus as a parameter for vestibular excitability as against eye speed, as there are too many variables involved.

For the first few caloric tests done with electro-nystagmography, 40 second irrigation times were used. This resulted in durations of 240 seconds or more and was time consuming and expensive in paper. For this reason, it was decided to modify the test to a 30 second irrigation. The effects of duration of irrigation at 30°C on various parameters was investigated in Experiment 10. Onset, duration, frequency of nystagmus, maximum eye speed, and time of maximum eye speed were recorded. The differences between a 10 and 20 second irrigation were not marked, but there was a great difference in all parameters at a 30 second irrigation. It was felt that a 30 second irrigation gave a response sufficient for differential caloric examination (a 3 minute duration and a 30° maximum eye speed).

CAWTHORNE and COBB (1954) showed that irrigating the ear with water at 30°C for 40 seconds, caused a peak change of temperature within the lateral canal of 0.67°C . It is remarkable to consider the sensitivity of the cupula and the efficiency of temperature exchange within the ear, when a 10 second irrigation with water at 30°C produces a mean eye speed of $23.3^{\circ}/\text{second}$ and a mean duration of 159.2 seconds; the peak temperature change within the lateral

Figure 40: Showing the effect of cold stimulation on eye speed and duration in Case 95.

canal with this must be very much less than 0.67°C , and yet a reaction of such intensity is produced. This may be taken as another factor pointing to the importance of extreme accuracy required in temperature control of routine caloric examinations.

Case 95, who had a normal left ear and a right radical mastoid cavity was tested with Dundas Grant stimulation for one minute (Figure 40). On the right side, the eye speed reached high values, but the reaction diminished and disappeared quite soon. The eye speed on the left was lower than on the right but quite within normal limits. The duration on the left was longer than that on the operated side. This was because although the same physical stimulus was used, the temperature on the operated side reached much lower levels more quickly because of the lack of obstruction by intermediate tissues, thus producing the high eye speed. This temperature change on the operated side recovered to normal more quickly for the same reason, and so the duration was shorter. Thus, if duration only is used as a parameter, then strong vestibular reactions may be overlooked. This can also be claimed as more evidence that duration is a measure of recovery of induced temperature change to normal. These results are in agreement with those of HENRIKSSON (1956).

Figure 41: Showing square waves at the end of a 44°C irrigation in the left ear (left beating nystagmus).

Figure 42: Case 200 (F.42) with vestibular neuronitis. Square waves seen in the supine position.

End Point of Caloric Nystagmus.

Theoretically, once nystagmus is induced it never reaches an end point as it diminishes by exponential decay. Fortunately, near the end point, square waves appear, and this is taken as the end point in all readings. Each square wave (Figure 41) consists of a quick movement in either direction separated by a pause; there is no slow component as there is in a nystagmus beat. According to McLAY, MADIGAN and ORMEROD (1957) this could be the co-existence of two nystagmoid movements in different directions.

Square waves do not occur, unfortunately, in every case. They are almost invariable in patients over the age of 40, but are rare in cases under the age of 30. If they do not appear, then deciding when the last nystagmoid beat occurs becomes very inaccurate and difficult. In young patients, occasionally, the rapid component of a nystagmus beat tends to become 'slower' and is no longer a true vertical line. In this case it is usual to take the last true nystagmus beat by examining the rapid phase to see if it is still vertical.

Square waves are occasionally seen in pathological subjects during the position tests (Figure 42). McLAY, MADIGAN and ORMEROD (1958) stated that they occurred in vestibular neuronitis. In this series, square waves have been seen in position tests in many different pathological

conditions. If they occurred in the upright position (which is the first test position after calibration), it could be surmised that they were memory movements from the calibration. The upright position, however, is the only one of the six positions in which square waves have not been seen. These waves have never been seen without nystagmus being present in at least two other positions. No identifiable pattern has come to light, however, but it seems probable that these waves are closely related to nystagmus, and the previously expressed view of McLay, Madigan and Ormerod (1957) seems tenable.

Directional Preponderance.

Whether a directional preponderance is of peripheral or central origin has been debated since 1891 when KORANYI and LOEB found differences in post rotational nystagmus in rabbits that had a unilateral hemispherectomy. BAUER and LEIDLER (1912) found that the directional preponderance occurred towards the operated side and they suggested that it was due to the central lesion; this view received support from DE BARENNE and DE KLEYN (1923), KOCH (1933), MITTERMAIER (1935) and BARRE (1938).

With their conception of canal paresis and utricular paresis, CAWTHORNE, FITZGERALD and HALLPIKE (1942) regarded a directional preponderance as an effect of a peripheral

lesion also. JONGKEES (1953), ANDERSEN (1954) and STAHLE (1958) agreed that peripheral lesions were also responsible for a directional preponderance.

Directional preponderance is now regarded as an expression of a faint or latent spontaneous nystagmus (JONGKEES, 1953; HALLPIKE, 1955; HAMERSMA, 1957; STAHLE, 1958; KOCH et al, 1959).

Of the 175 caloric tests done in the pathological series, there were 50 (29%) which showed a directional preponderance. Seventeen agreed as regards maximum eye speed and duration, and 33 disagreed. Of this group of 33 cases, 30 were judged to have a directional preponderance according to the eye speed, and only 3 with regard to duration. It seems from this that the maximum eye speed is a much better indication of a directional preponderance than the duration. According to HENRIKSSON'S (1956) normal series, however, one should take the duration to give the pathological result more often than the eye speed. A point to note, however, is that the values given in his normal series are higher than those of any other authors, and it may have been that the 'normals' were particularly sensitive.

Of the 50 cases of directional preponderance, 38 had a spontaneous nystagmus to the same side as the preponderance. Thirty one (62%) had spontaneous nystagmus in the supine position, i.e. the position in which the test was carried out.

Two cases had no spontaneous nystagmus and a similar number had nystagmus to the opposite side. The remaining eight cases had either a position changing spontaneous nystagmus or a direction changing nystagmus. Since 170/200 cases showed spontaneous nystagmus, this leaves 132 cases which showed a spontaneous nystagmus with no directional preponderance.

Thus, with electronystagmography, most of the cases of directional preponderance are related to nystagmus, but the majority of cases with nystagmus are not related to a directional preponderance. If the spontaneous nystagmus was intense, it usually showed up as a directional preponderance, but the more frequent cases with faint spontaneous nystagmus were not usually expressed by a directional preponderance. A possible explanation could be that in cases with faint spontaneous nystagmus, the spontaneous nystagmus was not enough to inhibit induced nystagmus in the other direction. A direction fixed nystagmus which was intense and did not show up as a directional preponderance, was nearly always due to a non-functioning canal on the opposite side.

While there is a close relationship between directional preponderance and spontaneous nystagmus, it does not mean that if a direction fixed spontaneous nystagmus is present one can expect a directional preponderance to that side. Only if function is present on both sides can a directional

preponderance be expected, and if this exists, then spontaneous nystagmus with the eyes closed, can be regarded as the earliest sign of imbalance of the vestibular system, whether of a central or a peripheral origin. HENRIKSSON (1959) describes a gradation of imbalance - first is a spontaneous nystagmus in the dark, then also a directional preponderance in the dark, next is a directional preponderance in the light, and lastly a spontaneous nystagmus in the light.

In central disorders, the directional preponderance is said to be directed towards the affected side (HALLPIKE, 1955; ANDERSEN, 1954) and in peripheral disorders most often towards the healthy ear. The direction of the preponderance in itself, did not, in this series, give any information on the site of the lesions, unless it was known whether it was central or peripheral.

In all 15 cases of directional preponderance in unilateral Meniere's disease and C.S.O.M., the directional preponderance was directed towards the healthy side. In the central lesions it was difficult to say on which side the lesion was in those cases with a directional preponderance, but one case of acoustic neuroma had a directional preponderance to the healthy side and one case of syphilitic ganglion neuritis had a directional preponderance to the affected side. As regards the vestibular neuronitis group, it is still not certain where the lesion is, but all 7 cases of directional

preponderance showed it towards the presumably healthy side. The diagnostic or topographical value of directional preponderance per se is very limited.

In many cases it was from the hot water reaction that a directional preponderance was revealed as regards eye speed. Consider the example of a left sided Meniere's disease with a spontaneous nystagmus to the right. The cold water irrigation will often disclose little or no difference in the maximum eye speed between the left and right sides, because the reactions from the right side (nystagmus to the left) will be counteracted by the spontaneous nystagmus to the right, thus producing a modest eye speed reaction. Stimulation of the left ear, however, will provoke a reduced reaction which is increased by the spontaneous nystagmus to the right. In the hot water irrigation, the reduced effect from the left ear is counteracted by the spontaneous nystagmus to the right, and this spontaneous nystagmus also increases the normal reaction from the right. Thus the hot water reaction exaggerates the real difference in sensitivity between the ears.

Canal Paresis.

Out of the 175 caloric tests performed there were 65 cases of canal paresis. As regards the maximum eye speed and duration, these agreed in 45 cases and disagreed in 20. In

this group of 20, only once did duration give the pathological result. Thus, again it is seen that the maximum eye speed gave a better indication of the intensity of a reaction than the duration. This fact confirms the opinions of RUDING (1953), VAN EGMOND and TOLK (1954), HENRIKSSON (1955, 1956), ASCHAN et al (1956), KOCH et al (1959) and JONGKEES and PHILIPSZOON (1964).

Twenty six had a spontaneous nystagmus to the opposite side, 15 (including 9 Meniere's, 8 of whom were bilateral) had a spontaneous nystagmus to the same side of the paresis, 12 had no nystagmus and 2 had a bilateral canal paresis. The remaining 10 cases had either a position changing or direction changing spontaneous nystagmus.

If a canal paresis is an indication of hypofunction in a unilateral peripheral lesion, then it is difficult to see how anything other than a spontaneous nystagmus to the opposite side could co-exist. In this series there were 15 which had a spontaneous nystagmus to the side of the paresis. At least 8 of these were bilateral cases, but there was one case each of unilateral Meniere's disease, disseminated sclerosis, mumps, post traumatic vertigo, vertebro-basilar insufficiency, cervical vertigo and vertigo causa ignota.

If a canal paresis exists with a spontaneous nystagmus to the same side, the sequence of events is as follows. Take the example of a left canal paresis and a left spontaneous

nystagmus. The cold irrigation will produce a reduced response from the left ear which is further counteracted by the spontaneous nystagmus. The right side will give a normal response, further enhanced by the spontaneous nystagmus to the left. The hot irrigation will cause a reduced response from the left side enhanced by the spontaneous nystagmus to the left. The reaction will be greater than that of the cold irrigation but not as great as that on the right side. The reaction on the right with hot water will be normal but counteracted by the spontaneous nystagmus. The result is an uncrossed mixed lesion. It is not enough, therefore, to see a direction fixed spontaneous nystagmus and then to do a quantitative caloric test with a Dundas Grant method to estimate the caloric result.

A canal paresis is a more severe lesion than a directional preponderance. In Case 165 (Bickerstaff's encephalitis), the first result from the caloric test was a left canal paresis with a spontaneous nystagmus which was direction changing in the position test. As the swelling of the myelin sheaths recovered, the hearing in the left ear, which showed initially a severe deafness, recovered to a 50dB non-recruiting loss. A caloric test repeated at this time showed recovery of function on the left side, a spontaneous nystagmus to the right in the supine position, and a directional preponderance to the right.

Different Wave Forms.

One obvious advantage of electronystagmography is that it provides a record of eye movements which can serve as an objective basis for discussion. Though the same record may be interpreted in different ways, discussion will be more profitable if the record is down on paper and not merely a written subjective description.

Electronystagmography also ascertains whether eye movements are true nystagmus or not. Many cases have been referred for electronystagmography with a clinical note that nystagmus is present, but on testing, it has proved not to be a true nystagmus with a quick and a slow phase.

While electronystagmography clears up the above problem well, it also creates problems of different wave forms which must be distinguished. The following are occasionally seen on position testing.

1. Blinks: This is usually obvious and shows as a large spike (Figure 43).

Figure 43: Showing a right beating nystagmus with three blink spikes.

