PAROXYSMAL POSITIONAL VERTIGO

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ABSTRACT

Two hundred and fifty five patients (Group I) were considered as suffering from paroxysmal positional vertigo because: (a) they gave a typical history or, (b) an attack was elicited by Hallpike's maneuver, as modified by Stenger, during the clinical examination. Most patients developed Type I nystagmus during the attack in contrast to the incidence reported by other authors (Stahle et al, 1965). This difference is discussed. No pathognomonic electronystagmographic (ENG) findings were obtained except when the actual attack was recorded in a small number of cases. In most patients no apparent cause of their syndrome and no other vestibular abnormalities as manifested by post-caloric labyrinthine preponderance were detected. These patients were classified as idiopathic and their age distribution raises the possibility that the syndrome may be part of the ageing process in this group. In four cases the syndrome coexisted with intracranial tumors. Cause-effect relationship is doubtful in all of them. Subsequently, in 11 patients (Group II) the nystagmus during the attack was observed and recorded by ENG in different gaze directions. Discrepancies between observed and recorded nystagmus are discussed. Further research is necessary to clarify a number of questions which arise from this study.

RESUME

On a constaté que 255 patients (Groupe I) avaient éprouvé de vertige de position paroxystique car: (a) l'anamnèse était typique ou (b) pendant l'examen clinique, une attaque était provoquée par la manoeuvre de Hallpike, selon la modification de Stenger. La plupart des patients ont manifesté le nystagmus de Type I pendant l'attaque, ce qui ne correspond pas avec les cas que d'autres auteurs ont rapportées (Stahle et al, 1965). Les raisons de cette différence sont discutées. L'électronystagmographie (ENG) n'a pas donné des résultats pathognomoniques qu'à l'exception de quelques cas, où une attaque actuelle était enregistrée. Chez la plupart des patients, on n'a pas trouvé d'autres troubles vestibulaires, manifestés par une préponderance labyrinthique post-calorique, ainsi que la cause de leur syndrome. Ces patients étaient désignés comme idiopathiques et leur distribution d'âge rend possible le fait que le syndrome de ce groupe est un trait de l'âge avancé. Quatre patients avaient en même temps des tumeurs intracrâniens. C'est bien possible qu'il n'y avait pas de relation entre les deux conditions. Dans la suite, chez 11 patients (Groupe II), on a observé et enregistré par ENG le nystagmus pendant l'attaque dans de directions de regard différentes. Les divergences entre le nystagmus observé et enregistré sont discutées. Des recherches supplémentaires sont nécessaires pour répondre à un nombre des questions posées par cette étude.

To

Doctor William J. McNally

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CHAPTER ONE

INTRODUCTION

Since the initiation of a special Clinic for Dizziness at the Royal Victoria Hospital, Montreal, Canada, late in 1973 it has become increasingly evident that paroxysmal positional vertigo is a frequent syndrome. This is a clinical syndrome characterized by recurring attacks of vertigo of acute onset and short duration, provoked by special head positions. These provocative head positions occur when the patient bends over, when he turns his head up or more frequently, when he lies in bed or when he turns from side to side in bed, or when he tries to get up from bed. Although the attack does not last for more than a few seconds, it is frequently violent and for most of the patients it is a frightening experience.

The attacks can be provoked over periods of time followed by remissions which occasionally may last even for years. On examination an attack may be elicited by a sequence of body positions which has been described by a number of authors (see Clinical Examination).

Barany (1921) was probably the first to describe this peculiar syndrome when he wrote "Until now it was not possible to diagnose a lesion of the otolith. I was recently able to make this diagnosis..... This was a twenty seven year old woman with headaches for one year. Hearing, caloric responses and

neurologic evaluation were normal. For the last fourteen days she had suffered from spells of vertigo. My assistant, Dr. Karlefors, noticed that these spells occurred only when the patient lay on her right side. She also showed a strong rotatory nystagmus to the right with a vertical component beating upwards. This nystagmus became purely rotatory on gaze to the right and purely vertical on gaze to the left. The attack lasted for about half a minute and was accompanied by strong vertigo and nausea. If immediately after the attack the patient turned her head to the right there was no new spell. To have another attack the patient had to lie on her back or her left side for a while. These observations are not new. Oppenheim, Burns and I have observed similar vertigo and nystagmus attacks for many years".

It was not until 1952 when Dix and Hallpike described more cases of paroxysmal positional vertigo and called this condition "Positional Nystagmus of the Benign Paroxysmal Type".

Since then many different terms have been used to define the syndrome including: postural vertigo (De Weese, 1952); paroxysmal postural vertigo (Lindsay, 1967); benign positional vertigo (Harrison, 1966); paroxysmal positional nystagmus (Stahle et al, 1965); paroxysmal positional vertigo (Preber et al, 1957, Gacek, 1974); benign paroxysmal positional nystagmus (Harbert, 1970); positional vertigo (Schuknecht, 1962); postural dizziness (Orma et al, 1957); positioning vertigo (Kornhuber, 1974) and cupulolithiasis (Schuknecht, 1969).

The syndrome may be confused with other syndromes causing positional vertigo and nystagmus. For example, as early as 1913 Barany wrote, presenting a case of multiple sclerosis: "The new observation, which I would like to present to you, consists of the fact that the patient shows little, if any, nystagmus with the eyes in midposition or gaze to the left (and the head in straight position). By tilting the head to the right, even when this is performed slowly, or when the patient rolls over slowly to the right from the straight supine position, intense horizontal and slightly rotatory nystagmus appears, which persists unchanged as long as the head is maintained in this position".

Occasionally a few authors give definitions which may not be in agreement with what has been defined above. Harrison et al (1975) and Dayal et al (1974, 1977) seem to call "paroxysmal positional nystagmus" nystagmus which intermittently appears in different head positions during electronystagmographic (ENG) recordings.

The etiology of paroxysmal positional vertigo has been a matter of controversy in the literature. As early as 1921, Barany attributed this syndrome to otolith dysfunction. He thought the rotatory nystagmus observed in his patient (see above) during the paroxysmal positional vertigo attack was eye counter-rolling (Gegenrollung). From earlier animal experiments by Magnus, it was already known that eye counter-rolling is a mani-

festation of otolith stimulation. "Therefore," Barany concluded, "further research on eye counter-rolling presents a renewed interst". Other authors reported cases of paroxysmal positional vertigo where the syndrome was thought to be caused by brainstem pathology (Barber et al, 1971; Rubin, 1973). McCabe (1975) reported cases with whiplash injury in which the clinical picture was similar to paroxysmal positional vertigo.

Schuknecht (1969) was the first to introduce the term cupulolithiasis and on the basis of experimental and clinical data he postulated that paroxysmal positional vertigo attack is generated by stimulation of the posterior semicircular canal from deposits derived from otoconia fallen off from the otoliths.

The present study confirms the hypothesis that the paroxysmal positional vertigo attack is generated by the activation of the posterior semicircular canal.

In the Dizziness Clinic, Royal Victoria Hospital, Montreal, the patients evaluated are a highly selected group. A number of them are in- and out-patients from the Montreal Neurological Hospital which is a large referral centre. Some other patients are in-patients of the Royal Victoria Hospital; others are referred by specialists or family physicians. There are very few patients who come to the Clinic without being referred. Therefore, most of the patients have already had their symptom "dizziness" screened by a physician. In this patients' population

paroxysmal positional vertigo is a frequent syndrome. It is estimated that for the time period mentioned below about 30% of the dizzy seen in the Clinic suffered from paroxysmal positional vertigo.

Between January, 1974 and January, 1977, 255 patients (Group I) with paroxysmal positional vertigo were evaluated. Subsequently, in eleven patients (Group II) the nystagmus of the paroxysmal positional vertigo attack was studied by direct observation and electronystagmographic (ENG) recording.

In this thesis, after a review of the literature, these data are presented and discussed.

CHAPTER TWO

LITERATURE REVIEW

CLINICAL FEATURES

History

The patient complains of the sudden onset of attacks of vertigo, frequently violent, which occur after he lies supine in bed, when he turns from side to side in bed, or when he looks upwards or bends forward (Dix and Hallpike, 1952; Schuknecht, 1962; Barber, 1964; Schuknecht, 1969; Schuknecht, 1974). There are no prodromal symptoms.

History reveals the following important points (Dix and Hallpike, 1952; Citron et al, 1956; Harbert, 1970; Drachman et al, 1972; Hart, 1973; Barber, 1973; Barber et al, 1973; Schuknecht et al, 1973; Rubin, 1973; Rubin et al, 1974; Schuknecht, 1975; Barber, 1975; Jongkees, 1975; Barber et al, 1976):-

1. Nausea and occasionally vomiting may accompany the attack (Harrison, 1966). These symptoms may persist for quite sometime after the attack is over.
2. There is a latent period, a few seconds elapsing from the time the head assumes the provocative position until the nystagmus and vertigo appear.
3. The nystagmus and vertigo do not last for more than thirty seconds.

4. These symptoms show marked fatiguability, their intensity diminishing on successive attempts to assume the vertigo-provoking head position.
5. There are intermittent periods, from days to months, when attacks can be provoked and remissions may last several years.
6. The attacks are accompanied by rotatory vertigo or disequilibrium which occasionally may be severe.
7. There may be associated conditions present (Table II) but the patient rarely feels that there is a cause-effect relationship with these conditions.

Clinical Examination

The nystagmus of the attack is mainly rotatory with a linear component which has been described as vertical, horizontal or oblique (Jung et al, 1964), and which depends upon the direction of gaze, being mainly rotatory on gaze towards the nystagmus fast phase direction and mainly linear on gaze in the opposite direction (Barany, 1921; Harbert, 1970).

When the patient assumes the sitting position or turns over to the other side immediately after an attack, vertigo and nystagmus may appear again after a few seconds but now the nystagmus beats in the opposite direction.

Positional testing to elicit an attack has been called Hallpike's (Barber et al, 1976), Cawthorne's (Hart, 1973) or Nylen and Barany's (Drachman et al, 1972). The patient sits on the examining table in a position such that when he lies down,

his head is over the edge of the table. From the sitting position he is brought comfortably but quickly to the supine position. The head is hyperextended to about 45° and the neck is rotated about 45° towards the examiner's side (Fig. 1). In this position the eyes are observed for one minute either with or without Frenzel's glasses. Then the patient is returned to the straight sitting position and the eyes are observed for another minute. The same sequence is then followed for the head-hanging position towards the opposite side. Some authors (Stahle et al, 1965; Stenger, 1957) also suggest examination in the supine position with the head hanging straight back.

Types of Paroxysmal Positional Vertigo Attack

Using such positioning tests, Stahle and Terins (1965) distinguished four types of "paroxysmal positional nystagmus" in their eightycases:

I. The Dix-Hallpike Type (26 cases). The attack is elicited in only one lateral head-hanging position and the nystagmus beats towards the lowermost ear. When the sitting position is assumed immediately after the attack is over, vertigo and nystagmus reappear frequently within a few seconds but the nystagmus beats towards the opposite direction.

II. The Stenger Type (Stenger, 1957) (14 cases). This is similar to Type I but the attack is elicited when from the sitting position the patient assumes the supine position with the head hyperextended straight back.

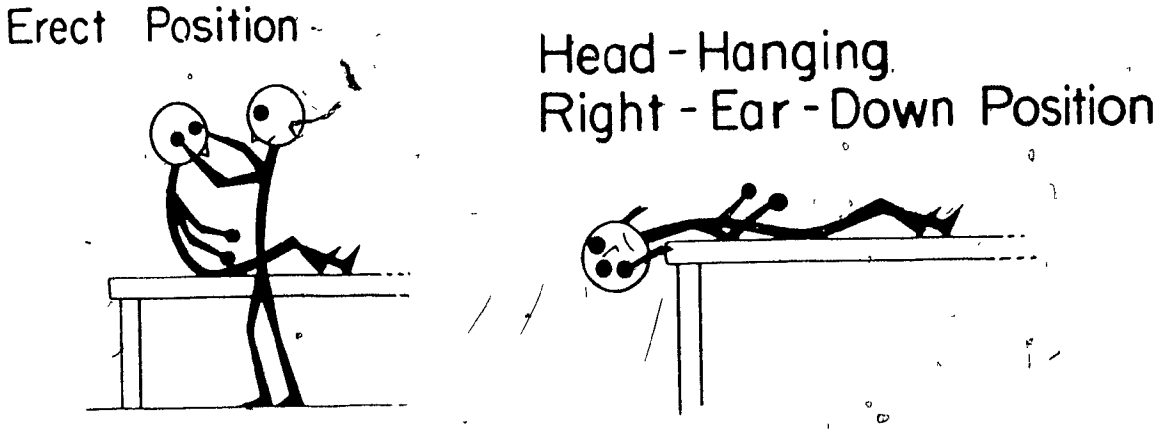

Fig. 1. Hallpike's positioning (from Schuknecht, 1975).

III. The Divergent Type (26 cases). The attack occurs in both lateral head-hanging positions when assumed from the supine straight position and the nystagmus beats towards the lowermost ear. In some cases the attack recurs when the patient returns to the supine or sitting position.

IV. The Convergent Type (12 cases). The nystagmus beats away from the lowermost ear and is elicited in one or both lateral head-hanging positions.

Two of their cases did not fit any of the above four types. One was a case of Meniere's disease which showed paroxysmal positional vertigo alternating between Type III and Type IV. The other showed a basilar artery deformity with attacks of alternating right beating and left beating nystagmus occurring on the right lateral head position.