Figure 45: Showing atactical eye movements in Case 17 (P.59) with vertebro-basilar insufficiency. Nystagmus was thought to be present clinically.

Figure 46: Showing pendular movements in Case 131 (M.67) who had a parietal lobe glioblastoma.

On one occasion it was so troublesome that it made interpretation of the caloric test quite impossible. Usually, if troublesome, it can be lessened by sticking the eyelids together with sellotape.

2. Eyelid tremor: This is usually seen in elderly patients and may be confused with nystagmus, but there is no slow or quick phase (Figure 44).

Figure 44: Showing an example of eyelid tremor (and two blink spikes) in Case 55 (F.55) with cervical vertigo.

In Figure 44 the tremor is easily recognisable as rounded humps, but when these become small spikes the differentiation becomes more difficult.

3. Atactical movements: These are most often seen in atherosclerosis and cases of vertebro-basilar insufficiency. They may be mistaken for nystagmus on clinical examination and are shown in Figure 45.
4. Pendular movements: This curious movement is a slow swing in both directions resulting in a wavy appearance (Figure 46).

In this series, this movement has been seen in case of brain tumour, patients who are lightly dozing and as a memory nystagmus.

The following three abnormal wave forms have been seen during caloric stimulation.

1. Inhibition of nystagmus: In some subjects, nystagmus does not reach its maximum speed until about 100 seconds (Figure 47).

Figure 47: Showing inhibition of nystagmus during and after right caloric stimulation at 30°C in Case 56 (F.73). The nystagmus does not begin until about 80 seconds and does not reach its maximum until about 100 seconds.

McLAY et al (1957) suggested that this was a subjective suppression due to the dislike of the tactile stimulus set up in the meatus by the douching. This view would explain those in whom nystagmus does not appear until the tubing is removed from the meatus, and in which the start of the nystagmus is sudden and of high speed and frequency. It does not explain the later onset of nystagmus. This is more likely to be

associated with the state of alertness.

2. Intermittent nystagmus: This is rather similar to inhibition of nystagmus. In these cases, the nystagmus begins normally and then ceases, only to restart again a few seconds later (Figures 48 and 49). This intermittency may be repeated over a period of up to one minute or more.

Figure 48: Showing intermittent caloric nystagmus between 60 and 90 seconds in Case 72 (M.56) with right Meniere's disease.

Figure 49: Showing intermittent caloric nystagmus in Case 100 (M.59) with right Meniere's disease. This patient could 'switch' the nystagmus on and off at will.

Recognition of both of these phenomena will stop one thinking that nystagmus has stopped during the latent phase.

Both of these phenomena can be abolished by asking the patient to do mental arithmetic (vide infra).

3. Dysrhythmia: This abnormality is an irregular nystagmus in which large oscillations alternate with small ones, fast frequency with slow frequency, and in which sometimes there are periods of jerks alternating with periods of rests (RIESCO-MAC-CLURE, 1964). This is seen in Figure 50.

Figure 50: Showing a dysrhythmic caloric response to cold irrigation in Case 163 (F.49) with disseminated sclerosis.

Normal post caloric nystagmus is usually regular in amplitude and frequency. Riesco-Mac-Clure (1964) has reported dysrhythmia particularly in lesions of midline structures of the posterior fossa such as medulloblastoma, astrocytoma, ependymoma and advanced disseminated sclerosis. In this series it has been seen in peripheral as well as central lesions and probably has no diagnostic value.

Figure 52: Case 85 (M.42) with vestibular neuronitis. Square waves appear at 150 seconds and secondary nystagmus at 187 seconds.

Two further anomalies may occur which could lead to an error in judging the end point.

1. After nystagmus: In some cases, once the square waves have appeared, some nystagmus in the same direction as the induced nystagmus may occur and continue for some time (Figure 51).

Figure 51: Showing after nystagmus at 290 seconds after a hot caloric in the right ear in Case 101 (M.62) with cervical vertigo.

It is easily recognised as after nystagmus, if it appears after square waves. If the square waves do not occur, however, then estimation of the end point of the nystagmus becomes impossible. Occasionally, after nystagmus has been seen for six minutes after cessation of douching. It is felt that after nystagmus could be due to memory (vide infra).

2. Secondary nystagmus: In a number of cases (normal and pathological), after the square waves have appeared secondary nystagmus occurs (Figure 52).

Secondary nystagmus is nystagmus in the opposite direction to the induced primary nystagmus. In one normal subject it has

been seen for up to two minutes after cessation of the primary nystagmus.

It is such a constant post-rotational response that it can, in the rotation test, be regarded as almost normal (McLAY et al, 1957). It presents no difficulties as regards end points, as, if it does not follow square waves, there is a short pause between the last of the primary nystagmus strokes and the first of the secondary phase.

Secondary nystagmus probably has a central cause (BUYS, 1924; DODGE, 1923; FISCHER & WODAK, 1924; LANGE, 1939 and MITTERMAIER, 1950). The fact that optokinetic tests can produce secondary nystagmus (ASCHAN & BERGSTEDT, 1955) suggests that secondary nystagmus is to be dissociated from ampullar stimulation. In this case it is a special form of oculogyral illusion (GRAYBIEL et al, 1946). Secondary nystagmus also, never occurs at extreme cupular deviations, but only after all after-sensation has gone. It is more frequently seen in hyperexcitable labyrinths.

Both after and secondary nystagmus are easily recorded and seen with the use of electronystagmography, but by direct observation of the eyes, gross errors in estimating duration could occur.

Figure 53: Showing normal sinusoidal movements occurring in a normal subject after watching a pendulum. E.C. = Eyes closed.

Figure 54: Nystagmus to the left after watching a pendulum. At the arrow the eyes closed.

Nystagmus and Memory.

Anyone who has had the misfortune to be travel sick is familiar with the phenomenon of after motion. By this is meant the sensation, after landing, of unsteadiness and dysequilibrium which may persist up to 24 hours, even after a short journey, if the stimulus has been severe. This dysequilibrium cannot be the result of angular or linear accelerative processes as these are no longer active.

In this respect, the experiments of KLIJN and EK (1959) are informative. They stimulated pigeons with a sinusoidal rotatory movement, and as a result of acceleration, eye movements occurred which had the same sinusoidal pattern as the stimulus. About four hours after the pigeons were no longer subject to the stimulus mentioned above, the sinusoidal pattern could still be found in the spontaneous head movements. The head movements were found to be identical to the response obtained under stimulation.

In Experiment 6, in which six normal subjects were asked to follow the movements of a pendulum for three minutes and then to close the eyes, there was evidence of a pendular memory eye movement after closure in three cases (Figure 53). In one subject, a brisk nystagmus to the left was induced (Figure 54).

That this was a cortical function was shown by the fact that the memory eye movements were abolished by asking the

subjects to do mental arithmetic (Figure 55).

Figure 55: Showing abolition of memory nystagmus by mental arithmetic. M.A. = point at which questions started.

If it is true that a recollection of eye movements exists and causes a continuation of eye movements after termination of the stimulus, it could be assumed that this memory function could also affect the duration of a caloric nystagmus.

When the cupula is no longer stimulated there can still be a nystagmus which is the result of memory. In this way, the after nystagmus, in the same direction as the primary nystagmus, is not the result of direct vestibular stimulation.

It is also possible that memory may artificially prolong a caloric nystagmus (disregarding the after nystagmus). If this is so, then it could be one of the factors to account for the fact that in a caloric test, the maximum eye speed of the slow phase has proved to be a more sensitive and more reliable criterion of the excitability of the labyrinth than the duration of the nystagmus.

According to McLAY et al (1957) the after nystagmus due to memory is seen very frequently after optokinetic stimuli.

Nystagmus and Alertness.

Loss of clear nystagmus during a caloric or rotation test and its later return without renewed vestibular stimulation (i.e. intermittent nystagmus) has been pointed out by SPIEGEL and ARONSON (1933), WENDT (1951), BENDER (1955), McLAY et al (1957) and LIDVALL (1961). The same authors have put forward the general idea that something like alertness is important in maintaining this effect. MOWRER (1934) and FISCHER and WOLFSON (1943) pointed out that tension factors might influence the caloric response.

The effects of eye closure on the central nervous system are not yet clearly known. It is known, however, that when the eyes close, alpha activity appears in the electroencephalogram. Caloric nystagmus is facilitated by two factors which inhibit alpha rhythm, i.e. eye opening and alerting.

MAHONEY, HARLAN and BICKFORD (1957) put forward two possibilities for intermittency. Firstly, when the eyes are closed they no longer stay in the central position, because the extraocular motor nuclei are then under the influence of stimuli from vestibular nuclei, uncontrolled by cortical inhibiting influences. With alerting, the tendency is for

Figure 56: This was a patient not included in this series who had a left sided total deafness and absence of vestibular function due to mumps many years previously. No nystagmus was shown until she was asked to do mental arithmetic (signified by the arrows).

Figure 57: Normal subject (M.30) who had no caloric nystagmus at all unless constantly asked to do mental arithmetic. During the phase where no nystagmus was shown no questions were asked.

Figure 58: Case 157 (M.53) with cervical vertigo. Patient showed no caloric nystagmus unless asked to do mental arithmetic. Q.S. = Questions stopped. M.A. = Mental arithmetic restarted.

the eyes to go back to the central position and restart the nystagmus. The alternative explanation would be that the eye centering mechanisms hold in check caloric nystagmus during eye closure only so long as the attention of the person can be entirely directed towards this end. Any alerting mechanism would interfere with this inhibition.

The experiments of COLLINS and GUEDRY (1962) showed that loss of clear vestibular nystagmus during periods of mental relaxation occurred in several ways, but the eyes were not simply displaced in the slow phase direction. The fast phase was replaced by a slow phase in the anticipated fast phase direction, or by a substantially reduced fast phase. During such periods, regardless of the speed of the fast phase, the eye movements in the anticipated slow phase direction were of lower velocity than during regular nystagmus when the subject was mentally active.

In the present series, suppression or intermittency of nystagmus has been seen on many occasions, and it is now routine to ask the patients to do simple mental arithmetic as an arousal stimulus, if no nystagmus is shown in a certain position or after a caloric test. Figures 56, 57 and 58 show three examples of the effect of arousal on nystagmus.

With an A.C. amplifier it is impossible to say to which side the eyes are deviated. Figures 57 and 58 show that some movement of the eyes has occurred. This is not shown in

Figure 59.

Figure 60.

Figure 56 however.

According to COLLINS and GUEDRY (1962) three types of eye movements exist - reduced slow phase with fast phase normal, reduced slow and fast phase and slow oscillation with no distinguishable fast phase. It may be that these represent different levels of alertness which influence differently the eye centering tendency. It is also obvious that the slow phase can vary with alertness as well as by cupular deflection.

The clinical significance of this finding as related especially to the caloric test is very important. As well as duration being affected by higher centres (memory), eye speed may also be affected (arousal).

Frequency.

From Experiment 8 it is seen that frequency of nystagmus increases with the eye speed as the temperature falls (Figure 59). The increase is not linear, however, and the frequency has almost reached its maximum at 33°C.

In a typical normal subject, the frequency follows eye speed during the course of a post caloric reaction quite closely but by no means exactly (Figure 60).

TOROK and NYKIEL (1962) felt that the significant information provided by the recording of the slow component

velocity could be omitted, as long as the frequency of the nystagmus is analysed. They attached a simple computer to the nystagmogram which provides a legible and comprehensive record of frequency.

Frequency is not as reliable a component as eye speed if a computer is not available, however. If the amplitude is regular, then frequency is easy to count, but if the amplitude varies and the frequency is fast, then some of the beats will be 'lost' in the increased time it takes the pen to cover the larger amplitude. Frequency is not reliable in a dysrhythmic caloric nystagmus or if the nystagmus is intermittent.

Errors in the Method.

Almost every poor record is due to faulty skin preparation, placement or fixation of electrodes. The interference takes the form of a 50 c.p.s. hum, which to an extent, can be filtered off. Even if this is successful, however, it usually precludes the use of higher gains on the machine.

In time, the electrodes become corroded by the use of electrode jelly and to delay this happening as long as possible, it is important to clean them thoroughly with methylated ether immediately after use.

Interference in the record is also liable to occur if the

recordings are made too near a physiotherapy, sterilising, radiotherapy or radiology department, due to the high voltage equipment used there.

It seems hardly necessary to mention two simple precautions, but once or twice in the process of this work they have been a source of trouble. Firstly, it is necessary to make sure the electrodes are placed on the correct sides. If they are placed on the wrong sides (i.e. red on the right and orange on the left), the record will be reversed, and a right beating nystagmus will be represented as left beating and vice versa. Secondly, it is necessary to label all records before starting, with the patient's name, gain used and speed of paper. If this simple procedure is not a routine, then one is liable to be left with two or three unlabelled, indistinguishable nystagmograms at the end of the day.

Disadvantages of the Method.

With a single channel recorder only eye movements in the recording plane are accessible. With a two channel instrument, however, simultaneous recordings of both horizontal and vertical nystagmus can be made. This is not such a drawback, however, as vertical nystagmus recordings give no added clinical information. There is no automatic time marker on

Figure 61: Case 126 (M.16) with gross
rotatory nystagmus. No evidence of this
is seen in the record.

the apparatus used in this work, and it is necessary to check the speed of the paper occasionally to see if the timing is correct.

Purely rotatory nystagmus cannot be registered with any method of electronystagmography. No other writers have been able to confirm the claims of DIETERLE and MONNIER (1955) that purely rotatory nystagmus can be recorded with the electrodes placed in a ring around the orbit. It is possible to pick up horizontal nystagmus with electrodes placed right and left as the eyes move around a vertical axis; with the electrodes placed above and below the eye, vertical nystagmus can be recorded as the eyes move around a horizontal axis. In rotatory nystagmus the eyes rotate about an axis parallel to the sagittal plane of the head, and so no movement of the corneo-retinal potential difference occurs; for this reason it is difficult to see how purely rotatory nystagmus could be recorded.

Figure 61 shows an attempt to record a gross rotatory nystagmus in Case 126 (M.16) who had had encephalitis at the age of 9 years, and who now had deafness, cerebellar ataxia and easily visible rotatory nystagmus.

Since this series was completed, an attempt has been made to record the movements in a case of nystagmus retractorius in which the eye moves in and out. Not surprisingly the attempt was unsuccessful.

If electronystagmography is to achieve a place in routine clinical practice, the time element is very important. Using the methods described in this thesis, to do the position and caloric tests with electronystagmography takes about 45 minutes. Three patients can, therefore, be done in an afternoon quite easily.

D.C. and A.C. Amplifiers.

When recording nystagmus with a D.C. amplifier and the subject looks to the right, the deviation from the base line is maintained. On the other hand, with an A.C. amplifier the shift is transient (Figure 62).

Figure 62: See text.

When recording an induced nystagmus, the direction of gaze influences the nystagmus. With electronystagmography, recording with the eyes closed, the direction of gaze is known

only with a D.C. amplifier. A.C. recordings, apart from the direction of gaze, give reliable information as to the other parameters.

In a D.C. amplifier, the resistance capacity coupling between the grids of the valves of the A.C. amplifier, is replaced by direct coupling. HALLPIKE, HOOD and TRINDER (1960) described how to get over the problem of random voltage variations within the valves, circuit and batteries of a D.C. amplifier (which may exceed the corneo-retinal potential changes). They used a chopper amplifier to convert the standing potential difference into an alternating waveform which is amplified by a conventional resistance capacity coupled amplifier. To reconvert the alternating waves into a direct current, a second chopper, operating in strict synchrony with the first, is introduced in the final stage.

According to HENRIKSSON (1965) the greatest difficulty that he had with D.C. recordings was the design and care of suitable electrodes to eliminate electrical artefacts from electrochemical potentials generated at the skin electrode interface.

If electronystagmography is to find a place in routine clinical practice in centres which are not fortunate enough to have a full-time physicist on the staff, then the future must lie in the use of A.C. amplifiers, which are much cheaper, much easier to control, more reliable and also give reliable

information on all parameters apart from the direction of gaze.

CONCLUSIONS

The Devices High Speed Recorder which has been used in this work fulfils all the criteria required for clinical electronystagmography. It is compact and portable, and is sensitive enough to pick up the average eye signal of 40 microvolts for 1° of eye movement quite easily. It is direct writing, it involves no mechanical interference with the eyes, it provides for recording eye movements under different visual conditions, it does not restrict the patient's movements, it is reasonably priced and it is reliable.

Electronystagmography gives an accurate documentation of the oto-neurological findings, and can give a reliable and comparable guide as to the course of a patient's condition; it also enables various cases to be compared.

The use of electronystagmography is the only certain way to distinguish ocular from vestibular nystagmus.

When a patient complaining of vertigo presents himself at a clinic, the main questions to be answered are:-

1. Is it functional or organic?
2. Is it central or peripheral?
3. If it is peripheral especially, which side is affected?

Position tests with electronystagmography give a reasonably reliable answer to the first question if the

criteria for pathological nystagmus are applied. Since electronystagmography shows nystagmus about eight times more often than it is visible clinically it is extremely useful in this role.

No reliable answer can be given to the second question. It can be said, however, that if direction changing nystagmus is seen, then the lesion is likely to be central. It is only possible to see this with electronystagmography.

Neither the position tests nor caloric tests on their own will show the side affected in a peripheral lesion unless there is a unilateral canal paresis.

A new classification is proposed for positional nystagmus in order to avoid ambiguity. The terms position changing nystagmus and direction changing nystagmus are redefined.

The caloric test is an unphysiological test of vestibular function. JONGKEES and PHILIPSZOOM (1964) stated that "in only a few cases will the caloric test give new information after the position test and audiogram have been performed." In this series no diagnosis was made from the result of the caloric test. A full history and examination, audiometry and position tests were usually enough to give a diagnosis.

No additional help was gained from the caloric test in deciding the side of the lesion. In unilateral lesions, the audiogram was usually the best guide as to the side affected. In no case in this series was the caloric test on its own, of

help in deciding whether a lesion was central or peripheral.

If the caloric test is used, however, it ought to be done with electronystagmography, and the maximum eye speed taken as the parameter of estimation of vestibular function rather than the duration. It has been shown from the temperature experiments and the results of the pathological series that the eye speed is a more precise criterion of measurement of vestibular function than duration.

Electronystagmography has shown how the higher centres affect the response to caloric stimulation with memory affecting duration and state of alertness affecting both the eye speed and the duration.

The only way to see the anomalies in the end point of a caloric nystagmus is with the use of electronystagmography.

A directional preponderance gives no clue as to the site of the lesion, whether peripheral or central; it is only a caloric manifestation of a spontaneous nystagmus.

Perhaps the most important clinical feature of this work has been the subdivision of patients complaining of transient bouts of vertigo on movement of the head, into true vertebro-basilar insufficiency and cervical vertigo. The latter group is amenable to therapy by either neck traction or a collar. The only way to differentiate these two groups adequately, is with the use of electronystagmography.

APPENDICES

FOREWORD

Since this thesis deals primarily with electronystag-mography, only the results of the position tests, audiometry and caloric tests have been tabled. The full symptomatology and physical findings in each of the 200 pathological cases have been left out in the interest of clarity.

The speed and direction of the nystagmus in each position of the position tests is given, as are the results in any further position tests under the heading 'Hallpike Position Test'.

As regards audiometry, it can be taken that all deafnesses are neurosensory in origin unless labelled conductive. Generally speaking, only the cases which showed positive recruitment are so labelled; occasionally, where interesting, a negative recruitment is signified.

The caloric results are given under the headings of canal paresis and directional preponderance; the percentages given refer to the results of the formulae mentioned in Section 4 as relating to both maximum eye speed and duration.

Many cases had position tests and/or caloric tests several times, but for clarity, only the results on first visit have been listed.

The following abbreviations have been used:-

U	=	upright
P	=	prone
S	=	supine

HR - head right

HL - head left

HD - head down

CP - canal paresis

DP - directional preponderance

ES - eye speed

D - duration

B - bilateral

R - right

L - left

RCT - recruiting

CSOM - chronic suppurative otitis media

Post op. - post operative

DS - disseminated sclerosis

HYST - hysterical vertigo

VUO - vertigo of unknown origin

APPENDIX 1

HORIZONTAL NYSTAGMUS

IN NORMALS.

Case No.	Sex	Age	U	P	S	HR	HL	HD
1	M	24	-	1°R	-	-	4°R	-
2	M	32	-	-	4°L	-	-	-
3	F	26	-	-	-	-	-	-
4	F	19	-	-	-	-	-	-
5	F	29	-	2°R	-	-	2°R	-
6	M	55	-	-	3°L	-	-	2°L
7	F	27	-	-	-	-	-	-
8	M	30	2°R	2°R	-	-	-	2°R
9	F	22	2°R	-	-	-	3°R	-
10	F	19	-	-	2°L	3°L	5°R	-
11	F	22	-	-	-	-	-	-
12	M	28	-	-	-	-	2°L	-
13	F	23	2°L	-	-	-	2°L	-
14	M	33	-	-	-	-	-	-
15	M	20	-	-	-	-	-	-
16	M	21	-	-	-	-	-	-
17	M	35	2°R	-	-	-	-	1°R
18	F	29	-	-	4°L	-	-	-
19	M	44	-	-	-	2°L	-	-
20	M	32	-	-	-	-	-	2°R
21	M	35	-	-	-	-	-	-
22	F	29	2°R	-	-	-	-	4°R
23	M	51	2°R	-	2°R	1°R	-	-
24	M	55	-	-	-	-	-	-
25	F	40	-	2°L	2°L	-	-	-
26	F	24	-	-	-	-	-	-
27	F	21	-	-	-	1°R	-	-
28	F	19	4°L	-	-	-	-	-
29	F	20	2°L	-	-	-	5°L	-
30	M	26	-	-	-	-	-	-
31	M	24	-	-	1°R	1°R	-	-
32	M	42	-	-	-	-	-	2°L
33	F	49	-	1°L	-	-	-	2°L
34	M	21	-	-	-	-	-	-
35	F	19	-	-	-	-	-	-

APPENDIX 2.Vertical Nystagmus in Normals.

Case No.	Sex	Age	U	P	S	HR	HL	HD
1	M	28	3°U	5°U	6°U	5°U	4°U	9°U
2	M	30	14°U	5°U	12°U	10°U	4°U	16°U
3	F	22	-	5°U	5°U	-	5°U	5°U
4	F	22	5°U	-	4°U	5°U	3°U	-
5	F	19	-	3°D	-	3°D	-	-
6	F	30	-	-	5°U	-	-	-
7	M	30	5°U	10°U	-	6°U	10°U	8°U
8	M	30	-	-	-	-	-	-
9	M	29	-	-	-	-	-	-
10	F	19	-	-	-	-	-	-
11	F	30	10°U	8°U	4°U	6°U	9°U	2°U
12	M	24	-	-	-	-	-	-
13	F	21	4°U	5°U	-	-	3°U	-
14	M	33	5°U	5°U	5°U	-	4°U	9°U
15	F	21	-	-	-	-	-	-
16	M	55	6°U	7°U	-	-	-	8°U
17	F	31	7°U	-	-	4°U	9°U	9°U
18	M	30	-	-	-	-	-	-
19	M	42	-	-	-	7°D	-	10°D
20	F	21	5°U	-	5°U	5°U	-	-
21	F	21	-	-	-	-	-	-
22	M	25	-	-	-	-	-	-
23	F	19	6°U	-	-	7°U	7°U	-
24	F	20	-	6°U	-	8°U	-	8°U
25	F	24	-	6°U	-	-	-	9°U

APPENDIX 3. LINEARITY OF CALIBRATION.

APPENDIX 4. LIGHTING EFFECTS ON CALIBRATION.

APPENDIX 5. 30° UPWARD DEVIATION OF THE EYES.

APPENDIX 3LINEARITY of CALIBRATION

Case No.	Sex	Age	20°	30°	40°
1	M	34	17	26	36
2	M	54	20	31	44
3	F	23	19	30	40
4	M	28	19	29	40
5	F	19	18	29	39
6	M	29	20	30	41
MEAN			18.9	29.1	40.