Electronystagmography

Electronystagmography (ENG) has shown in some cases that after the initial attack fades away the nystagmus reverses itself while the head is still in the vertigo position. This nystagmus is less intense and lasts longer than the initial attack and has been called "nystagmus of the secondary period" by Stahle and Terins (1965) although no explanation for its occurrence was offered. Barber et al (1976), using electronystagmographic techniques, noted that the direction of the recorded nystagmus during the attack is frequently opposite to that observed.

Occurrence

Dix and Hallpike (1952) found both sexes to be equally affected by paroxysmal positional vertigo. Barber (1964, 1973) reported it to be more common in females in their fifth decade of life. Lindsay (1967) found that it occurs most frequently after sixty years of age.

PATHOPHYSIOLOGY AND ETIOLOGY

The pathophysiology and etiology of paroxysmal positional vertigo have provoked much controversy in the literature (McNally et al, 1967). At times different structures or neural connections have been implicated as causing paroxysmal positional vertigo. Under the headings (a) the otolith, (b) the neck, (c) the brainstem, and (d) the semicircular canal, the available data are briefly discussed.

(a) The Otolith Organs

Barany (see Introduction) thought paroxysmal positional vertigo was due to an otolith disturbance. Dix and Hallpike (1952) were of the same opinion and described a patient who died of a brainstem glioma with histopathological evidence of utricular degeneration. Citron and Hallpike (1956) reported two cases treated by labyrinthectomy and one (1962) by intracranial VIII nerve section without recurrence of paroxysmal positional vertigo after the operation, despite which they raised the question whether the syndrome could be due to a disturbance of the central

otolith connections, at least, in some cases. Cawthorne and Hallpike (1957) presented a patient with utricular degeneration who also had small carcinomatous metastases in the cerebellum. Lindsay and Hemensway (1956) reported patients with sudden partial loss of vestibular function some of who subsequently developed symptoms compatible with paroxysmal positional vertigo; one showed histopathologic changes in Scarpa's ganglion and the nerves to the utricle, superior and horizontal semicircular canals on the affected side.

Despite these views expressed in the literature is there really evidence to support the idea that stimulation of the otolith is capable of generating such a violent vertigo attack and intense nystagmus?

The otolith organs (utricle and saccule) are sensors of linear acceleration and gravity. Tait and McNally (1934) made extensive studies through ablative procedures on the contribution of the otolith organs to frog's posture. It was obvious from their experiments that there is direct interaction between the otoliths and the semicircular canals.

What is the influence of the otolith signal upon the oculomotor system? In 1955, McNally wrote "The utricle has been shown to exert dominant influence over labyrinthine tonic reflexes, labyrinthine righting reflexes and labyrinthine compensatory eye movements. The best controlled experiments in which stimulation or ablation was strictly confined to the utricle

have not elicited eye nystagmus". However, it has early been recognized (Barany, 1921) and subsequent data supported the idea (Cohen, 1974) that utricular stimulation produces eye counter-rolling. Electric stimulation of the utricular nerve in cats activates extra ocular muscles (Suzuki et al, 1969; Tokumasu et al, 1971). Gernarndt (1970) was able to generate nystagmus in cats and monkeys by direct stimulation of the utricle. Fluur et al (1970a,b, 1971) were able to demonstrate in cats that electric stimulation of different areas of the utricle and saccule generated specific eye deviations. Experimental animals and humans exposed to linear acceleration responded by generating nystagmus-like eye movements (McCabe, 1964; Jongkees, 1967). Fluur et al, (1973a,b,c; 1974a,b) in a series of experiments on cats observed that nystagmus generated by sectioning one ampullary nerve was inhibited or enhanced depending upon whether the head was tilted away or towards the side of the lesion and on whether utricular function was present or absent. This was attributed to the otolith influence upon the semicircular canal nystagmus. It was thought that there is a close inter-relationship between the semicircular canals and the otolith organs. Furthermore, the same authors (Fluur et al, 1973d) concluded that utricular impulses generated either by sectioning one utricular nerve or by exerting pressure on the utricular macula (Owada et al, 1960) elicited nystagmus only by enhancing or inhibiting the function of the semicircular canals. Fluur (1973) gave the same explanation for the observation that

when patients with vestibular neuronitis and nystagmus beating away from the side of the lesion were placed with the head tilted towards the healthy ear inhibited their nystagmus and when placed with their head towards the site of the lesion enhanced their nystagmus.

Positional nystagmus due to alcohol ingestion has been described by many authors (Suzuki, 1972; Cohen, 1974). Positional alcohol nystagmus (PAN) manifests itself in two types - Type I with the fast phase beating towards the earth (geotropic) and Type II, the secondary phase of Type I, during which the fast phase beats away from the earth (apogeotropic). The site of origin of PAN is still a matter of controversy. There are arguments supporting the view that the semicircular canal is responsible for PAN (Money et al, 1974) and arguments that PAN arises in the otolith (Oosterveld, 1973). It is of interest (Odkvist, 1975) that in volunteers tested under increased gravitational forces (2-3g), after ingestion of alcohol, previously subsided PAN II reappears. Furthermore, some of the volunteers showed PAN II under increased gravitational load even without alcohol intake.

A number of otolith mathematical models have been devised (Young et al, 1967). In his model Young tried to take into account data showing that sustained tilt angle produces sustained steady otolith output. Mayne (1974) devised a model which combines the semicircular canal and otolith signals for the

determination of the vertical and the computation of linear velocity and displacement. According to his mathematical concept the data need central processing in order that information on the vertical and linear velocity and displacement be accurate. It is interesting that, according to the model, direction-fixed positional nystagmus "would probably point to central pathology".

This brief discussion shows that it is rather unlikely that otolith disturbances may be responsible for the nystagmus of the paroxysmal positional vertigo attacks. It is obvious that whenever nystagmus eye movements are generated by destruction or stimulation of otolith structures or nerves, they are not similar to this high velocity, fast fading nystagmus of the paroxysmal positional vertigo attack. Whenever intense nystagmus appears as a sequel of otolith activation it is from modification of nystagmus generated by one of the semicircular canals.

(b) The Neck (Torsion)

It has been repeatedly discussed in the literature whether whiplash injuries may cause positional nystagmus (Barber, 1964; Rubin, 1973). McCabe (1975) described two cases of whiplash injury with vertigo and bursts of high velocity slow phase nystagmus of short duration on head turning. He thought there was a cause-effect relationship between whiplash injury and vertigo. He called this vertigo, cervical. Van de Calseyde et al (1977) gave a number of normal individuals and patients with

different neck problems what they called "a neck torsion test". During the test, the individual under examination was sitting on a chair with his head fixed by a bitemporal fork. The chair could be rotated in both directions by hand. The body rotation could go up to 65° laterally. The nystagmus response to this neck torsion was from 1.2°/sec. to 4.1°/sec. with a mean of 2.7°/sec. of slow phase velocity for normals and from 0.94°/sec. to 6.79°/sec. with a mean of 3.18°/sec. of slow phase velocity for patients with neck problems. They concluded that although "neck torsion nystagmus" was more frequent in patients with neck problems, there were no striking differences between the groups of normals and patients. Is spinal input to the vestibulo-oculomotor system able to generate vertigo and nystagmus similar to the paroxysmal positional vertigo attack?

Spinovestibular afferents have been described by different authors (Pompeiano, 1972, Brodal, 1974). There are also neck afferents to the flocculus (Wilson, 1975a) where there is an interaction between labyrinthine, visual and neck inputs (Wilson, 1975b). Cervical input also influences the vestibulo-ocular reflex of the abducens motoneurons (Hisosaka, 1973). Dichgans et al (1974) studied the importance of neck afferents in monkeys during active eye-head turning. They showed that after bilateral labyrinthectomy one of the compensatory mechanisms was the increase in gain of the neck loop. Igarashi et al (1972) found no positional nystagmus after sectioning or blocking by local medication cervical dorsal roots in monkeys. However,

they observed a modification of the post-rotatory and opto-kinetic nystagmus.

It is obvious that eye movements due to neck afferent signals, either experimental or clinical, are possible. However, the generated eye movements are dissimilar from that of the paroxysmal positional vertigo attack. The same applies to cases of whiplash injuries. The two cases reported by McCabe (1975) are the only exception. It is interesting to note that both patients had typical histories of paroxysmal positional vertigo!

(c) The Brainstem (Dysfunction)

It is of interest to note that the two first cases reported of paroxysmal positional vertigo and utricular degeneration also showed brainstem pathology (Dix and Hallpike, 1952; Cawthorne et al, 1957). Cawthorne and Hinchcliffe (1961) reported six patients with subtentorial tumors who had what they called "nystagmus of the central type". This nystagmus appears as soon as the head assumes the provocative position and continues as long as the head position is maintained. Vertigo is only infrequently severe. None of these patients had symptoms typical of paroxysmal positional vertigo. A similar case was earlier described by Barany (see Introduction).

Barber (1975) did not encounter paroxysmal positional vertigo in his patients with acoustic neuroma but noted its occurrence in five patients with vertebro-brasilar insufficiency

(Barber et al, 1971). Rubin (1973, 1974) reported two cases of acoustic neuroma and two with "other central pathology" with symptoms of paroxysmal positional vertigo. Stahle and Terins (1965) reported five cases of paroxysmal positional vertigo attributed to central pathology. Harrison and Ozsakinoglu (1975) reported cases of paroxysmal positional vertigo due to central pathology, so did Dayal et al (1974, 1977). These authors, however, give a different definition for "paroxysmal positional nystagmus" (see Introduction).

It is well known that paroxysmal positional vertigo may appear after head injuries with or without fractures and with or without manifestations of other vestibular disorders (Gordon, 1954; Preber, 1957; Barber, 1964; Harrison, 1966). It is obvious that head injury does not necessarily mean brain-stem injury.

Is there any experimental evidence that brainstem dysfunction or stimulation gives nystagmus similar to that observed during the paroxysmal positional vertigo attack?

Nystagmus has been produced experimentally in animals with lesions affecting the vestibular nuclei (Uemura et al, 1973) and lesions or stimulation of different areas of the cerebellum (Cohen et al, 1969; Ron et al, 1973), but this nystagmus did not fulfill the criteria for paroxysmal positional vertigo. Fernandez et al (1960a, 1960b) were able to produce positional nystagmus in cats by ablating the nodulus of the cerebellum but the nystagmus differed from that of paroxysmal positional vertigo

in that it was direction-fixed regardless of positions, was mainly vertical and occasionally rotatory and, in some cases, showed a crescendo-decay but was present as long as the head position was maintained. In some cats it showed a long latency period (50 sec.) and lasted from 30 to 180 seconds and was compensated in all animals in ten days (except two where it took twenty days). Finally it did not recur although some animals were kept alive for seventy days. At this point it is unlikely that brainstem neurons would be able to produce the full picture of paroxysmal positional vertigo.

(d) The Semicircular Canal

Two lines of evidence point towards the posterior semicircular canal as the origin of paroxysmal positional vertigo: (1) Schucknecht's cupulolithiasis theory discussed below and (2) the similarity of the eye movements in paroxysmal positional vertigo to those produced by stimulation of the posterior semicircular canal.

Barany (1921) was the first to recognize that when the eyes were deviated towards the lowermost ear the nystagmus was rotatory and when deviated to the opposite direction, the nystagmus was vertical during the paroxysmal positional vertigo attack. This observation was confirmed by other authors, such as Harbert (1970), who by using neurophysiologic data and his own observations on forty patients tried to explain the nystagmus generated during the paroxysmal positional vertigo attack and he

postulated that the posterior semicircular canal was probably responsible for this type of eye movements.

Through a three neuron, bisynaptic pathway, the semicircular canals activate the extraocular muscles (Szentagothai, 1964). Each semicircular canal activates specific oculomotor muscles so that the eye movement caused by semicircular canal electrical stimulation is characteristic of this particular canal. More specifically, stimulation of the superior (anterior) semicircular canal activates mainly the ipsilateral superior rectus and the contralateral inferior oblique muscles. This results in an upward deviation of the ipsilateral eye and a rotatory movement of the contralateral eye. The horizontal (lateral) semicircular canal activates, mainly, the ipsilateral medial rectus and the contralateral lateral rectus muscles. This results in a conjugate deviation of the eyes, in the horizontal plane, away from the stimulated canal. The posterior semicircular canal mainly activates the ipsilateral superior oblique and the contralateral inferior rectus muscles. This results in a rotatory movement for the ipsilateral eye and a downward deviation of the contralateral eye (Cohen et al, 1964; Cohen et al, 1965; Cohen et al, 1966). Furthermore, the initial position of the eye in the orbit influences the movement to be described under the electrical stimulation of the particular semicircular canal. Fig. 2 shows the movements of the left eye when the left posterior semicircular canal is stimulated (Cohen, 1966). When the eye is deviated away from the stimulated side the compensating eye movement is almost purely vertical. As the eye turns towards

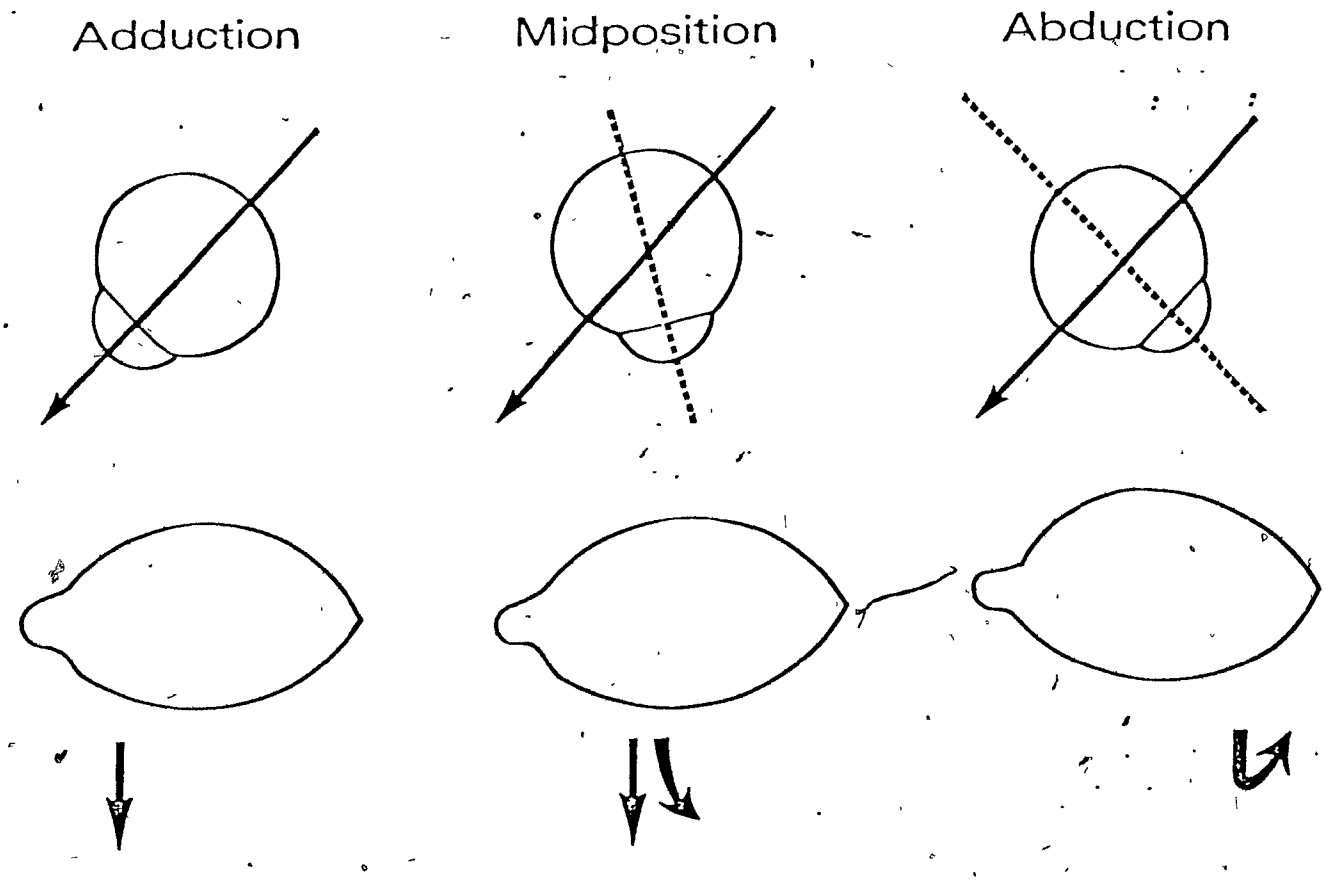

Fig. 2. Compensatory left eye movements resulting from left posterior semicircular canal electrical stimulation.

Top row shows left eye seen from above and bottom row as seen from in front. Solid line represents plane of posterior semicircular canal and dashed line that of visual axis. The lines coincide when the eye is adducted. Direction of movement indicated by arrows (from Coehn et al, 1966).

the stimulated side, a rotatory component appears and purely rotatory movement occurs when the eye is directed to the stimulated side. In other words, when the optic axis of the eye coincides with the direction of action of the muscle, in this particular case the superior oblique muscle, the eye movement will be almost purely vertical. As the eye moves from the adducted towards the abducted position, the optic axis becomes more and more nearly perpendicular to the direction of the action of the muscle, and the eye movement becomes increasingly rotatory.

When the two posterior semicircular canals are simultaneously stimulated the oblique muscles are totally or partially inhibited. Both inferior recti muscles are strongly activated and the eyes deviate downwards. When the anterior semicircular canal on one side and the posterior canal on the other side are simultaneously electrically stimulated the superior and inferior recti muscles are partially or totally inhibited and the eyes describe a purely rotatory movement. When the two anterior canals are simultaneously stimulated all four obliques are partially or totally inhibited and the eyes deviate upwards. In all these combinations the eyes move conjugately. Finally, if all three semicircular canals on the same side are simultaneously stimulated the superior and inferior recti muscles are partially or totally inhibited and the eyes describe a horizonal-rotatory movement in direction opposite to the stimulated side by the activation of ipsilateral medial rectus and superior oblique and contralateral lateral rectus and inferior oblique muscles (Cohen, 1964).

During all these semicircular canal stimulations in isolation or in combination other oculomotor muscles are activated but this is weaker than the activation of muscles described above (Lorente de No, 1932).

From this discussion it is obvious that the eye movements generated by the posterior semicircular canal are similar to those generated during the paroxysmal positional vertigo attack. What activates the posterior semicircular canal during the attack?

Schuknecht (1969, 1973, 1974, 1975) introduced the term "cupulolithiasis" and on the basis of clinical and experimental data he postulated that the posterior semicircular canal of the lowermost ear in the provocative head position, stimulated by deposits of otoconia or material probably deriving from disintegrated otoconia, is responsible for the paroxysmal positional vertigo attack.

Lim (1973) found that the mammalian otoconium is composed of an organic matrix and calcium carbonate. He also postulated that the vestibular dark cells which are supposed to be endolymph-secreting, seem to be capable of removing calcium from otoconia which have been attached on their surfaces. It is possible that these dark cells participate in a process of eliminating dislodged otoconia.

In the erect head position the posterior semicircular

canal is the most dependent structure of the labyrinth (Fig. 3). It is possible that in this position free floating material, heavier than the endolymph, accumulates in the ampulla of the posterior semicircular canal. In the provocative head position, the posterior canal assumes a more superior position, the gravitational force on those deposits is altered and, therefore, the degree of deflection of the cupula changes. Gacek (1974) was able to control the attacks of paroxysmal positional vertigo in a number of patients by transection of the posterior ampullary nerve.

Schuknecht (1973) thought the causes of paroxysmal positional vertigo to be labyrinthine concussion due to head blows, chronic otitis media, post-stapedectomy, occlusion of the anterior vestibular artery and degenerative changes of ageing. Can cupulolithiasis explain the excitation of the posterior semicircular canal during the paroxysmal positional vertigo attack?

The semicircular canals are sensors of angular acceleration. Their hydrodynamics are expressed by a second order differential equation described by Steinhausen and Van Egmond (Mayne, 1974). It has been shown (Melvill-Jones, 1972) that within a certain range of frequency, the semicircular canal signal, which reaches the brainstem, corresponds to that of head angular velocity. Malcolm et al (1970) introduced a modification of the Steinhausen model in an attempt to include the short term adaptation which takes place during the semicircular canal cupula stimulation process. The mean cupular restoration time constant

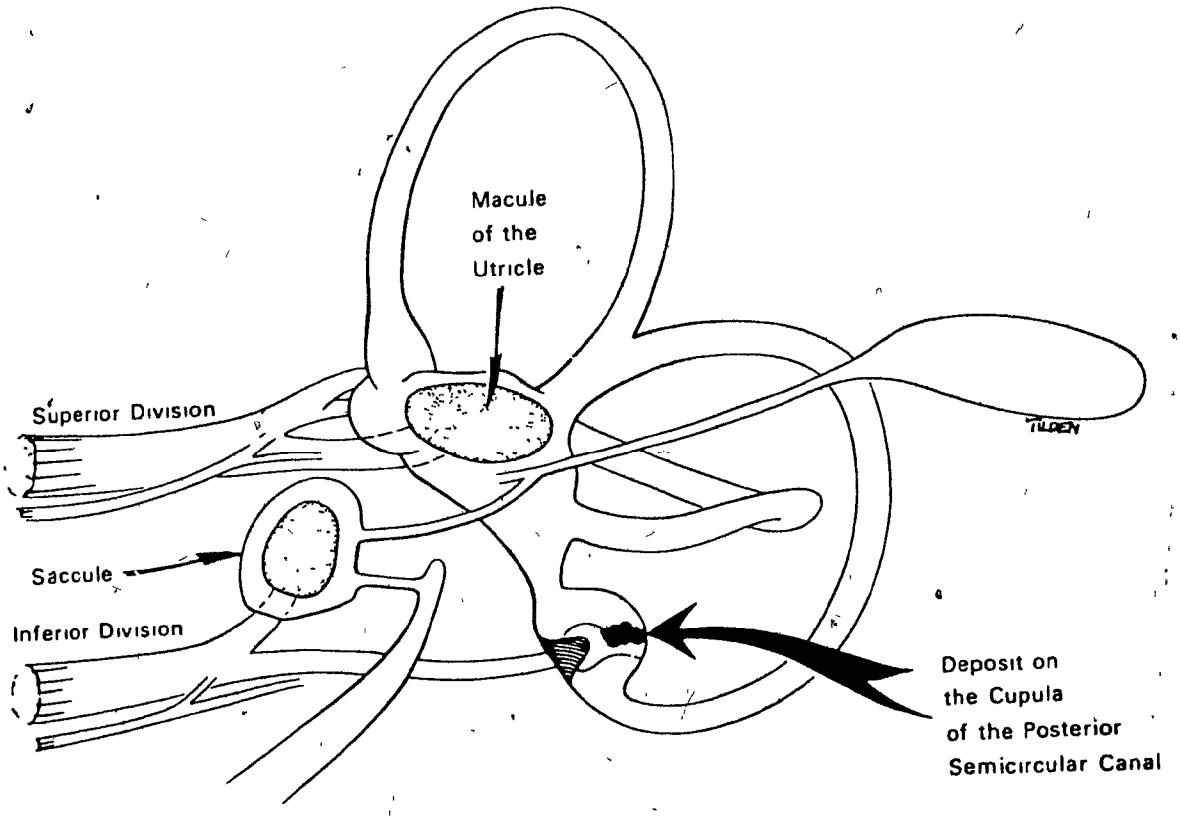

Fig. 3. The position of the labyrinth in an approximate erect head position (from Schuknecht, 1975).

for their healthy subjects was found to be 21 sec. and the mean adaptation time constant, 82 sec. Recently, Demers (1975) showed that continuous ear canal irrigation with water of constant other than the body temperature results in nystagmus which can be sustained for 45 min. Under these conditions one may assume that there is a maintained cupula deflection. This is in agreement with the explanation given by Money et al (1974) for the appearance of positional alcoholic nystagmus. These authors theorize that when alcohol diffuses into the endolymph the specific gravity changes and the cupula becomes more dense than the endolymph. When the head is tilted towards one side the cupula is deflected by gravity and stays deflected until the alcohol is eliminated from the endolymph.

It has been recently shown (Gonshor et al, 1976; Melvill-Jones et al, 1976) that the vestibulo-ocular reflex (VOR) has powerful adaptive capabilities to the point that under specific vision reversal conditions, the VOR may reverse.

From this discussion it is obvious that there are some points which support Schuknecht's theory:-

1. The eye movements generated during the paroxysmal positional vertigo attack are compatible with posterior semicircular canal activation.
2. Lim's observations on degeneration and resorption of otoconia.
3. The fact that Gacek was able to control the attacks in some patients by sectioning the posterior ampullary nerve.

4. The fact that the posterior semicircular canal is the most dependent structure of the labyrinth in the upright position.
5. The time course of the attack is compatible with cupula dynamics.

However, there are some points regarding the behaviour of these deposits in the ampulla of the posterior semicircular canal which are not explicitly explained by this theory -

1. If these deposits sit on the cupula in the erect head position (Fig. 3), there would be a maintained utriculofugal cupula deflection. This being the direction of normal excitation of the posterior canal (see Semicircular Canal, McNally et al, 1967) there should be a sustained nystagmus compatible with the excitation of that canal (Demers, 1975). In the provocative head position these deposits would be dislodged, the cupula would be restored and the nystagmus fast phase would be opposite to the nystagmus produced during the excitation of the canal.
2. If the deposits do not sit on the cupula in the erect head position then in the provocative head position they would fall on the cupula in a utriculofugal way and the cupula would be deflected as during the excitation of the canal. The generated nystagmus would be similar to that generated during the activation of the posterior canal. However, as the deposits would sit on the cupula producing a maintained deflection, the nystagmus would last as long as the head stays in the provocative position.

It is difficult to explain what happens during the paroxysmal positional vertigo attack with either assumption. Recently McClure et al (1977) divided their cases of paroxysmal positional vertigo in cupulolithiasis and idiopathic on the basis of cupula deflection time constant measurements according to Malcolm's (1970) model. However, these authors measured the responses of the lateral semicircular canal which, according to what has been discussed above, is not responsible for the paroxysmal positional vertigo attack. Also, they did not take into account the central adaptational processes (Page 23) which are very likely to come into play. Therefore, although changes of the time constant of the cupula of the affected semicircular canal are possible these authors' measurements seem to be incorrect.