APPENDIX 4LIGHTING EFFECTS on CALIBRATION

Case No	Sex	Age	E.O.	E.C.
1	M	27	18	18
2	M	54	22	20
3	F	23	22	24
4	M	31	16	18
5	M	27	19	20
6	M	29	20	18
MEAN			1.9	2.1

E.O. = Eyes open E.C. = Eyes closed.

APPENDIX 530° UPWARD DEVIATION of the EYES

Case No.	Sex	Age	10° extended	20° flexed.
1	M	34	15	16
2	M	54	17	20
3	F	23	18	18
4	M	29	19	21
5	M	27	18	21
6	F	19	20	20
MEAN			17.9	19.3

Figures expressed in millimetres of deviation on the paper.

APPENDIX 6.

Relationship of Eye Speed to Duration at Different Temperatures.

Case No.	Sex	Age	37°C		35°C		33°C		31°C		29°C	
			E.S.	D.	E.S.	D.	E.S.	D.	E.S.	D.	E.S.	D.
1	F	19	2	68	4	124	7	134	9	136	12	165
2	F	23	0	0	15	218	22	202	30	214	30	208
3	F	30	15	136	16	134	19	148	24	172	38	178
4	M	31	3	96	8	112	10	118	12	138	17	168
5	M	29	2	122	21	136	48	142	70	152	90	165
6	M	21	8	106	10	142	14	151	15	154	21	169
7	M	23	2	101	14	118	16	141	16	152	22	168
8	M	32	2	102	14	112	56	152	65	184	74	209
9	M	50	6	108	9	1130	11	141	16	146	16	165
10	F	21	6	112	13	121	20	182	21	166	29	205
11	M	35	11	168	19	214	35	205	36	194	42	191
12	M	55	4	132	10	171	16	178	18	162	23	174
13	M	30	7	141	14	155	33	166	38	173	45	182
14	F	19	6	110	11	121	13	130	19	150	21	170
15	M	27	2	101	5	115	12	130	24	147	35	159
MEAN			5.1	106.9	12.2	141.5	22.1	155.3	27.4	162.6	34.3	178.4

ES = eye speed in degrees per second

D.= durations in seconds.

APPENDIX 7

RELATIONSHIP BETWEEN EYE SPEED AND FREQUENCY AT DIFFERENT TEMPERATURES

Case No.	Sex	Age	37°C		35°C		33°C		31°C		29°C	
			ES	F	ES	F	ES	F	ES	F	ES	F
1	F	19	2	0.3	4	0.9	7	1.0	9	1.3	12	1.9
2	F	23	0	0	15	1.3	22	1.9	30	2.0	30	1.7
3	F	30	15	1.4	16	1.8	19	2.4	24	2.4	38	2.5
4	M	31	3	0.5	8	1.2	10	1.4	12	1.9	17	2.0
5	M	29	2	1.0	21	1.8	48	2.6	70	2.7	90	2.5
6	M	21	8	1.2	10	1.7	14	1.8	15	2.4	21	2.6
7	M	23	2	0.5	14	1.3	16	1.7	16	1.8	22	2.1
8	M	32	2	0.5	14	1.2	56	1.7	65	2.2	74	2.9
9	M	50	6	0.8	9	0.9	11	1.8	16	1.9	16	2.0
10	F	21	6	0.8	13	1.3	20	1.8	21	1.7	29	1.8
11	M	35	11	1.2	19	1.6	35	1.7	36	2.0	42	2.4
12	M	55	4	1.2	10	1.7	16	1.8	18	2.3	23	2.3
13	M	30	7	1.3	14	1.6	33	2.2	38	2.4	45	2.5
14	F	19	6	1.1	11	1.7	13	1.8	19	2.0	21	2.6
15	M	27	2	0.4	5	1.1	12.	1.8	24	2.0	35	2.5
MEAN			5.1	0.86	12.2	1.4	22.1	1.9	27.4	2.1	34.3	2.3

ES = Eye speed in degrees per second

F = Frequency in beats per second

APPENDIX 8

EFFECT OF TEMPERATURE ON ONSET OF NYSTAGMUS

Case No.	Sex	Age	37°C		35°C		33°C		31°C		29°C	
			O	ES	O	ES	O	ES	O	ES	O	ES
1	F	19	42	2	38	4	30	7	26	9	19	12
2	F	23	-	-	56	15	38	22	35	30	33	30
3	F	30	50	15	38	16	31	19	26	24	22	38
4	M	31	52	3	39	8	32	10	29	12	22	17
5	M	21	41	8	22	10	26	14	32	15	29	21
6	M	23	44	2	30	14	30	16	22	16	22	22
7	F	21	35	6	25	13	35	20	35	21	29	29
8	M	35	27	11	14	19	9	35	7	36	5	42
9	M	55	25	4	16	10	18	16	18	18	23	23
10	M	30	35	7	31	14	22	33	18	38	12	45
MEAN			35.1	5.8	30.9	12.3	27.1	19.2	24.0	21.9	21.1	27.9

O = onset expressed in seconds

ES = high speed expressed in degrees per second

APPENDIX 9.

THE EFFECTS OF DIFFERENT
DURATIONS OF IRRIGATION

10 Second irrigation

Case No	Sex	Age	Onset	Duration (seconds)	Frequency (bts per sec)	Maximum eye speed (degrees/sec)	Point of Max. eye speed
1	M	23	18	165	2.4	21	50
2	M	29	17	158	2.1	40	50
3	M	27	26	152	2.6	22	50
4	M	55	32	145	1.6	14	50
5	M	26	12	175	2.1	31	60
6	M	33	34	160	1.7	12	60
MEAN			23.2	159.2	2.1	23.3	53.3

20 Second irrigation

1	M	23	17	186	2.8	31	60
2	M	29	22	162	2.0	40	50
3	M	27	25	180	2.9	33	60
4	M	55	24	147	1.1	14	50
5	M	26	12	182	2.2	31	50
6	M	33	40	169	1.3	9	60
MEAN			23.3	171.	2.2	26.2	55

30 Second irrigation

1	M	23	15	210	2.9	32	50
2	M	29	17	176	2.1	55	60
3	M	27	25	209	3.0	35	60
4	M	55	17	149	1.7	16	60
5	M	26	16	182	2.1	27	50
6	M	33	20	236	1.8	40	70
MEAN			18.3	193.7	2.3	34.2	58.3

APPENDIX 10.

GROUP 1

Case No.	Sex	Age	V.S.N.	U	P	S	H.R.	H.L.	H.D.	Hallpike Posit. Tests	Audiometry	Caloric
3	F	15	no	-	-	2°L	4°L	-	4°L	-	50dB L. RCT.	DP (L) 17%D 21%ES
6	M	60	to L	4°L	3°L	4°L	4°L	4°L	5°L	-	50dB B. RCT.	Mixed LP & DP (R) total
10	M	39	no	4°L	5°L	4°L	2°L	6°L	2°L	-	60dB R. RCT.	CP (R) total
12	F	54	no	5°R	-	2°R	4°R	5°R	4°R	-	30dB low tone R	DP (R) 22%D 56%ES
14	M	53	to L	-	-	7°L	-	-	7°L	HRU - HLU - HRD 3°L HLD 5°L HRU 7°L HLU 9°L	60 dB L RCT.	DP (L) 62%ES 50%D
15	F	43	to L	-	-	5°L	10°R	8°L	8°L	-	70dB R RCT.	CP (R) total
19	M	44	no	-	4°R	7°L	-	-	4°L	HRU - HLU - HRD 2°R HLD - HRU 2°L HLU -	Total deafness L.	CP (L) 54%ES 35%D
22	M	53	no	2°R	1°R	-	5°R	-	-	-	25dB low tone R.	CP (R) 26%ES 0%D
23	F	42	no	1°R	-	-	2°L	3°R	3°L	HRU - HLU - HRD 5°L HLD - HRU 3°R HLU 2°L	50dB L. RCT.	CP (L) 41%ES 0%D
32	M	59	no	4°L	4°L	-	-	-	-	-	40dB L RCT.	CP (L) 38%ES 19%D
33	M	69	no	-	-	-	1°R	1°R	1°R	-	60dB R RCT.	CP (R) 56%ES 42%D
40	M	66	no	6°R	4°R	4°R	3°R	5°R	-	-	40dB R RCT.	N.A.D.
43	M	74	no	-	-	3°L	-	-	3°L	HRU 2°L HLU 1°L HRD 3°L HLD 1°L HRU 2°L HLU 1°L	60dB B RCT.	DP (L) 15%ES 21%D
44	M	55	no	5°R	2°R	4°R	2°R	4°L	2°R	HRU - HLU - HRD 3°R HLD - HRU - HLU 2°R	80dB L RCT.	CP (L) 51%ES 0%D
47	F	65	no	-	-	-	-	2°L	2°L	-	75dB R RCT.	DP (L) 36%ES 19%D
48	M	67	no	-	4°R	3°R	-	6°R	-	HRU 4°R HLU - HRD - HLD - HRU - HLU 3°R	70dB R RCT.	DP (R) 23%D 35%ES
51	F	54	no	2°L	2°L	3°L	-	2°L	-	-	50dB L RCT.	CP (L) 21%ES 11%D
57	M	22	no	-	-	5°R	-	-	-	-	20dB low tone R.	DP (R) 25%ES 19%D
63	M	52	to R	5°R	5°R	3°R	4°R	9°R	-	-	20dB low tone R. RCT.	CP (R) 35%ES 26%D
72	M	56	no	-	-	3°R	-	4°R	5°R	-	60dB R RCT.	CP (R) 68%ES 18%D
74	M	50	no	4°R	6°R	4°R	5°R	-	5°R	-	total deafness L. 80dB R. RCT.	DP (R) 21%D 17%ES
78	F	60	no	2°L	-	-	2°L	2°R	2°L	-	30dB low tone loss L. RCT.	DP (L) 20%ES 9%D

APPENDIX 10 Continued

GROUP 1

Cases 34 and 88 are cases
of Lermoyes syndrome.

Case No.	Sex	Age	V.S.N.	U	P	S	H.R.	H.L.	H.D.	Hallpike Posit. tests	Audiometry	Calorics.
82	M	57	no	-	-	2°L	3°L	-	1°L	-	30dB B low tone loss	DP (L) 15%ES 21%D
100	M	59	no	-	-	-	-	-	-	-	20dB low tone R. RCT.	CP (R) 12%ES 12%D
102	F	53	no	-	-	-	-	4°R	4°R	-	70dB R RCT.	CP (R) 6%D 33%ES
105	F	49	no	-	3°R	3°R	-	2°L	3°L	-	60dB R RCT.	CP (R) 80%ES 13%D DP (L) 35%D 0%ES
107	M	44	no	6°R	2°R	2°R	-	-	4°R	-	50dB L RCT.	DP (R) 22%ES 34%D
108	M	45	no	10°R 5°L	-	-	-	-	-	-	50dB R RCT.	DP (R) total
127	F	57	no	3°R	3°R	-	-	3°R	-	-	60dB R RCT.	CP (R) total DP (R)
146	F	61	no	3°R	-	6°R	4°R	2°R	6°R	-	45dB R RCT.	DP (R) 33%ES 57%D
154	M	47	no	-	-	-	6°R	3°R	-	-	50dB L RCT.	DP (R) 33%ES 21%D
155	M	50	no	5°R	2°R	5°R	4°R	2°R	4°R	HRU4°R HLU - HRD10°R HLD6°R HLU4°R HLU -	total deafness L.	CP (L) 70%ES 56%D
161	F	45	no	-	62	-	-	-	4°L	-	50dB R RCT	CP (R) 24%ES 28%D
166	F	61	no	2°L	4°L	6°L	-	-	2°L	-	40dB L RCT.	DP (L) 30%ES 26%D
172	F	73	no	-	5°R	-	-	3°L	2°R	-	70dB R RCT.	CP (R) 45%ES 16%D
176	M	40	no	1°R	-	3°R	4°R	3°R	6°R	-	60dB R 80dB L RCT. L.	CP (L) 47%ES 14%D
178	F	55	no	2°R	2°R	2°R	-	2°R	-	HLU1°L HRU2°R	50dB R RCT.	DP (R) 16%ES 7%D
179	F	52	no	1°R	-	3°R	-	-	-	-	40dB B RCT. R.	CP (R) 23%ES 5%D
180	F	52	no	-	3°R	-	2°R	-	6°R	-	60dB L RCT.	DP (R) 40%ES 22%D
185	M	24	no	4°L	-	-	3°L	6°L	4°L	-	40dB R RCT.	CP (R) 24%ES 20%D
188	M	55	no	-	-	-	2°R	-	4°R	-	20dB low tone L.	CP (L) 30%ES 22%D
191	M	15	no	-	-	-	-	-	-	-	30dB R RCT.	CP (R) 52%ES 17%D
194	M	61	no	-	-	-	-	-	-	-	40dB L RCT.	CP (L) total
198	M	26	no	3°L	-	2°L	-	2°L	2°L	-	total deafness L.	CP (L) 54%ES 14%D
34	M	45	no	-	-	4°R	-	-	-	-	50dB L	DP (R) 18%ES 23%D
88	F	57	no	-	2°L	2°L	2°L	-	3°L	-	total L 60dB R. Posit. changing R.	CP (L) 13%ES 48%D