TREATMENT

For most patients the rational treatment is reassurance and an explanation of how to avoid the provocative head position (Schuknecht, 1974). In rare, severe, intractable cases, labyrinthectomy (Citron et al, 1956) VIII nerve (Citron et al, 1962) or posterior ampullary nerve section (Gacek, 1974) have been reported to control the attacks. Occasionally antivertiginous medication may be needed for the first few days of the attacks. Some authors (Barber, 1973) advise their patients to go repeatedly through the head positions which trigger the attack every day, in the hope that this will shorten the time period over which the attacks may occur.

CHAPTER THREE

MATERIAL AND METHODS

Between January, 1974 and January, 1977 in the Dizziness Clinic of the Royal Victoria Hospital, Montreal, 255 patients (Group I), 80 males and 175 females, with paroxysmal positional vertigo were evaluated. All but 12 patients were referred by other physicians and significantly a large number had undergone thorough neurological evaluation.

This study generated a number of questions. In an attempt to find some details as to the type of nystagmus of the attack a second study was carried out during which the nystagmus of the paroxysmal positional vertigo attack was observed and recorded by ENG in different gaze directions in 11 patients (Group II). These patients were evaluated after January, 1977 and were selected out of a large group of patients suffering from this syndrome for the cool approach to their problem and their willingness to fully cooperate during the distressful paroxysmal positional vertigo attack. They all fulfilled the same criteria as the first group of patients.

A patient was considered to suffer from paroxysmal positional vertigo if:-

(a) He gave a history of episodic short-duration vertigo occurring when lying or turning from side to side in bed, when getting up

from bed, on hyperextending the head or on bending over or any combination of these circumstances, or

(b) A typical attack was elicited during the initial or subsequent examinations.

(a) History

During the first Clinic visit a thorough history was taken throughout which the following points were discussed -

1. Through one or two leading questions the patient was left to volunteer information.
2. The type of sensation he experienced; whether he felt that there was any rotatory sensation or not; if not, then what the sensation was; if yes, then whether he was turning around or the surroundings were turning around him; also, the direction of turning, whether vertical or horizontal and if horizontal, whether from right to left or vice versa.
3. The duration of the actual attack. This point always takes time to clarify for any kind of vertiginous patient. Frequently the patient confuses the duration of the attack with the duration of the after-effects which may be a sensation of rotation with slower velocity, unsteadiness or just light-headedness. These symptoms are occasionally accompanied by nausea which, however, is not as severe as during the actual attack.
4. The special circumstances which triggered the attack, as to body (head) position or special head movements.
5. The frequency of attacks, whether they occurred intermittently

or everyday; whether this was the first time or whether the patient experienced the same vertigo before.

6. An intriguing point was to find out whether the patient experienced other types of vertigo in the near or distant past and in which way this vertigo was different from his present problem.
7. The possibility of concomitant symptoms with special emphasis on ear problems such as hearing loss, tinnitus, or sensation of blockage in the ear.
8. Other medical problems and the medication the patient was taking, if any.
9. The patient's occupation and whether he was a driver.

(b) Clinical Examination

All 266 patients underwent complete neuro-otological examination by the author and 73 had neuro-ophthalmological evaluation by Dr. T. Kirkham, Neuro-ophthalmologist, Montreal Neurological Hospital. The neuro-otological examination consisted of an examination of the ears, nose and throat; the cranial nerves (from III to XII); the search for nystagmus in five eye positions: midposition, gaze to right, gaze to left, gaze upwards and gaze downwards; the testing of cerebellar function consisting of the search for dysdiadochokinesis or incoordination for all four extremities; the search for disequilibrium as manifested by the Romberg test and tandem gait with eyes open and closed. Hearing acuity was tested by three tuning forks (1024Hz, 2048Hz, 4096Hz).

Initially positional testing using Hallpike's maneuver (Page 7) was performed only for the right and left head-hanging positions. Later the examination also included the straight head-hanging position so that 171 out of 266 patients were examined for all three head-hanging positions. In all instances, except as specified otherwise (see Results), the patient was requested to keep his eyes on the examiner's finger, somewhere in midposition, during the attack and the eyes were observed without Frenzel's glasses and in a fully lighted examining room. Before Hallpike's maneuver was performed, detailed instructions and explanations were always given to patients as to what to do and what to expect from this type of examination. Subsequently to an unsuccessful Hallpike's maneuver, we invariably asked the patient to simulate the movements which triggered an attack in his everyday life.

(c) Audiologic Evaluation

An audiologic examination was performed for 190 out of 266 patients. This consisted of a pure tone threshold audiogram for air and bone conduction and speech reception threshold (SRT) and speech discrimination scores. If necessary, special tests were performed: Bekesy tracing, tone decay, recruitement tests and tympanometry.

(d) Vestibular Tests

An ENG was undertaken in 190 out of 266 patients. In our Laboratory horizontal and vertical eye movements were recorded using Beckman miniature silver-silver chloride skin electrodes. After AC amplification with a time constant of three seconds and a band width of 30Hz, records were made on a standard chart recorder. Calibration of the recording, which also gave the performance of the eyes during horizontal saccadic movements, was carried out by instructing the patient to follow with his eyes two successively flicking lights, the first in the midline at the primary position, the second at 10° to the right. This was followed by a similar process with the second light flicking at 10° to the left. The same sequence was followed for lights flicking at 10° above and 10° below the midposition. The vertical eye movements were recorded from the right eye and the horizontal eye movements through bitemporal leads. If appropriate pathology was suspected the horizontal eye movements were recorded separately for each eye.

Positional nystagmus (eyes closed) was recorded in nine head positions: in the caloric test position with head straight, tilted to the left, then to the right. The same sequence was followed in the sitting position and the supine position with 30° hyperextension. In all positions the patient's body (head) was positioned slowly by the ENG technician.

For caloric stimulation, water of 30°C and 44°C was used from baths with automatically controlled temperatures. The irrigations were of 20 seconds duration. As post-caloric response, we measured the velocity of the slow phase of nystagmus at the peak of the response in degrees per second with eyes closed. The bithermal caloric response which produced up to 20% directional or labyrinthine preponderance was considered as borderline normal. The test for the visual suppression of the post-caloric nystagmus was performed, for both cold calorics around 100 sec. from the beginning of the irrigation. At this point the patient was advised to open his eyes and fixate on a light in midposition.

In the 11 patients, Group II, the nystagmus of the attack was recorded by ENG with eyes open, the same recording technique was followed. Initially, the ENG recording was calibrated as described above. Then the patient was tested for gaze nystagmus with his eyes in midposition, 30° to the left and 30° to the right in the horizontal plane; 10° up and 10° down in the vertical plane. Next he was tested for positional nystagmus, eyes closed, in the caloric test position with his head straight only. Then he was given detailed instructions as to what to do during the attack and very slowly he was brought to the straight sitting position and subsequently, through the Hallpike's maneuver, to the provocative head-hanging position. He was asked immediately to fix his eyes on the examiner's finger in a gaze deviation of approximately 30°-40° towards the lowermost ear or in opposite

direction. In about the middle of the attack he was asked to turn his eyes, following quickly the examiner's finger, towards the uppermost ear in about the same gaze deviation. In the second gaze position the ENG recording continued till the nystagmus clinically disappeared. After a few seconds the patient was brought fast, as in Hallpike's maneuver, to the sitting position with his eyes fixed in mid-position. The ENG recording continued till the nystagmus clinically disappeared. After a few minutes of rest, an ENG recording was performed similar to that for the patients in Group I.

In three out of 11 patients the eye movements were recorded by ENG separately for each eye for the horizontal leads during the paroxysmal positional vertigo attack. In the remaining nine patients the horizontal eye movements were recorded through bitemporal leads.

All the patients were advised to refrain from using anti-vertiginous medications, central acting drugs, such as diazepam, barbiturates, etc. and alcohol at least 48 hours prior to the ENG examination.

(e) Other Tests

A number of patients were referred for skull x-rays or tomograms of the internal acoustic meatus when this was thought to be necessary.

RESULTS

GROUP I.

(a) History

The great majority of patients had their first attack either as soon as they lay down in bed at night or when they tried to get up in the morning. A few woke up in the middle of the night vertiginous as they tried to turn from side to side in bed. Some had their first attack during the day when, because of their occupation, they tried to hyperextend their heads (housewives checking high placed shelves) or bend over (mechanics, construction workers, etc.). Invariably they described their experience as frightening. They all had the impression that this was a sequel of a serious disease. Some thought they had brain tumors; other that they experienced a heart attack; others that they experienced a severe fainting spell, or epilepsy. Most of them could not understand why they felt better during the day at their regular occupations and the vertigo recurred every time they tried to sit down and relax or when they went to bed at night. Most of them volunteered explanations related either to other diseases they knew they suffered from or life situations obviously unrelated to their problem and its etiology. Some patients felt quite unsteady or nauseated and a few vomited during the first one or two days of the period when attacks could be elicited and a few had to stay in bed, but the great majority were able to carry on with their regular activities. Most patients knew that the vertigo lasted less than one minute but others thought the vertigo

attack was much longer until it was provento them during examination that this was not really the case. Some patients knew the initial provocative head position of the vertigo but most thought the vertigo occurred when they turned either way. Some thought the provocative head position was in the direction opposite to the real one. A number of patients thought the supine was the provocative position. Hallpike's position showed the provocative was one of the lateral head positions in most of the patients. Most of them complained of lightheadedness during the period of attacks. This sensation became worse when they turned the head around quickly.

Only 38 out of 255 patients were seen during their first period of attacks. All the others had experienced the same vertigo previously with remissions between attacks ranging from a few days to several years.

All patients who had experienced the vertigo before knew they were undergoing periods of remission of variable duration between periods when attacks could be elicited. Most of the patients seen during the first period of attacks reported that their vertigo was rapidly subsiding with the exception of one patient (See below).

(b) Clinical Examination

In 105 out of 255 patients the vertigo attack was not elicited by positional testing during the initial examination.

Sixty-three of these 105 patients returned when the vertigo recurred, at which time the Hallpike positioning test elicited the attack in 48 patients. In the remaining 15 patients positioning did not elicit any attack and in this group some noticed that their attacks occurred over a period of only a few hours.

The results of Hallpike's positioning test are summarized in Table I.

Two patients were able to generate an attack by themselves while Hallpike's positioning was unable to do so. One patient lay on his side quickly from the sitting position without twisting his neck. The attack was elicited only on one side and on the following sitting position. The other patient lay flat on his back and then he turned his head fast to the left then to the right, then to the left side again. Both patients were from the idiopathic group (See below).

All patients who experienced an attack of paroxysmal positional vertigo during the positioning test complained of vertigo on return to the sitting position; most of them considered this vertigo less severe. A number of them developed nystagmus, which was invariably in the opposite direction and less intense than the nystagmus in the head-hanging position. The paroxysmal positional vertigo attack was elicited with the affected ear in the lowermost position in all cases where we were able to establish and localize the cause.

TABLE I.

Results of Hallpike's positioning test

(255 Cases)

Type		No. of Patients
I	(Dix, Hallpike)	188
II	(Stenger)	2
III	(Divergent)	5
IV	(Convergent)	3
Attack not elicited		57

All patients showed a latent period of 2-5 sec. before the appearance of the attack in the provocative head position. In all patients the attack lasted from 5-30 sec. approximately. In all patients there was a strong rotatory component of the nystagmus during the attack. All patients examined on subsequent recurrences developed the attack in the same provocative head position and the nystagmus generated had the same direction.

All patients examined during a period when the attacks could be elicited, showed a degree of unsteadiness which was more obvious on tandem gait with eyes closed. Horizontal nystagmus with the fast phase towards the affected side on gaze to this direction was present only in patients suffering from severe attacks. Gaze nystagmus with the fast phase away from the affected side was suspected in a small number of patients. None of the patients showed gaze nystagmus in more than one direction.

(c) Etiology

When the clinical and laboratory data were correlated for all 255 patients it was possible to determine a causative mechanism in a number of patients. These possible causes are summarized in Table II.

Head Injury (43 cases)

In this group 12 patients only had skull fractures. Only eight showed post-head injury partial hearing loss. None

TABLE II

The most apparent causes of paroxysmal positional vertigo in 255 patients

Head injury	43
Vestibular neuronitis	5
Otosclerosis (no surgery)	2
Post-stapedectomy	2
Chronic otitis media	5
Sudden hearing loss	8
Menière's disease	3
Paroxysmal vertigo in childhood	1
Sudden inner ear pressure change	4
Vertebro-basilar insufficiency	2
Intracranial tumours	4
Immediate post-operative period*	3
Systemic degenerative disease	4
Idiopathic	169

*Other than ear surgery

of the patients showed bilateral hearing loss. In 21 patients the bithermal caloric stimulation gave symmetrical responses. Nine patients showed post-caloric directional preponderance; seven patients showed post-caloric labyrinthine preponderance and in 6 patients no ENG recording was performed. No correlation was established between the severity of head injury and paroxysmal positional vertigo. The first attack occurred a few hours after the head injury in 17 cases; a few days to a few months post-head injury in 12 cases; years later in six cases (in two cases, 5 years later). Obviously in the latter cases it was not possible to establish a cause-effect relationship. Finally, in 8 cases the interval between the head injury and the onset of paroxysmal positional vertigo was not established.

In all cases where there was hearing and/or vestibular function loss as manifested by post-caloric labyrinthine preponderance towards the other side, the paroxysmal positional vertigo attack was elicited with the affected ear in the lowermost position.

In four cases an attack was not elicited. Two cases showed a Type III attack; 37 cases showed a Type I attack. In all patients with immediate post-head injury onset of paroxysmal positional vertigo, who returned for follow up, a tendency for less severe attacks and shorter subsequent periods of attacks was noted. The youngest patient was 14 years of age, the oldest 81 years of age. Fig. 4 shows the age distribution of all 43 cases.

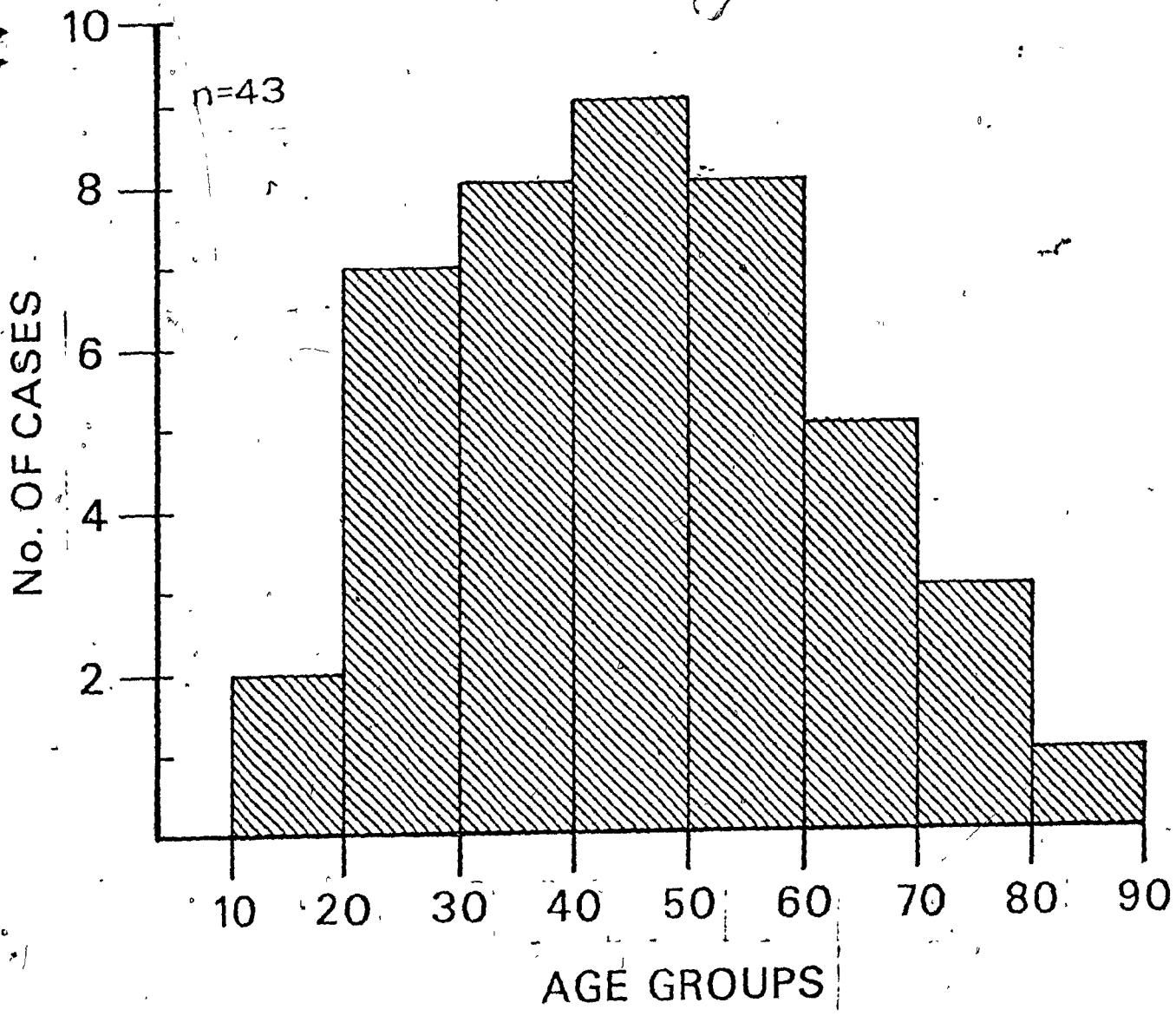

Fig. 4. Age distribution of patients with head injury.

Vestibular Neuronitis (5 Cases)

In all cases paroxysmal positional vertigo occurred years after vestibular neuronitis. Three patients recalled clearly that a few years previously they had experienced a type of vertigo which was different from that currently experienced. Two patients needed closer questionning to reveal that once in the past they had experienced a longstanding vertigo attack.

Otosclerosis - No Surgery (2 Cases)

It was interesting to note that although both patients had bilateral otosclerosis their paroxysmal positional vertigo was unilateral.

Post-Stapedectomy (2 Cases)

In these cases the first attack occurred two and four years respectively post-stapedectomy and both retained an excellent post-operative hearing result. Both were bilateral cases of otosclerosis operated only in one ear. The attacks occurred when the operated ear was in the lowermost position.

Chronic Otitis Media (5 Cases - 2 Post-Mastoidectomy)

Two patients had dry ears, three had intermittently discharging ears. There was no correlation between discharge and appearance of paroxysmal positional vertigo attacks.

Sudden Hearing Loss (8 Cases)

All patients were fully worked up despite the fact that all lost their hearing more than a year prior to onset of paroxysmal positional vertigo. No apparent cause was established for the hearing loss. Three had partial vestibular function loss in addition. In the latter cases a vascular problem was suspected.

Meniere's Disease (3 Cases)

One patient noticed the presence of paroxysmal positional vertigo as soon as he started to recover from his first Meniere's disease attack. The other two experienced the first attack years after the onset of Meneire's disease. It is interesting to note that in the latter two cases an examination of the tests combined with a superficial history taking could lead to the erroneous impression that Meniere's disease had recurred.

Paroxysmal Vertigo in Childhood (1 Case)

This patient in his mid-30's had a typical history of paroxysmal vertigo in childhood, a disease which subsided in his teens. For a number of years he did not experience any vertigo attacks. A few months prior to initial examination he suffered from recurrent vertigo with a typical history of paroxysmal positional vertigo. On examination the attack was elicited with the ear in the lowermost position which showed substantial vestibular

function loss on bithermal caloric stimulation. The remainder of the work-up revealed no further pathology.

Sudden Inner Ear Pressure Change (4 Cases)

These patients developed paroxysmal positional vertigo immediately after the following circumstances:- one patient, on landing from a flight, one after strenuous exercise, one immediately after diving and one following childbirth. None of these patients had any hearing or other vestibular deficiencies.

Vertebrobasilar Insufficiency (2 Cases)

One patient was clinical diagnosed, the other had a vertebral angiogram. We were not able to establish any correlation between paroxysmal positional vertigo and symptoms of vertebro-basilar insufficiency.

Intracrnaial Tumors (4 Cases)

1. The first patient had a temporal lobe glioma diagnosed and treated by radiotherapy about seven years prior to our initial examination. She developed paroxysmal positional vertigo six weeks prior to examination. On examination we were able to elicit an attack.
2. The second patient had a craniotomy for removal of an optic nerve glioma five years prior to initial examination. She developed paroxysmal positional vertigo four weeks prior to initial

examination. Over the last two years she had been examined repeatedly, and the vertigo was always present. The attack was elicited with the ear in the lowermost position which had very little vestibular function left. Further work up did not reveal any other pathology.

3. The third patient had a craniotomy for a successful removal of a meningioma ten years prior to initial examination. She developed paroxysmal positional vertigo over one year prior to initial examination with long remissions between periods of attacks. On examination the attack was easily elicited.
4. The fourth patient developed paroxysmal positional vertigo three months prior to initial examination. The periods of attacks were intermittent but she developed a slowly progressive unsteadiness which never subsided. Her clinical examination revealed no pathology except right beating nystagmus on right lateral gaze, mild bilateral hearing loss for the high tones, paroxysmal positional vertigo in the right head-hanging position with nystagmus having a strong rotatory component and unsteadiness on tandem gait disporportionate to the rest of the clinical picture. The audiogram confirmed the clinical impression. The ENG showed low amplitude right beating nystagmus, dysmetria on refixation saccades, symmetrical bithermal caloric responses and bilateral failure of suppressing the post-caloric nystagmus by fixation. The CT-Scan (Fig. 5) showed a mass in the posterior fossa on the right side. A craniotomy showed this tumor to be meningioma compressing the

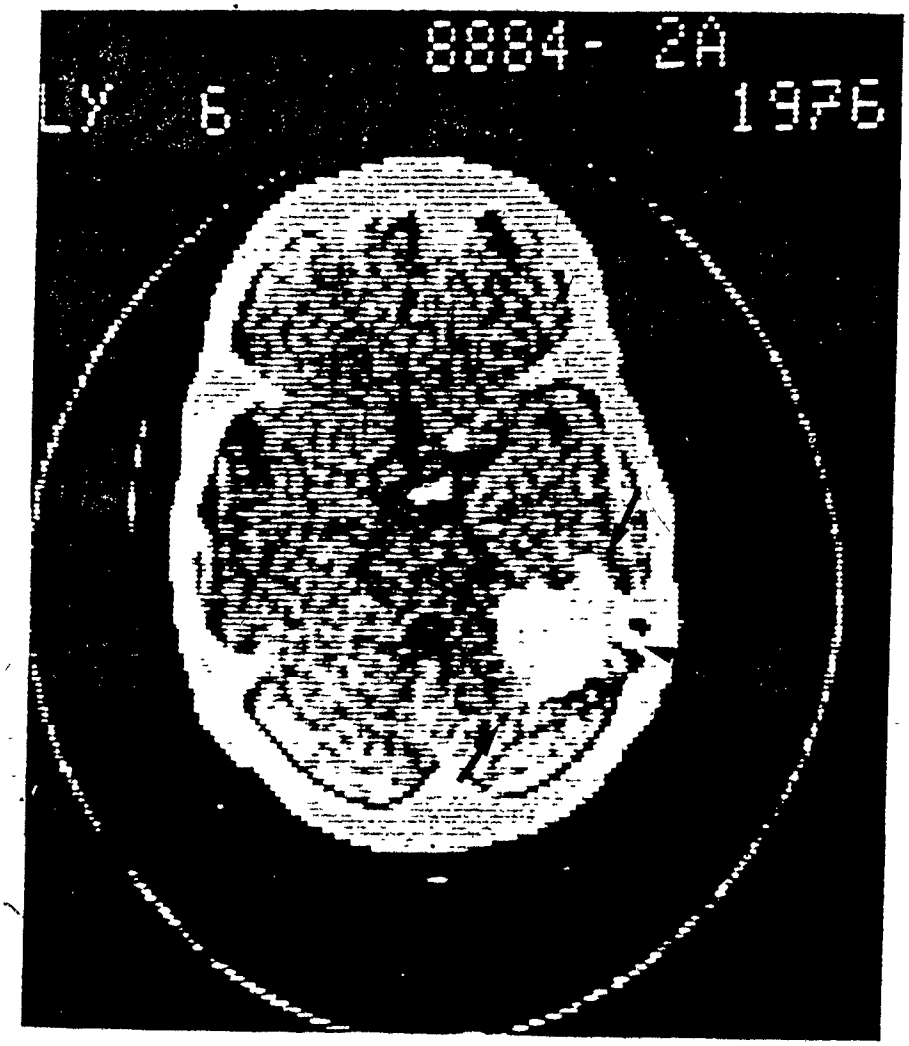

Fig. 5. CT-Scan showing meningioma (arrows) in posterior fossa. (Courtesy Dr. R. Ethier, Neuroradiologist, Montreal Neurological Institute).

right cerebellar hemisphere. The tumor was removed in toto uneventfully. One year after the operation the patient still experienced the attacks unchanged.

Immediate Post-Operative Period (Other than Ear Surgery - 3 Cases)

The first patient underwent a lumbar spinal operation; the second a kidney operation, the third a cholecystectomy.

Systemic Degenerative Diseases (4 Cases)

One patient had an extensive carcinoma of the breast without apparent brain metastases, one had encephalopathy of unknown origin, one had diabetic polyneuropathy and one had multiple myeloma. Conceivably there was no relationship between these diseases and the paroxysmal positional vertigo which the patients experienced.