APPENDIX 11A

GROUP 2A

Case No.	Sex	Age	VSN	U	P	S	HR	HL	HD	Hallpike Posit. tests	Audiometry	Caloric
1	F	60	no	-	-	2°R	1°R	-	2°R	HRU3°R HLU3°R	total deafness R. 55dB L	DP (R) 10%ES 19%D
17	F	59	? to R	-	-	-	-	-	-	-	N.A.D.	N.A.D.
24	F	60	no	-	-	-	-	1°R	2°R	HRU - HLU - HRD - HLD - HRU12°L HLU26°R	N.A.D.	DP (R) 41%ES 14%D
37	M	59	no	-	-	-	5°R	-	-	-	60dB R 40dB L.	DP (R) 14.5%ES 14.5%D
38	M	54	no	-	-	-	-	3°R	-	-	N.A.D.	N.A.D.
41	M	63	no	-	13°L	6°L	15°L	5°L	10°L	HRU - HLU - HRD11°L HLD15°L HRU - HLU -	30dB B	DP (R) 10%ES 16%D
49	M	55	no	-	-	3°R	-	-	-	HRU - HLU - HRD6°R HLD2°R HRU - HLU4°L	N.A.D.	DP (R) 22%ES 14%D
56	F	73	? to R	-	-	-	-	-	-	HRU - HLU - HRD - HLD4°L HRU9°R HLU4°R	30dB B	DP (L) 33%ES 0%D
58	M	63	no	-	-	-	-	-	-	HRU - HLU - HRD - HLD5°R HRU - HLU5°R	50dB B	CP (R) 38%ES 32%D
59	F	73	no	-	5°L	-	-	20°R to 5°L	-	HRU - HLU - HRD - HLD6°R to 4°L HRU - HLU -	30dB B	-
68	F	56	no	-	-	-	3°L	-	3°L	-	50dB B	DP (L) 15%D 0%ES
70	F	74	no	-	2°R	1°R	-	-	-	-	50dB B	CP(R) total. DP(R)
75	F	60	no	-	1°L	1°L	-	4°L	-	-	40dB B	CP (L) 15%ES 26%D
76	F	72	no	-	-	-	-	-	6°R	-	50dB B	CP (L) 31%ES 0%D
87	F	73	no	-	-	-	-	-	-	HRU - HLU - HRD4°L HLD2°L HRU1°R HLU8°R	N.A.D.	CP (L) 50%D 74%ES
98	M	59	no	1°R	-	1°R	-	-	1°R	-	N.A.D.	DP (R) 61%ES 44%D
113	F.	52	no	-	-	-	-	-	-	-	N.A.D.	CP (R) 37%ES 6%D
114	F	39	no	-	-	-	3°R	-	-	-	N.A.D.	N.A.D.
115	M	37	no	-	-	-	-	-	-	-	30dB Cond. B	N.A.D.
116	M	42	no	7°R	6°R	5°R	9°R	6°R	8°R	HRU3°R HLU4°R HRD10°R HLD6°R HRU7°R HLU5°R	20dB cond. B	CP (L) with Dundas Grant.
120	F	61	no	-	-	-	-	3°L	-	-	N.A.D.	N.A.D.
140	F	56	no	-	-	2°R	-	2°R	-	HRU5°R HLU4°R	N.A.D.	N.A.D.
147	M	55	no	-	-	-	-	-	-	-	20dB B	N.A.D.
148	M	60	no	-	5°R	-	-	-	-	-	30dB B	CP (L) 40%ES 25%D
152	F	55	no	6°R	5°R7	2°R	-	5°R	-	HRU7°R HLU9°R	30dB B	DP (L) 25%ES 7%D

APPENDIX 11A Continued.

GROUP 2A.

APPENDIX 11B

GROUP 2B.

Case No.	Sex	Age	VSN	U	P	S	HR	HD	HL	Hallpike Posit. tests	Audiometry	Caloric.
170	M	69	no	-	-	-	2°L	2°L	-	-	20dB B	N.A.D.
171	M	60	no	3°R	2°R	-	-	-	5°R	-	30dB B	CP (L) 1%ES 28% DP (R) 27%ES 5% N.A.D.
175	F	55	no	-	-	-	-	4°L	-	-	40dB B	N.A.D.
186	F	71	-	-	-	-	-	2°L	-	-	30dB B	DP (R) 13% 9%ES
192	F	54	-	5°L	-	3°L	-	-	4°L	-	25dB B	N.A.D.

APPENDIX 11 B

39	M	53	to R	10°R	6°R	5°R	4°R	3°R	5°R	-	60dB L	DP (R) 24% 15%ES
138	F	63	no	5°L	7°L	9°L	6°L	6°L	6°L	-	total deafness R.	DP (L) 65% 29%ES EP (R) 23% 61%ES
150	F	60	to L.	-	3°L	6°L	5°L	-	7°L	-	60dB R	DP (L) 48% 60%ES
160	F	64	no	5°R	3°R	6°R	4°R	2°R	8°R	-	total deafness L.	CP (L) total
197	M	29	no	2°L	2°L	5°L	14°L	5°L	4°L	-	total deafness R.	CP (R) total.

APPENDIX 11C.

GROUP 2C.

Case No	Sex	Age	V.S.N. U	P	S	HR	HL	HD	Hallpike Posit. tests	Audiometry	Caloric.	
4	F	60	to R	3°R	2°R	3°R	-	6°R	5°R	-	30dB R total L	CP (L) total
7	M	66	no	-	-	-	3°R	3°L	2°R	-	30dB B	DP (R) 40%ES 45%D
27	F	76	to L	3°L	3°R	3°L	-	5°L	-	HRU7°R HLU - HRD21°R HLD29°R HRU7°R HLU9°R head extended 7°R head flexed 3°L	40dB B	-
31	F	74	no	1°L	1°L	-	3°L	3°R	4°L	HRU - HLU - HRD - HLD6°R HRU - HLU4°L	total deafness R. 40dB L.	CP (L) 51%ES 20%D
36	F	60	no	-	-	8°R	4°R	4°R	4°R	HRU - HLU - HRD6°R HLD3°R HRU5°R HLU3°R	40dB B	DP (R) 11%ES 47%D
46	F	47	no	-	-	-	-	3°R	3°R	HRU - HLU - HRD3°R HLD2°R HRU - HLU3°R	NA.D.	DP (R) 27%ES 11%D
50	F	79	no	3°R	-	3°L	2°L	2°R	-	-	80dB L 40dB R	CP (L) 50%ES 9%D
52	M	66	no	8°R	8°R	-	17°R	-	6°L	Head extended 7°R Head flexed 5°L	total deafness L. 60dB R.	DP (L) 12%ES 31%D
54	M	63	no	-	-	-	3°R	2°R	-	-	60dB L 40dB R	DP (L) 44%ES 8%D
55	F	55	no	-	-	4°R	4°R	7°L	-	-	40dB B	Impossible to interpret
64	M	62	no	-	-	-	2°L	3°L	3°L	HRU - HLU - HRD3°R HLD20°R HRU - HLU -	30dB B -76°L	-
69	F	60	no	-	1°L	-	1°L	3°L	3°L	HRU - HLU - HRD3°L HLD4°L HRU2°L HLU3°L	N.A.D.	DP (L) 26%ES 7%D
71	M	56	no	2°R	-	2°R	12°L	13°R	2°L	-	N.A.D.	DP (R) 24%ES 21%D
101	M	57	no	3°R	-	3°R	1°R	3°R	1°R	-	20dB B	DP (R) 47%D 15%ES CP (L) 61%ES 26%D
106	F	73	no	1°L	1°L	-	2°R	1°L	-	HLU - HLU1°L HLD4°L HLD4°R HLU1°L HLU2°L	N.A.D.	-
121	M	61	no	-	-	3°R	2°R	4°R	2°R	-	N.A.D.	CP (L) 30%ES 0%D

APPENDIX 11C Continued.

GROUP 2C.

Case No	Sex	Age	VSN.	U	P	S	HR	HL	HD	Hallpike Posit. tests	Audiometry	Caloric
125	M	64	no	2°L	-	5°L	5°L	6°R	5°L	-	40dB B	DP (L) 36%ES 13%D
129	F	66	? to R	2°L	-	12°L	6°L	2°L	6°L	HRU ? HLU ? HRD14°L HLD4°L HRU7°L HLU -	50dB B	CP (L) 50%ES 45%D DP (L) 62%ES 50%D
145	M	69	no	3°L	-	4°L	3°L	3°L	6°L	HRU - HLU2°R HRD8°L HLD7°L HRU4°R HLD5°R	20dB B	DP (R) 31%ES 17%D
151	M	60	no	3°L	2°L	6°L	6°L	7°R	5°L	-	20dB B	CP (R) 25%ES 4%D
153	M	33	no	2°R	-	1°R	2°R to 2°L	2°R to 2°L	-	-	Cond R 40dB.	N.A.D.
157	M	53	no	-	-	-	2°L	-	4°L	-	40dB B	CP (R) 25%ES 0%D
158	M	40	no	3°R	-	2°L	5°R	3°R	HRU2°R HLU3°L HRD2°R HLD2°R to 4°L HRU 2°R HLU 3°L	N.A.D.	N.A.D.	
162	M	64	no	1°R	-	-	11°R	13°L	7°R	-	20dB B	N.A.D.
164	F	72	no	-	-	-	2°R	4°R	-	HRU2°R HLU -	30dB B	N.A.D.
168	M	50	to R	2°R	-	-	8°R	9°L	6°R	-	30dB B	N.A.D.
183	F	60	no	-	4°R	-	8°L	6°R	-	-	N.A.D.	N.A.D.
184	M	45	no	-	-	-	4°R	4°L	-	, -	15 dB B	N.A.D.
189	F	65	no	-	3°R	2°L	7°L	10°R	-	-	N.A.D.	CP (L) 33%ES 0%D DP (R) 12%D 0%ES
190	M	54	no	6°R	2°R	2°R	4°R	6°R	8°R	HRU 6°R HLU5°L	conduct 40dB L	CP (L) total.
195	M	54	no	2°R	2°R	-	-	3°R	3°R	HRU2°L HLU -	30dB B	CP (R) 31%ES 28%D

APPENDIX 12.

GROUP 3.