Idiopathic (169 cases)

All patients for whom no apparent cause was identified were classified as idiopathic. There were 49 males and 120 females. Fig. 6 shows the age distribution of male patients and Fig. 7 shows the age distribution of female patients at the time of onset of the initial period when attacks could be elicited. A number of patients in this group suffered from diseases characteristic of their age (high blood pressure, diabetes, etc.). A number of them had bilateral sensorineural hearing loss which could be attributed to presbycusis or noise induced damage in both

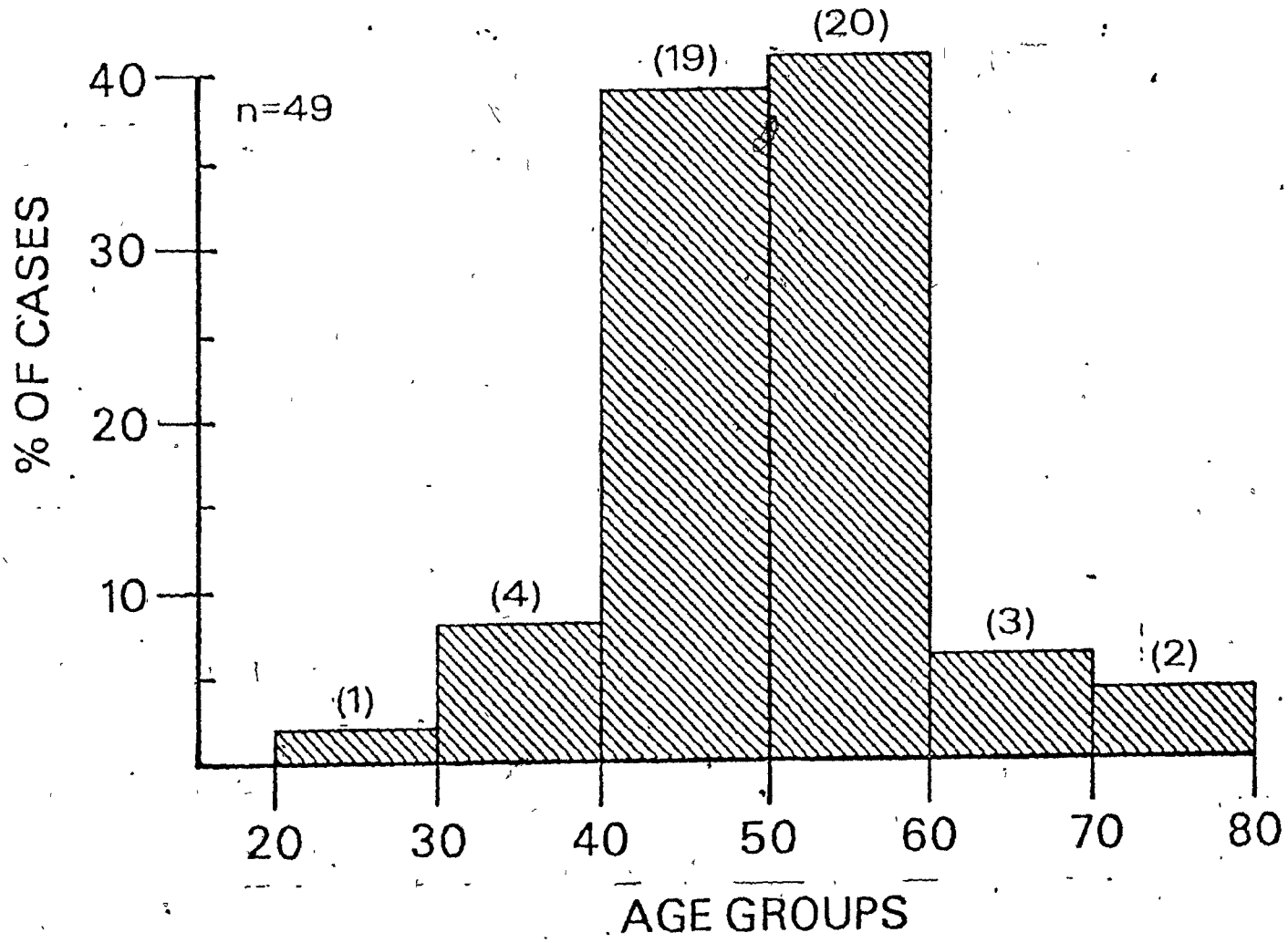

Fig. 6. Age distribution of idiopathic male patients.

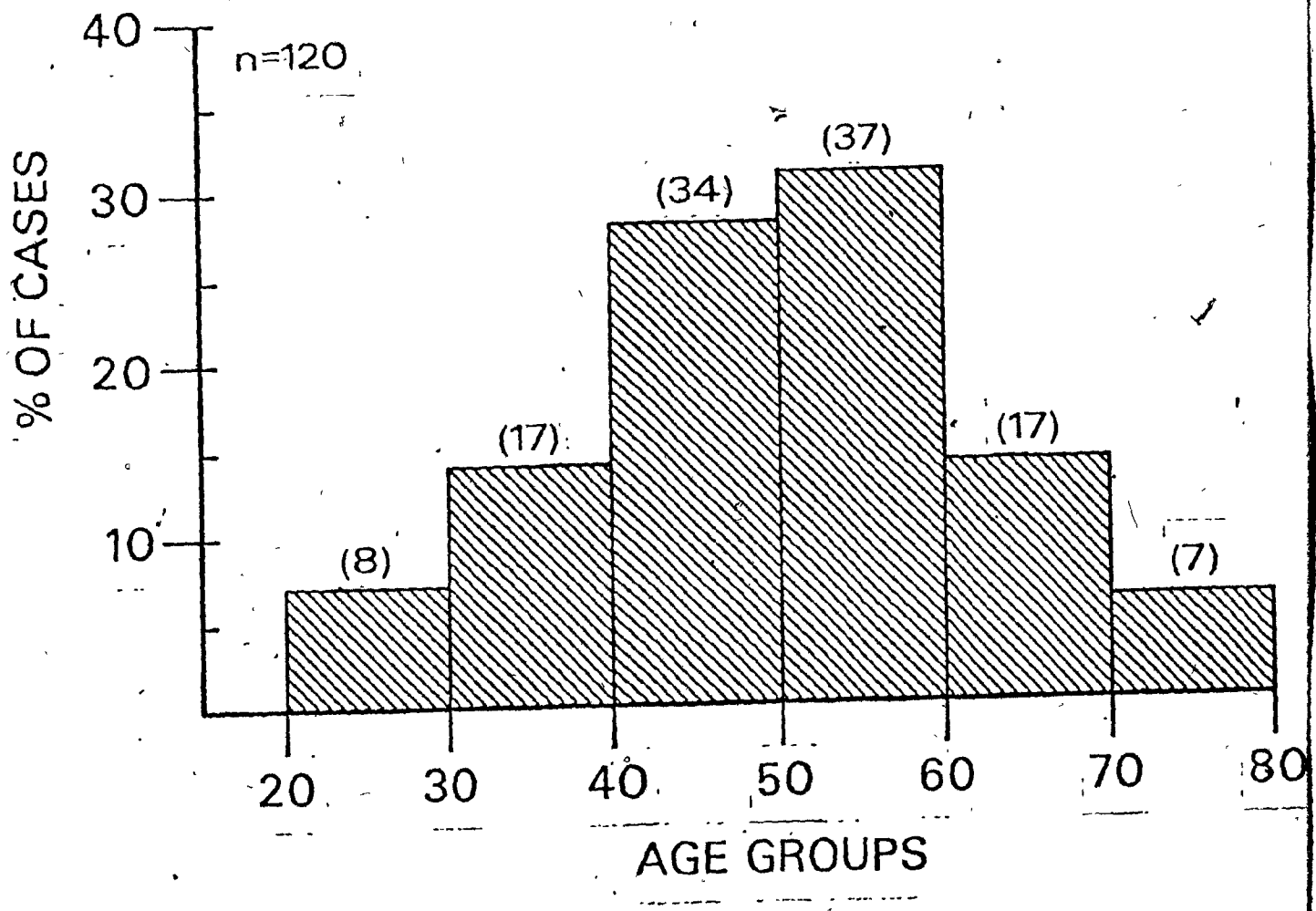

Fig. 7. Age distribution of idiopathic female patients.

ears. None of the patients showed labyrinthine preponderance on bithermal caloric stimulation.

(d) Treatment

Most of the patients thought they had a serious disease. Reassurance and an explanation to the patient of how to avoid the provocative head position allayed these fears. The more severe cases were treated with antivertiginous medication with or without small dosages of Diazepam for a few days. None of our patients thought the condition serious enough to warrant surgery, except one, who is presently under consideration for surgical treatment. He is an old Meniere's disease case.

(e) Electronystagmography

An ENG was undertaken in 179 out of 255 patients. A number of patients (Table II) showed concomitant vestibular dysfunction manifested as post-caloric labyrinthine preponderance. All patients who did not show post-caloric labyrinthine preponderance, and who were tested during a period when paroxysmal positional vertigo attacks could not be provoked, showed no abnormalities on their ENG recordings.

In 76 patients without post-caloric labyrinthine preponderance an ENG was performed during a period when attacks could be elicited. Nineteen patients showed direction-changing positional nystagmus; twenty-three showed direction-fixed positional nystagmus with the fast phase towards the ear which was in the lowermost position during the attack; twelve showed

direction-fixed positional nystagmus with the fast phase in the opposite direction and 22 showed practically no positional nystagmus. With the exception of the actual attack, the velocity of this positional nystagmus was 2^{o}-7^{o}/sec. except in five patients who showed positional nystagmus up to 20^{o}/sec. in a few positions. In all these five patients the positional nystagmus was of direction-fixed type, with the fast phase towards the ear which was in the lowermost position during the actual attack. Besides, all five showed post-caloric directional preponderance towards the same direction.

Out of 76 patients, 56 gave symmetrical bithermal post-caloric responses. Seventeen gave post-caloric directional preponderance towards the ear which was in the lowermost position during the attack and three gave post-caloric directional preponderance towards the opposite side.

In seven patients out of 255 an actual attack was recorded with eyes closed during the routine ENG examination. In two patients the direction of the recorded nystagmus during the attack was the same as the direction of the nystagmus observed clinically during another attack. In four patients the direction of the recorded nystagmus was opposite to the direction of the observed nystagmus and in one patient the direction of the recorded nystagmus reversed in the middle of the attack (Fig. 8).

The so-called nystagmus of the secondary period was recorded in two patients.

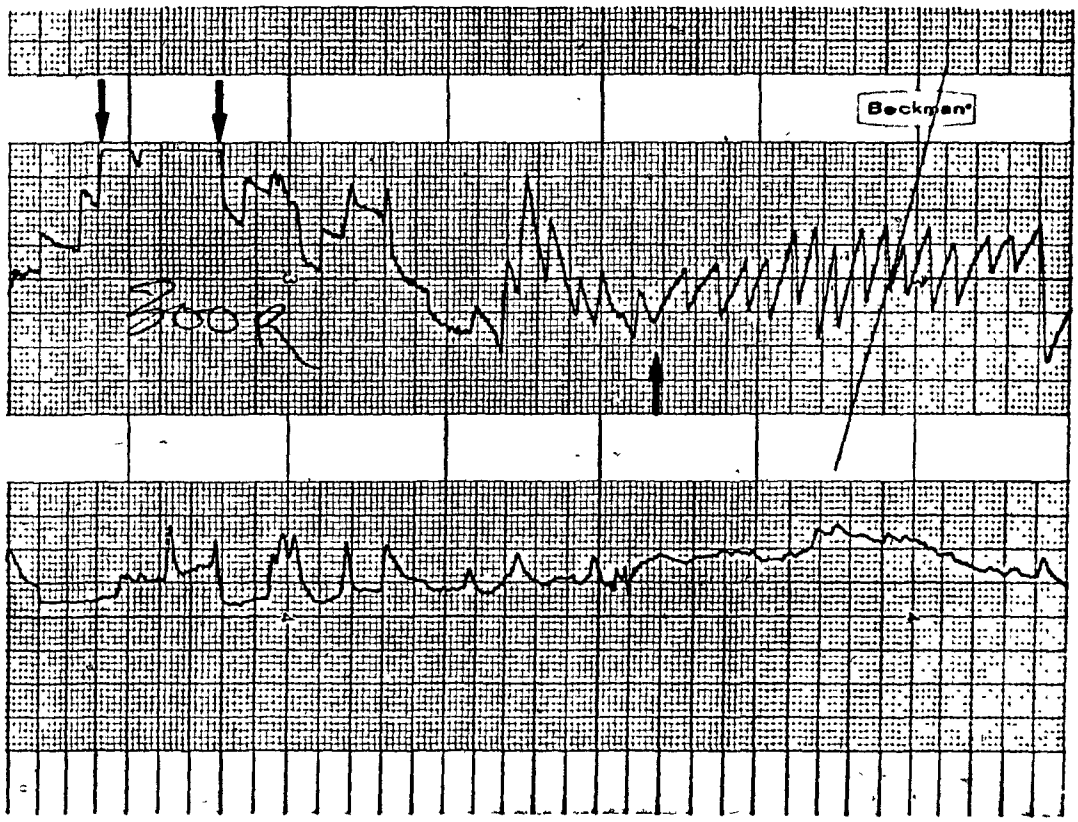

Fig. 8. ENG recording of paroxysmal positional vertigo attack. Time of right head hanging positioning 2 sec. (between arrows). The attack begins about 4 sec. later with right beating nystagmus which reverses direction some 2.5 sec. later (lower arrows). Upper recording shows horizontal bitemporal leads. Calibration 10°/sec. Paper speed 10mm/sec. Lower recording shows vertical lead from right eye. Eyes Closed.

GROUP II.

There were 11 patients in this group. They all met the same criteria for paroxysmal positional vertigo as the patients in Group I. They were all examined during a period when an attack could be elicited and they were all evaluated in the same way as the patients of Group I. Three patients were post-head injury cases, all of immediate onset, all seen within a month post-accident. One was an old Meniere's disease case; one post-vestibular neuronitis and six were of the idiopathic group. The youngest in the latter group was 43 years old, the oldest 60 years old.

On examination no abnormalities were found except:

1. In some patients a degree of hearing loss (See below);
2. A degree of unsteadiness on tandem gait with eyes closed, in some of them with eyes open as well;
3. Gaze nystagmus in three patients towards the ear which was lowermost during the attack;
4. Paroxysmal positional vertigo attack which they all developed in different provocative head positions during the Hallpike's testing.

They all showed Type I nystagmus during the attack, except one post-head injury patient who showed Type III nystagmus.

The audiogram of 10 out of 11 patients showed no hearing loss, or sensorineural loss affecting the two ears symmetrically. The Meniere's disease patient showed substantial sensorineural hearing loss in one ear; the hearing in the other ear was close to normal.

A routine ENG examination performed in all 11 patients gave no positional nystagmus in two idiopathic cases; direction changing nystagmus in three idiopathic cases; direction fixed nystagmus with the fast phase towards the ear which was lowermost during the attack in five cases (two post-head injury, one Meniere's disease, one vestibular neuronitis, one idiopathic). Strangely, the bilateral post-head injury case (Type III) showed also direction fixed positional nystagmus.

Four cases, all idiopathic, gave symemetrical caloric responses; three cases (one post-head injury, two idiopathic) gave post-caloric directional preponderance; four cases (two post-head injury, one Meniere's disease, one vestibular neuronitis) gave post-caloric labyrinthine preponderance.

None of the patients showed gaze nystagmus on the ENG recording. In none of the patients was the attack recorded during routine ENG recording.

The Paroxysmal Positional Vertigo Attack (Eyes Open)

The attack was elicited using Hallpike's maneuver.

During the attack the eye movements in different gaze directions were observed and recorded by ENG. The process was described in detail above (See Methods).

The direct observation of the eyes in all eleven patients during the attack confirmed the earlier observation reported in the literature. In all patients, when the eyes were deviated towards the lowermost ear the nystagmus was mainly rotatory; when they were deviated towards the uppermost ear the nystagmus was mainly vertical. Our impression was that the eye ipsilateral to the lowermost ear showed a stronger rotatory component of the nystagmus than the contralateral eye. In some patients a horizontal nystagmus component was also suspected.

Out of eleven patients' recordings, two were technically unsatisfactory. Finally, seven patients' tracings of bitemporal horizontal eye recording and two patients' tracings of separate horizontal eye recording were studied. Eight out of nine patients whose attack was successfully recorded developed an attack only in one head hanging position; one patient developed an attack in both head hanging positions. All the attacks had the characteristics already described. The slow phase velocity of the nystagmus at the peak of the attack was of variable magnitude. The lowest velocity was 25°/sec. and the highest velocity recorded was 135°/sec. (Fig. 9). This is the highest slow phase nystagmus velocity ever recorded in our ENG Laboratory

since early 1974.

Horizontal Bitemporal Leads

In four cases the fast phase of the nystagmus was towards the lowermost ear when the eyes were deviated towards this direction. When the eyes were deviated towards the uppermost ear the fast phase changed direction. In one case the nystagmus was recorded only when the eyes were deviated away from the lowermost ear. In this gaze direction the fast phase of the nystagmus was away from the lowermost ear.

In one case the fast phase was towards the uppermost ear when the eyes were deviated towards the lowermost ear. When the eyes were deviated towards the opposite direction the nystagmus remained unchanged.

In the bilateral case, in both head hanging positions, the fast phase was towards the lowermost ear when the eyes were deviated towards this ear. When the eyes were deviated towards the uppermost ear, the fast phase changed direction (Fig. 10, 11).

Vertical Leads

In one case vertical nystagmus was not recorded. In the bilateral case, when the eyes were deviated towards the lowermost ear, in one head hanging position, the nystagmus was downbeating (Fig. 10), in the other head hanging position, the

nystagmus was upbeating (Fig. 11). When the eyes were deviated towards the uppermost ear the nystagmus was upbeating in both head hanging positions. In two cases, the nystagmus was downbeating when the eyes were deviated towards the lowermost ear. When the eyes were deviated towards the opposite direction, no nystagmus was recorded. In three cases the nystagmus remained upbeating in both directions of eye deviation.

Separate Horizontal Eye Leads

In one case when the eyes were fixated in midposition, the fast phase of the nystagmus was towards the lowermost ear. When the eyes moved towards the lowermost ear the nystagmus remained unchanged in direction but a slow phase velocity difference between the two eyes appeared (Fig. 9).

In the other case when the eyes were deviated towards the uppermost ear, the nystagmus fast phase was towards this direction for both eyes. When the eyes moved towards the lowermost ear, the eye ipsilateral to this ear developed nystagmus fast phase towards this ear, while the nystagmus of the contralateral eye remained unchanged (Fig. 12).

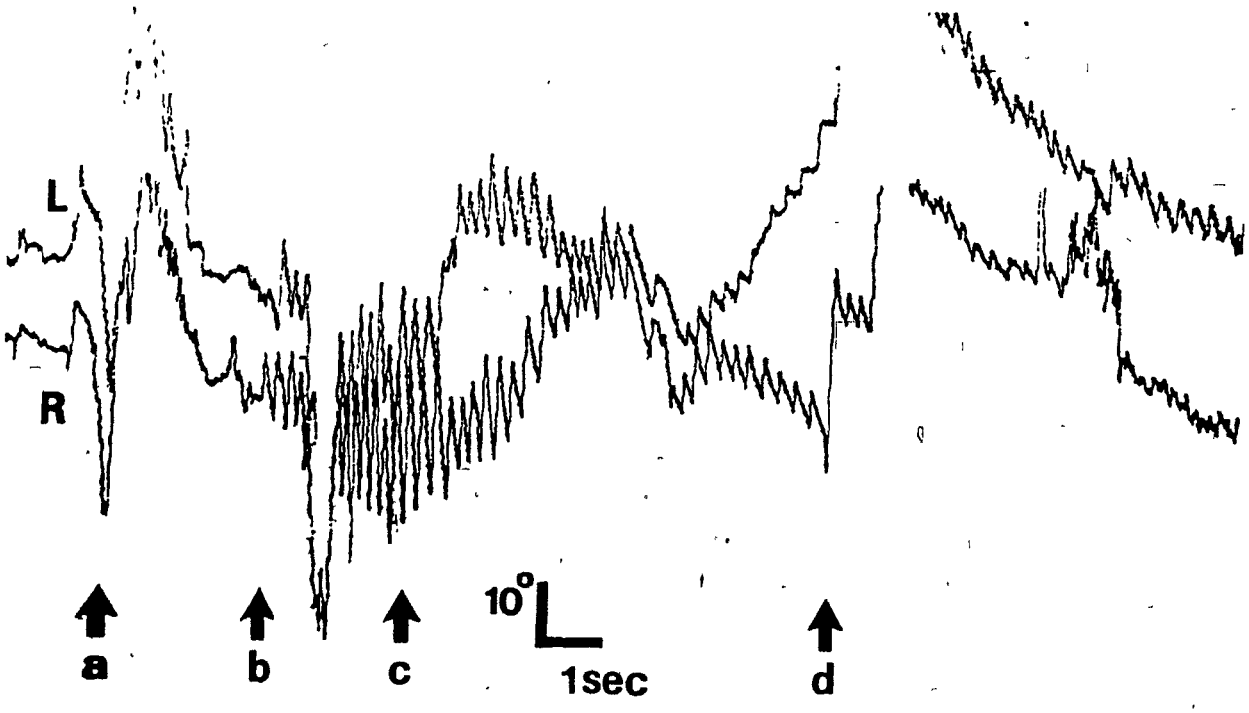

Fig. 9. L: horizontal right eye recording; R: horizontal left eye recording; a: left head hanging position; b: beginning of the attack; c: peak of the attack (eyes fixated in midposition); d: eyes towards lowermost ear (left).

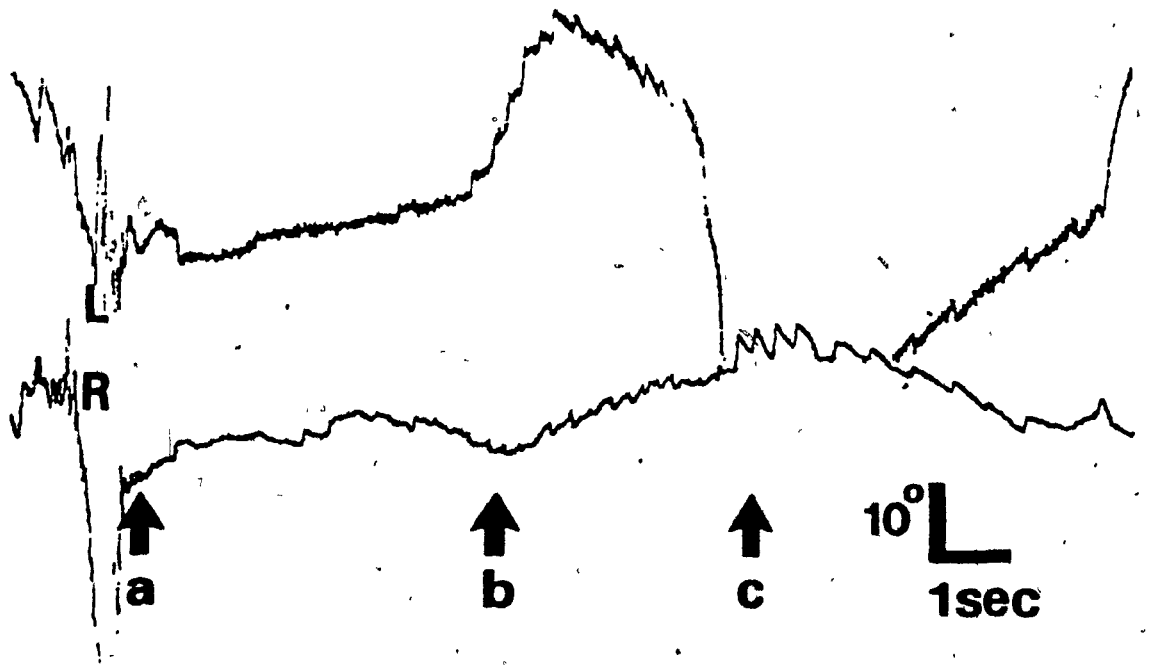

Fig. 10. L: bitemporal horizontal tracing; R: vertical tracing from right eye; a: right head hanging position completed; b: eyes fixated towards the lowermost ear (L: right beating nystagmus; R: down-beating nystagmus); c: eyes towards uppermost ear (L: left beating nystagmus; R: upbeating nystagmus).

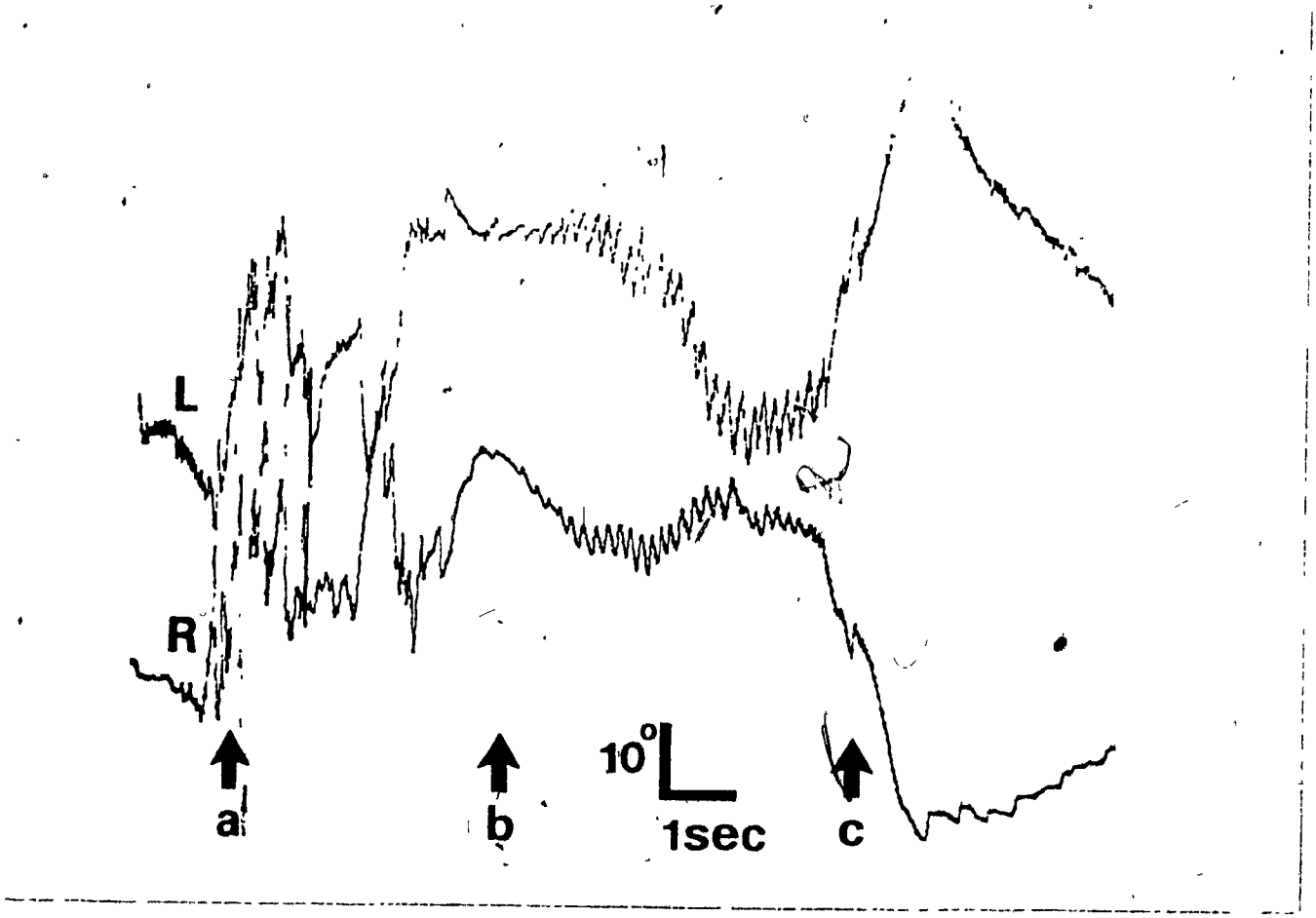

Fig. 11. L: bitemporal horizontal tracing; R: vertical tracing from right eye; a: left head hanging position; b: eyes fixated towards lowermost ear (L: left beating nystagmus; R: upbeating nystgmus); c: eyes towards uppermost ear (L: right beating nystagmus; R: upbeating nystagmus).