Case No	Sex	Age	VSN	U	P	S	HR	HL	HD	Audiometry	Caloric
9	F	58	no	-	-	8°L	-	2°L	2°L	N.A.D.	CP (R) total
18	M	24	no	-	4°R	3°R	-	-	-	N.A.D.	DP (R) 13%ES 15%D
26	M	52	no	3°R	-	-	-	-	-	N.A.D.	CP (R) 48%ES 1%D DP (R) 62%ES 1%D
79	F	43	no	-	6°R	-	-	3°R	-	N.A.D.	DP (R) 19%ES 0%D
84	F	47	to L	11°R	7°L	-	-	4°L	4°L	N.A.D.	N.A.D.
85	M	42	no	4°R	3°R	4°R	-	-	6°R	N.A.D.	DP (R) 37%D 30%ES
92	F	42	no	-	-	-	-	1°L	2°R	N.A.D.	DP (L) 14%ES 6%D
93	F	48	no	2°R	-	-	2°R	-	-	N.A.D.	DP (R) 21%D 7%ES
94	F	28	no	-	-	-	-	-	2°L	N.A.D.	DP (L) 17%D 6%ES
109	M	41	no	-	-	4°R	4°R	-	-	N.A.D.	DP (R) 53%ES 30%D
133	M	41	no	-	-	-	-	-	-	N.A.D.	DP (L) 56%D 52%ES
156	F	65	no	2°R	9°R	2°R	7°R	2°R	6°R	N.A.D.	DP (R) 45%ES 49%D
169	F	26	no	4°L	3°L	2°L	6°L	6°L	6°L	N.A.D.	DP (L) 56%ES 42%D
173	F.	53	no	-	-	-	-	-	3°L	N.A.D.	CP (R) 18%ES 20%D
187	M	59	no	1°L	6°L	2°L	3°L	6°L	5°L	N.A.D.	DP (L) 2%ES 4%D
200	F	42	no	-	-	-	3°L	3°L	5°L	N.A.D. DP	DP (L) 20%ES 6%D

Case

Sex

Age

VSN

II

D

APPENDIX 13.GROUP 4.

Case No	Sex	Age	VSN	U	P	S	HR	HL	HD	Hallpike Posit. tests	Audiometry	Caloric
5	F	58	to L	-	2°L	3°L	-	6°L	2°L	HRD Rotatory	40dB L total R	N.A.D.
77	M	44	no	-	-	8°R	4°L	-	7°L to 5°R	HRU - HLU - HRD9°R HLD - HRI6°R HLU -	Dip at 3KC B.	-
104	M	43	no	6°R	-	-	2°R	-	2°R	HRU - HLU - HRD12°R HLD - HRU5°R HLU -	N.A.D	DP (R) 29%ES 6%D
112	M	41	no	-	-	-	-	-	-	HRU rotatory	N.A.D.	N.A.D.
117	M.	41	no	3°R	-	3°R	7°R	6°L	6°R	HRU2°R HLU - HRD4°R HLD15°R HRU2°R HLU -	High loss L.	CP (L) total
119	M	36	no	5°R	-	-	5°R	-	8°L	HRD rotatory	Dip 3KC B	CP (R) 60%ES DP (R) 34%D
128	M	60	no	4°L	-	3°L	2°L	3°L	-	HRU - HLU - HRD 8°L HLD5°L HRU5°L HLU4°L	High loss L.	DP (L) total
142	F	65	to R	10°R	11°R	8°R	13°R	3°R	10°R	HLD rotatory	High loss L	DP (R) 41%ES 40%D
182	M	49	no	2°L	14°R	4°R	15°L to 5°R	7°R	5°R	HRU - HLU - HRD18°L to 7°R HLD - HRU - HLU -	N.A.D.	DP (R) 16%D 0%ES
196	M	61	to R	5°R	15°R	9°R	8°R	2°R	6°R	HRU9°R HLU4°R HRD12°R HLD9°R HRU12°R HLU7°R	40dB B	DP (R) total

APPENDIX 14.

GROUP 5.

Case No	Diagnosis	Sex	Age	VSN	U	P	S	HR	HL	HD	Audiometry	Caloric
21	C.S.O.M. E L. facial palsy		16	no	-	-	4°L	4°L	-	-	60dB L conductive	-
28	Post op. R. Lab- rynthitis	M	16	to L	8°L	20°L	7°L	-	6°L	-	60dB R conductive	-
61	CSOM. R	F	36	no	-	-	4°R	5°R	3°R	4°R	55dB R conductive	-
67	L. Eust. tube deafness	F	47	no	-	-	2°L	3°L	-	-	25dB loss L. conductive	DP(L) 25%ES 30%D
95	CSOM. fistula sign R	M	45	no	-	-	-	2°R	-	2°R	70dB R 40dB L	Function R with Dundas Grant.
97	Post op Stapedect- omy R.	M	23	no	2°R	2°R	-	2°R	2°L	-	total deafness R.	Function L with Dundas Grant.
110	CSOM R.	M	48	no	1°R	-	2°R	-	-	-	80dB R conductive	Both respond with Dundas Grant.
123	L Petros- itis	F	59	no	-	-	-	-	-	-	60dB L conductive	Both respond with Dundas Grant.
124	Post op Stapedect- omy L	F	60	to L	-	-	-	-	-	9°R	40dB L conductive 60dB R	-
130	Post op Stapedect- omy L	F	70	no	8°R	5°R	7°R	6°R	11°R	6°R	50dB B conductive	Function L with Dundas Grant
132	CSOM L.	F	70	no	7°L	-	10°L	12°L	12°L	21°L	total deafness L	Function L with Dundas Grant.
137	Post op Labyrinth- itis L.	M	40	no	-	5°L	3°L	7°L	3°L	5°L	60dB L conductive	-

APPENDIX 15.

GROUP 6.

[illegible]

APPENDIX 16.

GROUP 7.

Case No	Diagnosis	Sex	Age	VSN	U	P	S	HR	HL	HD	Audiometry	Caloric
134	Hyst	M	38	no	-	-	-	-	1°L	2°L	N.A.D.	N.A.D.
135	Hyst	F	26	no	-	-	-	1°R	2°R	-	N.A.D.	DP (R) 36%ES 18%D
149	Hyst	F	57	no	-	-	-	-	-	-	N.A.D.	-
174	Hyst	F	26	no	-	-	-	-	-	-	N.A.D.	N.A.D.
199	V.U.O. Haemophilia	M	23	to R	14°R	-	30°L	26°R	30°R	20°R	50dB L no RCT.	no reaction either side.
118	V.U.O. Vit A. deficiency	M	14	no	-	7°R	-	1°R	5°R	7°R	40dB B RCT.	DP (R) 39%ES 0%D
2	V.U.O.	F	64	no	-	-	-	-	-	-	60dB R total L.	N.A.D.
20	V.U.O.	F	40	no	-	-	2°L	3°L	2°L	-	total R	DP (L) 23%ES 14%D
25	V.U.O.	F	56	no	-	-	-	-	3°L	2°L	30dB low tone B no RCT	CP (L) 40%ES 5%D
30	V.U.O.	F	45	no	2°R	-	3°R	-	-	4°R	N.A.D.	CP (L) 26%D 0%ES DP (R) 26%ES 0%D
35	V.U.O.	M	47	to L	2°L	4°L	5°L	-	7°L	10°L	total deafness R. N.A.D. L.	CP (R) 66%ES 40%D
45	V.U.O.	M.	67	no	-	-	4°R	-	-	9°R	40dB B. RCT L.	CP (L) 89%ES 59%D
53	V.U.O.	F	60	no	4°L	-	-	-	-	2°L	N.A.D.	CP (R) 26%ES 0%D DP (R) 44%ES 32%D
60	V.U.O.	F	36	no	-	-	-	4°L	-	2°L	N.A.D.	CP (R) 58%ES 29%D
73	V.U.O.	M.	20	no	-	-	-	-	-	-	50dB L	CP (L) 28%ES 8%D
80	V.U.O.	F	49	no	-	-	8°R	4°R	-	-	N.A.D.	DP (R) 12%ES 53%D
89	V.U.O.	M.	69	no	5°L	-	-	-	-	-	30dB R conductive	DP (L) 20%ES 0%D
90	V.U.O.	M	25	no	-	-	-	-	-	-	N.A.D.	DP (R) 18 ES 30% D
96	V.U.O.	F	42	no	-	-	-	-	-	-	N.A.D.	CP (L) total
103	V.U.O.	F.	32	no	-	-	3°L	-	3°L	-	N.A.D.	CP (L) 46%D 5%ES DP (R) 13%ES 30%D
122	V.U.O.	M	44	no	-	-	3°R	3°R	2°R	-	30dB B	CP (L) 52%D 56%ES
136	V.U.O.	M	58	no	5°L	-	2°R	3°R	-	-	N.A.D.	N.A.D.
139	V.U.O.	M.	32	no	-	-	-	-	-	-	N.A.D.	CP (R) 33%ES 0%D DP (R) 27%ES 6%D.
167	V.U.O.	M	42	no	-	4°R	4°R	-	-	-	N.A.D.	N.A.D.

APPENDIX 17

OCULAR NYSTAGMUS

<u>Case No.</u>	<u>Sex</u>	<u>Age</u>	<u>Lesion</u>
16	F	34	Unknown
91	M	42	Tubercular Chorio- Retinitis
99	F	36	Toxoplasmotic Chorio- Retinitis
111	F	49	Toxoplasmotic Chorio- Retinitis
181	M	14	Albino

BIBLIOGRAPHY

- ALEXANDER: quoted by JONGKEES, L.B.W. (1953).
- ANDERSEN, H.C. (1954): Acta oto-laryng. (Stockh.), 44, 568.
- ARSLAN, M., CAMBRELIN, G., CRABBE, F., HALLPIKE, C.S.,
PORTMANN, G., & VAN EGMOND, A.A.J. (1951): Rev. Laryng.
(Bordeaux), 72, 290.
- ASCHAN, G. (1948): Acta oto-laryng. (Stockh.), 34, 1.
- ASCHAN, G., NYLEN, C.O., STAHL, J. & WERSALL, R. (1952):
Acta oto-laryng. (Stockh.), 42, 451.
- ASCHAN, G. (1954): Acta oto-laryng. (Stockh.), suppl. 116.
- ASCHAN, G. & BERGSTEDT, M. (1955): Acta Soc. Med. upsalien.,
60, 1.
- ASCHAN, G. (1955): Acta Soc. Med. upsalien., 60, 77.
- ASCHAN, G. (1955): Acta Soc. Med. upsalien., 60, 99.
- ASCHAN, G. (1955): Acta Soc. Med. upsalien., 60, 123.
- ASCHAN, G., BERGSTEDT, M., & STAHL, J. (1956): Acta oto-
laryng (Stockh.) suppl. 129.
- ASCHAN, G., BERGSTEDT, M., DRETTNER, B., NYLEN, C.O. &
STAHL, J. (1957): Laryngoscope, 67, 884.
- BARANY, R. (1906): Mschr. Ohrenheilk., 40, 193.
- BARANY, R. (1907): Mschr. Ohrenheilk., 41, 477.
- BARANY, R. (1910): Wien. med. Wschr., 60, 2033.
- BARANY, R. (1911): Vehr. Ges. dtsh. Hals.-Nas.-u Ohrenarz.
37, 64.
- BARANY, R. (1921): Acta oto-laryng. (Stockh.), 2, 434.
- BARANY, R. (1926): Rev. oto-neuro-ophtal., 3, 551.
- BARLOW, J.S. (1962) Quart. rep. Electronics, No. 65.
- BARRE, M.J.S. (1937): Rev. Oto-neuro-ophtal., 15, 353.
- BARRE, M.J.S. (1938): Rev. Oto-neuro-ophtal., 16, 419.
- BARRENE, J.G.D. de, & KLEIJN, A. de (1923): Graefes Arch.
Ophthal., 111, 374.
- BARTELS, (1911): Ber. dtsh. ophthal. Ges., 20, 214.
- BAUER, J., & LEIDLER, R. (1912): Arb. Neurol. Inst. Univ.
Wien, 19, 155.
- BEGBIE, H. (1965): Personal Communications.
- BEHRMAN, W. (1936): Acta Soc. Med. upsalien., 42, 67.
- BENDER, M. (1955): Arch. Neurol. Psychiat. (Chicago), 73, 685.
- BERLIN. (1891): quoted by Dohlman, G. (1925).