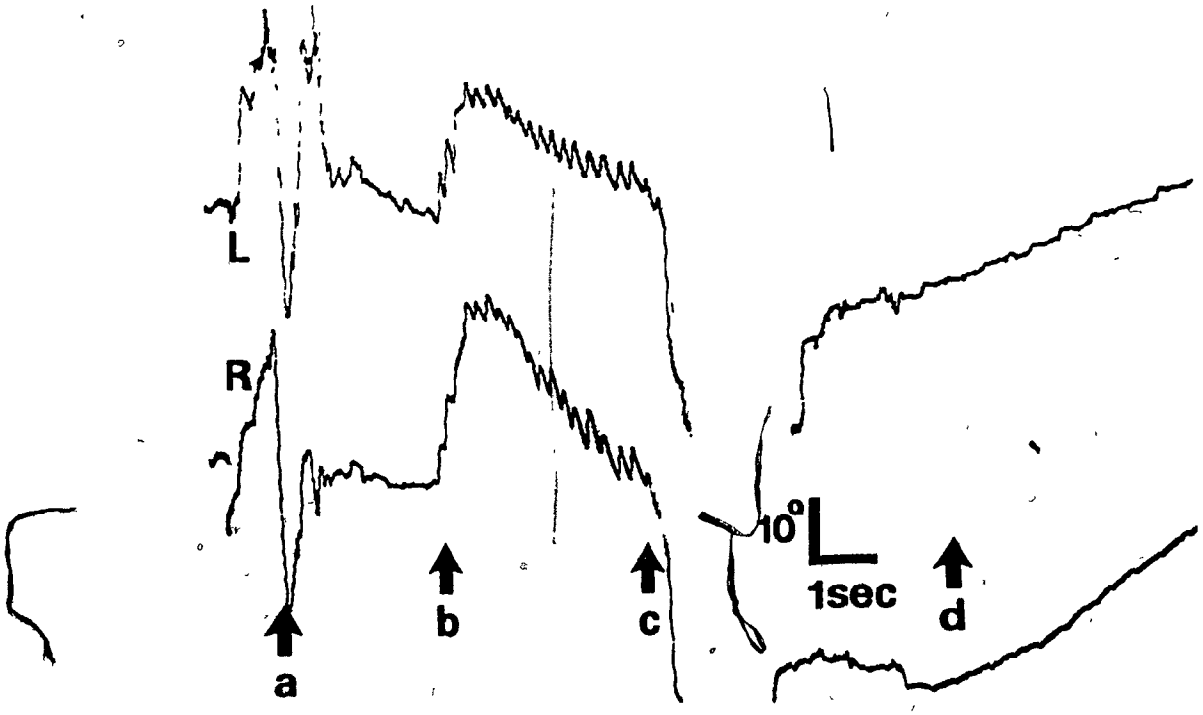

Fig. 12. L: horizontal left eye recording; R: horizontal right eye recording; a: left head hanging position; b: eyes deviated towards uppermost ear (both tracings showing right beating nystagmus); c: eyes moving towards lowermost ear; d: eyes deviated towards lowermost ear (L: right beating nystagmus; R: left beating nystagmus).

CHAPTER FOUR

DISCUSSION

Paroxysmal positional vertigo is a syndrome which has puzzled neuro-otologists for over half a century. It is a common disease which is easily overlooked or misdiagnosed.

History

There are three crucial points to be clarified from the history:-

(a) The duration of the actual attack. Concomitant symptoms such as nausea or vomiting and unsteadiness or lightheadedness may make the patient think that the attack is much longer than it actually is. The other interesting point is that in a number of patients the degree of panic is so high that they never try to stay in the provocative head position longer than a few seconds. In this way they try to get up, they feel dizzy and right away they lie in bed again. In a few minutes another attempt to get up from bed gives the same results. In this way they may stay in bed for many hours convinced that they cannot get up. Therefore, this point needs to be clarified.

(b) The provocative head position is also important. Most of the patients experience attacks more frequently when lying in bed, when trying to get up from bed, or turning from side to side in bed or any combination of these movements. There-

fore, they need to be asked specifically for these movements. A number of patients do not know the exact head position which triggers the attack but they know the vertigo occurs in relation to the flat body position.

(c) If another vestibular syndrome is present or it has been in the past, the patient usually knows that he expriences or has experienced another type of dizziness, although he may not volunteer this information.

Clinical Examination

The crucial part of the whole examination is the Hallpike's positioning. First the physician has to make sure that the vertigo attacks still occur on the day of the examination. Second, the maneuver, as modified by Stenger, needs to be performed quickly and accurately. However, there is a small number of patients who will not develop the attack when positioned by the physician. In these cases the patient should be asked to provoke an attack himself.

Etiology

From the discussion on etiology (See Literature Review) it is obvious that the most probable cause of paroxysmal positional vertigo is activaton of the posterior semicircular canal by a mechanism such as the one suggested by Schuknecht. Our four cases with intracranial tumors appear to contradict this assumption. However, on closer examination it is obvious that

in three of these cases the presence of paroxysmal positional vertigo was a mere coincidence. In the fourth, it might be argued that two possibilities arise: either the tumor compromised the blood circulation in the inner ear thus creating the necessary conditions for cupulolithiasis, or the fact that paroxysmal positional vertigo persisted unchanged one year after total removal of the tumor makes it seem likely that the two were unrelated.

From Fig. 6 and Fig. 7 it is obvious that the age distribution of idiopathic cases is different from the age distribution of head injury cases (Fig. 4). It is obvious that the distribution of the idiopathic cases is more dense in the 40 to 60 years group. It is possible, therefore, that the mechanism in the endorgan which causes paroxysmal positional vertigo is part of the ageing process. At this point there is no satisfactory explanation for the fact that paroxysmal positional vertigo seems to be more common among females than males in the idiopathic group.

Electronystagmography

Group I.

None of our ENG findings in this group either during a period of attacks or during a remission was pathognomonic. It is further astonishing that such a high number of patients gave no ENG abnormalities even during a period of attacks. Therefore,

it is obvious that the ENG does not help in the diagnosis unless the actual attack is recorded. It is useful, however, in ruling out other concomitant abnormalities.

The fact that in only seven patients was an attack recorded during routine ENG recording, is due to the fact that the patient's head (body) was slowly positioned in the nine tested head positions (See Methods). This shows again that Hallpike's positioning needs to be used for an attack to be provoked.

Group II

The routine ENG recordings in this group were not different from the recordings in Group I.

The ENG recordings with eyes open during the paroxysmal positional vertigo attack are of particular interest. Although all patients developed nystagmus during the attack which was identical to the observer, the recorded nystagmus was different. Bitemporal horizontal recording showed the fast phase of the nystagmus to change direction when the direction of eye deviation changed. Vertical recording showed upbeating nystagmus to become downbeating or vice versa when the horizontal eye deviation changed direction. Finally, and probably most impressively, separate horizontal eye recording showed the fast phase of the nystagmus to change direction only in one eye while in the other remained unchanged, when eye deviation changed direction.

How these observations could be explained? The nystagmus observed visually is complex, especially when the eyes are deviated towards the lowermost ear during the paroxysmal positional vertigo attack. In this case it appears that each quick phase involves a rotation of the eye about the visual axis, combined with motion in a more or less semicircular arc in an oblique direction. When the eyes are deviated towards the uppermost ear, the degree of rotation during each quick phase appears to be less, and the length of the semicircular arc along which the eye moves is reduced, so that the movement appears to be predominantly vertical.

Given the complexity of the eye movement, and the unreliability of human observation of quick movements of such short duration, it is not surprising that the ENG recordings are difficult to relate to visual observations. The ENG extracts only the linear horizontal and vertical components of eye motion, which are very difficult to assess visually since the rotational component is so striking.

Type of Observed Nystagmus During the Attack (Group I).

The frequency with which the different types of observed nystagmus in patients of Group I occurred during the paroxysmal positional vertigo attack is of special interest (Table I). All patients, except ten, developed Type I nystagmus during the attack. These results are in disagreement with those of Stahle and Terins (See Page 8). During the attack we asked the patient to fixate

his eyes on the examiner's finger which was in a more or less midposition (See Methods). In this eye position the nystagmus is a combination of rotatory and obliquely vertical nystagmus. Stahle and Terins used Frenzel's glasses in all of their patients. Frenzel's glasses are supposed to abolish fixation, but this is true only if the eyes are kept in midposition. If the eyes deviate towards either horizontal side, the electric bulb is clearly visible and the eyes may fixate on this light. Obviously Stahle and Terins' patients were able either to keep their eyes in midposition or to fixate on the light at either side and there is no reason to suppose that all did the same. Therefore, it is likely that these authors observed their patients' eyes under different gaze conditions which the patients chose by chance and without any control by the examiners. This may have given the impression of different direction nystagmus. It is possible that this explains the discrepancy of occurrence of Type I and Type IV of observed nystagmus in Group I and the series reported by Stahle et al. At this point, we do not have a satisfactory explanation for the difference of occurrence for Type II (Stenger) observed nystagmus in Group I and the patients of Stahle et al.

CONCLUSIONS

The data from our 255 patients (Group I) and 11 patients (Group II) suffering from paroxysmal positional vertigo lead to the following conclusions:

1. From the history the crucial points are: (a) the duration of the attack; (b) the special head (body) positions which trigger it; (c) whether this is the first experience or whether there were periods before, during which attacks could be elicited; (d) whether there is a history of another type of vertigo.

2. Next a detailed clinical examination is necessary to document concomitant symptoms and signs. The pathognomonic clinical test is the Hallpike's maneuver, as modified by Stenger, which must be performed quickly and carefully. If an attack is not elicited through this positioning the patient needs to be asked to go through the movement process which triggers the attack. A small number of patients will be able to elicit an attack although the Hallpike's maneuver failed.

3. The ENG recording does not show any pathognomonic signs and, therefore, does not help in the diagnosis unless an actual attack is recorded, which is rather unusual during routine ENG examinations. This makes the clinical examination even more important. ENG recording, however, is necessary for the detection of oculomotor or other vestibular abnormalities.

4. If the direction of gaze is controlled during the attack, the nystagmus to be observed is nearly always the same. Different types of nystagmus observed in other reported series may be explained from the patient's gaze direction during the attack.

5. The discrepancies between the observed and recorded nystagmus by ENG with eyes open and different gaze directions during the paroxysmal positional vertigo attack point clearly to the fact that ENG recordings are difficult to relate to visual observations for such complex eye movements. It is obvious that the ENG extracts only the linear horizontal and vertical components. This implies that in the presence of rotatory nystagmus, ENG recordings need to be interpreted with caution.

6. All available data point to the posterior semicircular canal as the possible site of origin of the paroxysmal positional vertigo attack.

7. Paroxysmal positional vertigo may be developed on a pre-existing endorgan problem.

8. Most of idiopathic cases belong to 40-60 years of age group for both sexes, suggesting that the idiopathic type is part of the process of ageing. At this point there is no satisfactory explanation for the observation that the idiopathic type is more common among women than men.

9. Although we believe that paroxysmal positional vertigo is a syndrome caused invariably by a disturbance of the endorgan which is in the lowermost position during the attack, we wish to draw the clinician's attention to the fact that this disturbance in the endorgan may rarely coexist with central pathology. It is not clear whether there is a cause-effect relationship in a few of these cases.

10. Although Schuknecht's theory of cupulolithiasis seems to explain most of the manifestations of this syndrome it is not yet clear what really happens to the cupula of the posterior semicircular canal during the attack. At this point a reconstruction of events during the attack under any possibilities seems to contradict itself.

11. Further research is needed on this syndrome. Four aspects in particular need clarification.

(a) A controlled prospective study as to the different head positions in which the attack occurs. During the retrospective study for Group I the question was why there are patients who are more troubled by movements related to the supine position and others who feel more frequently and more severely vertiginous when they bend over or when they move their head upward. At this point one wonders whether this implies another mechanism of disease such as a different excitation process of the cupula or different size of deposits in the ampulla of the posterior semicircular canal.
It is possible also that the diverse results as to positional nystagmus (eyes closed) and post-caloric responses obtained by routine ENG recordings during periods when attacks could be elicited in patients of Group I, may be related to different abnormalities of cupula excitation and restoration. It is conceivable that DC ENG recordings will help in clarifying whether these differences are due to different eye position

in the orbit or different vestibular signals.

(b) It is clear that high speed photography or other methods of recording complex eye movements are essential to obtaining an accurate quantitative description of this type of eye movements. Therefore, the nystagmus of the attack needs to be studied by such methods.

(c) To find new ways to measure the function of the affected cupula in the hope of distinguishing different possible processes of excitation and restoration.

(d) Long term follow up prospective study to find what happens to the duration of subsequent periods when attacks can be elicited and subsequent periods of remissions.

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