- BERGSTED, M. (1961): Acta oto-laryng. (Stockh.), suppl. 165.
- BIEMOND, A. (1939): Proc. kon. med. Akad. Wet., 42, 370.
- BIEMOND, A. (1940): Proc. kon. med. Akad. Wet., 43, 901.
- BJORK, A. (1956): quoted by ASCHAN, G. et al (1956).
- BLOMBERG, L.H. (1959): Nord. Med., 62, 1009.
- BOER, E. de., CARELS, J. & PHILIPSZON, A.J. (1963): Acta oto-laryng. (Stockh.), 56, 457.
- BOIS-REYMOND, E. du (1849): quoted by JONGKEES L.B.W. & PHILIPSZON, A.J. (1964).
- BORRIES, G.V.T. (1922): Acta oto-laryng. (Stockh.), 4, 8.
- BOURDON, B. (1914): Ann. Psychol, 20, 1.
- BREUER, J. (1874): Med. Jahrb. Osterr. Staates, P. 72.
- BREUER, J. (1889): Pflugers Arch. ges. Physiol, 44, 135.
- BRODAL, A. (1964): Acta oto-laryng. (Stockh.), suppl. 192.
- BROWN, C. (1874): J. Anat. Physiol, 8, 327.
- BROWN-SEQUARD, C. (1860): Course of Lectures on the Physiology and Pathology of the Central Nervous System, Philadelphia. P. 187.
- BRUNNER, H. (1921): in ALEXANDER, G. & MARBURG, O. Handbuch der Neurologie des Ohres. Urban & Schwartzberg. Berlin. P. 239.
- BUYS, E. (1924): Rev. Oto-neuro-ophtal., 2, 641.
- BYFORD, G.H., HALLPIKE, C.S., & HOOD, J.D. (1952): Acta oto-laryng. (Stockh.), 42, 511.
- CANEGHEM, D. Van (1946): Bull. Soc. belge Otol. Lar. Rhin., 88.
- CARMICHAEL, E.A., DIX, M.R., & HALLPIKE, C.S. (1954): Brain, 77, 130.
- CATALANO, G.B., & MADONIA, T. (1957): Clin. otorinolaring, 9, 113.
- CAWTHORNE, T., FITZGERALD, G., & HALLPIKE, C.S. (1942): Brain, 65, 115.
- CAWTHORNE, T., & COBB, W.A. (1954): Acta oto-laryng. (Stockh.) 44, 580.
- CAWTHORNE, T., DIX, M.R., HALLPIKE, C.S., & HOOD, J.D. (1956): Brit. Med. Bull., 12, 131.
- CITRON, L., & HALLPIKE, C.S. (1956): J. Laryng., 70, 253.
- COHEN, L. (1961): J. Neurophysiol., 24, 1.
- COLLINS, W.E., & GUEDRY, F.E. (1962): Acta oto-laryng. (Stockh.), 54, 349.

- CRAWFORD, J. (1952): in *When Doctors Are Patients*. Pinner, M. Miller, B.F., Norton, New York.
- DELAGE, Y. (1888): *Arch. zool. Exp.*, 4, 535.
- DETLEFSEN, J.A. (1923): *Proc. Am. phil. Soc.*, 57, 292.
- DIETERLE, P. & MONNIER, M. (1955): *Pract. oto-rhino-laryng.* (Basel), 17, 164.
- DIX, M.R., HALLPIKE, C.S., & HARRISON, M.S. (1949): *Brain*, 72, 241.
- DIX, M.R. & HALLPIKE, C.S. (1952): *Proc. roy. Soc. Med.*, 45, 341.
- DODGE, R. & CLINE, T.S. (1901): *Psychol. Rev.*, 8, 145.
- DODGE, R. (1923): *J. exp. Psychol.*, 6, 1.
- DOHLMAN, G. (1925): *Acta oto-laryng.* (Stockh.), suppl. 5.
- DOHLMAN, G. (1929): *Acta oto-laryng.* (Stockh.), suppl. 8.
- DOHLMAN, G. (1954): quoted by HENRIKSSON, N.G. (1955).
- DOHLMAN, G. (1957): *L'appareil Vestibulaire*, P.U.F., 315.
- DOLOWITZ, D.A., FORSSMAN, B. & HENRIKSSON, N.G. (1962): *Acta oto-laryng.* (Stockh.), 55, 496.
- DUNLAP, K. (1925): *J. Comp. Psychol.*, 5, 485.
- DUNLAP, K. & DORCUS, R.M. (1926): *J. Comp. Psychol.*, 6, 329.
- EGMOND, A.A.J. van, & JONGKEES, L.B.W. (1948): *J. Laryng.*, 62, 447.
- EGMOND, A.A.J. van, GROEN, J.J. & JONGKEES, L.B.W. (1949): *J. Physiol.* (Lond.), 110, 1.
- EGMOND, A.A.J. van, & TOLK, J. (1954): *Acta oto-laryng.* (Stockh.), 44, 589.
- ENGSTROM, H. (1958): *Acta oto-laryng.* (Stockh.), 49, 109.
- EWALD, J.R. (1892): *Physiologische Untersuchungen über des Endorgan des Nervus Octavus*. Bergmann. Wiesbaden.
- FEARING, F. (1926): *J. comp. Psychol.*, 6, 115.
- FEARING, F. (1941): *J. comp. Psychol.*, 31, 47.
- FENN, W.O. & HURSCH, J.B. (1937): *Amer. J. Physiol.*, 118, 8.
- FERNANDEZ, C., ALZATE, R., & LINDSAY, J.R. (1959): *Ann. Otol.* (St. Louis), 68, 816.
- FERNANDEZ, C., ALZATE, R., & LINDSAY, J.R. (1960): *Ann. Otol.* (St. Louis), 69, 94.
- FISCHER, M.H. & WODAK, E. (1924): *Pfugers. Arch. ges. Physiol.*, 202, 553.
- FISCHER, J., & WOLFSON, L.E. (1943): *The Inner Ear*. Grune & Stratton. New York.
- FITZGERALD, G., & HALLPIKE, C.S. (1942): *Brain*, 65, 115.
- FLUUR, E. & ERIKSSON, L. (1961): *Acta oto-laryng.* (Stockh.), 53, 486.

- FLUUR, E. (1962): Acta oto-laryng. (Stockh.), 54, 181.
- FORD, F.R. & WALSH, F.B. (1936): John Hopk. Hosp. Bull., 58, 80.
- FORSSMAN, B. (1963): Acta oto-laryng. (Stockh.), 56, 1.
- FRENZEL, H. (1938): Z. Hals-Nasen-u. Ohrenheilk, 44, 347.
- FRENZEL, H. (1955): Spontan-und Provokations-nystagmus als Krankheitssymptom. Springer-Verlag. Berlin-Göttingen-Heidelberg.
- FUJIMORI, S. (1924): Klin. Wschr., 3, 885.
- FUKUDA, T. (1959): Acta oto-laryng. (Stockh.), 50, 95.
- GEMERT, A.G.M. van., DUYFF, J.W., FRANCOIS, P.R., TOOREN, T.N.A. van., VOORHOEVE, P.E. & ZWANN, P.J. (1954): Acta physiol. pharmacol. neerl., 3, 299.
- GLORIG, A. & MAURO, A. (1950): Ann. Otol. (St. Louis), 59, 146.
- GRAHE, K. (1920): Beitr. Anat. etc. Ohr., 15, 169.
- GRAHE, K. (1921): Beitr. Anat. etc. Ohr., 17, 251.
- GRANIT, R. (1955): Receptors and Sensory Perception, Yale University Press, New Haven.
- GRAYBIEL, A., CLARK, B., MCCORQUODALE, K. & HUPP, D.I. (1946): Amer. J. Psychol., 59, 259.
- GRAYBIEL, A. & PATTERSON, J.L. (1955): J. appl. Physiol., 7, 666.
- GRIFFITH, C.R. (1920a): J. expt. Psychol., 3, 15.
- GRIFFITH, C.R. (1920b): Am. Nat., 54, 524.
- GRIFFITH, C.R. (1924): J. comp Psychol., 4, 137.
- GROEN, J.J., LOWENSTEIN, O., & VENDRIK, A.J.H. (1952): J. Physiol. (Lond.) 117, 329.
- GROEN, J.J. (1953): quoted by HENRIKSSON, N.G. (1955).
- GOULD, H.N. (1926): J. comp. Neurol., 41, 223.
- GUTTICH, A. (1914): Beitr. Anat. etc. Ohr., 7, 1.
- HALLPIKE, C.S., HARRISON, M.S. & SLATER, E. (1951): Acta oto-laryng. (Stockh.), 39, 151.
- HALLPIKE, C.S. & HOOD, J.D. (1953): Proc. roy. Soc. B., 141, 216.
- HALLPIKE, C.S. (1955): Pract oto-rhine-laryng. (Basel), 17, 301.
- HALLPIKE, C.S., HOOD, J.D., & TRINDER, E. (1960): Confin. neurol. (Basel), 20, 232.
- HALSTEAD, W. (1935): The Effects of Cerebellar Lesions upon The Habituation of post rotational Nystagmus. Northwestern University Press. Chicago, Ill.

- HALSTEAD, W., YACORZYNSKI, G., & FEARING, F. (1937): Amer. J. Physiol., 120, 350.
- HAMERSMA, H. (1957): The Caloric Test. A Nystagmographical study. Thesis (Amsterdam).
- HARA, T., TOTSUKA, G., & SUZUKI, J. (1960): Yokohama med. Bull., 11, 407.
- HASEGAWA, T. (1925): Mschr. Ohrenheilk, 127, 19.
- HEMMES, G.D. (1924): Over Hereditairen Nystagmus. H. Veenman & Zonen. Wageningen. P. 105.
- HENNEBERT, P. (1946): Bull. Soc. belge Otol. Lar. Rhin, 86.
- HENRIKSSON, N.G. (1955): Acta oto-laryng., (Stockh.), 45, 25.
- HENRIKSSON, N.G. (1955): Acta oto-laryng., (Stockh.), 45, 120.
- HENRIKSSON, N.G. (1956): Acta oto-laryng., (Stockh.), Suppl. 125.
- HENRIKSSON, N.G., KOHUT, R. & FERNANDEZ, C. (1961): Acta oto-laryng. (Stockh.), 53, 333.
- HENRIKSSON, N.G., DOLOWITZ, D.A., & FORSSMAN, B. (1962): Acta oto-laryng., (Stockh.), 55, 33.
- HENRIKSSON, N.G. (1965): Personal Communications.
- HERTZ, H. & RISKAER, N. (1953): Arch. Otolaryng., 57, 648.
- HIRSCH, C. (1940): Ann. Otol. (St. Louis), 49, 232.
- HOFFMAN, A.C., WEILMAN, B., & CARMICHAEL, L. (1939): J. exp. Psychol., 24, 40.
- HOGYES, A. (1881): quoted by FISCHER, J.J. in The Labyrinth, Grune & Stratton. New York. London. 1956.
- HOLSOPPLE, J. Q. (1923): J. comp. Psychol., 3, 85.
- HOOD, J.D., & PFALTZ, C.R. (1954): J. Physiol. (Lond.), 124, 130.
- HUIZINGA, E. (1931): Pflugers Arch. ges. Physiol., 226, 709.
- HULK, J. & JONGKEES, L.B.W. (1948): J. Laryng., 62, 70.
- JONGKEES, L.B.W. & GROEN, J.J. (1946): J. Laryng., 61, 529.
- JONGKEES, L.B.W. (1948): Arch. Otolaryng., 48, 402.
- JONGKEES, L.B.W. (1948): Arch. Otolaryng., 48, 645.
- JONGKEES, L.B.W. (1949): Arch. Otolaryng., 49, 594.
- JONGKEES, L.B.W. (1949): J. Physiol. (Lond.), 110, 447.
- JONGKEES, L.B.W. (1953): Fortschr. Hals-Nas-Ohrenheilk, 1, 1. S. Karger. Basel.
- JONGKEES, L.B.W., SAAS, J.P.M. & PHILIPSZON, A.J. (1962): Pract. oto-rhino-laryng. (Basel), 24, 65.

- JONGKEES, L.B.W., & PHILIPSZOON, A.J. (1964): Acta oto-laryng. (Stockh.), suppl, 189.
- JOSEPH, J. & NIGHTINGALE, A. (1952): J. Physiol (Lond.), 117, 484.
- JUNG, R. (1939): Klin. Wschr., 18, 21.
- JUNG, R. & TONNIES, J.F. (1948): Klin. Wschr., 26, 513.
- KASER, O. (1942): Schweiz. med. Wschr., 72, 846.
- KELTON, I.W. & WRIGHT, R.D. (1949): Aust. J. exp. Biol. med. Sci., 27, 505.
- KESTENBAUM, A. (1948): Clinical Methods of Neuro-ophthalmologic Examination. Heinemann Medical Books Ltd. London.
- KLEIJN, A. de. & STORM. VAN LEEUWEN, W. (1921): Graefes Arch. Ophthal., 107, 109.
- KLEIJN, A. de. & VERSTEEGH, C.R.J. (1927): J. Laryng., 42, 649.
- KLEIJN, A. de. & VERSTEEGH, C.R.J. (1930): Acta oto-laryng. (Stockh.), 14, 356.
- KLIJN, J.A.J. & EK, J. (1959): Pract. oto-rhino-laryng. (Basel), 21, 391.
- KOBRACK, F. (1918): Beitr. Anat. etc. Ohr., 10, 214.
- KOCH, J. (1933): Arch. Ohr.,-Nas-u. Kelk.-Heilk., 135, 232.
- KOCH, H., HENRIKSSON, N.G., LUNDGREN, A. & ANDREN, G. (1959): Acta oto-laryng. (Stockh.), 50, 517.
- KOIKE, Y. (1959): Acta oto-laryng. (Stockh.), 50, 377.
- KORANYI, A. von & LOEB, J. (1891): Pflugers Arch. ges. Physiol., 48, 423.
- KUILMAN, J. (1958): Arch. Otolaryng., 67, 424.
- LANDSBERG, M.P. (1962): Ned. T. Geneesk., 106, 398.
- LANGE, C. (1939): Arch. Ohrenheilk., 146, 302.
- LAUDENBACH, J.P. (1899): J. Physiol. Path. gen., 1, 946.
- LEDoux, A. (1958): Acta oto-rhino-laryng. belge., 12, 1.
- LEKSELL, L. (1939): Acta chir. scand., 82, 262.
- LIDVALL, H.F. (1961): Acta oto-laryng. (Stockh.), 53, 33.
- LINDSAY, J.R. (1951): Ann. Otol. (St. Louis), 60, 1134.
- LINDSAY, J.R. & HEMENWAY, W.G. (1956): Ann. Otol. (St. Louis), 65, 692.
- LORENTE de NO, R. (1931): Ergebn. Physiol., 32, 99.
- LORENTE de NO, R. (1933): Laryngoscope, 43, 1.
- LOWENSTEIN, O. & SAND, A. (1940): J. Physiol. (Lond.), 99, 89.
- LOWENSTEIN, O. & ROBERTS, T.D.M. (1949): J. Physiol. (Lond.), 110, 392.

- MACH, E. (1875): Grundlinien der Lehre von den Bewegungsempfindungen. Engelmann. Leipzig.
- MAHONEY, J.L., HARLAN, W.L. & BICKFORD, R.G. (1957): Arch. Otolaryng., 66, 46.
- MARAN, A.G.D. (1963): J. Laryng., 77, 1032.
- MARX, E. & TRENDELENBURG, W. (1911): Z. Sinnesphysiol., 45, 87.
- MAXWELL, S.S. (1920): J. gen. Physiol., 3, 157.
- MAXWELL, S.S., BURKE, U.L. & RESTON, C. (1922): Amer. J. Physiol., 58, 432.
- MAXWELL, S.S. (1923): Labyrinth and Equilibrium; Lippincott: London.
- MCCABE, B.F. (1960): Trans. Amer. Acad. Ophthal. Otolaryng., 64, 264.
- MCCOUCH, G.P., DEERING, I.D. & LING, T.H. (1951): J. Neurophysiol., 14, 191.
- McLAY, K., MADIGAN, M.F., & ORMEROD, F.C. (1957): Ann. Otol. (St. Louis), 66, 473.
- McLAY, K., MADIGAN, M.F., & ORMEROD, F.C. (1958): J. Laryng., 72, 48.
- McLAY, K., (1965): Personal Communications.
- MEYERS, I.L. (1929): Arch. Neurol. Psychiat. (Chicago), 21, 901.
- MILES, W.R. (1939): Proc. natn. Acad. Sci. U.S.A., 25, 25.
- MILES, W.R. (1940): Science, 91, 456.
- MITTERMAIER, R. (1935): Der. Nervenarzt, 8, 459.
- MITTERMAIER, R. (1950): Z. Hals-Nasen-u. Ohrenheilk., 29, 477.
- MITTERMAIER, R. (1954): Acta oto-laryng. (Stockh.), 44, 574.
- MITTERMAIER, R. & CHRISTIAN, W. (1954): Z. Hals. Nasen-u. Ohrenheilk., 33, 20.
- MITTERMAIER, R. (1955): Pract. oto-rhino-laryng. (Basel), 17, 179.
- MONTANDON, P. & MONNIER, M. (1964): Brain, 87, 673.
- MOTTA, G., BERGER, G.P. von & BARAVELLI, P. (1957): Boll. Soc. ital. Biol. Sper. 33, 58.
- MOWRER, O.H. (1934): Comp. Psychol. Monogr., No. 45.
- MOWRER, O.H. (1934): Arch. Otolaryng., 19, 46.
- MOWRER, O.H., RUCH, T.C. & MILLER, N.E. (1935): Amer. J. Physiol., 114, 423.
- NETTLESHIP, E. (1911): Trans. ophthal. Soc. U.K., 31, 159.
- NYLEN, C.O. (1922): Acta oto-laryng. (Stockh.), 3, 502.
- NYLEN, C.O. (1926): Acta oto-laryng. (Stockh.), 9, 325.

- NYLEN, C.O. (1927): Acta oto-laryng. (Stockh.), 11, 147.
- NYLEN, C.O. (1929): Acta oto-laryng. (Stockh.), 13, 302.
- NYLEN, C.O. (1931): Acta oto-laryng. (Stockh.), Suppl. 15.
- NYLEN, C.O. (1950): J. Laryng., 64, 295.
- NYMAN, H. (1945): Acta oto-laryng. (Stockh.), Suppl. 60.
- OHM, J. (1914): Z. Augen heilk., 32, 4.
- OHM, J. (1936): Proc. Acad. Sci. Amst., 39, 549.
- OHM, J. (1939): Z. Hals-Nas.-u. Ohrenheilk., 45, 22.
- ORMA, E.J. (1957): Acta physiol. scand., 38, 288.
- PEITERSEN, E. (1963): Arch. Otolaryng., 78, 59.
- PEITERSEN, E. (1963): Arch. Otolaryng., 78, 642.
- PEITERSEN, E. (1963): Arch. Otolaryng., 78, 797.
- PEITERSEN, E. (1964): Arch. Otolaryng., 79, 481.
- PEITERSEN, E. (1965): Personal Communications.
- PFALTZ, C.R. & RICHTER, R. (1956): Pract. oto-rhino-laryng. (Basel), 18, 263.
- PLANCK, M. (1936): The Philosophy of Physics. Allen & Unwin. London.
- POLLOCK, L.J. & DAVIS, L. (1927): Brain, 50, 277.
- POWSNER, E.R. & LION, K.S. (1950): Electronics, 23, 96.
- PREBER, L. (1958): Acta oto-laryng. (Stockh.), suppl. 144.
- PURKINJE (1853): quoted by JONGKEES, L.B.W. (1953).
- RADEMAKER, G.G.J. & BRAAK, J.W.G. TER (1948): Brain, 71, 48.
- RASMUSSEN, G.L. (1946): J. comp. Neurol., 84, 141.
- RASMUSSEN, G.L. & GACEK, R.R. (1958): Anat. Rec., 130, 361.
- RIESCO-MAC-CLURE, J.S. (1957): Rev. Otorrinolaring., 17, 42.
- RIESCO-MAC-CLURE, J.S. (1964): Ann. Otol. (St. Louis), 73, 829.
- ROSS, D.A. (1936): J. Physiol. (Lond.), 86, 117.
- RUDING, J.H.H. (1953): Int. Congr. Otol. P. 725.
- SAMUEL, E. (1965): Personal Communications.
- SCHMALTZ, G. (1932): Proc. roy. Soc. Med., 25, 359.
- SCHOTT, E. (1922): Dtsch. Arch. Klin. Med., 140, 79.
- SEIFERTH, L.B. (1937): Arch. Ohr.-Nas.-u. Kehlk.-Heilk., 168, 19.
- SMITH, K. & BRIDGMAN, M. (1943): J. exp. Psychol., 33, 165.
- SPIEGEL, E.A. & ARONSON, L. (1933): Arch. Otolaryng., 17, 311.
- STAHL, J. (1958): Acta oto-laryng. (Stockh.), Suppl. 137.
- STEIN, S. von (1904): Physiologische Russe, 3, 1.

- STEINHAUSEN, W. (1931): Pflugers Arch. ges. Physiol., 228, 322.
- SUZUKI, J.I. (1961): Acta oto-laryng. (Stockh.), 53, 381.
- TALPIS, L. (1927): Arch. Ohr.-Nas.-u. Kehlk.-Heilk., 116, 255.
- TERRACOL, J. (1927): Rev. Oto-neuro-ophtal., 5, 963.
- THIELEMAN, M.B. (1924): Beitr. Anat. etc. Ohr., 20, 213.
- THOMSEN, K.A. (1953): Acta oto-laryng (Stockh.), 109, 189.
- THULIN, C. (1953): Acta physiol. Scand., 28, Suppl. 103.
- TOROK, N., GUILLEMIN, V. & BARNOTHY, J.M. (1951): Ann. Otol. (St. Louis), 60, 917.
- TOROK, N. (1960): Confin. neurol. (Basel), 20, 266.
- TOROK, N. & NYKIEL, F. (1962): Acta oto-laryng. (Stockh.), 54, 363.
- TRAVIS, R.C. & DODGE, R. (1928): Physcho. Monogr., 38, 1.
- TSCHIRREN & VIESINGER (1947): quoted by MILOJEVIC, B. (1965), Laryngoscope, 75, 243.
- UNTERBERGER, S. (1938): Arch. Ohr.-Nas.-u. Kehlk.-Heilk., 145, 478.
- VEITS, C. (1931): Z. Hals-Nasen-u. Ohrenheilk., 29, 368.
- VISSER, S.L. (1962): Ned. T. Geneesk., 106, 2139.
- VOGEL, K. (1929): Dtsch. med. Wschr., 55, 268.
- VOOGD, J. (1946): Nederl. keel-neus-, en oorheilk. vereenig., 79, 3.
- VRIES, H.I. de. (1956): Prog. Biophys. biophys. Chem., 6, 208.
- WALSH, E.G. (1960): J. Physiol. (Lond.), 153, 350.
- WALSH, E.G. (1961): J. Physiol. (Lond.), 155, 506.
- WENDT, G.R. (1951): In STEVENS, S.S., Handbook of
of Experimental Psychology. John Wiley & Sons Inc. New York.
P.1191.
- WERSALL, J. (1956): Acta oto-laryng, (Stockh.), Suppl. 126.
- WINSTON, J., LEWEY, F.H., PARENTEAU, A., MARDEN, P.A. &
CRAMER, F.B. (1948): Ann. Otol. (St. Louis), 57, 738.
- WODAK, E. & FISCHER, M.H. (1922): Munch. med. Wschr., 69, 193.
- WODAK, E. (1957): Pract. oto-rhino-laryng. (Basel), 19, 587.
- ZILSTORFF-PEDERSEN, K. & PEITERSEN, E. (1963): Arch. Otolaryng., 77, 237.
- ZILSTORFF-PEDERSEN, K. & PEITERSEN, E. (1963): Arch. Otolaryng., 77, 353.
- ZILSTORFF-PEDERSEN, K. & PEITERSEN, E. (1963): Arch. Otolaryng., 77, 586.
- ZILSTORFF-PEDERSEN, K. & PEITERSEN, E. (1964): Arch. Otolaryng., 79, 600.